Building a systematic model of risk and protective factors for intimate partner violence against women: the role of long-term community and structural disadvantages

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Abbreviations

ALSPAC	Avon Longitudinal Study of Parents and Children
ARR	Adjusted relative risk
CI	Confidence interval
DAG	Directed acyclic graph
HIC	High-income countries
IPV	Intimate partner violence
IQR	Interquartile range
IRR	Incidence rate ratio
LMIC	Low- and middle-income countries
LSOA	Lower-layer super output area
MLM	Multilevel model
MSM	Marginal structural model
OR	Odds ratio
PTSD	Post-traumatic stress disorder
RCT	Randomized controlled trial
RR	Risk ratio
SE	Standard error
SES	Socioeconomic status
STI	Sexually transmitted infection
UK	United Kingdom
USA	United States of America
WHO	World Health Organization

Abstract

Building a systematic model of risk and protective factors for intimate partner violence against women: the role of long-term community and structural disadvantages

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D.Phil in Social Intervention, Trinity Term 2019

Background: Intimate partner violence (IPV) is estimated to affect at least 1 in 3 women, making it the most common form of violence perpetrated against women. While there is ample research studying IPV, there have been relatively few studies of patterns of risk over time and even less that have investigated the longitudinal relationship between IPV and community- or structural-level factors.

Objectives: (1) Systematically summarise the longitudinal risk and protective factors for IPV against women; and, using data from the Avon Longitudinal Study of Parents and Children (ALSPAC) in the United Kingdom, evaluate: (2) the measurement and prevalence of IPV; (3) the dynamic relationship between objective and perceived measures of neighbourhood environments, and (4) the effect of long term exposure to neighbourhood-level deprivation on the risk of experiencing IPV among women.

Method: This thesis used a variety of quantitative methods, including: a systematic, meta-analytic review; psychometric analysis; trajectory analysis; and marginal structural models using inverse probability weighting. I reviewed all available prospective-longitudinal studies of at least one risk or protective factor for IPV against women. My remaining empirical work used ALSPAC data from mothers and their children enrolled from birth between 1991-1992. When enrolled children were aged 21, they responded to a novel 8-item scale of physical, psychological, and sexual IPV experiences. Additionally, 18 years of data on neighbourhood environments, including the Indices of Multiple Deprivation, and family-level characteristics were available.

Results: Few prospective-longitudinal studies on IPV against women have been conducted outside the United States or that investigated any community- or structural-level risk or protective factors. Among women participating in ALSPAC for 21 years, 32% had experienced IPV between ages 18-21 alone, with variations in their exposure to neighbourhood-level deprivation over the study period – demonstrating the need for further etiological study using appropriate longitudinal methods. Accounting for time-varying socioeconomic characteristics and sample attrition, I found that long-term exposure to more versus less deprived neighbourhoods over the first 18 years of life was associated with experiencing more frequent IPV and a higher risk of experiencing any IPV in early adulthood among participating women.

Conclusions: This thesis synthesised, for the first time, the longitudinal literature on the risk and protective factors for IPV against women and contributed both methodologically and substantively to a major evidence gap identified: the association between neighbourhood-level deprivation and IPV. My findings demonstrate that long-term neighbourhood disadvantages predict women's increased risk of experiencing IPV and the value of longitudinal epidemiologic methods in advancing our understanding of IPV and its potential causes.

Chapter 1: Introduction

Gender-based violence against women is the intentional cause or threat of physical, psychological, or sexual harm to women.[1] Acts of gender-based violence, by definition, disproportionately affect women or are inflicted because the victim is a woman.[2] While men are more likely to experience violence from strangers or acquaintances, women are far more likely to be victimised by someone they know well, with violence committed by a current or former intimate partner (i.e., intimate partner violence or IPV) being the most common form of violence against women.[3,4] Although IPV is perpetrated by women or against men – and disagreements are entrenched in the field as to whether more men or women experience or perpetrate IPV[5,6] – it is nearly universally agreed that female victims experience the vast majority of severe negative outcomes at the hands of male perpetrators.[7,8] While death is the most drastic outcome of IPV, its nonfatal physical and psychological consequences are the most prevalent and greatest contributors to the health, economic, and social burdens it causes.[9,10] These include physical injury, alcohol and substance misuse, severe psychological disorders, and increased risk of chronic diseases and pain.[11,12]

In 1993, the United Nations officially recognised violence against women as an extreme violation of women's rights to equality, liberty, and security.[13] This recognition was brought on by decades of activism, particularly from feminist movements, with little but, where existing, noteworthy attention from academia, which focused on the qualitative experiences of 'battered wives', failure of health, legal, and welfare systems to support them, and the hypothesised patriarchal roots of these problems.[14] The international recognition of violence against women as a severe health and social problem helped

initiate the vast body of cross-sectional evidence that exists today on the prevalence and consequences of IPV against women.[15,16] However, to date there is an overall dearth of rigorous, longitudinal research into the complex factors that contribute to the occurrence and exacerbation of IPV against women. Resultantly, many evidence gaps remain in our etiological understanding of this violence.[17,18] This remains a barrier for the development of effective interventions and policies for tackling IPV, with significant uncertainty over what works and within which contexts. While on average, low- and middle-income countries (LMIC) have the highest rates of IPV, rates are unacceptably high globally including in high-income countries (HIC).[9] In response to this pervasiveness, there have been increasing international calls for greater implementation of preventive interventions to reduce violence against women.[19] However, if interventions are to be effective, we need a clear understanding of what the risk and protective factors for the victimisation of women are and by what pathways these operate.[20] While prior research has largely focused on correlates (factors associated with IPV against women), successful intervention requires targeting *causal* risk factors (correlates that can change and, when changed, increase the risk, or probability, of IPV) or protective factors (correlates that, when changed, decrease the risk of IPV) – of which we know far less.[21,22]

Internationally-recognised conceptions of violence have generally advanced into ecological or multilevel frameworks, where violence is seen as the result of a complex interplay between individual, relationship, community, and structural factors.[23-26] Implicit to this framework is the recognition that neither individual nor societal factors alone are sufficient to understanding the social patterns in victimisation and why individuals commit violence. However, many causal links between potential risk and protective factors and IPV – particularly those between violent outcomes and more distal structural and

community-level factors – although conceptually plausible, have not been validated with empirical evidence[25] or have been left neglected altogether in systematic reviews. This thesis sought to address these gaps in the evidence base by advancing a systematic understanding of the causal risk and protective factors for IPV against women, evaluating available measures to investigate the longitudinal relationship between IPV and community and structural exposures related to the neighbourhood environment, and apply advanced quantitative methods to clarify the contribution of neighbourhood-level deprivation, a particularly under-studied exposure, to IPV against women. To this end, the objectives of this thesis were to:

1. Systematically review prospective-longitudinal evidence of risk and protective factors for IPV against women at all ecological levels (individual, relationship, community, and structural) and identify evidence gaps; and
2. Using two generations of data from the Avon Longitudinal Study of Parents and Children (ALSPAC) in the United Kingdom (UK), evaluate:
    a) The overall prevalence and gender differences in experiences and impacts of IPV in early adulthood and psychometric properties of the IPV measure,
    b) The dynamic relationship between objective neighbourhood-level deprivation and perceptions of the neighbourhood environment over time, and
    c) The effect of long-term exposure to objective neighbourhood deprivation on the risk of experiencing IPV among women in early adulthood.

In applying advanced quantitative methods to develop, and identify gaps in, the longitudinal evidence base for the etiology of IPV against women, this thesis further

sought to make a methodological contribution to the field that would support future directions for research as well as the design of more effective interventions and policies to prevent IPV and support women experiencing this violence.

1.1 The burden of IPV against women

1.1.1 Definition and global prevalence

IPV is the act or threat of physical, psychological, or sexual harm by a current or former partner.[4] Although historically there has been conflation of IPV with the terms 'domestic violence' or 'family violence', modern definitions describe IPV as a category of domestic/family violence, which more broadly includes any violence threatened or committed by a member of the same household, family, or relationship.[27-29] In addition to IPV, domestic violence typically encompasses child and elder abuse.

Women's experiences of IPV may be extensive and varied. Physical IPV may include being pushed, shoved, grabbed, hit, slapped, kicked, choked, burnt, or beaten, or threats thereof.[27] It can also involve a partner using or threatening to use a weapon or object to cause injury. Sexual IPV entails being forced or coerced to have sex or engage in some other unwanted sexual contact or experience (e.g., behaving in a sexual way that is perceived as degrading). Emotional IPV (often referred to interchangeably as verbal or psychological IPV or intimate partner abuse) involves being intimidated, belittled, humiliated, or insulted, or experiencing controlling behaviours such as being isolated from friends and family, having one's whereabouts monitored, or having one's access to resources restricted. Women may experience only one of these forms of IPV to damaging

effect. However, when experiences of different types of IPV are investigated in the same sample they have typically been found to be correlated, suggesting that women often experience multiple types. For instance, in one of the largest international studies of the prevalence of IPV against women using standardised scales, Garcia-Moreno and colleagues found that in the majority of the 15 study sites, comprising 24,000 women in 10 countries (Bangladesh, Brazil, Ethiopia, Japan, Peru, Namibia, Samoa, the former State Union of Serbia and Montenegro, Thailand, and Tanzania), 30-56% of women who had experienced physical or sexual IPV experienced both.[30] Similar patterns of overlap in the experience of physical, sexual, and emotional IPV have been reported in national samples from Australia (N=2,629 women);[31] Canada (N=9,178 women);[32] the United States of America (USA, N=3,568 women);[33] and the UK (N=11,503 women).[34] This suggests the common multidimensionality of women's experiences of IPV and the need to consider the full array of types and patterns of IPV against women when seeking to understand contributing factors and consequences.

Despite increasing awareness of its harmful nature, the prevalence of IPV against women remains alarmingly high worldwide. In the only global systematic review of all available data on prevalence estimates, retrieved from 79 countries and two territories, one in three women who had ever had an intimate partner reported that they had experienced physical and/or sexual IPV in their lifetime.[11] As shown in Table 1.1 the estimated prevalence of IPV against women was highest in LMIC, particularly in Africa, the Eastern Mediterranean, and South-East Asia. However, this review also demonstrated that the average prevalence of IPV is universally high across all regions, regardless of economic status, with nearly a quarter of ever-partnered women in HIC reporting that they had experienced IPV in their lifetime.

Table 1.1 Lifetime prevalence of physical and/or sexual IPV among ever-partnered women aged 15 and over by World Health Organization (WHO) region

WHO region	Prevalence, %	95% Confidence Interval (CI), %
Low- and middle-income regions:		
Africa	36.6	32.7 to 40.5
Americas	29.8	25.8 to 33.9
Eastern Mediterranean	37.0	30.9 to 43.1
Europe	25.4	20.9 to 30.0
South-East Asia	37.7	32.8 to 42.6
Western Pacific	24.6	20.1 to 29.0
High-income regions	23.2	20.2 to 26.2

Note. Table adapted from Garcia-Moreno and colleagues (p. 17).[11] Countries and territories with available data were grouped into WHO regions as follows. Africa: Botswana, Cameroon, Democratic Republic of the Congo, Ethiopia, Kenya, Lesotho, Liberia, Malawi, Mozambique, Namibia, Rwanda, South Africa, Swaziland, Uganda, United Republic of Tanzania, Zambia, and Zimbabwe; Americas: Bolivia, Brazil, Chile, Colombia, Costa Rica, Dominican Republic, Ecuador, El Salvador, Haiti, Honduras, Jamaica, Mexico, Nicaragua, Paraguay, and Peru; Eastern Mediterranean: Egypt, Iran, Iraq, Jordan, and Palestine; Europe: Albania, Azerbaijan, Georgia, Lithuania, Republic of Moldova, Romania, Russia, Serbia, Turkey, and Ukraine; South-East Asia: Bangladesh, East Timor, India, Myanmar, Sri Lanka, Thailand; Western Pacific: Cambodia, China, Philippines, Samoa, and Vietnam; High income: Australia, Canada, Croatia, Czech Republic, Denmark, Finland, France, Germany, Hong Kong, Iceland, Ireland, Israel, Japan, Netherlands, New Zealand, Norway, Poland, South Korea, Spain, Sweden, Switzerland, UK, and USA.

In the UK, the focus of the empirical portion of this thesis, national estimates of IPV against women are similarly high. The most recent national estimates come from the 2018 Crime Survey for England and Wales (formerly the British Crime Survey), whose respondents were household residents chosen at random from the Royal Mail's list of addresses with approximately 74% response rate among N=35,000 households.[35] IPV in the survey is defined by self-reports of having experienced: physical force; being prevented from having a fair share of household money; being stopped from seeing friends or relatives; being repeatedly belittled to the extent of feeling worthless; threats to hurt the respondent or someone close to them; actual or attempted rape or sexual assault; unwanted sexual contact or exposure; or two or more incidents (causing distress, fear, or alarm) of receiving obscene or threatening unwanted letters, emails, text messages, or phone calls, having had obscene or threatening information about the respondent placed on the internet, waiting or loitering around respondent's home or workplace, or being followed or watched by a current or former partner.[36] The survey found that 23% of women had experienced IPV in their lifetime, almost identical to the lifetime prevalence found for the high-income region in the World Health Organization (WHO) global prevalence review (Table 1.1).[11] Moreover, 6% of women reported that they had experienced IPV in the last year alone.

Notably, both these figures were significantly larger than those for male respondents, among whom 11% indicated they had experienced IPV in their lifetime and 3% in the last year.

1.1.2 Consequences

To understand the gravity of the problem of IPV against women it is essential to consider the extent of its consequences for women. The evidence, which largely focuses on heterosexual relationships, demonstrates that women face the greatest burden of severe consequences following IPV compared to men and, specifically, that male-to-female IPV tends to produce more severe negative effects than female-to-male IPV. Additionally, the wide range of consequences of IPV against women exemplifies why it is both a health and social problem, and thus why conceptual frameworks from both these spheres are useful for developing theories of IPV and preventive interventions and policies.

Physical health

Mortality is the most severe outcome of IPV. In a systematic review of the highest quality estimates available from 66 countries, Stöckl and colleagues found that 14% (interquartile range (IQR) 9-18%) of all homicides globally were committed by an intimate partner.[7] Moreover, the proportion of all homicides committed by an intimate partner was more than six times higher when the victim was female (39%, IQR 31-45%) compared to male (6%, IQR 3-6%). These are conservative estimates as for 21% of homicides reported the victim-offender relationship was unknown and subsequently categorised by the researchers as non-intimate. Table 1.2 further summarises the relative proportions and

absolute numbers of homicides committed by an intimate partner in all countries for which data were available for both men and women. The results demonstrate that although a greater number of homicides had male victims overall, the estimated number of women killed by an intimate partner outweighs the number of men in every high-income country and all low- and middle-income countries, excluding only Brazil and Panama.

Table 1.2 Absolute number (derived) and relative proportion of female and male homicides committed by an intimate partner by country

Country	N all female homicides	N female IPV homicides (% total)	N all male homicides	N male IPV homicides (% total)
High-income:				
Poland	11	6 (54.55)	46	3 (6.52)
France	18	6 (33.33)	41	4 (9.76)
Estonia	26	7 (26.90)	77	2 (2.60)
Ireland	54	18 (33.33)	151	7 (4.64)
Denmark	66	43 (65.15)	71	7 (9.86)
Portugal	104	60 (57.69)	258	10 (3.88)
New Zealand	114	37 (32.46)	180	12 (6.67)
Germany	131	50 (38.17)	173	11 (6.36)
Czech Republic	140	114 (81.43)	214	14 (6.54)
Scotland	352	145 (41.19)	1 309	83 (6.34)
Hong Kong	358	84 (23.46)	663	28 (4.22)
Switzerland	429	246 (57.34)	638	54 (8.46)
Finland	692	298 (43.06)	1 716	82 (4.78)
Netherlands	739	332 (44.93)	1 810	142 (7.85)
China (Taiwan)	774	148 (19.12)	2 629	35 (1.33)
Australia	1 525	335 (21.97)	2 688	190 (7.07)
Canada	3 556	1 370 (38.53)	7 995	350 (4.39)
England & Wales	4 465	2 938 (65.79)	8 670	544 (6.28)
Japan	6 166	1 400 (22.71)	11 338	1 050 (9.26)
USA	92 582	28 552 (30.84)	327 035	10 236 (3.13)
Low- or middle-income:				
Tanzania	16	5 (31.25)	108	2 (1.85)
Nigeria	23	12 (52.17)	80	1 (1.25)
Mauritius	24	10 (41.67)	47	3 (6.38)
Brazil	37	15 (40.54)	367	24 (6.54)
Romania	45	15 (33.90)	126	4 (3.18)
India	80	47 (58.75)	344	3 (0.87)
Russia	100	20 (20.00)	100	4 (4.00)
Panama	221	8 (3.62)	2094	9 (0.43)
El Salvador	227	10 (4.35)	2119	0 (0.00)

Note. IPV homicide indicates a homicide committed by an intimate partner. N all female and male homicides and % female and male IPV homicides were taken from the Appendix from Stöckl, Devries (7). N IPV homicides was derived based on the proportion of all homicides committed by an intimate partner provided in the systematic review and was always rounded to the nearest whole number. Data are presented from all countries where estimates were available on both male and females.

Despite being the most common type of female homicide, IPV homicide is still a relatively rare event and thus difficult to study prospectively.[37] As such, the strongest evidence available on the risk of IPV leading to homicide tends to come from retrospective case-control studies. These have shown that IPV homicide typically occurs following a history of severe IPV incidents by a current or former intimate partner, rather than during a

first-time incident of severe violence.[38] For instance, in a study of 11 USA cities more female IPV homicide victims (N=220), compared to a control group of women experiencing IPV (N=343), had experienced extreme controlling behaviours (66% vs. 25%), degrading insults (78% vs. 48%), stalking (21% vs. 6%), frequent physical violence (60% vs. 26%), severe physical violence (64% vs. 20%), strangulation (56% vs. 10%), and rape (57% vs. 15%) by their intimate partner.[39] This study further found that women who had recently separated from their partner had higher odds of being killed compared to those still living with their partner (odds ratio (OR)=3.64, 95% confidence interval (CI) 1.71–7.78). The odds of women being killed after separation were even higher if their partner had also previously exhibited controlling behaviours (OR=8.98, 95% CI 3.25–24.83). This highlights the increased risk of severe violence and death women face when attempting to leave abusive relationships.[40] In addition, a partner's access to firearms has been found to consistently increase the risk that IPV will lead to female homicide.[38] This helps explain why the USA, with roughly 310 million firearms,[41] has such high numbers of IPV homicide victims as shown in Table 1.2.

If women are not killed by IPV, they may experience physical injury. Recent population surveys in Canada, the UK, and the USA have found that between 25-30% of IPV victims reported physical injuries from this violence, ranging from scratches and bruises to broken bones.[28,42,43] These surveys found that female victims of IPV experienced physical injury more often than male victims (with risk differences ranging between 16-28%) except the UK survey, which found a small difference (6%) with men (29%) reporting more physical injuries than women (23%).[28] Differences between these surveys may have been because American and Canadian participants reported on the prevalence of physical injuries caused by IPV in a particular time-period, whereas UK

participants reported on injuries caused by the most recent incident of IPV. The WHO multi-country study further estimated that the proportion of women who had ever experienced physical IPV and were injured is as high as 42% on average (19-55% among the 15 study sites).[44] In terms of the physical injuries most commonly caused by IPV against women, a systematic review of studies of women presenting to emergency departments found that women who had experienced IPV had significantly more head, neck, and facial injuries than women injured by other means (N=4 studies, n=846 women, pooled OR=24.0, 95% CI 15.0-38.0).[45]

IPV and the acute physical injuries women may experience can in turn lead to chronic conditions and pain, including heart attack, stroke, torn muscle tissue, decreased dexterity, back pain, migraines, and recurrent fainting and seizures.[12,15] Experiencing IPV can also increase engagement in negative health behaviours, such as smoking, alcohol or substance misuse, poor diet, or insufficient sleep or exercise, which in turn can lead to the development of chronic diseases. For instance, in a prospective cohort study of 5,593 women in Oslo, Norway, Stene and colleagues found that lifetime exposure to physical or sexual IPV was associated with smoking, abdominal obesity, unhealthy cholesterol levels, and high blood levels of fatty acids.[46] In turn, these women were more likely to have been prescribed medication for cardiovascular disease (hypertension) 4 to 9 years after their baseline report of IPV compared to women who had not reported IPV, adjusting for age, education, and baseline blood pressure (incidence rate ratio (IRR)=1.36, 95% CI 1.09-1.70). In the National Violence Against Women Survey, a USA population-based study of adult women (N=6,790) and men (N=7,122), physical or sexual IPV was associated with greater substance use among both women and men.[47] However only among women was IPV associated with greater reports of poor health (adjusted relative risk (aRR) 2.0, 95%

CI 1.3-2.9), heavy alcohol use (aRR 2.6, 95% CI 1.6-4.3), the development of a chronic disease (aRR 1.6, 95% CI 1.3-2.0), and extreme interferences in daily life because of a chronic condition (aRR 3.1, 95% CI 1.6-6.0). Indeed, using national data from Australia, Vos and colleagues[48] have shown that among women aged 15-44, IPV accounted for 8% (95% CI 6-10%) of the total disease and injury burden and was a greater risk factor for poor health than blood pressure, cholesterol, tobacco use, alcohol and substance use, and body weight.

Sexual and reproductive health

In addition to its general physical health effects, perpetrating IPV against women can result in unintended pregnancy by directly (e.g., through actual or threatened force) or indirectly (e.g., by creating difficulties or fears in negotiating contraception) coercing women into having unprotected sex.[49,50] The WHO multi-country study estimated that across all 15 study sites, the proportions of unintended pregnancy and abortion that could be attributed to physical or sexual IPV were 15% and 30%, respectively.[51] This means that IPV may have even further detrimental effects for women when they only have access to unsafe abortion, a leading cause of maternal mortality.[52] Likewise, in cases where abortion is not used, unintended pregnancies may have negative child and maternal health outcomes.[53,54]

If IPV involves forced sex, it can also cause genital trauma and chronic pain. In a systematic review of studies investigating IPV against women and sexual health outcomes, Coker[55] found that IPV was consistently associated with chronic pelvic or abdominal pain (9/10 studies), pain during intercourse (8/8), and painful menstruation (5/5). By increasing

the risk for or directly involving unprotected sex, IPV against women is also associated with increased risk for sexually transmitted infections (STIs). In Coker's systematic review, all three included longitudinal studies (all retrospective) found that more women who had a history of IPV had an STI compared to those without any IPV history. This may also be explained by the review's findings that women who have been victimised by IPV tend to engage in riskier sexual behaviours (e.g., alcohol use before sex, multiple sex partners; 8/10 studies) and be involved in relationships where she, her partner, or both are non-monogamous (8/8).

Mental health

Beyond physical health, IPV has important implications for women's mental health. For instance, two systematic, meta-analytic reviews of longitudinal studies by Devries and colleagues have shown that IPV against women is associated with incident depressive symptoms (N=6 studies, n=25,826 women, pooled OR=1.97, 95% CI 1.56-2.48)[56] and heavier subsequent alcohol use (N=5 studies, n=4,709 women, pooled OR=1.25, 95% CI 1.02-1.52).[57] The authors also found that IPV was associated with suicidal attempts among women in 2 of 3 studies, however, these studies only measured lifetime prevalence of suicide attempts (obscuring the temporality of the association) and the review authors did not compute a pooled effect size. These reviews only focused on physical or sexual IPV to the exclusion of psychological IPV, which may have important ramifications for victims' mental health.[47] A later systematic review that focused on all three types of IPV, but analysed both cross-sectional and longitudinal studies and did not meta-analyse effect estimates, also found that IPV tended to be associated with depression as well as post-traumatic stress disorder (PTSD), anxiety, and somatisation (physical

symptoms resulting from psychological distress).

There remain gaps in the evidence base for the mental health consequences of IPV against women, given the relatively few prospective longitudinal studies and limited systematic reviews. Longitudinal studies that measure change in mental health status following IPV are essential for understanding these effects given that women with existing mental health problems may be at greater risk for being victimised by IPV.[58] Nevertheless, there are plausible pathways for the mental health consequences of IPV against women. For instance, IPV against women may entail a traumatic incident of actual or threatened injury or near-death that causes fear or helplessness; actual or fear of repeated victimisation may lead to re-traumatisation, anxiety, and depression; psychological degradation may decrease self-worth and increase depression; and women may rely on negative coping strategies like alcohol and substance use.[59,60] At least one prospective longitudinal study in New Zealand lends additional support for these relationships.[61] The authors found that, when controlling for each mental health outcome at Age 18, being in a physically abusive relationship between Ages 24-26 was associated with a major depressive episode (OR=2.46, 95% CI 1.16-5.26), marijuana dependence (OR=10.14, 95% CI 3.62-28.39), and PTSD (OR=6.42, 95% CI 2.28-18.04) among women (N=449) at Age 26. In contrast, none of these relationships were clinically meaningful for men, with small and imprecise estimates of association.

These findings support the findings of reviews of gender differences in mental health following IPV, which have generally found that women experience more negative mental health consequences compared to men or no significant differences,[56,62] although no known comparative meta-analyses have been conducted. In their Australian study of

the contribution of IPV to the burden of disease and injury among women, Vos and colleagues found that 73% of the burden caused by IPV was associated with depression, anxiety, and suicide, highlighting both the psychological distress that IPV can create for women as well as the negative impacts on physical health this can have.[48] An additional 22% of this burden was accounted for by harmful health behaviours – namely alcohol, tobacco, and illicit drug use – which may also be correlated with poorer mental health among women.[63]

Socioeconomic impact

Not only can IPV affect women's health, but it can increase their economic and social vulnerabilities. For one, experiences of IPV may affect women's ability to attain or maintain employment. This may occur because of the perpetrator's direct interference in the woman's employment – through preventing her from going to work, sabotaging her efforts to gain employment, or stalking her at work – or indirectly by limiting her productivity – from late arrivals to work, missed days, or difficulties concentrating – due to the physical and/or psychological effects of the violence.[64] For instance, in a qualitative study of 10 women (aged 20-47) living in a domestic violence shelter in North Dakota, USA, the majority of women indicated that their violent partners significantly disrupted their work lives through their jealousy and anger about the women's work relationships, harassment through phone calls or coming to the women's workplace, costing the women work opportunities, and lowering the women's self-confidence in her work abilities.[65] Additionally, nine of the 10 women interviewed indicated they had no control over their own finances, increasing their economic dependence on their violent partner and entrenching their losses upon leaving the relationship.

In relying on a shelter sample, the above study likely represents the effects of the most severe forms of IPV. In a fixed-effects analysis of prospective longitudinal data from a broader sample of women receiving welfare in the USA (N=598), physical or sexual IPV victimisation was associated with fewer annual work hours, controlling for all time-invariant factors (e.g., age), mental health problems, physical health problems, cohabiting status with partner, number of young children, household size, transportation problems, and child health problems (coefficient=-136.85, standard error (SE)=59.02).[66] It may be the case that women with lower income or living in poverty are most vulnerable to the economic impacts of IPV. Indeed, early cross-sectional studies showed that having a lower paying job or receiving welfare was associated with staying in an abusive relationships for longer, which may further exacerbate women's economic dependencies on their partners.[67]

Given the high potential for economic dependency, attempting to leave following IPV may result in housing instability and, at worst, homelessness. In a cross-sectional study in a random sample of adult Californian women (N=3,619), Pavao and colleagues found that past-year experiences of IPV were associated with past-year housing instability (late rent or mortgage payments, homelessness, or difficulties finding safe, adequate, and affordable housing), independent of age, race/ethnicity, education, poverty status, marital status, and children in the household (OR=3.98, 95% CI 2.94-5.39).[68] Although no prospective longitudinal studies could be found, evidence for the relationship between IPV and homelessness is perhaps best described by the number of women in domestic violence and emergency shelters. For instance, from 2014-2015 in England alone, 6,189 women were supported by refuge services, from a sub-sample of 162 services that responded to the survey (out of a total of 356 domestic abuse services in the country).[69] Although far

from a perfect measure of the number of women requiring emergency or long-term refuge following IPV, this figure demonstrates that housing instability affects a significant number of female victims. It also must be noted that given women's fewer economic resources overall compared to men, the socioeconomic impacts of IPV are most strongly felt by them as opposed to male victims.[67,70]

The economic costs of IPV against women are high for both those who experience it as well as society. An analysis of cases of IPV against women in the USA from 1992-1996 estimated that the annual total costs were: $4 billion for medical and mental healthcare, $728 million from lost productivity from employment, and $131 million from lost productivity from housework, resulting in a total cost of $6 billion.[71] This study further estimated that victims were responsible for 25% to 35% of healthcare costs. Evaluations of the costs of IPV are similarly high in the UK. According to the most recent estimates (based on 2008 data), services for victims (criminal justice, health, social, housing, and civil legal) cost £4 billion, lost economic output costs £2 billion, and the human and emotional costs total £10 billion, for an overall annual cost of £16 billion for the state, employers, and victims.[72] These figures represent the costs of IPV against both women and men, however, they are based on findings from the British Crime Survey (now the Crime Survey for England and Wales) self-completion module, which consistently finds that women experience more IPV than men.[28] Together, these economic evaluations serve to illustrate that in addition to the vast burden of psychosocial and health outcomes that women may face following IPV, IPV places a serious economic burden on both the women who experience this violence and society at large.

1.1.3 Measurement of IPV

Official data: the complexity of help-seeking and underreporting

Given its many negative consequences, there is a clear need to reduce the problem of IPV against women. However, to effectively monitor and reduce the problem of IPV against women, we need to know how best to measure it – a notoriously difficult task. Official data, including police reports and hospital visits, tend to grossly underestimate the incidence and prevalence of IPV, as the vast majority of women who experience this violence do not contact formal services.[73] For instance, among women who indicated that they had experienced IPV in the last year in the 2015 Crime Survey for England and Wales (latest available estimates of help-seeking), only 43% told someone in an 'official position' (e.g., police, health professionals, legal professionals, local council department, or other government agency).[28] Similarly, only 32% told another support or professional organisation, such as a therapist, victim support, helpline, or support service. This suggests that if we solely rely on contacts to these formal services, we would only capture the tip of the iceberg of IPV incidents against women.[74]

There are many reasons why women may choose not to report on or seek help following IPV. In a mixed-methods study of women who reported ever experiencing physical or sexual IPV, randomly sampled from one urban and one rural area in New Zealand (N=956), only 31% of women who experienced IPV had told formal services.[75] Women's odds of contacting formal services were higher when they had experienced severe physical violence as opposed to sexual violence (OR=3.7, 95% CI 2.2-6.7) or moderate physical violence (OR=3.1, 95% CI 2.0-4.7), controlling for socioeconomic

factors and marital status. In giving reasons for why they did not seek help from formal services, 63% of women said they felt the violence was 'normal or not serious', 14% reported feeling embarrassed, 10% felt that they should deal with it on their own, and 6% said that they 'feared the consequences'. Moreover, even though most women (77%) had told at least a friend or family member about the violence they experienced, less than half (40%) reported that anyone tried to help them.

These results illustrate that women may not seek help following IPV because it has been normalised for them or out of fear or embarrassment. This is perhaps more strongly felt regarding sexual as opposed to physical IPV and more so when the physical violence is moderate as opposed to severe (in which case women may have less of a choice about disclosure due to resulting injuries). Furthermore, the inadequate help received by women upon disclosure may dissuade them from reporting further victimisation in the future. These themes were well-reflected in a meta-synthesis of qualitative studies on women's experiences with healthcare professionals following IPV, which found that barriers to disclosure were the women's feelings regarding her abuse (such as shame, embarrassment, self-blame, denial, and humiliation), fear of the repercussions of disclosing, and perceptions of the professionals as not respecting confidentiality, being judgmental, putting pressure on the women, lacking compassion, and not validating the women's experiences.[76] All of these factors result in not only the inadequate treatment and further prevention of IPV against women but also the underestimation of the problem in official data.

Survey data: quality of methods and scales

In contrast, survey data on IPV from women are typically thought to be more accurate, as women are more likely to self-report experiences of IPV than seek help from formal services.[77] However, there is wide variability in the quality and accuracy of IPV estimates obtained from different survey methods. Firstly, how the survey is conducted will influence whether participants will disclose experiences of IPV. For instance, the Crime Survey for England and Wales conducts both a self-completion module and face-to-face interviews on IPV, which allows for direct comparison of the two methods. An analysis of the 2012/2013 survey round showed that only 19% of women who disclosed incidents of IPV in the self-completion module reported these incidents in their face-to-face interview.[28] This suggests that women felt more comfortable disclosing their experiences privately than to an interviewer, where they may have felt their confidentiality and anonymity were at risk or been affected by factors similar to those described above, such as personal feelings and perceptions of the interviewer.

Nevertheless, it should not be assumed that all interview methods will underestimate IPV. On the contrary, researchers argue that face-to-face interviews will be effective in measuring IPV among women when the interviewers are female, experienced and trained in discussing sensitive issues, and IPV in particular, and able to show compassion, empathy, and emotional maturity.[78] These methods were applied in the WHO multi-country study, which found that across study sites between 21-46% of women who had never disclosed their experience of IPV did so for the first time in the study interview. This suggests that the trained interviewers, most likely in contrast to the Crime Survey for England and Wales, built the trust and support required for disclosure. Taken together, these findings show that trained interviewers and self-completed questionnaires can be an effective method for collecting data on IPV, although the extent of underreporting can

never be truly estimated. These results further demonstrate the risk of misapplied study methods, which can skew understanding of the problem of IPV against women.

The quality of IPV data, regardless of survey mode, will further be contingent on the quality of the measure used. There exists considerable variability in the survey measures used for IPV.[33] The field has generally moved towards using measures of specific behaviours that constitute IPV (e.g., partner hit woman, used weapons against her, or used force to make her have sex), as opposed to questions that use vague or loaded terms such as 'violence', 'abuse', or 'rape'.[16,77] This is because women may not identify their experiences of IPV with these latter terms – for instance, a woman may perceive her experiences of IPV as normal and therefore not through the especially negative or abnormal lens of 'violence' – and thus these terms are more likely to underestimate occurrences of IPV.

At present, the most widely used measure for IPV is the Conflict Tactics Scale[79] or its revised version,[80] which ask respondents to indicate whether they have experienced specific behaviours by a current or previous dating, cohabiting, or marital partner in the context of conflicts, disagreements, or 'settling differences' within a set period of time (e.g., the last 12 months). Both these versions have sub-scales on the use of reasoning or negotiation (revised scale: 6 items, e.g., "My partner suggested a compromise to an argument"), physical assault (revised: 12 items, e.g., "My partner pushed or shoved me"), and psychological violence (revised: 8 items, e.g., "My partner called me fat or ugly"). The difference between these two scales is that the revised version has new or revised items for each of these three sub-scales as well as two new sub-scales on sexual coercion (7 items, e.g., "My partner used force to make me have oral or anal sex") and physical

injury resulting from physical assault (6 items, e.g., "I was cut or bleeding").

Studies that do not use the Conflict Tactics Scale typically use different behavioural measures (i.e., other inventories of specific violent behaviours) or single items (e.g., "Has your partner slapped, hit, or pushed you in the past year?"). While single items are generally insufficient to measure the complexity and range of IPV, multi-item scales vary in terms of their content and length. Many scales – including the Conflict Tactics Scales as well as alternatives such as the Composite Abuse Scale,[81,82] Abusive Behavior Inventory,[83] Severity of Violence Against Women Scale,[84] and Measure of Wife Abuse [85] – have demonstrated adequate internal consistency (α>.70) and evidence of validity (e.g., convergent or factorial).[33] However, these scales differ in terms of the breadth of acts measured and the circumstances under which they occur, from the context of conflict resolution (Conflict Tactics Scale) to the impact of each act on women's wellbeing. This is perhaps more pronounced in scales for psychological IPV, which has been studied less than physical or sexual IPV, due to the greater complexities in operationalising the different types of psychological violence, which range from verbal abuse to controlling behaviours.[86,87]

Although it is the measure used most widely in IPV research, the Conflict Tactics Scale is not without criticism for many of the reasons touched on above. The most extensive criticism lodged against the scale is that it does not measure the attitudes, emotions, and intent behind the violent behaviours it measures.[88] As a result, the scale essentially equates initial acts of violence with those of self-defense or retaliation and treats each violent act as equivalent, regardless of severity, consequence, or intent.[5] Indeed, at least one validity study found that participants completing the Conflict Tactics

Scale recorded acts of play or mock violence as well as self-defense.[89] Moreover, the Conflict Tactics Scale only asks about violence under the pretext of 'conflict resolution', thereby limiting measurement of IPV that may be part of an overall pattern of control and domination of one partner over the other, as opposed to (solely) a reaction to conflict.[90] Finally, the scale does not measure acts of stalking, controlling behaviours, or any other type of sexual coercion beyond forced or coerced intercourse, which can be an important component of women's experiences of IPV.

Straus, the creator of the scale, has repeatedly denied the validity of these criticisms.[6,80,88,91] He argues that because the measure sub-divides physical assault into 'severe' items (e.g., punching, kicking, or choking) versus 'moderate' (e.g., slapping or throwing things at partner) it precludes equivalency among all items. However, this serves to falsely distinguish the severity of acts without considering their consequences, which is the true marker of severity. For instance, throwing an object at a person could be a very severe act of violence, depending on the strength of the throw and magnitude of the object, whereas kicking someone could be more or less severe depending on the strength, placement, and intent of the kick.[90] Straus argues, however, that criticisms against the Conflict Tactics Scale's lack of measuring context and consequences are redundant as the scale need only measure the occurrence of violent behaviour, not why it occurs or its effects. Specifically, he equates this to criticising a reading ability test for indicating only that a child reads poorly not why. However, this argument remains inadequate: the severity and consequences of violent acts are integral to their definition and the burden of the problem, and are essential to measure and monitor so that we can ensure we are reducing the most impactful forms of IPV. Failing to do so, as Kimmel illustrates:

> *Is more akin to a teacher who does not look at how far off the spelling mistakes are or whether there is a pattern in the mistakes that might point to a physiological*

> *problem such as dyslexia or some other learning disability rather than academic laziness and thus leaves the learning problems untouched and misdirects funds away from remediation toward punitive after-school programs for lazy students […] Can one imagine any other issue in which causes and consequences are thought to be irrelevant? (p. 1346).*[90]

If the Conflict Tactics Scale fails to accurately capture the severity and impact of the violent acts it measures, it may skew our understanding of the burden of IPV, just as other insensitive study methods have done.

1.1.4 Typologies of IPV and the debate on gender asymmetry

Debates on the Conflict Tactic Scale and more broadly the measurement of IPV are critical because they represent a larger theoretical divide in the field. On one side are researchers who believe that IPV should be defined as another type of criminal or family violence that is equally a problem among both women and men.[6,92] These researchers tend to cite family conflict surveys, which typically use the Conflict Tactics Scale and find roughly equivalent prevalence estimates of IPV among women and men.[93] In contrast, other researchers argue that IPV is part of a broader group of violent acts perpetrated against women within a social context that values men over women; in other words, there are gendered components to IPV and women are especially at risk.[14,24,73,94] These researchers tend to cite crime victimisation surveys and clinical studies, whether defined by police or judicial reports, healthcare visits, or domestic violence shelter use, where the vast majority of victims are women and perpetrators are men.[90,93] At least three measurement-based reasons have been given for this discrepancy. First, general or student population surveys on family conflict typically include only intact dating, cohabiting, or married couples, while crime victimisation and clinical surveys typically include former partners as well, who we know pose a serious risk for more severe IPV against women

(especially immediately after separation) as was discussed in Section 1.1.2.[95] Second, women experiencing severe IPV may be less inclined to participate in a general survey on family or relationship violence out of fear that their partners would find out and male perpetrators may likewise not respond so as not to criminalise themselves – whereas contact with formal services may be less of a choice.[96] Third, crime victimisation surveys may explicitly or implicitly illicit fears or threats to safety as a frame of reference for violent behaviours, generating estimates of more severe violence, whereas the family conflict surveys, using scales like the Conflict Tactics Scale, measure a wider range of behaviours used in contexts of conflict.[97]

Beyond measurement differences, researchers have suggested that these discrepant results exist because there is more than one typology of IPV. The most widely discussed categorisation is by M. P. Johnson, who originally put forward two types of IPV[96] and then revised this to include four types:[98] (a) intimate terrorism, where one partner is violent and controlling and the other is not; (b) violent resistance, where one partner is violent and controlling and the other is violent but not controlling; (c) situational couple violence, where one or both partners are violent but neither are controlling; and (d) mutual violent control, where both partners are violent and controlling. Johnson's central argument is that family conflict surveys most commonly capture situational couple violence, which suggests that violence is perpetrated by both men and women (i.e., gender symmetric). In contrast, clinical data and crime surveys capture mainly intimate terrorism and violent resistance – showing that men are most often the violent, controlling partner towards women – and, less commonly due to its low prevalence, mutual violent control (which, by definition, will be gender symmetric in heterosexual couples). Johnson confirmed his 4-category typology of IPV using a case-control dataset from Pennsylvania (women selected

from shelters and court cases matched with women in the same neighbourhoods, N=274), using seven measures of control that showed factor validity to differentiate types of violence: threats, economic control, use of privilege or punishment as coercion, using children for coercion, isolation tactics, emotional abuse, and sexual control.[98,99] He found that 97% of intimate terrorism was committed by husbands, 96% of violent resistors were wives, while situational couple violence was more gender symmetric (56% husbands and 44% wives), confirming his gender (a)symmetry hypotheses.

Since its conception, testing of Johnson's typology and gender asymmetry has been limited by the fact that few datasets exist that are likely to capture the entire typology of violence and control hypothesised by Johnson.[95] Where studies have been able to access both clinical and general survey data, they have found support for Johnson's typology and its gender predictions.[99,100] However, these studies have been criticised for being tautological in the sense that they have selected participants likely to satisfy the four patterns of IPV predicted by the typology; thus, *a priori* testing of this typology is still needed.[101,102] Perhaps more crucially, cross-sectional studies have also found that controlling behaviour and variation in the use and severity of physical violence are correlated: in other words, controlling behaviour may be a predictor or marker of severe physical or sexual violence, rather than a strict distinguisher of different classes of IPV.[103-105] Controlling behaviours may still be used but perhaps to a lesser extent in situational couple violence, suggesting that rather than mutually exclusive classes of IPV, there is a multi-dimensional spectrum of controlling behaviour, physical violence, and severity, along which male-to-female violence may tend to congregate at the more extreme end.[106]

Studies that are used to refute this argument have often produced findings to

support it upon further inspection. For instance, a widely cited New Zealand study, which claimed to find that men and women (N=955) were equally involved in clinically abusive relationships, defined clinical abuse based on whether *either* the man or woman was physically injured or sought help.[107] As a result, a man being classified as involved in a clinically abusive relationship may actually have been in a relationship in which only the woman experienced IPV with clinical consequences. Moreover, the study found that while on average both women and men reported that women perpetrated any violence in their relationships, they disagreed as to whether women or men perpetrated *more* violence, with women reporting that men perpetrated more and men reporting the opposite. Despite this disagreement, however, both genders agreed that men perpetrated *more* violence in clinically abusive relationships than in relationships without clinical consequences. Moreover, with male perpetration, IPV lasted for more months and involved more incidents per month, more men than women were convicted of domestic violence, more women than men required medical care for injuries, and women, but not men, experienced more controlling behaviours in clinically abusive relationships compared to those without clinical consequences. Finally, and perhaps most critical to conclusions of gender asymmetry, when only women were reported to have perpetrated violence in the relationship, these clinical consequences did not occur (p. 268). These findings thus support the heterogeneity of IPV severity, with women experiencing more severe and controlling violence on average.

Other typologies have been suggested to explain heterogeneity in IPV prevalence findings that instead classify perpetrators of IPV into different categories. For instance, Holtzworth-Munroe and Stuart reviewed 15 typologies of male perpetrators of IPV against women and determined that perpetrators tended to vary on three common dimensions: the

severity of IPV perpetrated, the scope of their violence beyond the family, and the presence of psychopathology or personality disorders.[108] They resultantly developed a typology with three categories of male IPV perpetrators: (a) family-only perpetrators, who engage in the least severe violence and are least likely to engage in psychological or sexual IPV, are least likely to perpetrate violence beyond their family members, and have little psychopathology; (b) borderline-dysphoric, who engage in moderate to severe IPV, commit violence mainly within their home, and are the most psychologically distressed and emotionally volatile; and (c) generally violent-antisocial, who engage in moderate to severe IPV, engage in the most violence outside the home, and are the most likely to have antisocial tendencies or extreme psychological disorders. The family-only perpetrators would be most similar to Johnson's situational couple violence, whereas borderline-dysphoric and generally violent-antisocial perpetrators would be classified as two types of intimate terrorists.[99] However, further tests of this typology have found that it does not classify all male perpetrators of IPV[109,110] and these subtypes do not always remain stable over time.[110,111] These results suggest that male perpetration of IPV is likely a varying construct, but that static typologies may not be able to address the changes in relationships that occur over time. Similar to the conclusions above regarding Johnson's typology, the authors have suggested the utility of a dimensional as opposed to a typological approach to defining perpetrators of IPV, where antisocial and borderline personality characteristics predict trajectories of future violence (e.g., low-level, escalating, or severe).[110]

Critical summary and steps forward

While the debate on gender symmetry and typologies in IPV prevalence remains unresolved, there are important lessons that can be gleaned to improve etiological research

of IPV against women and which will be adopted in this thesis. First, as is clear from the discussion above, the debate has highlighted that IPV is not a unitary phenomenon, and etiological analyses should consider the spectrum of acts and consequences involved. This is well reflected in the discussion in Section 1.1.1 on many women's multidimensional experiences of IPV (involving physical, sexual, and psychological acts), which are not encapsulated when we only consider whether any act of IPV has occurred. Second, even when severe violence is being perpetrated against a woman by an intimate partner, this does not necessarily entail that the woman experiencing abuse is passive: bidirectional violence within intimate relationships is not an uncommon phenomenon.[93,107] Nevertheless, the occurrence of bidirectional violence should not automatically equate the motivations, severity, and consequences of each partner's violence.[112] This emphasises why it is beneficial to consider the relationship-level factors that contribute to IPV against women, rather than just the woman's or man's circumstances on their own, as well as the community and structural-level factors that may create more stressful or toxic relationship environments.

Despite the intransigence of researchers on each side of the gender (a)symmetry debate on IPV prevalence, there are several factors that continue to support the utility of developing etiological theory for IPV against women in particular. First, researchers in both 'camps' nearly universally acknowledge the gender asymmetry in outcomes, detailed in Section 1.1.2.[6,113] Specifically, that women face a greater burden of the consequences of IPV than men, including death, injury, poor health, mental health problems, socioeconomic impact, as well as more fear of their partner. Second, where meta-analyses have been conducted on risk and protective factors for IPV victimisation among both women and men, gender differences have been found (e.g., for depressive symptoms,[56]

marital satisfaction,[114] and witnessing or experiencing violence in childhood[115]). Additionally, there is evidence that the risk factors for more severe IPV, which may more commonly occur from men to women rather than vice versa may be different.[105,107,110]

Finally, what is perhaps most often neglected or underestimated by family conflict proponents is that IPV does not occur in a social vacuum: rather IPV against women occurs in a societal context where overall women have lower social and economic power and status as compared to men.[70,116,117] For one, this makes it more difficult for women to leave violent relationships than men on average, given the typically fewer resources at their disposal and greater childcare responsibilities. These gender disparities may also affect how and why violent behaviours are used. For instance, in a cross-sectional study of women and men in shelters, prisons, and universities in England (N=113), Graham-Kevan and Archer found that while some women used controlling behaviours to intimidate and threaten partners, those in the most severely violent relationships only used controlling behaviours to gain economic resources, whereas men used controlling behaviours to intimidate and threaten their partner, undermine their partner's self-image, and restrict their partner's personal and economic freedom.[104] Furthermore, internalised gender norms may differentially affect the occurrence and interpretation of IPV against men and women (e.g., some male perpetrators may feel it is their right to control the relationship, female victims may normalise this violence).[90,118]

This thesis thus adopts the perspective that the gendered aspects of IPV against women should be recognised (given the greater number and severity of consequences women experience because of this violence compared to male victims and evidence of the differing risk/protective factors for these two groups). However, doing so does not

necessitate the denial that IPV occurs against men and by women – indeed this should not be denied. It does suggest though that there may be important etiological and clinical differences between these problems overall and it is reasonable to investigate separate problem definitions and intervention strategies for IPV against women. As is detailed in Chapter 2, the empirical analyses of this thesis evaluate gender differences in the prevalence and impacts of IPV in the primary sample and Chapter 7 includes a broader discussion of future directions for the IPV research and intervention fields considering these results and the broader spectrum of gender and sexual identities.

1.2 Theories of IPV

The ongoing debates on the heterogeneity of IPV prevalence findings demonstrate well that many questions remain in our etiological understanding of IPV. An important reason for this is the huge production of cross-sectional studies on IPV prevalence and correlates, and a lack of focus on (or capacity for) prospective theory testing of causal models. While a systematic review of evidence on the risk and protective factors for IPV against women is presented in detail in Chapter 3, below is an introduction to the main theories that exist today on the occurrence of IPV against women and their limitations.

1.2.1 Structural versus individual theories

Theories about the causes of IPV have historically fallen along similar lines to those drawn in the debates regarding IPV heterogeneity: on one side were feminist theorists who tended to emphasise the societal structures that perpetuate violence against women, while on the other side were theorists who tended to focus more on individual

characteristics of victims or perpetrators.[24,119,120] Feminist theorists have posited that IPV against women is the result of the patriarchal structure of society, which assigns greater power and resources to men over women and thus directly and indirectly supports the exploitation of women and condones their subordination.[14] In other words, IPV against women is viewed as a method for men to dominate and control women within their intimate relationships, mirroring men's dominance and control of women in broader society. However, this traditional feminist theory of IPV, for one, fails to explain why some men use IPV and most others do not.[24]

In contrast, a number of individual-level theories have been posited to account for individual differences in IPV perpetration and victimisation. First, based on Bandura's *social learning theory*,[121] sociologists and psychologists have suggested that IPV is a learned method of conflict resolution that begins with children being exposed to aggression and violence in their family of origin, either through family members abusing the child or the child witnessing abuse or violence between her/his family members (e.g., parents).[122] This results in the 'intergenerational transmission of violence' via the normalisation of violence or its positive reinforcement (e.g., because of agency gained, ability to control, or anger expression).[123] Second, several research groups have suggested that individual psychopathology and personality characteristics increase the likelihood of becoming a perpetrator of IPV. For instance, both Dutton[124] and Holtzworth-Munroe and Stuart[108] have posited that borderline personality – characterised by unstable affect, poor impulse control, and intense anger – and antisocial personality – a long-term pattern of self-centred behaviour that involves manipulating and using others for personal gain – are associated with increased risk of IPV perpetration. These behaviours and personalities are thought to develop largely through genetic propensities and childhood exposures (e.g., a

history of insecure attachment to other individuals characterised by fear of rejection). On their own, however, these individual-level theories fail to account for the fact that many perpetrators do not have mental health problems or histories of witnessing or experiencing abuse and vice versa.

In addition to individual and structural theories, family-level theories have been proposed by family violence researchers, largely from sociology. These theorists view IPV as one type of family violence, which all have a common set of causes. *Systems theory*, for instance, posits that conflict is a normal part of families, but whether the conflict will escalate into violence depends on the structure of the family system.[125] A central tenet of this theory is thus that violence is not an aberration or the result of psychopathology; rather family violence is produced by a combination of family conditions – such as socialised roles of family members, cognitive appraisals, and stress levels – that interact and provide positive (violence promoting) or negative (violence deterring) feedback. Similarly hypothesising about family structures, *social exchange/control theories* begin by positing that in all family interactions, as in all human behaviour, actors are seeking to gain benefits and avoid costs. Family violence then occurs when the benefits of this behaviour outweigh the costs, there is a lack of social controls in the family to promote social order and deter violence, and family and social structures (e.g., gender and power inequality in the family, attitudes that family matters are private, and conceptions of masculinity as entailing violence and aggression) serve to reinforce violence rather than deter it.[120,126]

Finally, *resource theory* views violence as another resource available for family members to attain their goals: if a partner has few resources available to them they will use violence to meet their goals and gain power.[127] *Relative resource theory* further posits that

disparity in resources between partners – i.e., if the woman has higher status over the man, for instance in terms of income or employment – increase the woman's risk of experiencing violence, particularly in lower-income families with fewer available resources overall.[128] However, it has similarly been argued that if a woman has lower status in the relationship, her risk of victimisation will increase because of her economic dependence on the relationship, which makes it difficult for her to negotiate change or leave the relationship.[129] Altogether, in their consideration of the interaction between individual and structure (at the family level), these family-level theories represent an improvement upon theories that only consider IPV in the context of the former or latter. However, in failing to acknowledge the broader ideological, contextual, and structural factors that affect the occurrence of IPV (e.g., when resource disparities may deter violence or exacerbate it), family-level theories remain insufficient to account for the complex etiology of IPV.[130]

1.2.2 'An integrated, ecological framework'

To address the growing need for a theory of IPV against women that can integrate individual, family, and structural factors, ecological theories have been proposed.[24,131,132] These are based on Bronfenbrenner's *ecological theory of human development*,[133] which posited that human development is the product of the environments within which people are nested. Bronfenbrenner proposed four levels at which environmental factors may interact with the developing person or each other: (a) the microsystem, which involves interactions between the person and her/his immediate settings (e.g., family); (b) mesosystem, interactions between the microsystems (e.g., between family, work, and peers); (c) exosystem, interactions between social structures that do not contain the

developing person (e.g., the neighbourhood, the workplace, informal social networks) but influence the microsystems in which they reside; and (d) macrosystem, cultural institutions (e.g., laws, cultural values) that determine the micro-, meso-, and exosystems.

Belsky was the first to apply Bronfenbrenner's model to violence – specifically, child maltreatment.[134] In his seminal paper, he replaced the mesosystem with an alternative level: ontogenic development, representing the personal history that each parent who mistreats their child brings with them to the family setting. The intuitive appeal of this multilevel, multifactorial model led to its application to a variety of social and health problems, including IPV. The most well-known application of ecological theory to IPV against women is by Heise,[24] whose 'integrated, ecological framework' guides the public health conception of IPV risk today.[23,26] Heise used the model put forward by Belsky and conceptualised the ecological model of IPV to involve: (a) the personal history of the target person that affect her relationships and behaviour, (b) microsystem, which encompasses the immediate context within which the abuse takes place (i.e., the relationship), (c) exosystem, which involves the informal and formal social structures and institutions in which the microsystem is situated (i.e., work, neighbourhood, social networks), and (d) macrosystem, representing the sociocultural views and organisation of society at large. In modern presentations, the ecological theory of IPV is typically conceived of as shown in Figure 1.1, with individual, relationship, community, and structural/societal risk and protective factors.[19,23,25,26,101] Consequently in the current thesis, *community-level risk and protective factors* refer to any characteristics of women's communities (whether residential neighbourhoods, school, or workplace) that may be determined from routine or census data (e.g., neighbourhood crime) or self-reported; whereas *structural risk and protective factors* include resource distribution, policies, and

societal norms and values, which would typically be defined at a contextual level (e.g., routine or census data, aggregated individual-level data, legislation). Notably, these levels are not necessarily mutually exclusive: for instance, a community-level factor such as neighbourhood deprivation is also related to structural-level resource distribution, and so may be reasonably defined as both a community and structural factor.

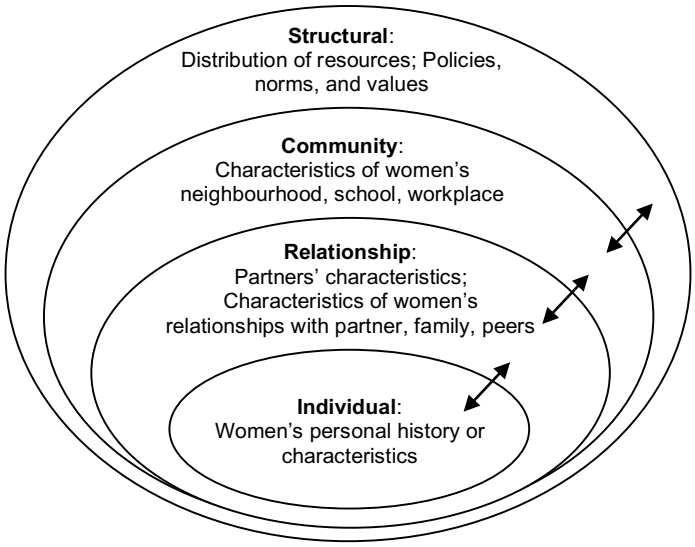

Figure 1.1 Ecological model of risk and protective factors for IPV against women[19,23,25,26,101]

The strength of the ecological framework of IPV against women is that it allows for the integration of the individual, family, and structural focused theories. Consequently, researchers using this framework can recognise the contribution of each of these levels of factors to the occurrence and maintenance of IPV but also that no level of factors on its own is sufficient for explaining this phenomenon. This is particularly important for the field of IPV, because it allows for common ground between feminist and family violence researchers: whereby individual personality and psychopathology, developmental history, and relationship/family dynamics are recognised as important contributors to IPV, but as are structural factors such as gender norms and resource inequities that interact with and

help shape these more proximal factors.[120]

Nevertheless, the wide application of this framework in etiological research has also had two major drawbacks. First, accepting the ecological model has often led to limited theorising about the causal pathways of the multi-level risk and protective factors proposed. Most of the theories described above only apply to individual or relationship-level factors; but the ecological theory posits that community and structural factors matter for IPV, providing little theory as to how. Resultantly, studies that have tested the associations between community and structural risk and protective factors often provide little in the way of theory development and instead operate as a 'checklist of associations'.[135,136] In addition, failing to explicitly consider or account for these causal pathways in study design has led to inadequate measurement or analytic control of the variables most likely to bias estimates of these associations.[137,138]

Second, because of the intuitive appeal of the theory, there has been little attempt to systematically review it in its entirety and across contexts. Consequently, all modern reviews that attempt to review the ecological model for IPV against women without context restrictions have been non-systematic.[25,139,140] Taken together, both of these drawbacks have contributed to the large majority of both primary and systematic review evidence for IPV against women being focused on individual and relationship-level factors, with a relative dearth of rigorous studies on community and structural factors. As a result, the field has been left with a poor understanding of the causal role of community and structural risk factors in IPV against women and how these should be integrated into an overall theoretical model.

1.3 Potential role of community and structural environments

The following section describes in further detail the potential of community and structural environments to affect IPV against women. In particular, it describes the history of contextual effects in public health and criminological research and the relevance of these to IPV. The methodological challenges of investigating these effects – including how communities should be defined, the difference between contextual and compositional effects, and confounding bias – are discussed in Chapter 2.

1.3.1 Scientific interest in community and structural effects

The hypothesis that the community and structural contexts in which people reside affect their behaviour, health, and wellbeing has a long history[141] and over the last three decades has grown in popularity in sociology, criminology, and public health.[142-149] This research has demonstrated that negative health outcomes (such as mortality and disease) and criminality (including homicide, violent victimisation, and delinquency) are not distributed evenly, but rather are related to social inequalities and concentrated in neighbourhoods and communities characterised by greater poverty, high unemployment, lower social status (e.g., due to migrant status or race/ethnicity), and greater residential instability. In what follows neighbourhoods and communities are considered to be spatially defined areas, although what the borders should be and whether (and how) social as opposed to only physical spaces should be included are discussed further in Chapter 2.

Several theories have been put forward as to why these community and structural effects exist. First, *social disorganisation theory*[141] posits that it is more difficult to

establish and sustain social bonds in neighbourhoods with high poverty, ethnic heterogeneity, and residential instability and thus, without this social cohesion, negative outcomes like crime and delinquency will be left uninhibited without informal social controls. However, many researchers have taken issue with social disorganisation theory, arguing that strong or dense social ties are not always positive – consider, for instance, criminal networks – and likewise weak social ties do not always lead to worse outcomes (and may in fact be more realistic in today's large urban centres).[147] Sampson and colleagues consequently adapted social disorganisation theory to develop a theory of *collective efficacy*, which maintained that social control is essential to reducing crime and other negative outcomes, but that it is established through mutual trust between community members and the shared will to intervene for the sake of the common good, rather than through dense social ties alone.[150] Finally, two related theories have been proposed that particularly incorporate a developmental component to the association between communities, societies, and violence. The *subculture of violence* theory suggests that violence and crime are concentrated in specific areas where there is a normative value system that supports violence and crime.[151] People living in the subculture learn these values and norms that are accepting of violence over time through socialisation, so continuing the cycle. Similarly, Sampson and Wilson hypothesised that these values and norms represent a *cognitive landscape* within the community that establish a perceived or learned standard of behaviour and conduct that is considered acceptable.[152] Together, these theories provide potential mechanisms via which sustained social inequalities and exposure to neighbourhood disadvantage may cause violence and crime – at least in the public sphere.

1.3.2 Relevance of community and structural contexts to IPV: theoretical pathways

The application of these structural and community-level theories to violence in the private (i.e., domestic) sphere has been relatively less prolific and even less so for IPV in particular. Community-level variation in child maltreatment was conceptualised earlier,[134,153] with an older non-systematic literature review already yielding 25 studies on the association between child maltreatment and neighbourhood-level characteristics such as poverty and residential instability.[154] The investigation of community-level effects and IPV has been slower to evolve, with two recent systematic reviews of peer-reviewed evidence finding 36 and 17 eligible articles, respectively, almost entirely from the last 15 years.[135,136] Both reviews found that the vast majority of evidence was cross-sectional and from the USA, yielding a total of only three longitudinal studies (all USA based).[155-157] The variables examined across all studies included neighbourhood socioeconomic status (e.g., unemployment, income, poverty, education), neighbourhood disorder or collective efficacy, community violence, and community norms regarding violence and women's status. Both sets of authors noted the lack of theory used to explain community-level variation in IPV and, that where theory was discussed, it was largely based on social disorganisation or collective efficacy. The current evidence base on community-level factors will be discussed and appraised in Chapter 3, but these reviews both highlight the lack of rigorous longitudinal research on whether there are community and structural effects on IPV against women, particularly outside the USA, and the insufficient attention paid to their causal mechanisms.

Defining the causal mechanisms via which social and structural contexts may affect IPV presents challenges, as the theories described above (e.g., social disorganisation

and collective efficacy theories) were developed for *public* forms of violence and crime (e.g., vandalism, burglary). Can the community monitor a *private* behaviour like IPV? Will community members view violence between partners as deviant and feel the need (or have the capacity) to intervene? Browning hypothesised that social disorganisation and collective efficacy may still affect the occurrence and continuation of IPV against women because these will dictate: (a) how safe people feel contacting services or seeking help and how effective they expect these services will be, (b) people's willingness or capacity to guide women away from relationships with known violent men or support women in leaving these relationships, (c) the level of impunity perpetrators of IPV may feel and (d) the level of trust women in violent relationships feel to disclose this violence to community members.[158] Importantly, however, Browning and other researchers note that whether people are aware of and feel inclined to intervene in cases of IPV against women will depend on the cognitive landscape of norms in the community regarding both whether violence is acceptable and the status of women.[135,136,158-160]

Related to Browning's extensions of social disorganisation and collective efficacy theory, sustained exposure to community and structural violence has also been hypothesised to normalise aggression, which then increases the risk of violence in intimate relationships.[157] In addition, sustained exposure to and marginalisation resulting from social inequalities like poverty, racism, and unemployment – and the crime, violence, and delinquency they cause – may increase trauma and stress among community members. This trauma and stress may in turn increase the propensity to use violence or vulnerability to victimisation, for instance by minimising socioeconomic and psychosocial resources to leave or avoid violent relationships.[136,159,161] Trauma and stress can likewise increase strain and tension in the relationship or exacerbate individual risks (such as substance and

alcohol misuse), which serve to further increase the probability of IPV.[162] Relatedly, one last structural factor that could affect the occurrence of IPV against women is alcohol outlet density, which may increase the likelihood of alcohol misuse and thus reduce impulse control and increase the risk for IPV – in addition to increasing the social interaction between those who misuse alcohol and may support violence.[161,163]

Even if IPV varies by community or structural contexts, what relevance does this have for the prevention of IPV against women? Understanding whether and how negative outcomes are related to community or structural-level exposures, or concentrated in certain community or structural contexts, can inform the development of effective strategies for both population-wide and targeted preventive interventions. The benefits of population-wide or community-level interventions were well articulated by Geoffrey Rose.[164,165] Rather than a binary outcome, Rose conceptualised negative health conditions, and their causes, as continuously distributed across a population, with people being more or less affected. He further demonstrated that the population's mean level of exposure or outcome will be associated with the prevalence of the most severe or high-risk cases. This overall level varies between populations (i.e., dictating the position of the population distribution), unlike the variation in the exposure or outcome within the population (i.e., the shape of the distribution), which may be constant. Rather than trying to reduce the most extreme, negative end of the distribution of causes or outcomes (i.e., the high-risk group), Rose argued it may be more effective to shift the mean level of exposure/outcome, and thus the entire population distribution, in the positive direction through interventions directed at the entire community or societal population – demonstrating the importance of community- and structural-level interventions.

Since the formulation of Rose's hypothesis, simulation studies have shown that both population-wide and targeted interventions play an important role in reducing and preventing public health problems.[166-168] For instance, one study comparing population-wide versus targeted interventions at the neighbourhood level found that improving a community-level exposure (collective efficacy) by a small amount among all neighbourhoods, in a region of New York City, was expected to have an equivalent or greater effect on violent victimisation than increasing collective efficacy by a large amount in targeted, high-violence neighbourhoods.[167] Moreover, when considering each neighbourhood as a population, an additional simulation study showed that the combination of a neighbourhood-wide intervention (hot-spot policing) with a targeted, individual-level intervention (cognitive behavioural therapy for those with violence-related PTSD) was most effective in reducing the population prevalence of violent victimisation and violence-related PTSD than either intervention alone.[168] This has important implications for preventive strategies for IPV, especially given the discussion in Section 1.1.4 about moving towards conceptualising IPV on a spectrum of severity as opposed to a simple binary outcome. Targeting community and structural-level factors that increase the average level of IPV in the population or delivering IPV interventions to community-level (e.g., neighbourhood) populations may not just reduce IPV overall, but also the prevalence (or risk) of severe IPV among women – especially when in concert with interventions targeted to high-risk individuals, which on their own are likely to have a smaller overall benefit.

These studies, and others, further raise an additional third reason for understanding the relationship between community-level exposures and IPV.[166-168] That is, the most effective strategy to reducing social disparities in violent outcomes (e.g., related to

socioeconomic status, race/ethnicity) is targeting their structural determinants.[167] A better understanding of the community-level exposures (e.g., neighbourhood deprivation) likely to be causally associated with IPV thus also informs the structural determinants of that exposure (e.g., tax policies) that ought to be targeted for maximal impact. Indeed, understanding and minimising such social and structural disparities in negative health outcomes has long been recognised as a foundational goal in public health to improve the health of populations rather than individuals.[23,144,169-174] These ideas underscore the central aims of this thesis to systematically summarise, build upon, and advance the research methods for future contributions to the existing evidence base on the association between modifiable community and structural-level factors and IPV against women.

1.4 Current state of interventions for IPV

As is clear from the above discussion, IPV against women is a multifactorial problem for which many evidence gaps remain in our etiological understanding. Although not the empirical focus of this thesis, the limited effectiveness of available preventive interventions for IPV further demonstrates these gaps, as briefly summarised below based on the latest systematic review evidence. As this thesis is mainly concerned with informing the prevention of IPV against women in the first instance (i.e., primary prevention) – as opposed to the prevention of repeated victimisation (secondary prevention) or treatment of negative consequences following IPV (tertiary prevention)[175,176] – this section focuses on existing interventions for primary prevention.

The most recent summary of intervention effectiveness for primary prevention of IPV comes from a recent systematic review of reviews on preventive interventions for

violence against women and girls.[175] This meta-review, which was later republished with a systematic search for additional studies not included in the eligible reviews,[17] found 58 studies that evaluated interventions for IPV against women (using randomised-controlled trials or RCTs, or quasi-experimental methods), more than 85% of which were from North America (mainly the USA). Despite the greater preponderance of HIC studies, a similar number of studies evaluated interventions for primary prevention in HIC and LMIC, with a greater proportion of LMIC studies evaluating primary prevention. Of all the evaluations identified, only 26% (N=15) showed any positive effects for IPV against women. Limited still, only four of these were primary prevention interventions in HIC; one of which, upon further inspection, was found to be a secondary prevention strategy (clinic-based screening for IPV victims and subsequent education on reproductive coercion).[177] Of the remaining three primary prevention interventions, one was a home visitation programme for mothers in the 3-year post-partum period[178] and two evaluated a preventive programme for dating violence among at-risk youth aged 14-16, one community-based[179] and one school-based.[180] As such no effective interventions were identified for the universal primary prevention of IPV against adult women in HIC, again demonstrating the currently weak evidence base for IPV interventions.

The remaining evidence for the primary prevention for IPV against women comes from LMIC. Ellsberg and colleagues,[17] as well as an additional systematic review of peer-reviewed studies of structural interventions for IPV against women in LMIC,[181] found a total of 16 studies of primary prevention interventions in LMIC. Only 56% (N=9) of these evaluations showed any significant positive effects on IPV outcomes. Of these effective evaluations, eight used random allocation of the intervention (either as a RCT or through randomised roll-out of the intervention), a more reliable evaluation method that controls

for both unmeasured and measured systematic differences between control and intervention groups.[182] These included: a microenterprise programme for women in Uganda;[183] national unconditional cash transfers in Ecuador;[184] school- and work-based participatory education on gender, relationships, HIV/STI risk, and violence for men in China;[185] a community mobilisation programme to change attitudes and norms combined with HIV prevention and counselling that addressed IPV in Uganda;[186] a savings intervention and gender training for women and their partners in Côte d'Ivoire;[187] and a combined microfinance and gender and HIV training programme for women and their partners in South Africa.[188] Similarly, a recent systematic review of cash-transfer programmes in LMIC largely found positive evidence for reductions in IPV.[189] Evidence from LMIC thus demonstrates the promise of community and structural interventions for reducing IPV against women but the availability of effective interventions is clearly inadequate given the burden of the problem. There may be scope for adapting and testing effective LMIC interventions in HIC but both contexts would benefit from a more rigorous understanding of the causes of IPV that should be targeted in interventions. Specifically, a better understanding of how community and structural factors may exert effects is needed given the initial, albeit limited, promising evidence of interventions at these more distal levels.

1.5 Chapter summary

This chapter has defined IPV and described the burden of the problem among women both globally and in the UK context, including its severe health and socioeconomic consequences. This chapter addressed the challenges of measuring IPV, debates regarding gender asymmetry in IPV prevalence, and how these have affected its conceptualisation.

Theories of IPV against women were discussed and the prominence of ecological frameworks was highlighted. The lack of systematic evidence on these ecological models and the limited evidence available on the community and structural effects they propose were presented, but as was the relevance of such contextual effects and potential theoretical pathways. The chapter concluded with a summary of the current state of interventions for the primary prevention of IPV against women, which illustrated the dearth of effective strategies available but the promising potential of community and structural interventions. This chapter thus established the current landscape of IPV research and demonstrated the potential contributions of the proposed thesis aims: to systematically review evidence of the causes of IPV against women within the ecological framework and advance the methodology for investigations of IPV and community and structural factors, including a novel evaluation, using a UK-based sample. Chapter Two expands upon the proposed research objectives and describes the methods that were used throughout the D.Phil.

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Chapter 2: Method

As discussed in Chapter 1, IPV against women has received significant attention from academic, advocacy, and political communities as well as the general public over the past few decades and has been clearly demonstrated to be a severe global problem. However, many gaps remain in our understanding of the etiology of IPV and few interventions have been shown to effectively prevent this violence in the first instance, particularly in HIC. This is partly due to the relative dearth of longitudinal studies – important to evidencing the causes of IPV – and the lack of a systematic summary of the full etiological evidence base for the most prominent public health model of IPV – the ecological framework. This thesis sought to address these significant gaps in the literature by: establishing a systematic understanding of the risk and protective factors for IPV against women based on the most rigorous evidence; and advancing the measures and methods, including conducting an original investigation therein, of the potentially causal effects of one of the most poorly understood set of risk and protective factors to date: community and structural factors. The specific research objectives and methodology of the thesis overall are detailed below.

2.1 Restatement of research objectives

As first articulated in Chapter 1, the objectives of this thesis were to:

1. Systematically review prospective-longitudinal evidence of risk and protective factors for IPV against women at all ecological levels (individual, relationship, community, and structural) and identify evidence gaps; and
2. Using two generations of data from the Avon Longitudinal Study of Parents and Children (ALSPAC) in the United Kingdom (UK), evaluate:

a) The overall prevalence and gender differences in experiences and impacts of IPV in early adulthood and psychometric properties of the IPV measure,
b) The dynamic relationship between objective neighbourhood-level deprivation and perceptions of the neighbourhood environment over time, and
c) The effect of long-term exposure to objective neighbourhood deprivation on the risk of experiencing IPV among women in early adulthood.

These research objectives map onto the thesis structure shown in Table 2.1. As can be seen, the thesis provides a thorough literature review, both through the background research conducted on the problem of IPV against women and developments in the field presented in Chapter 1 and the systematic review on risk and protective factors presented in Chapter 3. The thesis then proceeds with a series of empirical analyses that utilise UK-based data from ALSPAC that: evaluates the measurement and summarises the extent of IPV in the sample (Chapter 4); interrogates the relationship between different community-level measures collected in ALSPAC over time (Chapter 5); and tests, for the first time, the effect of long-term exposure to neighbourhood-level deprivation on the risk of experiencing IPV among women (Chapter 6). The thesis closes with a discussion chapter (Chapter 7) that summarises how the results across the systematic review and empirical work speak to each other, directions for future research, and the implications for practice and policy decisions.

Table 2.1 Thesis outline

Chapter	Research question/objective	Content
1	Summary of the IPV field and justification for the thesis	Literature review
2	Overall methodology of the thesis	Research questions and aims; Epistemological and epidemiologic framework; ALSPAC study design
3	What are the risk and protective factors for IPV victimisation among women?	Systematic and meta-analytic review of prospective longitudinal data
4	Is the IPV measure used in ALSPAC valid and reliable? Are there differences in the prevalence and impacts of IPV experiences among women and men in the sample?	ALSPAC analysis: Psychometric analysis, descriptive analysis, analyses of gender differences
5	How do neighbourhood and community exposures vary over time? To what extent does objective neighbourhood-level deprivation overlap with perceived measures of the neighbourhood environment over time?	ALSPAC analysis: Group-based dual trajectory analyses, multinomial logistic regression
6	What is the effect of long-term exposure to objective neighbourhood deprivation on the risk of experiencing IPV among women in early adulthood?	ALSPAC analysis: Marginal structural models estimated by inverse probability weighting, adjusted generalised linear models
7	Discussion and conclusions: What are the strengths and limitations of this thesis? What are the contributions to and implications for research, theory, and policy?	Interpretation of combined results; Directions for future research; Implications for theory and policy/intervention decisions

2.2 Evidence-informed interventions: why *causal* risk and protective factors matter

Understanding the risk and protective factors for IPV against women is essential for designing interventions that can effectively reduce the problem.[1] This understanding is known as a *problem theory* and when it is clearly conceptualised – including a full conception of the risk and protective factors that are associated with the outcome and the mechanisms that underlie these associations – it indicates which risk and protective factors should be targeted to produce the greatest reduction in IPV.[2] Without a robust problem theory, interventions may be ineffective (thus wasting valuable resources) or, at worst, harmful.[3] Although also influenced by factors such as political will and resource availability, the effects of an insufficiently defined problem theory in the case of IPV against women can be clearly seen in the lack of effective interventions available for primary prevention.

Of particular importance to developing a problem theory and subsequently intervention design is knowing the *causal* risk and protective factors of a problem. This is because if a condition is simply correlated with IPV but the association is ultimately not

causal then targeting this condition will be ineffective in reducing IPV. Bradford Hill defines nine criteria for determining whether an existing association is likely to be causal:[4]

1. Strength: Although small effects can still be causal, the presence of a large effect is likely to indicate a causal relationship;
2. Consistency: Associations that are consistently observed with different samples, methods, and contexts are more likely to be causal;
3. Specificity: If a single factor is associated with a specific effect, the relationship is likely to be causal, however, absence of specificity does not rule out causality;
4. Temporality: The factor must occur before the outcome if the relationship is causal;
5. Dose-response: If greater exposure to the factor leads to greater change in the outcome, this strengthens the evidence for a causal relationship, although again absence of this dose-response relationship does not negate causality;
6. Plausibility: There should be a plausible theoretical explanation for the causal relationship being hypothesized;
7. Coherence: Interpreting the relationship as causal should not conflict with what is already known on the phenomenon or its context;
8. Experiment: The strongest evidence for causality is if the condition is altered in an experimental context and the outcome changes in the predicted direction;
9. Analogy: Other similar causal relationships having been confirmed would increase the likelihood of causality.

As alluded to in these criteria, the strongest study design for evaluating the causality of an association is a RCT, whereby the groups being evaluated are systematically the same except that the risk or protective factor under consideration is randomly allocated to one and not the other (or the randomly allocated intervention targets a single risk or protective factor).[5] However, experimental designs are not always possible in risk/protective factor research: for instance, some factors cannot be randomised and are difficult to separately target in an intervention. For this reason, the next strongest and more common design for rigorously evaluating causal risk or protective factors is a prospective-longitudinal study. In such studies, it is possible to measure the risk or protective factor before the outcome occurs (fulfilling the criterion of temporality) and test whether change in the risk/protective factor leads to change in the outcome (temporality/dose-response).

This last point highlights a key matter in identifying causal risk and protective factors: we are primarily interested in identifying *malleable* factors, or factors that can change. While understanding risk factors that cannot be changed (i.e., those that are *fixed*, such as age and race) can be useful in terms of informing how a problem is distributed and who should be targeted in interventions (i.e., who is most at risk), by definition these fixed factors cannot change and therefore can cause no change in the outcome.[6] Resultantly, preventive interventions must target at least one malleable risk factor (even if distributed unevenly based on a fixed factor) in order to cause change in the outcome of interest. The investigative work of this thesis has thus been undertaken entirely with prospective-longitudinal studies and largely focused on malleable risk and protective factors hypothesised to be causal in order to inform the design of effective primary prevention strategies for IPV against women.

It should be noted that there is wide variation across disciplines in the terminology used regarding etiological studies and particularly around 'risk and protective factors'. Some researchers, especially those in medical fields, refer to these instead as etiological factors or prognostic variables.[7] Other researchers, especially in the social sciences, instead consider two separate kinds of protective conditions: promotive and protective factors – although the definitions for these vary still. Some define promotive factors as those associated with an increased likelihood of a positive outcome and protective factors as those associated with a decreased likelihood of a negative outcome.[8] Others refer to promotive factors as those directly associated with a decreased likelihood of a negative outcome and protective factors as those associated with a decreased likelihood of a negative outcome among people at risk or those that interact with and diminish the effects of risk factor(s).[9] For the purpose of this thesis, where the outcome of interest is negative (i.e., violence), the terms 'risk' or 'protective' factors are used, where the latter is defined as any condition or behaviour directly associated with a decreased likelihood of IPV.

It is also important to highlight a major criticism of risk/protective factor research at the outset: that much of it results in mere ‘checklists’ of factors associated with the outcome, without considering the theoretical mechanisms that underlie these associations.[6] This has clearly been the case in previous etiological research on IPV (especially on community and structural-level factors), as was discussed in Chapter 1, where little theory building and testing has occurred. Hypothesising and eventually testing potential causal mechanisms is essential for building a rigorous problem theory, as these mechanisms and pathways ultimately indicate how an intervention can be expected to have an impact on the outcome.[2] In other words, without properly understanding whether and how a causal risk or protective factor exerts its effect on an outcome, we cannot design an intervention that

will effectively disrupt (in the case of a risk mechanism) or promote (in the case of a protective mechanism) this pathway. It is beyond the scope of this thesis to fully identify and test the potential causal pathways for all of the risk factors for IPV against women considered – nonetheless, a primary aim of the empirical work has been to develop explicit causal assumptions, create analytic models based on these assumptions, and advance future directions for theory testing based on my results. This is described further below and in the following results chapters.

2.3 An introduction to the chosen methods

The specific methods used to achieve each objective of this thesis are described in detail in the corresponding results chapters (Chapters 3-6). In what follows, I introduce some of the main epidemiologic concepts underlying the chosen methods as initial justification for the framework of this thesis and demonstration of the novel contributions this thesis makes to the literature.

2.3.1 Systematic review

The first research objective of this thesis was to systematically review all available prospective longitudinal evidence on the risk and protective factors for IPV against women (Chapter 3). This entailed systematically searching for, screening, and appraising published and unpublished prospective-longitudinal studies of risk and protective factors for IPV victimisation among women. Where two or more studies tested the same risk or protective factor, I conducted random-effects meta-analyses, planned a priori. Systematic reviews, and where possible meta-analyses, of high quality studies most suitable for the

research question (here, prospective longitudinal studies) are the most rigorous form of evidence because they reduce the bias inherent in single studies, which will each have their own set of flaws and that are at greater risk of spurious effects.[10] Systematic reviews also allow for the identification of evidence gaps in the literature and likewise estimation of the quality of evidence available for specific risk and protective factors and the overall strength of these effects. Furthermore, systematic reviews and meta-analyses can illustrate how effects may vary across context or specific study characteristics (e.g., sampling, length of follow-up). Finally, systematically reviewing both published and unpublished studies helps estimate more accurate effect sizes because published studies on their own are more likely to contain positive results as opposed to negative or null, thus skewing effect estimates.[11] Prior to undertaking the systematic review presented in Chapter 3, there had been no systematic review in the past 10 years of all risk and protective factors for IPV without geographic or publication restrictions, highlighting the contribution of this review to the field and its relevance to policymakers and practitioners.

2.3.2 Measurement

Sound measurement is foundational to rigorous social epidemiology.[12] This entails thoughtful consideration of the constructs that matter most and how these relate to each other, followed by the use of valid and reliable measures. As outlined in Chapter 1, measurement of IPV has been a notoriously difficult endeavour. A key objective of this thesis was to empirically investigate an under-studied community-level exposure on IPV against women – neighbourhood deprivation – using data from a birth-cohort study (ALSPAC, described further below). However, despite the richness of the cohort data and

the availability of longitudinal, repeated-measures, it was critical to first consider the validity of the IPV measure available in the study and, subsequently, of theoretical and practical interest to summarise the extent of the problem in this sample using this measure. This work, presented in Chapter 4, entailed psychometric analyses of the ALSPAC IPV scale – including exploratory factor analysis to determine dimensionality, a correlational assessment of convergent validity, and evaluation of the internal consistency of items to establish reliability. I further used descriptive statistics to determine the overall prevalence of IPV in the ALSPAC sample and conducted analyses of gender differences in both the prevalence and impacts of this violence. Of note, this work serves as the first psychometric evaluation of the IPV measure used in ALSPAC, which was a unique short-form measure of physical, psychological, and sexual IPV, and the first assessment of the extent of IPV experienced by the birth cohort sample.

On top of the importance of understanding the measurement and extent of IPV in ALSPAC, due consideration of the measurement of neighbourhood deprivation was likewise essential (Chapter 5). The English Indices of Multiple Deprivation, the government's official measure of relative neighbourhood-level deprivation, were available in ALSPAC for every year over the study period.[13] Briefly, this is a robust measure of deprivation across seven domains: income; employment; health and disability; education, skills, and training; crime; barriers to housing and services; and living environment deprivation. Scores are constructed from indicators at the level of the lower-layer super output area (LSOA) in England, which are small areas of similar population size (approximately 1500 residents or 650 households). The more than 32,000 LSOAs in England are then ranked based on their overall deprivation scores, creating a distribution

of relative deprivation. This means that, in using this measure, the context of interest was defined as a small physical area (i.e., the LSOA) in which the participant resided at each time point, approximating neighbourhoods of residence.

How to define community (or neighbourhood or place) is of primary concern in analyses of contextual effects: these definitions tend to vary across studies and can lead to drastically different results in the same analysis – otherwise known as the *modifiable areal unit problem*.[14,15] The selected measure matters in terms of what the results mean for intervention design and delivery (e.g., is the area defined in space and likely to be a viable/suitable target for interventions?).[16,17] Furthermore, it also matters in regards to whether the environment as defined is expected to have the greatest contextual effect on the outcome. This may not simply be a matter of determining the radial distance around a person's residence that should be accounted for; other physical environments in which the person inhabits (e.g., the workplace) and their social or relational environment (e.g., the quality and quantity of their relationships) may also be influential, beyond the physical residential neighbourhood.[18-22] The LSOA is not necessarily the most meaningful definition of neighbourhood or community for study participants, as its borders are unlikely to align with what participants would conceptualise as their neighbourhood or community.[21] However, it does offer a suitable proxy for participants' immediate physical neighbourhood that allows for investigating the effects of accumulated deprivations,[23,24] a central concept in theories of neighbourhood effects,[25] with census-defined boundaries that can be easily translated to intervention design. Moreover, the ALSPAC setting has some of the most deprived and least deprived LSOAs in the country,[26] which provides for suitable variation in analyses.

Chapter 5 of this thesis thus acknowledges the uncertainties and complexities involved in defining neighbourhood effects and their mechanisms by exploring how the Indices of Multiple Deprivation, as an objective measure of the neighbourhood environment, mapped onto participants' perceptions and experiences of their neighbourhoods. This consideration of the extent of overlap between the objective and perceived neighbourhood measures is not only valuable to contextualising analyses of neighbourhood effects using the objective measure (Chapter 6); it further offers implications for whether studies in the field using these different measures are in fact tapping into different neighbourhood/community constructs (as opposed to a single construct) and effect mechanisms. Additionally, I explore the socioeconomic and psychosocial factors associated with variations in participants' perceptions of their neighbourhoods, establishing the intertwining of the individual and the neighbourhood in self-reported neighbourhood measures. Finally, this chapter establishes whether (and how) the different neighbourhood and community exposures in ALSPAC vary over time – which is critical to conducting (and designing) longitudinal analyses,[27-29] a primary aim of this thesis. These analyses use group-based dual trajectory modelling[30] and serve as one of the first investigations to model the dynamic (as opposed to point-in-time) relationship between perceived and objective neighbourhood measures over time.

2.3.3 Neighbourhood effects: relevance of time-varying confounding and the application of marginal structural models estimated by inverse probability weighting

Chapter 6 addresses the final objective of this thesis: to estimate the effect of long-term exposure to neighbourhood deprivation on the risk of experiencing IPV in early

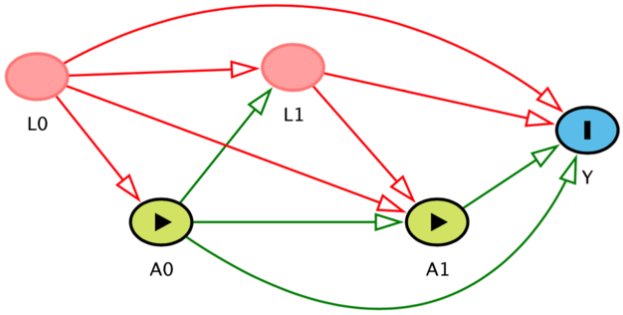
adulthood in the ALSPAC sample. In analyses of community-level or more specifically neighbourhood effects, two main challenges are often discussed. The first is whether the effect of the environment is *contextual* (i.e., whether there is a unique contribution of environmental characteristics to the outcome) or *compositional* (i.e., whether the effect of the environment is only observed because of the individuals that make up that environment – in other words, adjusting for individual factors will partial out the observed environmental effect).[16,22,31-35] The second, related challenge is the extent to which non-random selection of people into neighbourhoods is really the driver of observed neighbourhood effects (i.e., confounding bias).[25,29,36-38] That is, people move out of or live in particular communities over time not by chance but because of factors at the individual level, which likewise vary over time. As a result, in analyses of neighbourhood effects, researchers are typically expected to analytically parse out the unique effect of the neighbourhood from the more proximal characteristics that selected people into those neighbourhoods in the first place.

To address these challenges, researchers often compute multilevel models with one level representing the neighbourhood-level variables and the second level representing the individual level.[12,34,39] When the neighbourhood level does not account for variation in the outcome above and beyond the individual level, it is typically concluded that there is no valuable contribution of the neighbourhood context. However, these models are often over-adjusted and lead to false conclusions regarding the importance of context for several reasons.[16] First, just as people may live in certain neighbourhoods due to personal or family-level characteristics, a person's personal or family characteristics may be influenced by the characteristics of neighbourhoods (e.g., a person's social class may be dependent on the local labour market).[29,40,41]

Second, there is an overdependence on finding *direct* effects of exposures, given that the causes of social problems are multifactorial and operating in multilevel *pathways*.[42] Individual-level factors may very well underlie or *mediate* the associations between contextual factors and outcomes. In such cases, it would be valid to conclude that there are contextual effects on the outcome but that these operate indirectly via more proximal factors. Treating these individual factors as confounders (i.e., variables that are related to both the independent and dependent variables and therefore would cause a spurious association between the latter two), as is often the case in multilevel studies, would be inaccurate. As Macintyre and colleagues point out, this highlights the overall failure of many such studies to conceptualise the theoretical pathways via which environments may affect individual health and wellbeing.[16] In other words, researchers who aim to parse out compositional effects often fail to account for how neighbourhoods actually cause the outcome. It is unlikely that contextual and compositional effects are truly distinct and instead more plausible that the individuals who make up the neighbourhood and the contextual characteristics of the neighbourhood itself feedback to each other over time and the former lies on the causal pathway of the latter's effects.[16,43]

This feedback between neighbourhoods and individuals, however, creates a challenging analytic scenario. That is, individual-level socioeconomic and psychosocial characteristics predict the neighbourhoods that people move to or live in. In turn, future values of these individual-level characteristics are affected by the neighbourhood environments in which people live. Finally, these individual characteristics may additionally affect the risk of experiencing IPV in early adulthood.[29,44] This amounts to a classic case of *time-varying confounding affected by past exposure*, the simplest example

of which is depicted below, where A is the exposure variable measured at time 0 (T0) and time 1 (T1), L is a set of time-varying covariates at T0 and T1 and Y is the final study outcome:



Formally, time-varying confounding affected by past exposure occurs when a time-varying exposure is both affected by the level of prior time-varying covariates and predictive of future values of those time-varying covariates – all of which then predict the outcome of interest.[27] The result of the above depiction as well as more complex scenarios is that, when estimating the effect of exposure on the outcome, controlling for the values of time-varying covariates using conventional regression methods would lead to over-adjustment (partialling out part of the total effect of the exposure on the outcome) and potentially *collider-stratification bias* (i.e., when a spurious association is created between exposure and outcome as a result of conditioning on a variable that is a common effect of the exposure and a confounding variable).[28,45] However, not controlling for these covariates would result in bias due to confounding.

Acknowledging these methodological complexities, Chapter 6 presents a novel and advanced set of analyses of the long-term effect of neighbourhood deprivation on IPV using *marginal structural models* estimated by *inverse probability weighting*. Marginal

structural models allow for the estimation of a causal effect of a time-varying exposure in the presence of both time-varying confounders and mediators using observational data (under the assumptions of exchangeability, consistency, positivity, and correct model specification – which are defined in Chapter 6).[45] Of note, these assumptions are the same as those required by conventional regression methods when estimating causal associations. In fact, the latter require the additional assumption that measured time-varying covariates are not affected by prior exposure – which is not required of marginal structural models.[29] The model is referred to as *marginal* because it estimates the average effect of exposure based on the marginal distribution of the counterfactual variables or potential outcomes (e.g., comparing the outcome if the entire sample was exposed versus unexposed at time t) and *structural* because the estimate is of a causal effect.[46]

In this thesis, I estimate marginal structural models using inverse probability weighting. This essentially involves a two-step process, where I (1) predict each participant's cumulative probability of experiencing the exposure history she actually experienced conditional on her covariate history and (2) run a crude analysis (i.e., without time-varying covariates) in a sample where each participant is weighted by the inverse of this probability. The result of this process is that participants whose exposure histories are more common given their covariate histories are proportionally under-weighted in the analytic sample whereas participants whose exposure histories are less common given their covariate histories are proportionally over-weighted. Intuitively, this creates a *pseudo-population*, where the probability of exposure is made comparable across levels of the confounders at each time point – mimicking the concept of exposure occurring at random (assuming no unmeasured confounders). As a result, running a crude model in this weighted sample (the marginal structural model) accounts for nonrandom selection of

participants into neighbourhoods (e.g., due to socioeconomic variables) that may otherwise confound effect estimates but do not partial out the indirect effects of neighbourhood exposures via these variables at later time points.[29]

In addition to accounting for confounding without potentially biased adjustment, marginal structural models are easy to interpret for researchers used to more conventional methods (e.g., generalised linear models) as they are simply an extension of these models to weighted samples.[46] Of further benefit for use with cohort data, inverse probability weights can also be constructed for censoring or attrition, whereby, in addition to the exposure weights, participants in the analytic sample are also weighted by the inverse probability of remaining in the sample given their prior covariate and exposure histories – thus accounting for non-random attrition conditional on observed covariates. The investigation presented in Chapter 6 represents the first time these models have been applied to the etiological study of IPV against women, demonstrating the methodological contribution of this work to the field – particularly in its rigorous and transparent conceptual and analytic framework.

2.4 ALSPAC: sample and setting

The empirical analyses in this thesis use quantitative data from ALSPAC, an ongoing UK birth cohort study that has collected rich health and social data from 15,247 families since 1990. Data were reported by mothers, their partners, and their children until young adulthood, providing a robust set of perspectives and high reliability of measures. ALSPAC is the most detailed study of its kind in the UK and potentially the world,

offering a unique opportunity for prospective longitudinal analysis of community- and structural-level exposures, individual and family-level confounders, and IPV over time.

ALSPAC was originally set up as part of a large-scale project of birth cohort studies across Europe, the European Longitudinal Study of Pregnancy and Childhood, to investigate malleable risk and protective factors for child health and development.[47,48] The study was likewise originally named the Avon Longitudinal Study of Pregnancy and Childhood but was renamed to end with 'Parents and Children' as the study focus shifted to following children and their parents over their lifetimes. As clear from its name, ALSPAC focuses on the former administrative county of Avon. All pregnant women resident in one of the three health districts shown in Figure 2.1 who had an expected due date between 1 April 1991 and 31 December 1992 inclusive were eligible to participate. These three health administration districts covered the South-West Regional Health Authority, which eventually became the Bristol and District Health Authority, and include both urban and rural areas. The resulting children of the participating pregnant women were likewise eligible to participate and formed the 'Children of the Nineties' cohort.

Figure 2.1 NHS District Health Authorities (DHA) sampled for ALSPAC. This figure is taken from Boyd et al. (p. 3).[47]

The estimated total sampling frame for ALSPAC was 20,248 eligible pregnancies.[47,48] The study team invited a total of 16,734 known pregnant women to participate through media, community visits, and routine antenatal and maternity health services. Of those, 14,541 women provided data and were enrolled in ALSPAC, making the initial response rate of the study 87%, which accounted for 72% of the total sampling frame. When the enrolled children were age 7, the study team received additional funding to track the women who were eligible for initial participation in the study but who had not responded or been contacted. As a result, an additional 706 women and their eligible children were enrolled, increasing the total sample size to 15,247 families. The Children of the Nineties sample consists of 14,775 children who were born alive to the participating

mothers (76% of the 19,600 eligible live births). Participants remain in the study regardless of their history of providing data (or lack thereof) or if they move away from the study area (questionnaires are sent worldwide); moreover, if participants withdraw from the study they remain eligible to re-enroll.

To assess representativeness, researchers at the University of Bristol and the ALSPAC executive compared the sample at the 8-month postnatal questionnaire to mothers with an infant less than 1 year in Avon and the whole of Great Britain (based on the 1991 census).[48] The results of this comparison are shown in Figure 2.2. Compared to mothers in Avon and Great Britain, a greater proportion of ALSPAC participants had a car in their household, were married, lived in owner-occupied accommodation, and were white. Notably, however, a greater proportion of mothers in Avon also had a car in their household, lived in owner-occupied households, and were white compared to mothers in Great Britain, indicating that in part, the ALSPAC sampling frame had slightly higher socioeconomic characteristics and were less ethnically heterogeneous than the national population on average. In contrast to their generally higher socioeconomic position, ALSPAC participants tended to more often live in overcrowded conditions (an average of more than one person per room) than mothers in Avon or Great Britain.

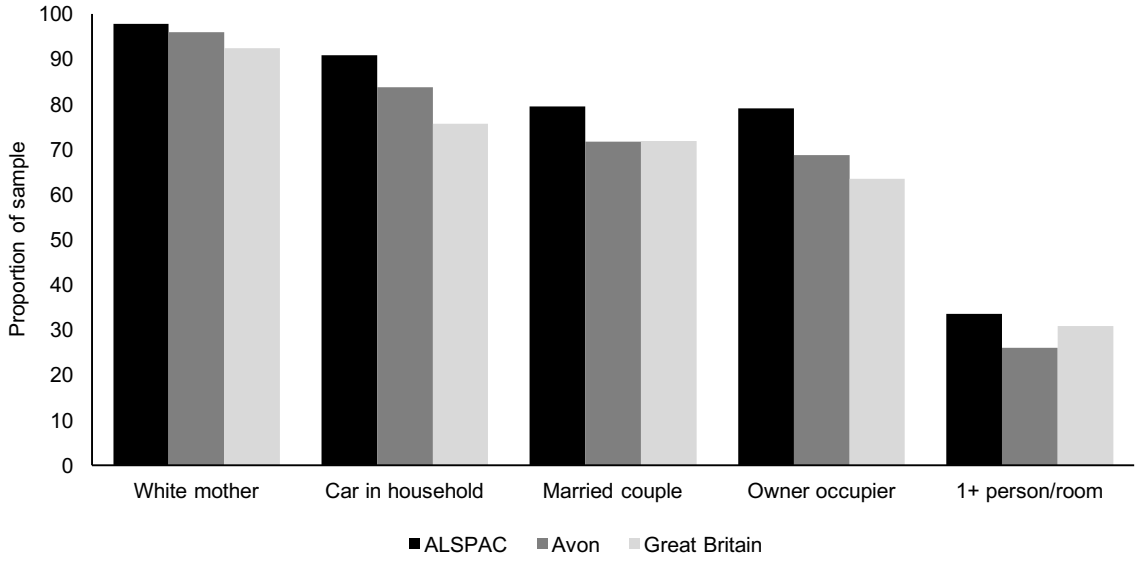


Figure 2.2 Summary of the sociodemographic characteristics of the ALSPAC sample at the 8-month time point versus mothers in the whole of Great Britain and Avon in 1991. Data for mothers in Great Britain and Avon from the 1991 census and based on women with an infant <1 year of age. Data from ALSPAC based on questionnaire responses at 8 months postnatal collected between 1992 and 1993; 80% of the enrolled pregnancy cohort from ALSPAC completed this questionnaire. Data are from Fraser et al.[48]

Despite the overall higher socioeconomic position of the ALSPAC sample compared to the national average, there was still substantial variation in the deprivation levels of residential neighbourhoods represented in the sample. For instance, Bristol contains some of the most deprived neighbourhoods in England and some of the least.[26] Exposure to neighbourhood deprivation in the sample is described in depth in Chapter 5. Additionally, a significant proportion of the ALSPAC sample has experienced mental health problems and exhibited risky behaviours, providing rich variation in psychosocial wellbeing.[49-52] Nonetheless, it is worthwhile to emphasise at the outset that the higher socioeconomic position of the sample likely means that the prevalence of IPV (Chapter 4) and the association between IPV and neighbourhood deprivation (Chapter 6) estimated in this thesis is likely to be conservative compared to similar populations with greater socioeconomic diversity.

2.4.1 Study data

ALSPAC data are primarily collected through paper-based questionnaires (sent via post) as well as in-person clinics. There were 68 data collection time points between the children's birth and when they reached age 18.[47] The study is ongoing, with the last round of data collection occurring when children were age 26, and expected to continue for the children's lifetimes. Mothers have self-completed questionnaires about their lives throughout the study's duration and also completed questionnaires on their children until the children were age 16. Children have self-completed questionnaires from age 5 onwards. This thesis used data from the children enrolled in ALSPAC from birth (i.e., the Children of the Nineties cohort) and followed into young adulthood, when their experiences of IPV were measured. Additionally, data on exposures related to the neighbourhood environment and potential confounders related to the family psychosocial and socioeconomic environment were also used from the young persons' mothers over the study period.

In addition to questionnaire data, and of relevance to this thesis, ALSPAC also maintains an annually updated address database for its participants so that, where available, participants' residential addresses are linked to standardised neighbourhood deprivation measures. Initial address data were collected at the start of the study when participants were recruited. Following recruitment, address data were recorded by the study team based on participants' self-recorded residence changes. As a result, the data are post-hoc, which may mean that some participants' residential changes were not recorded. However, this process also increases the temporal accuracy of the data as compared to if it was only collected once every few years.[53] Furthermore, as noted by the research team,

inaccurate residential details are less likely to affect analyses when linked with questionnaire data, as was the case for all analyses in this thesis, given that participants would be unlikely to return a completed questionnaire if sent to the wrong address. ALSPAC has validated 99% of the total sample's addresses as accurate, further indicating sound validity and suitability of using these address data. Participants' address data were then linked, by ALSPAC, to the English Indices of Multiple Deprivation.

2.4.2 Attrition and missing data

One of the most important limitations of ALPSAC, and indeed most long-term prospective longitudinal studies, is sample attrition. Figure 2.3 shows the permanent attrition that has occurred due to death, withdrawal, and those otherwise permanently lost to follow-up at each phase of the study until age 18. However, even more impactful to the study has been the temporary attrition that occurs at each time point, wherein participants do not provide data for that round of the questionnaire. For instance, only 3,049 participants responded to all child-based questionnaires up to age 18 – not accounting for missing data within each questionnaire assessment.[47] This means the sample used in most analyses in this thesis is considerably smaller than the total number of participants in ALSPAC, but is still a substantial number to detect even small effects. Missing data and strategies to account for these are rigorously explored in Chapter 6 to account for potential selection bias.

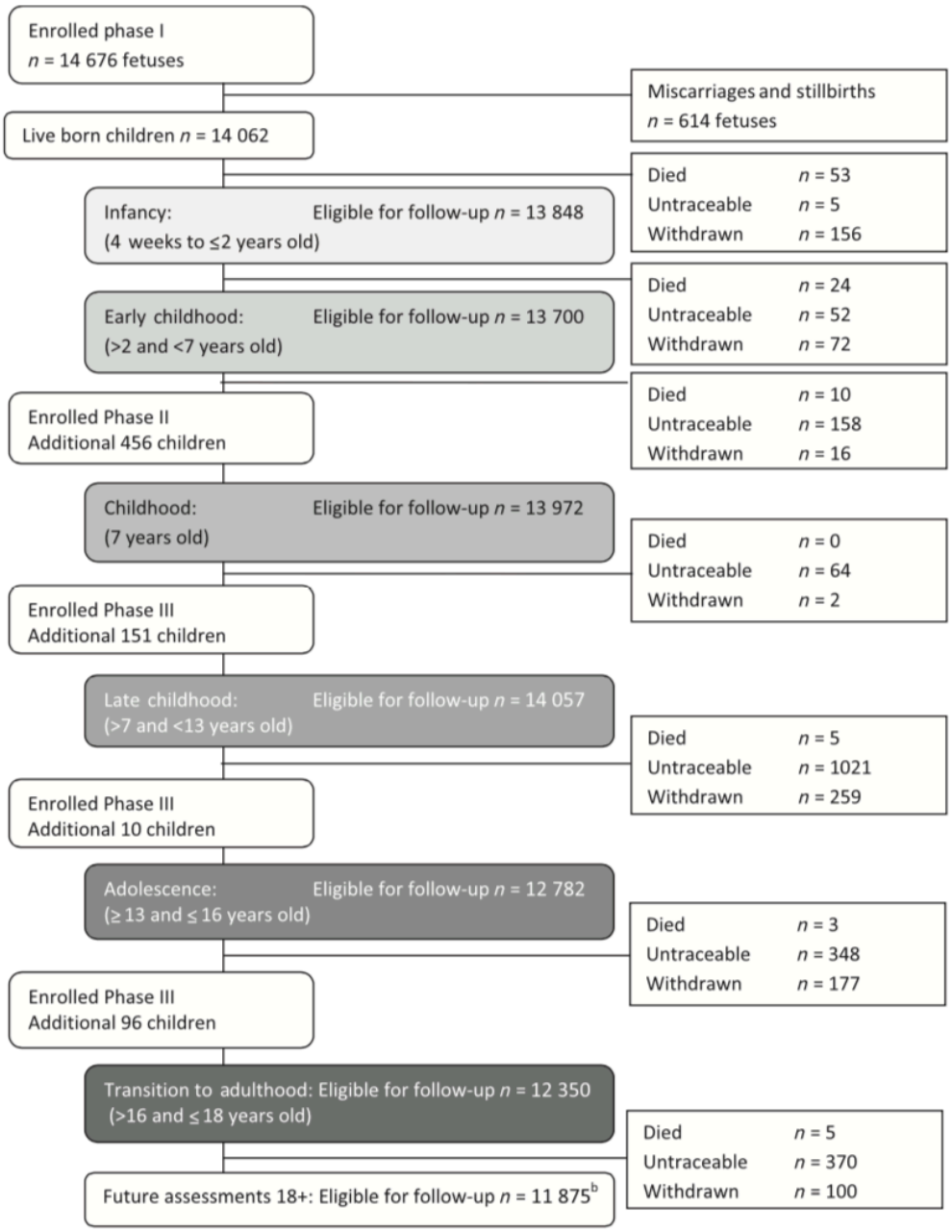


Figure 2.3 ALSPAC attrition flow diagram. Diagram is taken from Boyd, Golding (47, p. 10). [a]Individuals excluded from follow-up due to a 'permanent' status change that remained in place up to the age of 18 years (e.g. individuals became untraceable and were not found again before the age of 18 years). Individuals for whom a change in status meant they were temporarily excluded from follow-up are not included and, therefore, this diagram under-represents attrition at individual data collection time points. [b]Post-18-year age tracing and recruitment initiatives may have resulted in some individuals being included in future follow-up.

2.4.3 Ethics

The dataset used in this thesis was fully anonymised by ALSPAC prior to receipt. Ethical approval for ALSPAC was initially obtained from the Bristol and Weston Health

Authority (E1808 Children of the Nineties: Avon Longitudinal Study of Pregnancy and Childhood, 28th November 1989), Southmead Health Authority (49/89 Children of the Nineties – "ALSPAC", 15th April 1990), and Frenchay Health Authority (90/8 Children of the Nineties, 28th June 1990). All subsequent questionnaire content was approved by the ALSPAC Ethics and Law Committee.

2.5 Chapter summary

This chapter described the epistemological and methodological framework of the proposed thesis and re-stated the research objectives. The structure of the thesis was outlined and justification for the overall methods and contributions of each component were detailed. Finally, the setting and sample of ALSPAC was introduced.

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Chapter 3: Risk and protective factors for intimate partner violence against women: systematic review and meta-analyses of prospective-longitudinal studies*

3.1 Introduction

The objective of this chapter was to systematically review the current longitudinal evidence base for the risk and protective factors for IPV against women. As outlined in Chapter 1 (Section 1.1.1), IPV – actual or threatened physical, psychological, or sexual violence by a current or former partner – is the most common violence perpetrated against women.[1] Lifetime prevalence estimates are high for all WHO regions: ranging from 23–38% among ever-partnered women.[2] IPV is associated with severe negative health outcomes for women, including death, injury, psychological disorders, sexually transmitted infections, and chronic diseases.

As also discussed in Chapter 1 (see Section 1.2.2), the *ecological framework* conceptualising IPV as the result of the interplay between individual, relational, community, and structural factors is widely accepted in public health (Appendix A, Figure A1).[3,4] Yet there has been no complete, systematic assessment of this model. Prior reviews have conducted non-systematic searches, pooled mainly cross-sectional studies with longitudinal, and only focused on physical IPV.[5,6] Other reviews have included only subsets of risk factors (e.g., alcohol use)[7-11] or subpopulations (e.g., high-income countries).[12] These reviews have also largely included only peer-reviewed evidence, which tends to overestimate associations.

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Hundreds of cross-sectional studies have examined factors associated with IPV against women at all ecological levels, including substance misuse (at both the individual and relational levels), childhood exposure to violence (relational), and traditional gender norms (structural).[4] While these studies identified research priorities and population targets for interventions, developing effective interventions for preventing IPV in the first instance requires knowing which conditions will, when changed, increase the risk of IPV (risk factors) or decrease this risk (protective factors) – best evidenced by prospective-longitudinal studies, which measure exposures and outcomes over time.[13]

In this chapter, I thus conducted the first systematic review and meta-analyses of prospective-longitudinal risk and protective factors for different types of IPV against women at all ecological levels. I aimed to estimate the strength of each association, to inform programmatic decisions, and identify evidence gaps across contexts and types of IPV.

3.2 Method

3.2.1 Search strategy and selection criteria

The review protocol was registered with PROSPERO (CRD42016039213). I undertook a rigorous search strategy, developed in consultation with two information specialists (see Appendix A, Tables A1 and A2 for complete strategy). I searched 16 electronic databases from inception to June 2016: MEDLINE, Embase, Global Health, CINAHL, PsycINFO, Web of Science, Google Scholar, ASSIA, ProQuest Dissertations

and Abstracts, OpenGrey, National Criminal Justice Reference Service Abstracts Database, Cochrane Library, Sociological Abstracts, World Bank Open Knowledge Repository, WHO Institutional Repository for Information Sharing, and WHO Prevent Violence Evidence Base and Resources. Reference lists of all systematic reviews identified were screened for eligible studies and authors were contacted wherever their manuscripts mentioned but did not present eligible data (e.g., gender-disaggregated analyses).

Selection criteria were chosen iteratively in two stages given the extensive literature on IPV.[9] In the initial stage, titles and abstracts were screened by myself and a second reviewer for studies examining at least one risk or protective factor for IPV against women, using any quantitative design. After this initial screening, we limited our focus to prospective studies given that these by design will produce the best evidence for risk and protective factors. The remaining studies were then screened using these final selection criteria by myself and two independent reviewers, first by titles and abstracts followed by full texts as needed (Appendix A, Table A3). Studies were included that were published or unpublished, available in English, and had quantitatively analysed at least one risk or protective factor for physical, sexual, or psychological IPV in a population, community, or birth cohort sample, controlling for at least one other variable. Participants had to be at least 19-years-old at outcome assessment, according to the WHO definition of adulthood.[14] Studies had to include women's self-reports of IPV victimisation (including those that used both partner- and self-reports or conducted gender-disaggregated analyses). Studies that only measured self-reported perpetration were excluded, since people tend to self-report experiencing IPV more than their partners self-report perpetrating violence.[15] Finally, only prospective-longitudinal studies were included, defined as having at least two assessment waves, where exposure to the risk or protective factor was measured before the

outcome. Data on risk or protective factor(s) were excluded when: they were measured at the same time-point as the outcome without analysing change; they were measured retrospectively (e.g., retrospective history of child abuse); or the outcome was lifetime as opposed to current IPV prevalence (e.g., within the last year). In each of these cases it is not possible to determine whether the exposure preceded IPV without, for instance, recall bias.

3.2.2 Data extraction and quality assessment

I extracted study characteristics and effect estimates for eligible risk/protective factors using a piloted form. At each stage of screening and extraction, a random selection of studies (5–10%) were double-coded by two reviewers: there were no substantive discrepancies. Study quality was appraised using the Cambridge Quality Checklists,[16] which assesses the quality of correlational evidence for risk and protective factors (based on sampling, participation rates, sample size, and measurement reliability), temporal evidence (whether data are cross-sectional, retrospective, or prospective), and causal evidence (whether there is variation in the risk/protective factor, change in outcomes is analysed, and confounding is accounted for) (Appendix A, Table A4). A percentage score for each study was computed based on the study's total score across all three checklists. I discuss common sources of low scores below.

3.2.3 Data analysis

When two or more studies investigated the same risk or protective factor using similar measures, I conducted a random-effects meta-analysis in Stata 13.1 (StataCorp LP,

College Station, TX) using the method-of-moments estimator.[17] In contrast to a fixed-effects meta-analysis, which assumes there is *one* true effect, a random-effects meta-analysis assumes that the study effect estimates approximate a *distribution* of true population effects which vary due to factors beyond chance, including study methodology (e.g., duration of follow-up) and context (e.g., age of sampled women).[18] The method-of-moments estimator (also known as the DerSimonian and Laird method) is a standard method of estimating the random-effects meta-analysis which involves weighting studies by the inverse of the variance of the study effect estimate, adjusted to incorporate the variance between study effect estimates.[17,18] A random-effects model was selected a priori given the expected methodological and contextual differences among relevant studies.

Studies that did not use similar methods (e.g., diverse variable definitions) were deemed clinically heterogeneous and not combined. Pooled odds ratios were computed as studies typically reported binary outcomes. Log odds and standard errors for each study were used in meta-analyses, with data transformations conducted wherever necessary and possible (see Appendix A for conventions). When studies did not provide precision estimates or p-values, I conducted the meta-analysis twice: once with standard error estimated based on a liberal p-value (p=.060) and once assuming a conservative p-value (p=.500). Categorical and continuous measures of the same exposure in different studies were meta-analysed separately according to best practice.[19] Effect estimates from analyses of within-participant change were combined with those from analyses of independent groups wherever unstandardised estimates were provided or could be derived to ensure comparable metrics.[20] I^2 indicated the percentage of total variation among study effects due to study heterogeneity rather than chance and τ^2 indicated between-study variance. Sources of heterogeneity were not explored quantitatively given the small number of

studies (usually <4) per meta-analysis. Meta-analyses were summarised using forest plots (Appendix A, Figures A2–A4), which indicate the number of covariates controlled for in each study; however, no systematic pattern was descriptively observed regarding effect direction or significance based on the level of covariate control.

To describe all available evidence, regardless of meta-analysis eligibility, I also summarised all included studies using harvest plots, which graphically synthesise heterogeneous effect estimates.[21] These plots provide a novel summary that illustrates: where evidence gaps exist; whether the weight of evidence suggests positive, statistically non-significant, or negative effects for each type of IPV (physical, psychological, and/or sexual); and study quality. When there were multiple effect estimates using the same data, a decision-making algorithm was followed based on prior reviews[22] to select the most rigorous and relevant data at greatest maturity (Appendix A, Figure A5). Methods for combining multiple effect estimates from a single sample (e.g., robust variance estimation) were not used due to the low number of such cases, which produces inaccurate pooled effect estimates.[23]

3.3 Results

After screening 18,608 titles and abstracts, 309 studies were eligible for full-text review (Figure 3.1). Sixty studies satisfied all selection criteria, totalling 35 cohort samples.[24-83] Effect estimates for 35 of the 60 studies were subsequently meta-analysed. Reasons for exclusion from meta-analyses included: only examining variables not eligible for meta-analysis (e.g., early union, where only one study was available) (n=10);[29,37,38,42,49,52,53,64,65,71] not providing data required for transformations (n=6);[57-60,72,73]

using the same sample as higher-ranked studies, as per the algorithm (n=4);[28,54,78,80] and defining exposures heterogeneously to other studies (n=3).[32,75,82] Additionally, one study used a 56-day diary design too heterogeneous to combine with other studies[76] and another stratified relevant outcome data across multinomial categories that were not pooled[70] – thus both were excluded.

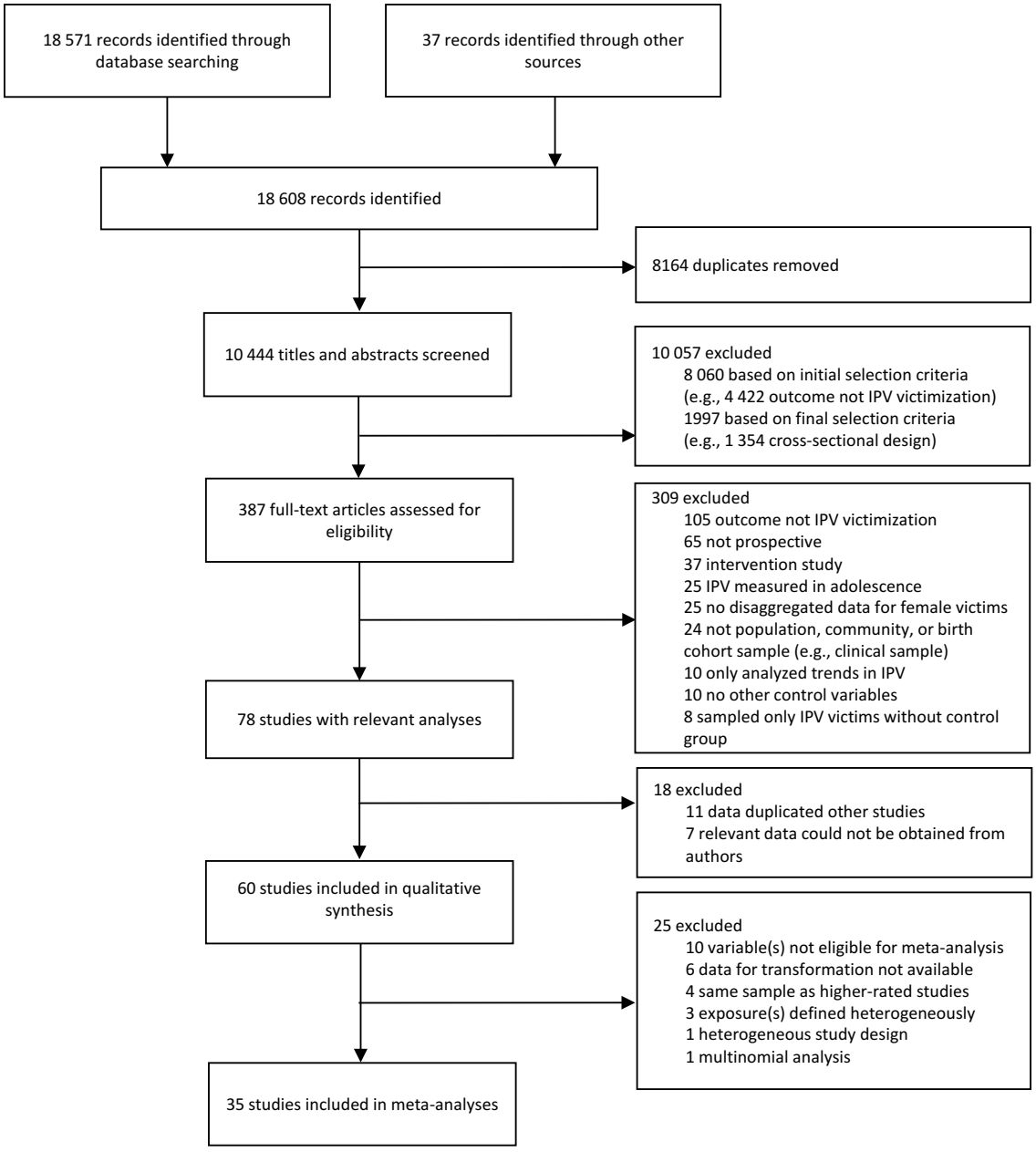

Figure 3.1 Study selection

The 60 included studies were largely USA-based (n=48), with only seven other countries represented – three of which were low- or middle-income countries (LMIC) (Table 3.1). Sample sizes ranged from 118–34,653 participants (M=3,126) and study duration ranged from 56 days–40 years (M=9 years), with an average of 6 follow-ups. Most studies began with an adult sample and just under half (n=29) followed women until a mean age of 30 years or less. Over half of the studies (n=32) used analysis of change when defined liberally; however, this was largely by controlling for baseline IPV (n=23). When defining analysis of change more stringently (analysis of within-subject variation), only nine met this condition. All studies but one analysed physical IPV, whereas only 21 analysed sexual IPV and 17 psychological; seven studies measured all three types. Most studies measured IPV via women's self-report only (n=40). The majority of studies analysed past-year prevalence of IPV, ranging from 1.5% (criminal incidents of physical or sexual IPV[82]) to 80.0% (any physical, psychological, or sexual IPV[83]) (see Appendix A, Table A7 for all study outcomes).

Table 3.1 Summary statistics for 60 included studies

Characteristic	N studies (%)	Mean (SD)	Median (Range)
Year of publication			
1986-1999	8 (13.3)		
2000-2010	31 (51.7)		
2011-June 2016	21 (35.0)		
Country			
Canada	1 (1.7)		
India	3 (5.0)		
New Zealand	4 (6.7)		
South Africa	1 (1.7)		
South Korea	1 (1.7)		
Uganda	1 (1.7)		
UK	1 (1.7)		
USA	48 (80.0)		
Sample size*		3 126.2 (5 705.1)	634 (118-34 653)
Length of follow-up (years)		9.3 (8.8)	7.0 (0.2-40.0)
Number of time-points		5.7 (7.9)	3.0 (2.0-56.0)
Developmental stage at baseline			
Birth	3 (5.0)		
Childhood (0-9 years)	4 (6.7)		
Adolescence (10-19 years)	19 (31.7)		
Adulthood (>19 years)	34 (56.7)		
Demographic area			
Urban	26 (43.3)		
Rural	4 (6.7)		
Both	30 (50.0)		
Relationship status at baseline			
Currently partnered	20 (33.3)		
Currently cohabitated or married	14 (23.3)		
Currently married	13 (21.7)		
Ever partnered	8 (13.3)		
Undefined	5 (8.3)		
Analytical method			
Generalised estimating equation	4 (6.7)		
Multilevel analysis	4 (6.7)		
Path analysis/Structural equation model	6 (10.0)		
Regression:			
Linear	9 (15.0)		
Binary logistic	22 (36.7)		
Multinomial logistic	7 (11.7)		
Ordered	1 (1.7)		
Poisson	1 (1.7)		
Probit	1 (1.7)		
Proportional hazards	1 (1.7)		
Other	4 (6.7)		
Analysis of change			
Yes:	32 (53.3)		
Controlled for baseline IPV	23 (71.9)		
Analysed within subjects (e.g., change scores)	9 (28.1)		
No	28 (46.7)		
Outcome type			
Physical only	29 (48.3)		
Sexual only	1 (1.7)		
Psychological only	0 (0.0)		
Physical and/or sexual	13 (21.7)		
Physical and/or psychological	10 (16.7)		
Physical, sexual, and/or psychological	7 (11.7)		
Outcome reporter			
Self-report	40 (66.7)		
Couple report	17 (28.3)		
Self-report and observation	1 (1.7)		
Couple report and observation	2 (3.3)		
Outcome measure			
Conflict Tactics Scale	30 (50.0)		
Inventory of specific behaviours (e.g., pushed or shoved)	22 (36.7)		
Other scale (e.g., Severity of Violence Against Women)	4 (6.7)		
General items (e.g., experience of 'violence' or 'assault')	4 (6.7)		

N=number. SD=standard deviation.
*Where relevant, couples are counted as a single unit.

Overall, studies scored highly on the quality assessment, largely due to their prospective nature: ranging from 63%–92% (M=76%, SD=7%) (complete scores are in Appendix A, Table A8). Common sources of lower scores were poor or inadequately reported participation rates (n=44) and no analysis of change (n=28), even when defined liberally as controlling for baseline IPV. Twenty-eight studies did not use random sampling and 19 studies had less than 400 participants. Finally, 21 studies used a behavioural inventory to measure IPV with poor or inadequately reported reliability or validity.

3.3.1 Risk and protective factors

Seventy-one risk and protective factors were analysed in the 60 included studies. Most were at the individual (n=27) or relational level (n=37); only seven community-level and no structural-level factors were analysed. Twenty-eight factors were ineligible for meta-analysis due to clinical heterogeneity among studies (n=23) or data not being available for transformations to log odds (n=5). Additionally, for 18 factors, only one study provided data appropriate for meta-analysis. All effect estimates, including the operationalisation of exposures, and forest plots are in Appendix A.

Meta-analyses showed few factors were associated with IPV against women with statistical significance (Table 3.2). Significant protective factors were: at the individual level, being older and, at the community level, living in a disadvantaged neighbourhood. At the relational level, being married was protective against women experiencing IPV, but only when one heterogeneous study that examined being married and monogamous compared to single, separated, or married and polygamous[50] was excluded (Appendix A,

Figure A3). At the individual level, experiencing an unplanned pregnancy was a significant risk factor for IPV against women, which remained stable in sensitivity analyses that also included estimates for unwanted pregnancy (Appendix A, Figure A6). At the relational level, having parents with less than a high school education was a risk factor. Sensitivity analysis with a study that instead compared women whose parents had less than a college education to those with more[44] showed a non-significant pooled association due to heterogeneity (Appendix A, Figure A3).

Most of my meta-analyses had high heterogeneity and/or few included studies, which likely contributed to the paucity of statistically significant findings. However, there were several clinically meaningful (based on strength of effect size and/or confidence interval) associations (Table 3.2). At the individual level, there was an overall trend between non-White racial/ethnic identity and greater odds of experiencing IPV. However, meta-analyses for Black and Latina identity had substantial heterogeneity, potentially because Huang and colleagues[43] studied a higher-risk sample of unmarried mothers, half of whom were welfare recipients. Nonetheless, women who identified as White had 28% lower odds of experiencing IPV than those who did not (95% CI 0.49, 1.05). Additional individual-level factors that showed positive associations were: women's experiences of unwanted pregnancy, binge drinking, marijuana use, experiences of child abuse, and adolescent antisocial behaviour. Women with traditional gender role attitudes had higher odds of experiencing IPV, regardless of whether a liberal (OR=1.11 [95% CI 1.01, 1.21]) or conservative (OR=1.16 [95% CI 0.94, 1.42]) p-value was assumed for the study missing precision estimates.[30]

Table 3.2 Summary of meta-analyses

Factor	N women	N studies	OR (95% CI)	p-value	I^2 (τ^2)
Individual level					
Racial identity:					
Black (vs. all else)	30 669	8	1.15 (0.73–1.81)	.552	89.2% (0.38)
Latina (vs. all else)	27 137	6	1.11 (0.73–1.70)	.615	80.7% (0.19)
White (vs. all else)	3 599	2	0.72 (0.49–1.05)*	.083	0.0% (0.00)
Women's education: less than high school	28 561	5	1.40 (0.98–1.99)*	.064	71.1% (0.10)
Age (continuous)	**19 623**	**10**	**0.96 (0.93–0.98)**	**<.001**	**68.7% (0.00)**
Pregnancy status:					
Recent pregnancy	13 406	3	0.89 (0.64–1.24)	.507	62.1% (0.05)
Unwanted pregnancy	6 874	2	1.24 (0.91–1.69)*	.181	0.0% (0.00)
Unplanned pregnancy	**3 085**	**2**	**1.66 (1.20–2.31)**	**.002**	**0.0% (0.00)**
Youth victimisation	5 572	2	1.67 (0.59–4.72)*	.333	81.8% (0.47)
Alcohol use:					
Binge drinking (binary)	4 604	3	1.20 (0.96–1.49)*	.113	0.0% (0.00)
Greater use (continuous)	2 633	4	1.08 (0.91–1.28)	.400	67.0% (0.02)
Substance use:					
Marijuana use	4 192	3	1.23 (0.93–1.64)*	.153	0.0% (0.00)
Other substance use	4 192	3	1.43 (0.81–2.54)	.221	71.1% (0.18)
Experience of child abuse	1 397	4	1.30 (0.93–1.80)*	.121	0.0% (0.00)
Antisocial behaviour:					
In adolescence	1 342	3	1.15 (0.96–1.37)*	.136	0.0% (0.00)
In childhood	880	2	1.03 (0.89–1.19)	.717	88.1% (0.01)
Traditional gender role attitudes	865	2	1.16 (0.94–1.42)*	.167	0.0% (0.00)
Relational level					
Relationship status:					
Married	**6 410**	**3**	**0.93 (0.87–0.99)**	**.021**	**0.0% (0.00)**
Cohabiting	1 231	2	1.52 (0.90–2.55)*	.114	69.1% (0.10)
Women's family structure (single parent)	3 268	4	1.57 (0.86–2.88)	.140	44.0% (0.16)
Women's higher relationship satisfaction	2 703	3	0.79 (0.56–1.12)	.182	89.9% (0.09)
Positive woman-parent relationship	2 474	2	0.96 (0.91–1.02)*	.182	77.8% (0.00)
Women's parents' education: less than high school	**2 452**	**3**	**1.55 (1.10–2.17)**	**.012**	**0.0% (0.00)**
Greater partner alcohol use	1 550	2	1.07 (0.97–1.17)*	.205	0.0% (0.00)
Women's association with deviant peers	949	2	0.86 (0.66–1.12)	.254	0.0% (0.00)
Women's family SES (lower)	813	2	1.58 (0.78–3.20)	.206	0.0% (0.00)
Partner's experience of greater parental monitoring	365	2	1.69 (0.65–4.39)	.280	76.7% (0.38)
Community level					
Neighbourhood disadvantage	**2 807**	**3**	**0.93 (0.87–1.00)**	**.050**	**0.0% (0.00)**
Residential instability	4 955	2	0.45 (0.06–3.33)	.430	83.7% (1.81)
Higher social support	6 068	3	0.96 (0.88–1.04)*	.311	68.9% (0.00)

Bolded factors are those that were statistically significant ($p \leq .05$).
*Factors with larger effect sizes and/or precision estimates that trended in a positive or negative direction but were not statistically significant.

At the relational level, factors that showed clinically meaningful risk associations with women's IPV experiences were: cohabiting and partners' greater alcohol dependence and consumption. There was a statistically non-significant, protective association between women's IPV experiences and positive parent-relationships. Additionally, each of two studies found that women whose partners had experienced less parental monitoring in childhood had significantly greater odds of experiencing IPV in adulthood.[47,79] However, the pooled estimate was not statistically significant due to substantial heterogeneity. This may have resulted from measurement differences: Theobald and colleagues' measured parental monitoring by child self-report at Age 10 only,[79] whereas Kerr et al. used both

child and parental reports at multiple time-points.[47] Finally, women who had less than a high school education had 40% higher odds of experiencing IPV in adulthood (95% CI 0.98–1.99). At the community level, there was a statistically non-significant, protective association between having more social support and experiencing IPV.

Results were graphically summarised for all but three of the 71 risk and protective factors, even those not meta-analysed. The three factors too diverse to synthesise were women's personality characteristics, partners' personality characteristics, and women's autonomy. Of these, the following were associated with higher odds of IPV against women: women's defensiveness[68] and partners' greater problem-solving tendencies, lower withdrawal tendencies, less femininity, and greater impulsivity.[27,57] Additionally, in an India-based cohort, women whose financial autonomy remained constant across time and who had constant or improved freedom of movement were at lower risk of experiencing IPV.[25]

Figure 2 shows the harvest plots for the remaining 68 factors. Women's race, age, alcohol use, and antisocial behaviour were the most frequently studied, demonstrating a focus on women's characteristics in relation to their experiences of IPV as opposed to characteristics of their partners or contexts. Most factors, regardless of ecological level, were studied in four or fewer studies: 18 of 26 individual-level factors (69%), 28 of 35 relational (80%), and all 7 community factors (100%). For 16 factors, only physical IPV was analysed as an outcome, including partners' substance use, women's life stress, and collective efficacy in women's communities. Factors not meta-analysed but that showed evidence for risk associations with IPV across studies (based on effect sizes and interval estimates) were: at the individual level, women's depressive symptoms and aggressive

tendencies; and at the relational level, couples' lower relationship satisfaction and partners' identification with non-White races, traditional gender attitudes, antisocial behaviour, and unstable employment.


A
Negative effect
Null
Positive effect
Women's race (any non-White effect)
Women's age (older)
Women's alcohol use
Women's marijuana use
Women's substance use
Women's depressive symptoms
Women's broader mental health (E.g., suicide attempt, PTSD)
Women's physical health (E.g., high BMI, HIV status)
Women's traditional attitudes (E.g., approval of marital aggression)
Women's sexual risk (E.g., experience of sexual assault)
Women's pregnancy status
Women's unwanted pregnancy
Women's unplanned pregnancy
Women's religiosity (E.g., importance of religion)
Women's aggressive tendencies
Women's antisocial behaviour
Women's earlier puberty
Women's youth victimization
Women's experience of child abuse
Women's exposure to inter-parental IPV
Women's unemployment
Women's education (higher)
Women's education achievements
Women's school enrolment in adolescence
Women's SES (lower class)
Women's occupation
-90
-80
-70
-60
Legend: IPV outcomes analyzed
Physical
Sexual
Physical and/or sexual
Psychological
Physical and/or psychological
Physical, psychological, and/or sexual
B
Negative effect
Null
Positive effect
Partner's race (any non-White effect)
Partner's age (older)
Age difference between partners (partner older)
Partner's alcohol use
Partner's substance use
Partner's depressive symptoms
Partner's traditional attitudes (E.g., approval of marital aggression)
Partner's sexual risk (Alcohol use before sex)
Partner's aggressive behaviours
Partner's antisocial behaviour
Women's life stress (E.g., child(ren) died)
Partner's negative avoidance coping)
Parenting status
Early union
Relationship status (Greater commitment)
Relationship duration (Longer relationship)
Women's relationship satisfaction
Partner's relationship satisfaction
Family structure (Difference from two-parent family)
Women's association with deviant peers
Women's parent relationship quality (positive)
Partner's parent relationship quality (positive)
Women's experience of low parental monitoring
Partner's experience of low parental monitoring
Partner's unemployment
Partner's education (higher)
-90
-80
-70
-60
Quality score (%)
Panel B continued below

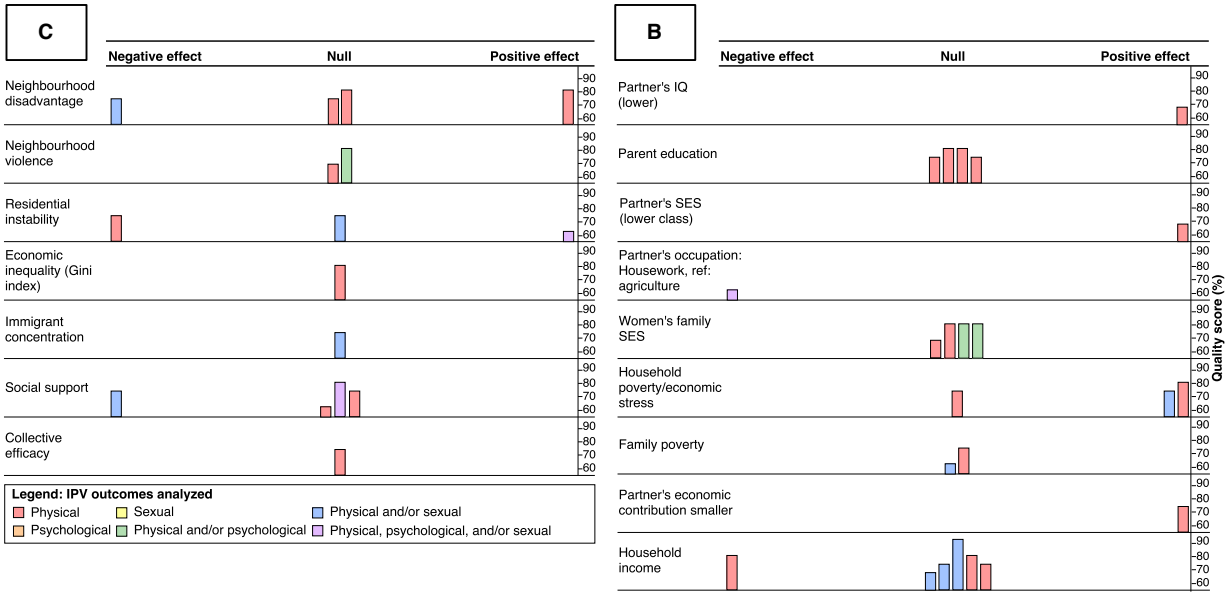

Figure 3.2 Harvest plots of all factors at the individual (A), relational (B), and community (C) level. Each bar represents an individual study, with the position of the bar indicating whether the findings suggested a negative (risk), null, or positive (protective) effect of the factor on IPV against women. Null (i.e., statistically non-significant) results may become statistically significant when pooled together and should not be taken to indicate clinical insignificance. Where necessary, study findings were re-interpreted so that all studies in a single category could be interpreted in the same direction. The height of the bars indicates the quality score limited to the range among included studies (60-100%). The colour of the bar indicates the IPV outcome analysed in the study.

3.4 Discussion

This chapter represents the first systematic review of the longitudinal evidence for all risk and protective factors for IPV against women. I identified 60 prospective-longitudinal studies that investigated 71 risk and protective factors: 64 were individual or relational-level factors, with only seven community-level and no structural factors. Most factors were investigated in four or fewer, largely US-based studies – illustrating the persisting dearth of prospective evidence in LMIC.

The strongest evidence available for modifiable risk factors for IPV against women was for: unplanned pregnancy and parents having less than a high school education, a plausible proxy for lower socioeconomic status.[84] Surprisingly, I found evidence that living in more disadvantaged neighbourhoods may be protective; however, this finding

could be susceptible to residual confounding or selection bias, with no included study hypothesising or explaining this relationship.[39,40,44] Nevertheless, while the association of neighbourhood disadvantage with 'public' criminal behaviours (e.g., vandalism) is well-established and theorised (e.g., social disorganisation theory), the applicability of these criminological theories to more private forms of violence like IPV is less certain.[9,10] This potentially spurious finding highlights the need for further testing of this association prospectively across contexts, with greater attention to the theoretical mechanisms that may link neighbourhood disadvantage to IPV, before policy implications are drawn.

Being older or married were also protective against women experiencing IPV. Since age and marital status are not amenable to intervention, this informs who should be targeted in preventive efforts – younger women who are single or separated and their partners – rather than which factors should be targeted. Several factors also showed positive but not statistically significant risk associations with IPV and should be further investigated. These include: partners' alcohol use and experiences of low parental monitoring as well as women's identification as non-White, unwanted pregnancy, binge drinking, marijuana use, experiences of child abuse, antisocial behaviour, traditional gender role attitudes, cohabitation with partners, negative parent relationship, low social support, and low education.

This review systematically identified the gaps in the prospective-longitudinal literature on IPV, which ultimately limits the generalisability of the above findings. The lack of prospective studies outside the USA means little can be said on contextual differences in the etiology of IPV against women. For instance, parental education may be an important marker for socioeconomic status among women in the USA and other high-

income countries,[84] but potentially less important in LMIC: a hypothesis that should be tested. The statistically or clinically significant findings in this review indicate priority exposures to measure in future prospective studies of IPV in LMIC and other high-income countries beyond the US.

In addition to its USA-bias, most of the literature analysed individual-level risk and protective factors. Twenty-seven factors were related to women's characteristics or behaviours, whereas only 15 were related to those of their partners. Additionally, only ten studies investigated any community factor. Although this is substantially more than the three longitudinal studies identified by prior community-focused systematic reviews,[9,10] no community-level factor was investigated outside the USA and most were measured at only one time-point, limiting conclusions regarding their importance to IPV against women. There were no investigations of structural-level factors, such as alcohol outlet density, societal norms, and gender disparities, despite widespread acknowledgment in public health of the role of social structures in determining outcomes.[1] Prospective research on IPV is thus needed that moves beyond women's characteristics and investigates partner and contextual factors to inform preventive interventions.

Despite the literature's focus on how women's characteristics relate to their own victimisation, these findings should still not be taken to mean that interventions or the responsibility to change should be targeted to women. The complexity of the risk factors identified and broader socio-ecological contexts in which they are embedded, including the overall greater social and economic power and status among men compared to women, must be considered when designing preventive programs. Although the strongest evidence appeared at the individual and relational levels, the best opportunities to lever change may

not be at these levels. Determining the most appropriate intervention strategies requires further qualitative and quantitative appraisals of the nature of each factor and the pathways via which it may causally relate to IPV among women.

An additional gap in the evidence base is the tendency for studies to analyse physical IPV to the exclusion of psychological and sexual IPV. It is consequently unclear how different types of IPV may vary etiologically, which future research should explore. Moreover, most studies began in adulthood and many followed women only until age 30 or less; although resource-intensive, more studies are needed that follow women throughout their lifetime to test early-life factors and the persistence of the associations identified. Indeed, only eight of 25 cohorts meta-analysed (32%) were specifically established to measure women's intimate relationships or experiences of violence, all of which were USA-based[27,31,39,46,68,69,77,83] except one Canadian study.[81] This may indicate a lack of funding for these prospective studies that drives the evidence gaps on IPV against women, especially in LMIC.

Finally, the available prospective-longitudinal studies do not allow for issues of reverse causality to be resolved: only nine measured changes in exposures and outcomes within participants in a repeated-measures design. This means that some included studies did not account for participants' experiences of IPV before being exposed to risk/protective factors and the hypothesised causal relationships may operate in reverse. For instance, the observed association between unplanned pregnancy and higher odds of IPV could operate causally via feelings of anger and jealousy. Yet sexual IPV and coercive control may directly cause unplanned pregnancy, which has been suggested by cross-sectional studies, but analysed in few, if any, prospective-longitudinal studies.[85]

Prospective-longitudinal studies that measure first experiences of IPV and/or changes in violence and exposures over time are thus necessary to clarify our causal understanding of the associations identified in this chapter.

This chapter demonstrates that many popular etiological hypotheses for IPV have been under-studied prospectively. For instance, few included studies investigated the hypothesis that violence is transmitted across generations and results were inconclusive. I found a positive but imprecise relationship between experiencing child abuse and subsequent IPV and little available evidence on exposure to inter-parental IPV. A recent meta-analysis similarly found only small effects between early domestic violence experiences and subsequent IPV, although this included mainly cross-sectional studies.[11] Further limiting conclusions, I identified only USA-based studies that prospectively investigated experiencing child abuse or inter-parental IPV in relation to women's self-reported victimisation. The question thus remains whether these associations exist prospectively and meaningfully across contexts.

Some of the findings in this chapter differ from previous meta-analyses, largely due to my focus on prospective community cohorts and use of modern best practices for meta-analysis. For instance, one meta-analysis of women's alcohol use and subsequent IPV victimisation found a significant positive association among three studies.[7] However, the authors combined categorical[61] and continuous[77] exposure measures and included a hospital-based study not eligible in the current review. Additionally, the authors combined adjusted effect estimates with Martino and colleagues' unadjusted estimate,[61] which showed a positive association between women's alcohol use and IPV; whereas I obtained

from the authors the adjusted, cross-lagged effect estimate, which was not statistically significant.

This chapter has several limitations. First, I only searched and included studies in English, which may have missed some relevant studies. Second, many meta-analyses had high heterogeneity, which I did not have enough statistical power to explore. This is a greater problem when meta-analysing observational studies, which tend to use more diverse methods and adjust for different covariates, as opposed to randomised-controlled trials.[19] Although I summarised the number of covariates controlled for in each study, it was beyond the scope of this chapter to cover qualitative differences, though no systematic pattern was observed. Longer studies may have been more affected by poor covariate control, given unmeasured time-varying confounders, however in descriptive checks no differences in the strength of effect estimates were observed between studies above versus below the median follow-up of seven years. Third, by focusing solely on studies where women self-reported experiencing IPV, I excluded studies that only measured self-reported perpetration of IPV against women. This was necessary to minimise the review's scope to the most sensitive and rigorous studies,[15] however, it means that some relevant risk and protective factors may not have been identified. While there are studies in the field that measure perpetrator characteristics beyond this scope, this chapter highlights that more prospective-longitudinal studies are needed that measure the characteristics of not just women but their partners as well and include both in measuring IPV against women. This will develop more rigorous evidence for informing preventive interventions that accounts for women's experiences of IPV but also the critical role of perpetrating partners' circumstances.

Despite its limitations, this chapter presents the first systematic, meta-analytic review of all risk and protective factors for IPV against women without location, time, or publication restrictions. Accordingly, I retrieved and screened thousands more records than prior reviews and found more prospective-longitudinal studies than previously identified.[5,6,8,12] By only including prospective-longitudinal studies I located the strongest available evidence for risk and protective factors. I also adopted rigorous methods to ensure maximum but appropriate inclusion of studies in meta-analyses. Finally, by adapting harvest plots I presented all included evidence, regardless of meta-analysis eligibility, for a novel and complete summary.

Although the burden of IPV against women has been recognised globally, few effective, preventive interventions exist.[86] Interventions for improving education and preventing unplanned pregnancies may be beneficial, at least in the USA and other high-income countries. Additionally, young and unmarried populations may require targeted interventions. Most conclusively, however, further cross-national, prospective research on the etiological relationships for all types of IPV is needed to inform intervention development worldwide, especially regarding partner and context-related risk and protective factors.

3.5 Chapter summary

This chapter systematically summarised the longitudinal evidence base on the risk and protective factors for IPV against women. Although this review highlighted that many risk and protective factors have been investigated in longitudinal studies, few of these factors had definitive evidence regarding the strength or even direction of their association

with women's experiences of IPV. Most notably, this chapter demonstrated the relative dearth of longitudinal studies investigating community- or structural-level factors for IPV against women or sampling women from outside the USA. In this regard, a surprising protective association was observed between neighbourhood disadvantage and IPV against women; however, theoretical and methodological rigour was lacking in the meta-analysed studies of this association, which were likewise all from the USA. The results of this chapter thus demonstrate the substantial work required to develop a meaningful ecological model for the etiology of IPV against women. Chapters 3-6 build upon these limitations and in particular advance methods and empirical knowledge on the prospective association between neighbourhood disadvantage and IPV against women.

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Chapter 4: A psychometric evaluation of the measurement of IPV in ALSPAC and summary of prevalence, impacts, and gender differences*

4.1 Introduction

As demonstrated in Chapter 3, there have been relatively few prospective-longitudinal studies on the etiology of IPV experiences among women outside of the USA. The remaining empirical work in this thesis thus relies on a long-term cohort study in the UK with rich health and social data available (ALSPAC) which was first introduced and described in Chapter 2. As outlined in Chapter 1, the most recent estimates for the UK indicate that IPV, especially among women, should be a public health priority, with 23% of women and 11% of men reporting any physical, psychological, or sexual IPV in their lifetime.[1,2] However, designing interventions for IPV requires accurately measuring and understanding its burden. Unlike many public health problems, official (e.g., police or hospital-reported) data typically provide poor estimates since most people do not contact formal services after experiencing IPV.[3]

Although survey data on IPV tend to be viewed as more accurate, measurement quality varies widely. While single-term and vague items such as *violence* are insufficient to measure the complexity of IPV, multi-item scales vary in content and length. As outlined in Chapter 1, the most commonly used measure in the field is currently the Conflict Tactics Scale,[4-6] which measures specific behaviours by a current or previous dating, cohabiting, or marital partner. However, the Conflict Tactics Scale has been

*This chapter is adapted from: Yakubovich, A. R., et al. (2019). Intimate partner violence victimisation in early adulthood: psychometric properties of a new measure and gender differences in the Avon Longitudinal Study of Parents and Children. *BMJ Open, 9*(3), e025621. doi:10.1136/bmjopen-2018-025621

criticised for measuring IPV only within the context of conflicts or disagreements and not measuring the intent (e.g., self-defense or harm) or impact of violence.[7,8] Other validated scales include the Composite Abuse Scale,[9] WHO multi-country survey,[10] Abusive Behavior Inventory,[11] Severity of Violence Against Women Scale,[12] Measure of Wife Abuse,[13] and the Extended-Hurt/Insult/Threaten/Scream tool.[14] However, several of these do not measure psychological IPV (including controlling behaviour)[10,12,14] and most are relatively long (>30 items),[9,11-13] risking response burden in larger or repeated-measures surveys.

In recent years, in response to the criticisms and limitations of existing measures, short-form measures of physical, psychological, and sexual IPV have been developed with emerging evidence of validity (e.g., among Canadian women: the short-form Composite Abuse Scale[15]). This chapter presents the first psychometric evaluation of a short-form measure for physical, psychological, and sexual IPV developed in the UK and used for the first time in its current form in ALSPAC, which uniquely also collected data on the impacts of this violence among adult women and men. The aims of this chapter were therefore to: (a) evaluate the psychometric properties of this new instrument; (b) estimate the overall prevalence of IPV and its impacts; and (c) test for gender differences in this UK-based birth-cohort study. This is essential to developing rigorous etiological evidence for IPV against women, which, as demonstrated in Chapter 3, is severely limited outside the USA.

4.2 Method

Data were from ALSPAC, which has established trust among participants, who

have been self-completing questionnaires since age 5 (now in early adulthood), which is ideal for measuring IPV. As detailed in Chapter 2 (Section 2.4), the sampling frame included all pregnant women resident in one of three health districts in Avon, UK due between 1 April 1991 and 31 December 1992.[16,17] The initial number of pregnancies enrolled was 14,541. When participating children were approximately age 7, eligible cases not in the study were contacted, increasing the sample to 15,427 pregnancies, with 14,775 live births (76% of eligible live births) – these children are my target sample. Ethical approval was obtained from the ALSPAC Ethics and Law Committee and Local Research Ethics Committees. A searchable data dictionary is available at http://www.bristol.ac.uk/alspac/researchers/our-data/.

4.2.1 Measuring IPV

At age 21, 3,458 participants completed the online questionnaire, of whom 3,273 (2,128 women, 1,145 men) provided any data on IPV, making this my starting sample. The IPV measures described below (see Table 4.1) were based on items from a previous National Society for Prevention of Cruelty to Children (NSPCC) questionnaire used with an adolescent population in Bristol[18] and a prior study of adolescent populations in five countries,[19] with modified wording and additional items based on a clinical questionnaire used in Bristol.[20] The development group consisted of IPV researchers (Christine Barter, Marianne Hester, Eszter Szilassy, and Gene Feder); prior to use, the questionnaire was piloted for acceptability with the ALSPAC participant advisory group.

Main instrument: IPV victimisation

Eight items measured physical, psychological, and sexual IPV victimisation. A ninth item (feeling scared) was relevant to the impact of this violence and is therefore included with the impact items. Participants indicated the frequency of each item (0=never to 3=often) and whether the behaviour occurred before and/or after age 18, allowing for measurement of temporality.

Impacts of IPV

Ten items measured the psychosocial impacts of IPV. Eight items indicated negative impacts (e.g., upset). One item measured whether the violence had no effect and two measured so-called positive effects (e.g., feeling loved).

Table 4.1 IPV victimisation and impact items

Order	Victimisation items: How often altogether have any of your partners ever done any of the following to you and how old were you?	Type of IPV
1	Told you who you could see and where you could go and/or regularly checked what you were doing and where you were (by phone or text)?	Psychological
2	Made fun of you, called your hurtful names, shouted at you?	Psychological
3	Used physical force such as pushing, slapping, hitting or holding you down?	Physical
4	Used more severe physical force such as punching, strangling, beating you up, hitting you with an object?	Physical
5	Pressured you into kissing/touching/something else?	Sexual/ psychological
6	Physically forced you into kissing/touching/something else?	Sexual
7	Pressured you into having sexual intercourse?	Sexual/ psychological
8	Physically forced you into having sexual intercourse?	Sexual
Order	**Impact items: How did you feel after they these things happened to you?**	**Dimension**
1	Did any of the above make you feel scared or frightened, or did any partner make you feel frightened in any other way?*	Negative
2	Upset/unhappy	Negative
3	Affected my work/studies	Negative
4	Made me feel sad	Negative
5	No effect/not bothered	Null
6	Anxious	Negative
7	Made me drink more alcohol/take more drugs	Negative
8	Felt loved/protected/wanted	Positive
9	Thought it was funny	Positive
10	Angry/annoyed	Negative
11	Depressed	Negative

Note. For each victimisation item, participants indicate the frequency of occurrence – where 0=never, 1=once, 2=a few times, 3=often – and age of occurrence, where 1=under 18, 2=over 18, 3=both. The question prompt included the following definition for partner: 'By partner we mean anyone you have ever been out with or had a relationship with, long-term or short-term (including one night stands)'. For each impact item, participants indicated 'yes' or 'no' as to whether this is how the IPV they experienced affected them.
*This item was asked along with the victimisation items and was therefore measured on the 'frequency' response scale.

4.2.2 Analysis

For the first aim of this chapter, I evaluated the internal consistency, dimensionality, and convergent validity of the IPV victimisation scale. The internal consistency of scale items – in other words, the degree of interrelatedness among the items – is a measure of scale reliability.[21,22] To determine internal consistency, I computed Cronbach's alpha (which is a function of the number of scale items and their average inter-correlation) for the eight IPV victimisation items using the polychoric (rather than Pearson) correlation matrix, which accounts for variables being ordinal rather than continuous.[23,24] Scale dimensionality indicates the number of underlying constructs the scale measures; as this was unknown for the ALSPAC IPV scale, I conducted an exploratory factor analysis using the polychoric correlation matrix.[25] I decided the appropriate number of factors based on their eigenvalues (using Kaiser's criterion that >1 indicates a viable factor), scree plot, and theoretical plausibility.[26] If a two (or more) factor solution was favourable, I decided a priori to use oblique rotation (which allows for correlation between the factors) since I expected differing dimensions of abuse to correlate. To test for possible gender differences in factor solutions, I also ran the exploratory factor analysis separately for women and men. To assess convergent validity – the extent to which measures of theoretically related constructs are correlated – I computed the Pearson correlation between the average frequency of IPV experiences and sum total of negative impacts among those who had experienced any IPV.[27] For this step, I first confirmed (via polychoric correlation) that the negative impacts of IPV (e.g., felt scared) were correlated in the same direction with each other and in the opposite direction from feeling no effect, more loved, or amused (see Table 4.1).

For the second aim of this chapter, I computed the mean of participants' scores across the eight IPV items (reflecting the average frequency of IPV experiences, 0-3), the mean number of IPV acts experienced at least once (0-8), the mean number of negative impacts (0-8), the proportion of participants who experienced any IPV, and the prevalence of any IPV with at least one negative impact. For the third and final aim, I tested for gender differences in each item and summary variable using chi-squared or two-sided t-tests, as appropriate. For the latter, when the Levene's test suggested that the variances of women's and men's scores were unequal, I computed a two-sided t-test for unequal variances.

4.2.3 Patient and public involvement

The IPV measure used in ALSPAC was based on prior questionnaire items used with adolescent populations,[18,19] developed with a young person's advisory group, and the PROVIDE survey,[20] which was discussed with the PROVIDE patient and public involvement group. Additionally, ALSPAC has an advisory panel of >30 participants who meet bi-monthly to advise on study design, methodology, and acceptability. ALSPAC communicates with participants via regular newsletters and has an active website and social media presence.

4.3 Results

Table 4.2 summarises sample characteristics by gender. Women and men were very similar on baseline sociodemographic characteristics: most were White and had characteristics of higher socioeconomic status. At age 21, most women and men saw

themselves as completely heterosexual (83% women, 85% men), followed by a smaller proportion reporting at least some same-sex preferences (16% women, 13% men) and a small number indicating asexuality (<1%). More women (72%) than men (59%), however, had been in relationships longer than 3 months by age 18 and, by age 20, more women (12%) than men (6%) were living with partners or children.

Table 4.2 Sociodemographic characteristics of the sample by gender

	N (%)	
Baseline	**Women**	**Men**
Ethnicity		
Non-White	134 (3.64)	138 (3.74)
White	3,545 (96.36)	3,552 (96.26)
At least one parent had higher than O-level education		
Yes	3,224 (55.29)	3,400 (54.76)
No	2,607 (44.71)	2,809 (45.24)
At least one parent part of lower social class (partly or unskilled occupation)		
Yes	1,150 (23.76)	1,167 (22.87)
No (Both parents in professional, managerial, or skilled occupations)	3,690 (76.24)	3,936 (77.13)
Mother married		
Yes	4,807 (75.30)	5,100 (74.53)
No	1,577 (24.70)	1,743 (25.47)
Lived with both biological parents		
Yes	4,489 (90.29)	4,830 (90.26)
No	483 (9.71)	521 (9.74)
Early adulthood (Ages 18-21)		
Longest relationship (at Age 18)		
More than 3 months	1,632 (72.18)	1,034 (58.78)
Less than or equal to 3 months	629 (27.82)	725 (42.22)
Living arrangements (at Age 20):		
One or both parents	1,200 (48.21)	819 (51.51)
Partner and/or children	307 (12.33)	98 (6.16)
Other	982 (39.45)	673 (42.33)
Sexual preference (at Age 21)		
Asexual	8 (0.37)	6 (0.51)
Any same-sex preferences	358 (16.63)	160 (13.72)
100% heterosexual	1,787 (83.00)	1,000 (85.76)

4.3.1 Reliability and validity

Correlations were strong between all IPV scale items, ranging from .57 (between experiencing humiliation/name-calling/shouting and forced sexual touch) to .92 (between forced and coerced touch) (Appendix B, Table B1). The alpha coefficient was .95, indicating strong internal consistency.

The exploratory factor analysis suggested a one- or two-factor solution (see Appendix B, Table B2 for factor loadings). Only the first factor had an eigenvalue more than 1 (5.8). All items loaded highly onto this factor (ranging from 0.8-0.9), which suggests that using a factor-based score for experiences of IPV overall is a valid analytic method in this sample. The scree plot plateaued between the second and third factor, and as the second factor had an eigenvalue close to 1 (0.8), I also attempted a two-factor solution with oblique rotation. This two-factor solution fit the data well, indicating plausible dimensions for (a) physical and psychological IPV and (b) sexual IPV. This suggests that analyses using a latent variable approach could reliably analyse these two factors. The factor analysis did not support a three-factor solution: the third factor had a low eigenvalue (0.2) and no items with a loading greater than 0.3. Overall, results were similar when factor analyses were run separately by gender (Tables B3-B4, Appendix B): all items loading highly onto a single factor and the same two-factor solution was identified for women and men.

As expected, the eight adverse impacts of IPV (e.g., felt scared, upset – see Table 4.1) were all correlated in the same direction with each other (ρ=.30-.89, Appendix B, Table B5). These items were further correlated in the opposite direction with IPV having no impact, seeming funny, or increasing perceptions of being loved, protected, or wanted (ρ=-.26 to -.86). Finally, these latter three impacts were correlated in the same direction with each other (ρ=.42-.64). I therefore correlated the sum total of the adverse impacts of IPV with the average frequency of IPV experiences among those who had experienced any IPV. As expected, experiencing more frequent IPV was strongly correlated with experiencing more negative impacts (N=1,111): r=.58, p<.001.

4.3.2 Overall prevalence

As shown in Table 4.3, the most frequently experienced IPV was psychological (e.g., 25% reported humiliation, name-calling, or shouting) and the least experienced was sexual (e.g., 4% reported forced sex). Among those who experienced any IPV, the majority of violent acts (>78%) occurred after age 18 (see Table B6, Appendix B for more detail). Most participants reported at least one negative impact following IPV, with the most common being feeling upset (78%) or angry (75%). The least common impacts of IPV were the positive ones: 13% of participants reported that the violence made them feel loved, protected, or wanted; 14% found the violence amusing.

Overall, 37% of participants reported experiencing any IPV and 29% experienced any IPV after age 18. The mean number of IPV acts experienced among those who experienced any violence, ranging from 1 to 8, was 3.0 (SD=2.1) overall and 2.2 (SD=1.6) after age 18. The mean number of negative impacts, ranging from 0 to 8, was 4.0 (SD=2.4) among those who had experienced any IPV and 2.9 (SD=2.6) among those who had experienced IPV after age 18.

Table 4.3 Frequencies of 8 IPV victimisation and impact items

		N (%)			
Victimisation items	**Total N**	**Never**	**Once**	**A few times**	**Often**
Told you who you could see and where you could go and/or regularly checked what you were doing and where you were (by phone or text)	3,268	2,544 (77.85)	124 (3.79)	322 (12.91)	178 (5.45)
Made fun of you, called you hurtful names, shouted at you	3,253	2,422 (74.45)	170 (5.23)	530 (16.29)	131 (4.03)
Used physical force such as pushing, slapping, hitting or holding you down	3,255	2,768 (85.04)	193 (5.93)	235 (7.22)	59 (1.81)
Used more severe physical force such as punching, strangling, beating you up, hitting you with an object	3,252	3,075 (94.56)	81 (2.49)	68 (2.09)	28 (0.86)
Pressured you into kissing/touching/something else	3,255	2,981 (96.58)	96 (2.95)	146 (4.49)	32 (0.98)
Physically forced you into kissing/touching/something else	3,250	3,115 (95.85)	68 (2.09)	49 (1.51)	18 (0.55)
Pressured you into having sexual intercourse	3,242	2,876 (88.71)	181 (5.58)	152 (4.69)	33 (1.02)
Physically forced you into having sexual intercourse	3,239	3,118 (96.26)	80 (2.47)	32 (0.99)	9 (0.28)
Impact items	**Total N**	**Never**	**Once**	**A few times**	**Often**
Scared or frightened in any way	3,221	2,711 (84.17)	191 (5.93)	234 (7.26)	85 (2.64)
Impact items: Only those who experienced at least 1 act of IPV	**Total N**	**Yes**		**No**	
Upset/unhappy	1,148	900 (78.40)		248 (21.60)	
Angry/annoyed	1,139	857 (75.24)		282 (24.76)	
Made me feel sad	1,142	813 (71.19)		329 (28.81)	
Affected my work/studies	1,141	799 (70.03)		342 (29.97)	
Anxious	1,133	495 (43.69)		638 (56.31)	
Depressed	1,138	418 (36.73)		720 (63.27)	
No effect/not bothered	1,133	206 (18.18)		927 (81.82)	
Made me drink more alcohol/take more drugs	1,138	168 (14.76)		970 (85.24)	
Thought it was funny	1,132	158 (13.96)		974 (86.04)	
Felt loved/protected/wanted	1,135	148 (13.04)		987 (86.96)	

4.3.3 Gender differences

As shown in Table 4.4, for all IPV victimisation items, regardless of whether lifetime or early adulthood (ages 18-21) was considered, significantly more women experienced violence than men. The largest percentage difference was for the lifetime prevalence of coerced sex (15% women, 4% men). Moreover, significantly more women than men reported experiencing all negative impacts of IPV, apart from substance use where there was no difference. The greatest percentage difference was in feeling scared because of their partner (56% women, 14% men in their lifetime). In contrast, more men than women reported that the IPV they experienced was funny or had no effect on them. Finally, every test indicated that women experienced more frequent and severe IPV overall than men, in both their lifetimes and early adulthood (Table 4.5): women experienced more frequent and more acts of IPV compared to men; more women than men experienced any IPV (with or without negative impact); and, among those who had experienced any IPV, women experienced more negative impacts than men.

Table 4.4 Gender differences in IPV victimisation and impact items

Victimisation items	Lifetime Women (n=2,050)	Men (n=1,108)	χ^2 (p)	Ages 18-21 Women (n=2,014)	Men (n=1,092)	χ^2 (p)
Told you who you could see, where you could go, or regularly checked what you were doing and where you were	510 (24.88)	196 (17.69)	21.41 (<.001)	346 (17.18)	152 (13.92)	5.59 (.018)
Made fun of you, called you hurtful names, shouted at you	596 (29.07)	210 (18.95)	38.75 (<.001)	443 (22.00)	166 (15.20)	20.74 (<.001)
Used physical force such as pushing, slapping, hitting or holding you down	362 (17.66)	106 (9.57)	37.31 (<.001)	245 (12.16)	82 (7.51)	16.29 (<.001)
Used more severe physical force such as punching, strangling, beating you up, hitting you with an object	142 (6.93)	31 (2.80)	23.68 (<.001)	96 (4.77)	22 (2.01)	14.67 (<.001)
Pressured you into kissing/touching/something else	240 (11.71)	26 (2.35)	81.70 (<.001)	144 (7.15)	20 (1.83)	40.05 (<.001)
Physically forced you into kissing/touching/something else	125 (6.10)	7 (0.63)	53.65 (<.001)	72 (3.57)	5 (0.46)	28.46 (<.001)
Pressured you into having sexual intercourse	313 (15.27)	43 (3.88)	93.25 (<.001)	192 (9.53)	36 (3.30)	40.49 (<.001)
Physically forced you into having sexual intercourse	114 (5.56)	5 (0.45)	51.79 (<.001)	64 (3.18)	5 (0.46)	24.12 (<.001)
Impact items (Among those who experienced any IPV)	**Women (n=800)**	**Men (n=292)**	**χ^2 (p)**	**Women (n=552)**	**Men (n=221)**	**χ^2 (p)**
Scared	444 (55.50)	40 (13.70)	151.47 (<.001)	279 (50.54)	31 (14.03)	87.61 (<.001)
Upset/unhappy	684 (85.50)	179 (61.30)	75.58 (<.001)	465 (84.24)	141 (63.80)	38.92 (<.001)
Angry/annoyed	625 (78.12)	195 (66.78)	14.72 (<.001)	441 (79.89)	152 (68.78)	10.91 (.001)
Made me feel sad	621 (77.62)	157 (53.77)	59.44 (<.001)	425 (76.99)	122 (55.20)	36.22 (<.001)
Affected my work/studies	275 (34.38)	51 (17.47)	29.21 (<.001)	187 (33.88)	38 (17.19)	21.28 (<.001)
Anxious	406 (50.75)	73 (25.00)	57.60 (<.001)	272 (49.28)	60 (27.15)	31.53 (<.001)
Depressed	329 (41.12)	69 (23.63)	28.27 (<.001)	231 (41.85)	52 (23.53)	22.82 (<.001)
No effect/not bothered	109 (13.63)	89 (30.48)	40.94 (<.001)	74 (13.41)	59 (26.70)	19.57 (<.001)
Made me drink more alcohol/take more drugs	127 (15.88)	34 (11.64)	3.05 (.081)	92 (16.67)	30 (13.57)	1.14 (.287)
Thought it was funny	64 (8.00)	92 (31.51)	96.53 (<.001)	48 (8.70)	67 (30.32)	58.26 (<.001)
Felt loved/protected/wanted	101 (12.62)	41 (14.04)	0.38 (.538)	80 (14.49)	32 (14.48)	0.00 (.996)

Note. Victimisation items were coded as 1=experienced at least once, 0=never experienced. Impact items were 1=yes, 0=no.

Table 4.5 Summary statistics for comparisons between women and men on overall IPV victimisation and impact

Item	Lifetime Women N	M (SD) or N (%)	Men N	M (SD) or N (%)	t(df) or χ^2	p	Ages 18-21 Women N	M (SD) or N (%)	Men N	M (SD) or N (%)	t(df) or χ^2	p
Mean frequency of IPV experiences (SD)	2,128	0.28 (0.50)	1,145	0.12 (0.25)	12.61 (3,252.18)	<.001	2,128	0.19 (0.39)	1,145	0.10 (0.24)	7.58 (3,219.11)	<.001
Mean number of IPV acts experienced (SD)	2,024	1.41 (2.19)	1,096	0.60 (1.22)	13.16 (3,115.22)	<.001	2,014	0.75 (1.47)	1,092	0.42 (0.97)	7.55 (2,996.44)	<.001
Any IPV (N, %)	2,128	884 (41.54)	1,145	330 (28.82)	51.62	<.001	2,128	683 (32.10)	1,145	275 (24.02)	23.47	<.001
Any IPV with a negative impact (N, %)	2,126	788 (37.06)	1,145	228 (20.63)	93.41	<.001	2,126	608 (28.60)	1,145	200 (17.47)	49.57	<.001
Mean number of negative impacts of IPV (SD)	800	4.39 (2.27)	292	2.73 (2.21)	10.75 (1,090)	<.001	746	3.21 (2.72)	279	2.24 (2.24)	5.77 (602.84)	<.001

Note. All t-tests were two-group t-tests with unequal variances, apart from 'number of negative impacts of IPV' for the overall sample, which did not have unequal variances between men and women (i.e., the Levene's test was statistically non-significant).

4.4 Discussion

This chapter estimated the prevalence of physical, psychological, and sexual IPV in a UK birth cohort (ALSPAC) during early adulthood using a novel measure. The prevalence of IPV was high: 37% of participants had experienced any IPV in their lifetime and 29% had experienced IPV between ages 18 to 21. As in previous research, the most commonly experienced violence was psychological and the least commonly experienced was sexual.[28] Over three-quarters of those who had experienced IPV had experienced this violence when they were aged 18 or older. This aligns with the broader IPV literature, as demonstrated in Chapter 3, which showed that early adulthood is an especially high-risk period for experiencing IPV. Most participants who had experienced IPV reported more than one negative psychological impact, with the most common being feeling upset or angry. The least common outcomes of IPV were finding the violence amusing or feeling more loved, wanted, or protected.

I found strong and consistent gender differences: for all types of violent behaviours, women experienced more frequent IPV than men, both in their lifetime and early adulthood. As in other prevalence surveys, the most dramatic differences between women and men were on sexual violence items.[28] For instance, the proportion of women who had ever experienced coerced sex was more than four times that of men. Moreover, significantly more women than men reported experiencing negative psychosocial impacts from IPV. For example, the proportion of women who felt afraid of their partner was more than four times that of men. Similar proportions of women and men reported that their alcohol and substance use increased after experiencing IPV. The evidence on whether there are gender differences in substance use following IPV is inconsistent;[29] one possible

explanation for similar proportions is the greater psychological impacts of IPV among women balance with the greater baseline tendency among men to use substances.

In contrast, the proportion of men who found their experiences of IPV amusing was more than three times that of women. More than double the proportion of men also reported that this violence did not affect them. Together with the gender differences in the negative impacts of IPV, this suggests that women experience more severe IPV than men, which is more difficult to trivialise and more likely to cause psychological harm. This extends a large body of evidence, outlined in Chapter 1, which demonstrates that women experience the majority of severe consequences of IPV and are more likely to have controlling, violent partners.[30,31] At the same time, participants' reporting on the impacts and interpretations of their IPV experiences may have been influenced by gender-role socialisation. Women may have more readily reported the negative consequences of violence from their partners whereas men may have felt more pressure to minimise their experiences and deny negative consequences due to internalised concepts of masculinity, for instance, as strong and powerful and femininity as vulnerable and weak.[32-34] Future survey research could explore this hypothesis by including questions on traditional gender-role attitudes.

The findings of this chapter contribute to the broader debate on gender asymmetry in IPV. Studies using the Conflict Tactics Scale or family conflict surveys tend to find equivalent prevalence estimates among women and men;[35] whereas crime or clinical surveys tend to find that women experience more IPV than men.[8,35] The data used in this chapter came from a community-based birth-cohort study, using a measure without reference to crime or conflict resolution, which minimises these priming biases. My

findings demonstrate that women experience more frequent and severe IPV than men, but also confirm that a considerable number of men experience violence from their partners. Therefore, my results support the continued research and advocacy enterprise for IPV against women in particular, while also demonstrating the need for resources to continue to be developed for both women and men.

The new measure for IPV used in ALSPAC and evaluated in this chapter showed excellent internal consistency and a strong, positive correlation with negative impacts of IPV, indicating convergent validity. The exploratory factor analysis suggested that the measure could be reliably analysed as a single dimension of IPV or as two – (a) physical or psychological IPV and (b) sexual IPV. As the scale items all loaded highly onto a single factor, analysing a factor-based score is appropriate and has the benefit of maintaining the measure's original scaling for more intuitive interpretation.[36] Overall, factor structures were equivalent among women and men. This should be confirmed in new samples, including tests of gender invariance, which overall has been understudied in the literature.[37]

The limitations of this chapter include, firstly, that the age of first occurrence of IPV before age 18 among participants could not be considered as this was not measured in ALSPAC. Second, the definition of intimate partner used in ALPSAC was broad, from casual sex partners to long-term relationships. Since this could capture sexual violence by an acquaintance, future research should use a more constrained definition (e.g., dating or marital partner). Third, the ALSPAC instrument did not measure specific instances of IPV or specific relationships; it is therefore unclear whether IPV was experienced by multiple perpetrators or repeatedly during a single relationship. I was also unable to determine

which types or instances of IPV caused the impacts reported based on the available data. Although more time intensive, it would be useful if future uses of the ALSPAC instrument allowed participants to indicate the perpetrator(s) and impact(s) of each experience of IPV. Obtaining more detailed information on IPV events and the relationship context would help determine intent and precipitants to inform directions for intervention research. Relatedly, data from participants' partners or an equivalent measure of IPV perpetration were not collected in ALSPAC. However, in the absence of sampling partners, self-reported victimisation is a more sensitive measure of IPV than self-reported perpetration.[38,39] Moreover, although IPV experiences may have involved the use of violence as well, that a greater proportion of women experienced nearly every measured negative impact from IPV compared to men suggests important differences in the severity and experience of IPV that remain critical to consider both for research and clinical practice.

Fourth, the IPV impacts measured in ALSPAC were mainly psychological, to the exclusion of further physical (e.g., injury) or socioeconomic consequences (apart from work or studies being affected). Additionally, the impact items were largely one-word terms such as *depressed* and *anxious*, which are more vulnerable to social desirability bias and internalised gender concepts as opposed to a scale of items measuring depression or anxiety.[40] Nevertheless, in longer surveys such as those used in ALSPAC, it may not be feasible to include more exhaustive measures of IPV impacts. Fifth, ALSPAC did not include alternative IPV measures to further evaluate the measure's convergent validity. Assessing convergence with long-form IPV measures in particular may be useful to determine if scale length or breadth has any impact on sensitivity or gender differences. Finally, as first discussed in Chapter 2 (Section 2.4), higher socioeconomic positions and

White persons are over-represented in this sample: the generalisability of this chapter's results to the greater UK population or other contexts requires further investigation (the implications of this are further discussed in Chapter 7).

Despite these limitations, this chapter has a number of strengths. The IPV measure used was brief and could therefore be implemented in surveys measuring a rich set of potential IPV predictors. Both women and men were sampled, allowing for analyses of gender differences. Although age of first occurrence was not measured in ALSPAC, participants indicated whether IPV occurred before and/or after age 18, which made it possible for me to compute a current prevalence of IPV (last three years). This further allows for analyses of temporal relationships between antecedents and early adulthood IPV using ALSPAC data, as conducted in this thesis in Chapter 6. Finally, my analyses were thorough and demonstrated consistent results, allowing for firm conclusions on the reliability of the IPV measure and gender differences in IPV in the ALSPAC cohort.

4.5 Chapter summary

This chapter showed that more than one-third of participants engaged in a cohort study for over 20 years in the UK had experienced IPV by early adulthood, demonstrating the public health import of this violence. Women consistently experienced more frequent and severe IPV than men by all measures, suggesting important gender differences in the burden of this violence and justifying the focus of this thesis (as first articulated in Chapter 1) on IPV against women as a unique clinical problem. The ALSPAC measure for IPV victimisation further showed strong indicators of reliability and validity, demonstrating its appropriateness for further etiological study in the current thesis as well as future

etiological studies. The investigation of neighbourhood disadvantage and IPV against women presented in Chapter 6 builds on the psychometric knowledge of the scale developed in this chapter. Before this, Chapter 5 explores the longitudinal trajectories and relationships between the available measures of neighbourhood disadvantage in ALSPAC to inform this final investigation.

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Chapter 5: Investigating the dynamic relationship between perceived and objective measures of the neighbourhood environment over time*

5.1 Introduction

As first outlined in Chapter 1 (Section 1.3), etiological theories of health and criminological outcomes typically hypothesise that structural and community contexts, including neighbourhood environments, affect individuals' health, wellbeing, and behaviour.[1-11] However, a fundamental issue in investigating neighbourhood effects is how to measure the neighbourhood environment.[12] *Objective* measures of neighbourhoods allow us to capture distinct aspects of the physical and social environment (e.g., available facilities or services, crime or unemployment rates, ethnic or racial composition) within defined physical areas (e.g., census units). In theory, this allows for conclusions regarding whether and how these features or phenomena relate to health outcomes, informing intervention and policy targets. In practice, these measures are also often routinely collected and thus convenient to access and use.

There are at least two important limitations of these objective measures. First, they fail to capture experiential or relational aspects of the neighbourhood environment, which are often hypothesised as part of the mechanisms of neighbourhood effects.[12-14] For instance, as discussed in Chapter 1, one of the most prolific neighbourhood theories posits that collective efficacy, or social cohesion between members of the neighbourhood and their perceived willingness to intervene on behalf of the 'common good' (i.e., informal social control), mediates the positive relationship between neighbourhood disadvantage

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and various health and criminological outcomes.[1,11,15] Measuring these social constructs requires reports from neighbourhood members, typically not collected in routine 'objective' data. Second, the definitions of neighbourhoods used (e.g. administrative tracts) in objective measures do not always correspond with individuals' own conceptions of their neighbourhoods and/or community contexts.[13,16-19] These varying definitions of neighbourhoods can lead to dramatically different results when used in analyses of neighbourhood effects—the so-called modifiable areal unit problem (discussed in Chapter 2, Section 2.3.2).[20]

Perceived measures of neighbourhoods can address these limitations by allowing participants to report on the social contexts of their neighbourhoods that are more difficult (or impossible) to capture by objective measurement, as well as the physical components of their neighbourhoods as they conceive and experience them. These perceived measures are often thought to correlate with objective measures of neighbourhoods: both in theories, like collective efficacy theory, which directly hypothesise mediating mechanisms, as well as the general expectation that perceptions of the environment will be influenced by its objective conditions.[3,15,21] The extent to which perceived and objective measures of neighbourhoods are in fact related, and the factors that underlie varying perceptions, has important implications both for how we should study the etiology of neighbourhood effects, what our results mean, and the development of intervention targets.

The extent and determinants of overlap between individuals' perceptions of their neighbourhood and its objective conditions has largely been studied in the context of physical activity.[22-29] These studies have found relatively low agreement between perceived and objective measures of, for instance, walkability or the proximity of

neighbourhood features (e.g., sidewalks), with sociodemographic and psychosocial factors (e.g., age, gender, ethnicity, residential mobility, socioeconomic status, marital status, and depression) underlying variation in people's perceptions of the built environment. Both within and beyond the physical activity literature, studies have also demonstrated low to moderate correspondence between police-reported and perceived crime and their unique impacts on health and wellbeing.[26,30-35] A more limited number of studies have further investigated and found differential impacts of perceived and objective measures of broader neighbourhood conditions on health and behavioural outcomes.[36-41]

Across these studies, however, a major limitation has been the primary use of point-in-time measures of neighbourhoods. As a result, available studies have not accounted for how the relationship between perceived and objective measures may vary over time, or more specifically over the life course, nor the factors underlying dynamic differences in neighbourhood perceptions. This chapter sought to address these evidence gaps. Using 18 years of data, I had two aims: (a) evaluate the dynamic nature of the relationship between perceived and objective measures of neighbourhood environments and (b) determine how social factors are related to neighbourhood perceptions over time. These inquiries are further enriched by considering broader experiences of neighbourhood environments (e.g., perceived neighbourhood disorder or social cohesion) rather than the built environment or crime alone.

5.2 Method

Data were from ALSPAC, the study methods for which were detailed in Chapters 2 and 4. Briefly, the total ALSPAC sample included 14,775 live births (76% of eligible live

births) between 1 April 1991 and 31 December 1992 and their mothers. In this chapter, I used data across ten time points from the sample of participating mothers whose children were also enrolled in the study (see Table 5.1). As shown, the time points in ALSPAC are labelled by the age of the enrolled children, which I maintain in this chapter. Mother-reported data were used in this chapter because, as can be seen from Table 5.1, children were aged 0-18 over the study period and longitudinal self-reported data on their neighbourhood environments are not yet available in ALSPAC. The ALSPAC Ethics and Law Committee and local research ethics committees provided ethical approval.

Table 5.1 Timing of measurement for neighbourhood variables

	Time point									
Age of mother's eligible child	**0**	**1.75**	**3**	**5**	**7**	**10**	**12**	**14**	**16.5**	**18**
Year	**1990**	**1992**	**1993**	**1994**	**1997**	**1999**	**2002**	**2006**	**2007**	**2010**
Objective neighbourhood deprivation	■	■	■	■	■	■	■	■	■	■
Perceived neighbourhood variables:										
Neighbourhood opinion	■	■	■	■	■	■				■
Social cohesion	■	■	■	■	■	■				■
Neighbourhood disorder		■	■	■	■	■				■

Note. Shaded box indicates variable was measured at time point indicated.

5.2.1 Measures

Objective neighbourhood measure

As discussed in Chapter 2, available in ALSPAC was England's official measure of neighbourhood deprivation: the Indices of Multiple Deprivation.[42] ALSPAC participants' addresses were linked to the Indices for ten time points from baseline (pregnancy) to age 18 (Table 5.1), based on the enrolled children's addresses, which were recorded annually for sending regular communications and questionnaires. I thus used the Indices of Multiple Deprivation as the measure of objective neighbourhood deprivation in this chapter. This requires the assumption that the addresses of mothers and their enrolled child were the

same, which is reasonable in most cases given the children's ages during the study period (0-18 years) and the fact that mothers were typically their primary caregivers.

I used the 2010 Indices of Multiple Deprivation, which are composed of 38 indicators across seven domains of deprivation (income, employment, education, health, crime, housing, and living environment) (see Table C1, Appendix C). Each lower-layer super output area (LSOA) in England – small census areas of ~1500 residents or 650 households that approximate residential neighbourhoods – is assigned a domain-specific and total deprivation rank score relative to all other neighbourhoods.[43] I had access only to participants' quintile ranks for each domain and the total deprivation index – determined by ALSPAC to protect anonymity – so that at each time point (see Table 5.1), I knew the quintile of deprivation each participant's residential neighbourhood fell into relative to all other neighbourhoods in England (i.e., not just within ALSPAC). Due to the underlying exponential distribution of the deprivation scores (see Figure C1, Appendix C), differences in deprivation are greater between the most deprived quintiles compared to the least (i.e., there is less variation in deprivation between the less deprived quintiles). To account for this, I computed a binary variable at each time point, where 1=deprivation quintiles 4 and 5 (i.e., living in the top 40% most deprived neighbourhoods in England at that time) and 0=otherwise. This allowed for a comparison of exposure to more versus less objective neighbourhood deprivation while still maintaining response variation (the proportion of participants in quintile 5 decreased to ~6% over time).

Perceived neighbourhood measures

All items used in each composite variable are in Table C2 (Appendix C). Mothers reported on three aspects of their neighbourhoods from pregnancy until participating children were aged 18 (Table 5.1). Mothers rated their neighbourhood as a place to live, where 1=very good place, 2=fairly good place to live, 3=not a very good place to live, or 4=not at all a good place to live. At later time points few mothers scored their neighbourhoods as not a very good place to live (3) or not at all a good place to live (4). Therefore, to maximize cell counts, I dichotomised *negative neighbourhood opinion* as 0=very good place (1) and 1=otherwise (2-4). *Poor social cohesion* was measured as the mean score on seven items (e.g., how often neighbourhood people visit the home) on a 5-point scale (0=always, 4=never) (α=.78-.82 at each time). *Neighbourhood disorder* was the mean score on 11 items (e.g., the size of the problem in the neighbourhood of noise from other homes) on a 3-point scale (0=no problem or opinion, 2=serious problem) (α=.78-.82). At the age 18 time-point, the neighbourhood disorder items changed slightly (e.g., badly fitted doors/windows removed; traffic added) (Table C2, Appendix C). All variables were measured at seven times apart from neighbourhood disorder, which was not measured in pregnancy.

Sociodemographic and psychosocial variables

To investigate the social factors potentially underlying participants' perceptions of their neighbourhoods, I selected variables based on the literature and data availability;[22-29] more details on measurement are included in Appendix C. I used the following variables reported by mothers at baseline: parental education (higher than standard high school qualifications), marital status (married versus not), lower social class (either mother or partner in partly or unskilled occupations), mothers' race/ethnicity (non-White versus not),

and residential mobility (mothers had moved house since becoming pregnant), maternal depressive symptoms (Edinburgh Post-Natal Depression Scale,[44] α=.85), maternal social support (ALSPAC Social Network Index, α=.79), and the sum total of household difficulties affording food, clothing, heating, accommodation, or items for child(ren).

5.2.2 Analysis plan

The first aim of this chapter was to evaluate the dynamic nature of the relationship between the perceived and objective neighbourhood measures over time. To this end, I conducted dual trajectory analyses between greater versus lesser neighbourhood deprivation and each perceived neighbourhood measure across the study period using the Stata *traj* plugin.[45,46] This group-based trajectory modelling approach is a finite mixture modelling method that identifies latent classes of individuals in the sample who follow similar trajectories on a particular measure over time in order to approximate the unknown trajectories followed by members of the population. The benefits of this method are that I was able to quantitatively evaluate the dynamic relationship between perceived and objective neighbourhood measures, account for heterogeneity in within-participant change, and make use of both the binary (using a logit model) and scale measures (using a censored normal model). Results include participants' estimated trajectories for each of the perceived and objective measures and the joint and conditional probabilities of trajectory group membership across the perceived and objective measures.

I determined the appropriate number of trajectory groups for each neighbourhood measure separately based on the Bayesian Information Criterion (greater values indicate better fit), parameter estimates (weak estimates indicate little additional value), and the

substantive contribution of each additional group (favouring a parsimonious and theoretically meaningful model).[45,46] Trajectory shapes (i.e., polynomial orders) were determined from the size and precision of the parameter estimates and model fit. I ran dual trajectory analyses between the objective measure model and each perceived measure model to quantify their relationships. At each stage, I considered additional model fit diagnostics according to standard best practice: (a) average posterior probability of group membership for individuals assigned to each group $\geq .7$ (i.e., the probability of belonging to a trajectory group among its group members), (b) odds of correct classification ≥ 5 (i.e., the odds of belonging to the trajectory group based on the posterior probabilities relative to the odds of actually being a group member), (c) estimated and observed probabilities of membership for each group are similar, and (d) non-overlapping confidence intervals of estimated trajectories.[45] Group-based trajectory modelling uses maximum likelihood estimation, which will produce unbiased parameter estimates as long as data are missing at random (i.e., the likelihood of being missing is related to the observed data but not the missing values themselves). I further excluded participants from the trajectory analyses if they did not have at least three time points of neighbourhood data available to improve group classification.[47] Models remained robust when I allowed more or less missing data, but diagnostics worsened or less common trajectories became more variable, respectively.

The second aim of this chapter was to determine how trajectories of neighbourhood perceptions are correlated with socioeconomic and psychosocial factors. To this end, I conducted multinomial logistic regression where, for each perceived neighbourhood variable, trajectory group membership was regressed onto mothers' baseline covariates, controlling for baseline objective neighbourhood deprivation exposure. This method is more prone to misclassification bias compared to estimating covariate associations

simultaneously with trajectory group parameters;[48] however, the latter also often leads to models that fail to converge.[49] This was the case for one of my three models (perceived neighbourhood disorder). Nevertheless, for the two models that did converge, comparisons of the covariate associations between models estimated with and without simultaneously estimating the trajectory groups showed consistent conclusions, with the latter analyses (presented in this chapter) tending to be slightly more conservative. Results are discussed according to clinical significance (i.e., the size or precision of the estimate).[50]

5.3 Results

Table 5.2 describes the sample's sociodemographic and psychosocial characteristics. A small proportion of mothers were non-White (2%). At baseline, 11% of mothers had moved since becoming pregnant. Most mothers (or their partners) had higher than standard high school qualifications (58%) and most were married (78%). Just over one-fifth of mothers (or their partners) were part of a lower occupational social class (22%). Mothers' average financial difficulties score was 2.74 (SD=3.44), with 62% experiencing any financial difficulty at baseline. Their mean depressive symptoms score was low at 6.71 (SD=4.70) (clinical cut-off is 13) and on average they had strong social networks.

Table 5.2 Sociodemographic and psychosocial characteristics of the sample of mothers at baseline

	N responses	Possible range (min-max)	N (%) or M (SD)
Non-White ethnicity, N (%)	10,513	-	230 (2.19)
Recent move, N (%)	10,287	-	1,158 (11.26)
Mother or partner education >O-level, N (%)	10,243	-	5,937 (57.96)
Mother or partner lower social class, N (%)	8,665	-	1,875 (21.64)
Married, N (%)	10,716	-	8,348 (78.38)
Household financial difficulties, M (SD)	10,270	0-15	2.74 (3.43)
Depressive symptoms, M (SD)	9,903	0-30	6.71 (4.71)
Social support, M (SD)	10,292	0-30	22.46 (3.79)

Note. Data are only presented for participants with at least three time points of neighbourhood data. N is number. M is mean. SD is standard deviation.

Table 5.3 summarises the neighbourhood measures. The proportion of participants living in the most deprived neighbourhoods decreased over the study period, partly due to attrition and partly due to participants moving to less deprived neighbourhoods on average. Likewise, the proportion of mothers who reported a negative opinion of their neighbourhood decreased over time. In contrast, mothers' experience of poor social cohesion in their neighbourhood improved from baseline to the age 5 assessment but then worsened from age 10 to 18. Mothers' perceptions of neighbourhood disorder improved slightly from baseline to age 5 and then remained relatively stable.

Table 5.3 Summary of perceived and objective neighbourhood measures

Objective measure	**Baseline**	**Age 5**	**Age 14**	**Age 18**
Exposure to more deprived neighbourhoods, N (%)	3,794 (33.99)	1,682 (24.75)	1,075 (18.58)	554 (17.26)
Perceived measures (possible range)[1]	**Baseline**	**Age 5**	**Age 10**	**Age 18**
Negative neighbourhood opinion, N (%)	6,008 (57.26)	4,311 (48.24)	3,394 (42.62)	1,426 (34.20)
Poor social cohesion (0-4)	2.15 (0.58)	1.90 (0.58)	1.92 (0.55)	2.22 (0.48)
Neighbourhood disorder (0-2)	0.36 (0.33)[2]	0.30 (0.28)	0.28 (0.28)	0.29 (0.31)

Note. Values are M (SD) unless otherwise noted.
[1]Only including participants with at least three time points of neighbourhood data.
[2]Measured at age 1.75 time point.

5.3.1 What is the nature of the relationship between perceived and objective neighbourhood measures?

I first discuss the trajectories identified for each objective and perceived neighbourhood measure (based on the final dual trajectory analyses for consistency, which used the univariate models as starting values). This is followed by discussion of the central contribution of the dual trajectory analyses: the overlap in trajectory group membership between objective neighbourhood-level deprivation and each perceived neighbourhood variable.

Trajectories of neighbourhood measures

Figure 5.1 shows the trajectory plots for each dual trajectory analysis, with parameter estimates and diagnostics for each model included in Tables C3-C8 (Appendix C). All models exceeded the diagnostic criteria, apart from two trajectory groups in the neighbourhood opinion model where the average posterior probabilities of group membership were near the threshold (≈.7) but the odds of correct classification based on these probabilities far exceeded the threshold (>5).

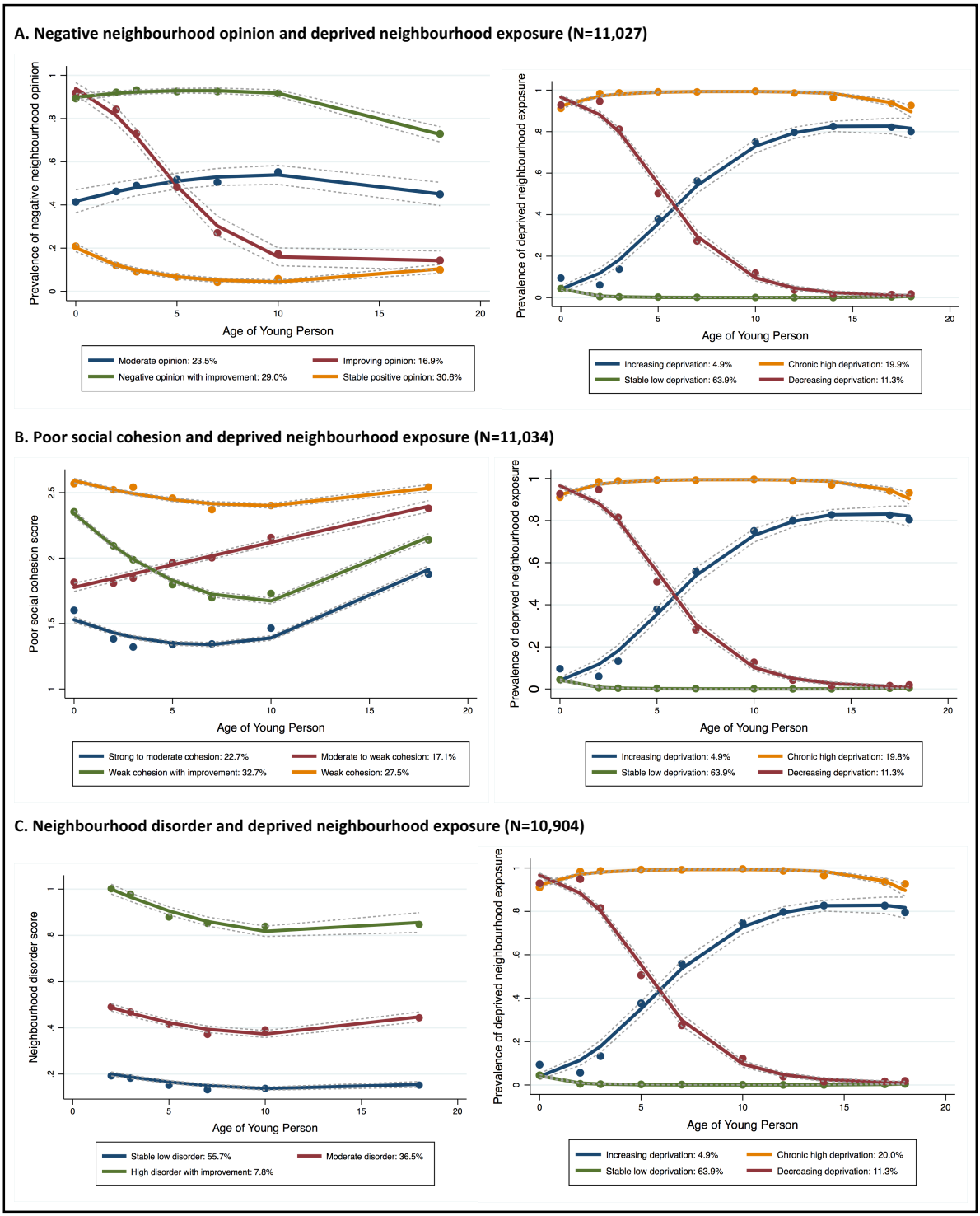
I identified four groups of participants that experienced markedly different patterns of objective neighbourhood deprivation over time (Figure 5.1). Summarising across the dual trajectory analyses, most participants experienced stable exposure to lower neighbourhood deprivation during the study period (64%). One-fifth of participants experienced chronic, higher-level deprivation. The remaining participants experienced changing exposure over time: 11% experienced a decrease in the severity of their deprivation exposure whereas 5% experienced an increase.

As shown in Panel A of Figure 5.1, I identified four trajectories of mothers' opinions of their neighbourhoods. Just over one-third of participants (31%) had a stable, positive opinion of their neighbourhood over time, while just under one-third (29%) had a negative opinion that improved slightly by the end of the study period. An additional 24% of participants held a moderate opinion of their neighbourhood over time. The final 17% of participants experienced a large improvement in their neighbourhood opinion during the study period.

Panel B of Figure 5.1 shows the four trajectory groups identified for poor social cohesion. The largest group of participants (33%) experienced weak social cohesion with a

period of improvement mid-study. The next most common trajectories among participants were consistently weak social cohesion (28%) and social cohesion that progressed from strong to moderate levels (23%). The final group of participants experienced moderate to weak social cohesion over the study period (17%). All four trajectory groups experienced a near-parallel worsening in perceived social cohesion from the age 10 to age 18 time points. Panel C shows the three, relatively stable trajectory groups identified for perceived neighbourhood disorder, which from most to least common among participants were: perceptions of low disorder (56%), moderate disorder (37%), and high disorder with improvement over the study period (8%).

Figure 5.1 Dual trajectory models for each perceived neighbourhood variable (maternal report) and objective neighbourhood deprivation



Note. Solid lines are the estimated trajectories, dots are the observed group means for participating mothers at each assessment (measured by the age of the young person participants).

5.3.2 The relationship between perceived and objective neighbourhood trajectory groups

Figure 5.2 shows the proportion of participants in each objective deprivation trajectory group who were members of each perceived measure trajectory group. In other words, this is the distribution of neighbourhood perceptions within each objective deprivation trajectory group observed in the sample. Appendix C (including Figure C2 and Table C9) provides additional description of how the perceived and objective measure trajectory groups were related.

Neighbourhood opinion and objective neighbourhood deprivation

Overall, holding more negative opinions of the neighbourhood was related to exposure to greater objective neighbourhood deprivation over time. As shown in Panel A of Figure 5.2, members of each objective neighbourhood deprivation trajectory group tended to also be members of the closest corresponding opinion trajectory group. That is: 47% of participants exposed to decreasing neighbourhood deprivation over time reported improving opinions of their neighbourhoods; 44% of participants who experienced stable low deprivation had stable positive opinions of their neighbourhoods; and 66% of participants exposed to chronic high deprivation held more negative opinions of their neighbourhoods. In contrast, there was no clear correspondence between the increasing deprivation group and the estimated neighbourhood opinion trajectories. Namely, among participants whose exposure to objective neighbourhood deprivation *increased* over time, 44% held (stable) moderate opinions of their neighbourhoods while 32% had negative opinions that, conversely, *improved* over the study period.

Perceived social cohesion and objective neighbourhood deprivation

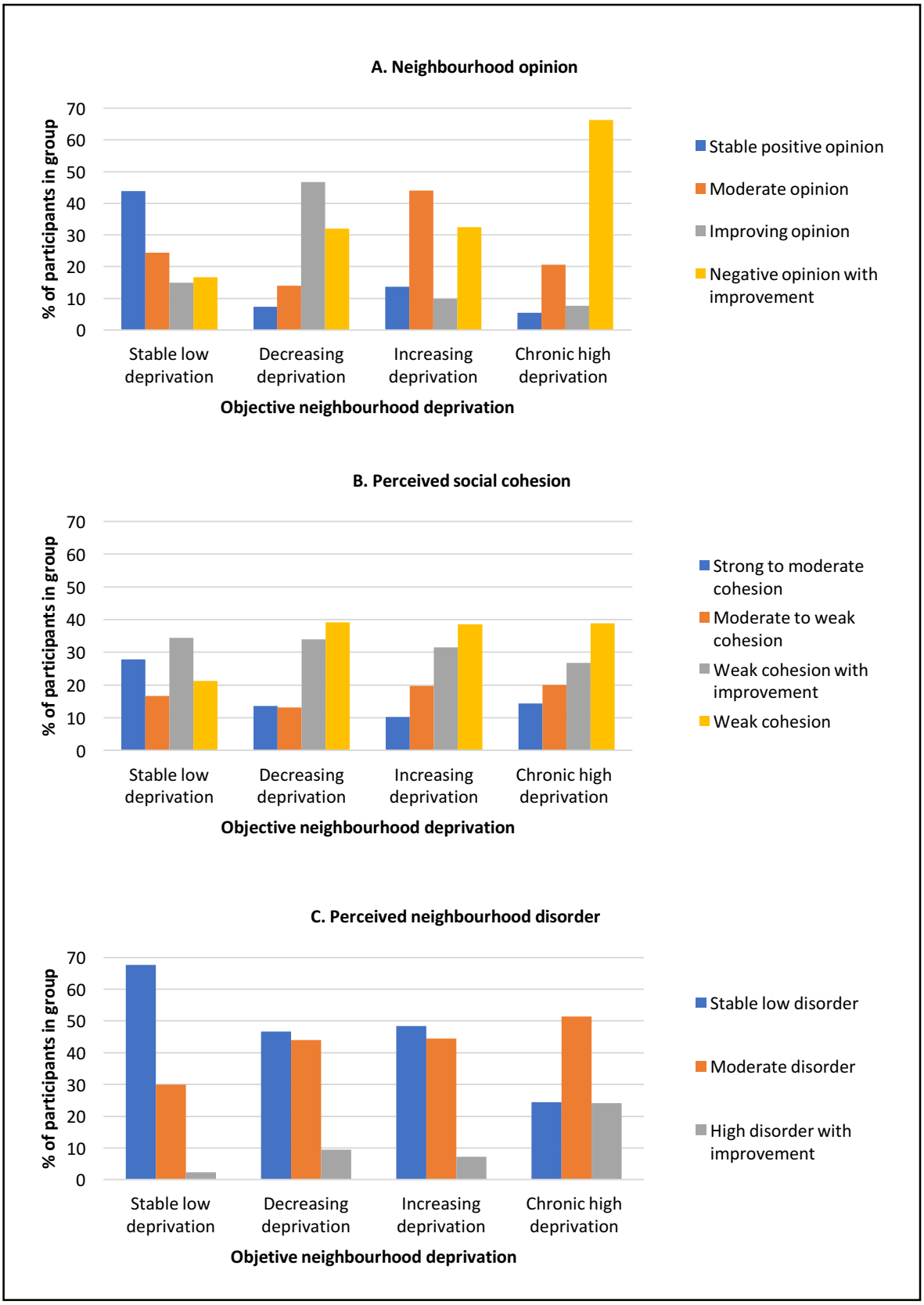
The results in Panel B of Figure 5.2 suggest a negative but weak relationship between objective neighbourhood deprivation and perceived social cohesion. For instance, a large proportion of participants who experienced low levels of objective neighbourhood deprivation over the study period had strong to moderate perceptions of neighbourhood social cohesion over time (28%). In contrast, however, most of the participants in this low deprivation group perceived weak social cohesion with some improvement over time (34%). Moreover, the patterning of participants' perceptions of social cohesion was very similar in each of the three remaining objective deprivation groups (increasing deprivation, decreasing deprivation, and chronic high deprivation): most participants in each of these groups perceived weaker social cohesion over time. This similarity in perceptions of social cohesion across the objective neighbourhood deprivation groups further weakens the evidence for the correlation between these two variables over time.

Perceived neighbourhood disorder and objective neighbourhood deprivation

Similar to the results for neighbourhood opinion, perceived neighbourhood disorder showed a stronger, positive relationship with objective neighbourhood deprivation over time but with significant areas of divergence. Panel C in Figure 5.2 shows that most participants who were exposed to increasing or decreasing objective neighbourhood deprivation over the study period perceived either stable or moderate neighbourhood disorder over time, with a roughly even split. As expected, more participants exposed to stable low neighbourhood deprivation perceived stable low neighbourhood disorder (68%)

as opposed to moderate (30%) or high disorder (2%) over time. Surprisingly, however, most participants in the chronic high deprivation group perceived moderate disorder (51%), with a roughly equal split between stable perceptions of low (25%) or high neighbourhood disorder (24%).

Figure 5.2 Percentage of participants in each objective neighbourhood trajectory group conditional on perceived neighbourhood measure trajectory group membership



5.3.3 Which sociodemographic and psychosocial factors are associated with perceptions of neighbourhood environments?

There was a clear social patterning in the trajectories of mothers' perceptions of their neighbourhood environments, even after controlling for objective neighbourhood-level deprivation (Table 5.4). Compared to mothers with stable positive opinions of their neighbourhoods over time, mothers with more negative opinions had more depressive symptoms, less social support, and greater financial difficulties and tended to not be married at baseline. Similarly, compared to those with positive opinions, mothers in the moderate or negative opinion groups tended to have lower educational qualifications, be non-White, and be in lower occupational social classes.

Compared to those who perceived strong to moderate social cohesion in their neighbourhoods over time, participants who perceived weaker social cohesion (across all groups) tended to have lower education and weaker social support. Furthermore, those in the weakest social cohesion groups (weak cohesion and weak cohesion with improvement) tended to be unmarried at baseline compared to participants in the stronger cohesion group. Finally, those whose perceptions of social cohesion progressed from moderate to weak over the study period experienced more financial difficulties and were in lower occupational social classes at baseline than those whose perceptions progressed from strong to moderate.

Participants who perceived moderate or high neighbourhood disorder over time reported more depressive symptoms and financial difficulties as well as weaker social

support than those who perceived lower disorder. Likewise, these participants were more often unmarried at baseline, non-White, and part of lower social classes.

Table 5.4 Covariates associated with trajectory group membership for perceived neighbourhood variables

	Log odds (95% CI)	p	Log odds (95% CI)	p	Log odds (95% CI)	p
	Prevalence of negative neighbourhood opinion (referent: stable positive opinion)					
	Moderate		**Improving**		**Negative opinion with improvement**	
Depressive symptoms	0.03 (0.01, 0.04)	.001	0.03 (0.01, 0.05)	.001	0.06 (0.05, 0.08)	<.001
Recent move	-0.01 (-0.25, 0.23)	.916	0.03 (-0.22, 0.29)	.797	-0.14 (-0.38, 0.10)	.242
>O-level education	-0.15 (-0.30, 0.00)	.048	0.03 (-0.14, 0.19)	.744	-0.22 (-0.37, -0.07)	.004
Married	-0.38 (-0.59, -0.17)	<.001	-0.53 (-0.75, -0.32)	<.001	-0.69 (-0.88, -0.49)	<.001
Non-White ethnicity	0.64 (0.02, 1.25)	.042	-0.50 (-0.17, 1.17)	.141	0.70 (0.10, 1.30)	.023
Low social class	0.27 (0.08, 0.45)	.005	-0.10 (-0.32, 0.11)	.344	0.20 (0.02, 0.39)	.029
Social network index	-0.04 (-0.06, -0.02)	<.001	-0.04 (-0.06, -0.02)	<.001	-0.08 (-0.10, -0.06)	<.001
Financial difficulties	0.09 (0.07, 0.12)	<.001	0.10 (0.07, 0.13)	<.001	0.14 (0.12, 0.17)	<.001
	Poor social cohesion score (referent: strong to moderate cohesion)					
	Moderate to weak cohesion		**Weak cohesion with improvement**		**Weak cohesion**	
Depressive symptoms	0.00 (-0.02, 0.02)	.697	0.01 (-0.01, 0.02)	.359	0.02 (0.00, 0.03)	.047
Recent move	-0.12 (-0.42, 0.18)	.423	0.01 (-0.22, 0.24)	.944	0.13 (-0.11, 0.38)	.287
>O-level education	-0.29 (-0.48, -0.11)	.002	-0.43 (-0.58, -0.28)	<.001	-0.59 (-0.75, -0.43)	<.001
Married	-0.10 (-0.35, 0.15)	.427	-0.39 (-0.58, -0.19)	<.001	-0.43 (-0.64, -0.22)	<.001
Non-White ethnicity	0.13 (-0.57, 0.83)	.710	-0.09 (-0.69, 0.51)	.766	0.39 (-0.20, 0.97)	.193
Low social class	0.17 (-0.05, 0.39)	.126	-0.06 (-0.24, 0.12)	.528	0.03 (-0.17, 0.22)	.793
Social network index	-0.11 (0.14, -0.09)	<.001	-0.11 (-0.13, -0.08)	<.001	-0.20 (-0.22, -0.17)	<.001
Financial difficulties	0.04 (0.01, 0.07)	.003	0.01 (-0.02, 0.03)	.516	0.00 (-0.02, 0.03)	.726
	Neighbourhood disorder score (referent: stable low disorder)					
	Moderate disorder		**High disorder with improvement**			
Depressive symptoms	0.04 (0.03, 0.06)	<.001	0.10 (0.07, 0.12)	<.001		
Recent move	-0.08 (-0.27, 0.10)	.379	0.25 (-0.08, 0.59)	.142		
>O-level education	0.04 (-0.08, 0.16)	.522	0.11 (-0.14, 0.36)	.397		
Married	-0.21 (-0.36, -0.06)	.006	-0.66 (-0.92, -0.39)	<.001		
Non-White ethnicity	0.62 (0.18, 1.05)	.005	0.88 (0.19, 1.56)	.012		
Low social class	0.13 (-0.01, 0.27)	.064	0.21 (-0.05, 0.48)	.115		
Social network index	-0.02 (-0.04, -0.01)	.003	-0.07 (-0.10, -0.04)	<.001		
Financial difficulties	0.08 (0.07, 0.10)	<.001	0.16 (0.13, 0.19)	<.001		

Note. Each panel is a multinomial logistic regression where the outcome is trajectory group membership and the regressors are baseline covariates and exposure to objective neighbourhood-level deprivation.

5.4 Discussion

This chapter investigated the dynamic relationship between an official objective measure of neighbourhood deprivation (the Indices of Multiple Deprivation) and perceptions of neighbourhood environments over time. My trajectory analyses revealed that, while the majority of participants experienced a stable trajectory of low-level neighbourhood deprivation (64%), the remainder experienced worsening (5%), improving (11%), or continuously high (20%) levels of deprivation over time – differences that would have been masked had I only considered the average values of neighbourhood deprivation exposure in the sample. This adds to a growing evidence base that demonstrates how exposure to objective neighbourhood deprivation varies over time and, by extension, the importance of accounting for this in analyses of its effects on health and wellbeing.[51-55] My results further corroborate and extend prior latent class growth analyses of neighbourhood measures in ALSPAC,[56,57] which have only considered time-variation in either perceived or objective neighbourhood measures. This chapter enriches our understanding of these measures by comparing participants' longitudinal experiences of objective neighbourhood deprivation with their longitudinal perceptions of their neighbourhood environments. I found diverse trajectory patterns for objective neighbourhood deprivation compared to each of perceived social cohesion, neighbourhood disorder, and opinion of the neighbourhood as a place to live – illustrating the divergence in participants' objective neighbourhood conditions and their interactions with and perceptions of their environments over time.

Overall, I found a negative relationship between objective neighbourhood deprivation and perceived social cohesion; however, this relationship was the weakest of

all observed. Although prior studies have found that weaker social cohesion is related to neighbourhood disadvantage,[15,58] researchers have also recognised that these two constructs do not inevitably co-exist in neighburhoods.[13,59,60] Instead, neighbourhood effects on health and behaviour may require or be exacerbated by the presence of both objective deprivation and weak social cohesion. My findings add to the caution against assuming that deprived neighbourhoods lack social cohesion (or that less deprived neighbourhoods are necessarily more cohesive) and highlight the importance of articulating the hypothesised neighbourhood mechanisms before selecting one (or both) of these constructs as the exposure measure(s). Likewise, when health effects of either neighbourhood deprivation or social cohesion are observed, hypothesised mechanisms involving one or the other construct should be explicitly tested as opposed to assumed. Although many studies of objective neighbourhood deprivation and outcomes related to health and behaviour (including IPV) use social disorganisation or collective efficacy theories[3,15,61] – which are centred on social cohesion and informal social control – to explain effect mechanisms, my results demonstrate the importance of broader consideration of other potential indirect pathways. These may include but are not limited to: service or resource access or availability, psychological stress, and social norms.[1,5,7,62]

Perceived neighbourhood disorder and overall opinion of the neighbourhood as a place to live showed stronger relationships with objective neighbourhood deprivation, although again there were areas of divergence. One reason underlying these differences is that participants' perceptions of their neighbourhoods were considerably influenced by personal factors. That is, extending prior studies, I found that participants with more negative perceptions of their neighbourhoods over time (weaker social cohesion, greater neighbourhood disorder, and more negative opinions) tended to be unmarried and non-

White and have greater depressive symptoms, lower socioeconomic status, and weaker social networks – over and above their exposure to objective neighbourhood deprivation.[21-25] This demonstrates the inextricability between the neighbourhood and the individual in self-reported perceived neighbourhood measures and the need for careful analytic consideration and robustness checks (e.g., to avoid same-source bias) if associations with self-reported outcomes are of interest.[63,64] Interestingly, the perceived neighbourhood measures overall (particularly neighbourhood disorder) also tended to show less variation over time than the objective measure (e.g., relative differences between most perceived trajectory groups were fairly stable over time) – perhaps as a result of these initial individual differences in psychosocial and economic status.

Nevertheless, there were notable patterns in these perceived measures across the life course that should be considered in future studies. In particular, across all social cohesion trajectory groups, there was a near-parallel decline in perceived social cohesion among mothers from when their children were 10 to 18 years old. This may have been due to children getting older and there resultantly being fewer opportunities for mothers to socialise within the neighbourhood (e.g., the 'empty nest' transition).[65] Inferences are limited here as there were no assessments between ages 10-18 and, additionally, mothers may have had other, younger children apart from their participating child. Nonetheless, this parallel decline was not found for any other perceived variable, suggesting a unique life course component to the experience (and thus measurement) of perceived social cohesion. This adds further to the established call in the literature to consider the complexity of neighbourhood exposures beyond simple binary point-in-time variables: not only in terms of considering the *duration* of exposure, as discussed above, but also its

timing (e.g., when in the life course exposure occurred) when evaluating potential neighbourhood effects.[51,53,55]

Limitations of this chapter are that, first, without access to participants' address data, I was not able to investigate neighbourhood-level aggregates of the perceived meaures. Aggregated neighbourhood perceptions are a stronger neighbourhood measure because they are less biased (although not entirely so) by individual-level factors[13,63,64] – although these factors were of interest in this chapter. Moreover, some of the observed differences between the perceived and objective measures may have been due to measurement insensitivity in the perceived measures, which should be explored in future longitudinal research using validated measures. Second, I did not have access to participants' continuous deprivation scores, which would have allowed me to analyse greater heterogenity in objective deprivation exposure and may have partly driven some of the observed divergence with the perceived measures.

Third, objective deprivation was measured using the enrolled child's address not the mother's; therefore, some differences between the mothers' perceived and objective neighbourhood data could be the result of the mother and enrolled child not living in the same household. Additionally, I used one iteration of the deprivation measure rather than multiple iterations over time. This improved the measurement of participants' neighbourhood mobility but means I could not account for changes in neighbourhoods over time. Nonetheless, changes in relative neighbourhood deprivation in the study area were minimal over the study period and thus unlikely to bias the observed findings.[66] Fourth, the sample is predominantly White with higher socioeconomic status than the national population: generalisability to other contexts should be explored. Fifth, the

estimated trajectory groups should not be interpreted as literally distinct population subgroups but rather approximations of an unknown distribution of trajectories experienced by the population.[46,67] Finally, results are correlational and I do not asssume a causal interpretation.

Despite its limitations, this chapter used 18 years of robust neighbourhood and social data to interrogate the measurement of neighbourhood environments over time. I found consistent areas of convergence and divergence between perceived and objective neighbourhood measures and a distinctive patterning in participants' perceptions of their neighbourhoods by individual-level social factors. Perceived neighbourhood measures, and especially measures of neighbourhood-level social processes such as social cohesion, should be considered distinct constructs from objective neighbourhood deprivation. Researchers of neighbourhood effects on health must carefully consider their hypothesised mechanism(s) when determining the most appropriate measures of the neighbourhood environment, and the level (neighbourhood or individual), timing, and duration for which exposures should be measured, to maximise relevance to both theory and intervention development.

5.5 Chapter summary

This chapter demonstrated variation over time in exposure to objective neighbourhood-level deprivation, measured by the Indices of Multiple Deprivation, among ALSPAC participants. This was important to establish for my investigation of the association between neighbourhood disadvantage and IPV against women (Chapter 6) given the dominance of point-in-time measures of neighbourhood environments in prior IPV studies, as demonstrated in Chapter 3. Mother-reported measures of the perceived

neighbourhood environment also showed time-variation, however, the relative differences between most trajectory groups remained fairly constant over time (particularly for perceived neighbourhood disorder). Moreover, these perceived measures were strongly related to individual differences in psychosocial and socioeconomic wellbeing, even after adjusting for objective neighbourhood deprivation, demonstrating the difficulty of parsing out the individual from the neighbourhood in perceived measures. Informed by these findings, as well as Chapters 3 and 4, the following chapter presents my investigation of the effect of long-term exposure to objective neighbourhood-level deprivation, as measured by the Indices of Multiple Deprivation, on IPV against women. I return to the implications of this chapter, in the context of the empirical work as a whole, in the final chapter of this thesis (Chapter 7).

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Chapter 6: Long-term exposure to neighbourhood-level deprivation and the risk of experiencing intimate partner violence among women*

6.1 Introduction

The aim of this final empirical chapter was to investigate the longitudinal association between objective neighbourhood-level deprivation and IPV against women in ALSPAC. Cross-sectional studies, including innovative spatial analyses,[1-4] have largely established a positive association between neighbourhood disadvantage and IPV against women.[5-8] In contrast, as demonstrated in Chapter 3, there has been a relative dearth of longitudinal studies of this association, which have shown uncertain findings – limiting causal conclusions and implications for preventive interventions.

As first outlined in Chapter 1 (Section 1.3.2), the most frequently hypothesised mechanisms for the relationship between neighbourhood deprivation and IPV are grounded in social disorganisation theory[9] or, its extension, collective efficacy theory.[10] These postulate that in neighbourhoods facing greater socioeconomic disadvantage and residential instability, it is more difficult to establish the social ties and informal social control that minimise violence and maximise intervention capacity. While developed to explain neighbourhood-level variations in *public* criminality (e.g., vandalism, burglary),[10-13] these theories have been extended to *private* forms of violence like IPV: for instance, with stronger neighbourhood social ties and support structures expected to 'steer women away' from violent partners, provide resources for leaving violent relationships, and create

*This chapter is adapted from: Yakubovich, A. R., et al. (under review – revised and resubmitted). Long-term exposure to neighbourhood-level deprivation and the risk of experiencing intimate partner violence among women: a birth-cohort study in the United Kingdom. *Epidemiology.*

a more inhibitory environment (e.g., where neighbourhood members are perceived as able and willing to regulate private violent behaviour).[14] Of relevance to a developmental context, sustained exposure to neighbourhood disadvantage has further been hypothesised to increase the risk of IPV by normalising aggression, for instance via higher rates of neighbourhood violence.[5-8] Likewise, this exposure is expected to increase trauma or stress and thus vulnerability to future victimisation by exacerbating individual-level risks such as substance misuse or social isolation, increasing relational strain, or inhibiting help-seeking behaviours.[5-8,15]

In Chapter 3, my meta-analysis of the three available and combinable longitudinal studies of neighbourhood disadvantage and IPV against women found a small but unexpected protective association, underscoring the need for further investigation.[16-18] To my knowledge, there are two additional prospective studies that were not included in this meta-analysis. One of these additional studies was not appropriate to combine with the rest (due to a heterogeneous exposure)[19] and the second was published after my search (in 2018).[20] Table 6.1 summarises the five available studies. Four of five of these studies were USA-based and all adjusted for post-exposure or cross-sectional covariates that may be on the causal pathway (e.g., socioeconomic status) – potentially underestimating the total effect of neighbourhood disadvantage or, at worst, inducing collider-stratification bias (discussed further in Section 6.2.1).[21-23] Additionally, four studies did not account for duration of exposure.[16-18,20] The fifth study measured exposure and outcome over two contemporaneous time points, limiting causal conclusions.[19]

Table 6.1 Summary of prior prospective-longitudinal studies of association between neighbourhood disadvantage and IPV against women

		Neighbourhood disadvantage exposure			Covariates adjusted for that may be on causal pathway			
Authors (Cohort)	**Country**	**Measure or construct (source)**	**N time points**	**IPV measure**	**Post or cross-sectional with exposure**	**Post or cross-sectional with exposure and outcome**	**Other relevant selection criteria**	**Association (analysis)**
Benson et al.[19,a] (National Survey of Families and Households)	USA	Top 25% most disadvantaged neighbourhoods measured by concentrated disadvantage index (census): 0=advantaged area both waves; 1=disadvantaged wave 1, advantaged wave 2; 2=disadvantaged both waves	2	Binary: Any physical IPV	-	Exposure is contemporaneous with outcome and all adjusted covariates, including: - Employment instability - Income-to-needs ratio - Financial strain	Only participants who were cohabiting or married to the same partner in wave 2 as in wave 1: a common effect of living in less disadvantaged neighbourhoods and causes of IPV	B=0.31, SE=0.13 (binary logistic regression, exposure is ordinal variable analysed continuously)
Giordano et al.[16,b] (Toledo Adolescents Relationship Study)	USA	Sum of neighbourhood problems (parent self-report)	1	Binary: Any IPV	-	Respondent and partner's: - Delinquency - Controlling behaviours - Trait and relationship-based anger	-	OR=0.98, 95% CI 0.86, 1.10 (binary logistic regression)
Gomez et al.[17,b] (National Longitudinal Study of Adolescent Health)	USA	Concentrated disadvantage (census)	1	Ordinal: no, less severe, more severe physical or sexual IPV since age 18 over entire study period	-	- Retrospective child abuse - Parental income - Educational attainment	-	OR=0.91, 95% CI 0.84, 0.99 (ordered logistic regression)
Jain et al.[18,b] (Project on Human Development in Chicago Neighborhoods)	USA	Concentrated disadvantage (census)	1	Continuous: Sum of frequency of physical IPV items	- Perceived community violence - Collective efficacy	-	By outcome measurement, >50% of the sample had moved from original neighbourhoods	B=-0.02, SE=0.33 (multilevel linear regression)
Leddy et al.[20,c] (HPTN 068 trial cohort)	South Africa	Wealthier (less unemployment, higher SES) and more permanent residents (census)	2	Binary: any physical IPV	- Collective efficacy - High school enrolment[d] - Household assets[d]	-	-	RR=0.99, 95% CI 0.95, 1.03 (modified GEE poisson regression)

USA is United States of America. B is unstandardised regression coefficient. SE is standard error. OR is odds ratio. CI is confidence interval. SES is socioeconomic status. RR is risk ratio. GEE is generalised estimating equation. Studies were identified through the systematic search described in Chapter 3, which was completed in June 2016. Following the completion of this search, automated alerts on relevant literature were set up via GoogleScholar and additional focused searches were conducted in March 2019.

[a]A series of studies were conducted using data from the same cohort but either using one time point of cross-sectional neighbourhood exposure and IPV outcome data[24-26] or using two time points of data but analysing any IPV at both time points as the outcome.[27] As the study by Benson et al. provided the strongest longitudinal design, only this study is summarised above. The association between concentrated disadvantage and IPV against women was positive in all of these studies.

[b]Studies included in the meta-analysis in Chapter 3 of the longitudinal association between neighbourhood disadvantage and IPV against women.

[c]Identified in updated searches (published in 2018).

[d]Unclear if adjusted covariate was measured contemporaneously/after exposure (as opposed to prior).

I aimed to build on this evidence base by investigating, for the first time, the effect of long-term exposure to neighbourhood-level deprivation on IPV against women using 21 years of ALSPAC data from the UK. As first discussed in Chapter 2 (Section 2.3.3), socioeconomic and psychosocial characteristics of the family environment affect the neighbourhoods that families live in and are in turn affected by neighbourhood environments; these family characteristics may additionally affect the risk of experiencing IPV in early adulthood.[23,28,29] As a result, adjusting for socioeconomic indicators, as in prior studies, may partial out part of the effect of long-term neighbourhood deprivation on IPV and induce collider-stratification bias (see below for further discussion), whereas not controlling for these indicators would result in confounding.

I therefore used marginal structural models, where I weighted each participant by the inverse of the probability of experiencing their observed neighbourhood exposure at each time point given their prior exposure and covariate history.[22,30,31] As detailed in Chapter 2, this creates a *pseudo-population*, where the probability of exposure is comparable across covariate levels at each time point. Analyses of the exposure-outcome association within this weighted sample account for confounding by covariates measured before each exposure, without removing the indirect effects of exposure to neighbourhood deprivation via these covariates at later times.

6.2 Method

Data were from ALSPAC, the study methods for which were described in Chapters 2 and 4. In this chapter, my target sample was the 7,219 girls enrolled at birth (between 1 April 1991 and 31 December 1992). I also used data from mothers on family-level

characteristics as detailed below. Mothers provided written informed consent regarding their own participation. Primary caregivers provided consent for child participation until children were age 16, after which children provided written informed consent. The ALSPAC Ethics and Law Committee and local research ethics committees provided ethical approval.

6.2.1 Measures

IPV

Of the 7,219 women with baseline data, 2,128 responded at age 21 to an 8-item scale on physical, psychological, and sexual IPV experiences (from 0=never to 3=often) before and/or after age 18 (Table 6.2, α=.95) – which was validated in Chapter 4. To delineate temporality, I only analysed IPV between ages 18-21. As discussed in Chapter 4, participants who experienced any IPV also self-reported on experiencing any of eight negative impacts from this violence: scared, upset, work/studies affected, sad, anxious, increased alcohol or substance use, angry, or depressed.

Table 6.2 IPV items

Item	How often altogether have any of your partners ever done any of the following to you and how old were you:	Type of IPV
1	Told you who you could see and where you could go and/or regularly checked what you were doing and where you were (by phone or text)?	Psychological
2	Made fun of you, called your hurtful names, shouted at you?	Psychological
3	Used physical force such as pushing, slapping, hitting or holding you down?	Physical
4	Used more severe physical force such as punching, strangling, beating you up, hitting you with an object?	Physical
5	Pressured you into kissing/touching/something else?	Sexual/psychological
6	Physically forced you into kissing/touching/something else?	Sexual
7	Pressured you into having sexual intercourse?	Sexual/psychological
8	Physically forced you into having sexual intercourse?	Sexual

[a]For each victimisation item, participants indicate the frequency of occurrence – where 0=never, 1=once, 2=a few times, 3=often – and age of occurrence, where 1=under 18, 2=over 18, 3=both. The question prompt included the following definition for 'partner': 'By partner I mean anyone you have ever been out with or had a relationship with, long-term or short-term (including one night stands)'.

I analysed two primary outcomes: (1) participants' sum scores across the eight IPV items (0-24), which reflected the average frequency of violence across all IPV experiences, and (2) any IPV experience between ages 18-21. These outcome operationalisations accounted for the right-skewed, discrete distribution of the IPV frequency scores, with the latter allowing me to estimate differences in the risk of any IPV and the former allowing me to further consider variations in the intensity of IPV.

Neighbourhood-level deprivation

I measured exposure to neighbourhood-level deprivation using the 2010 Indices of Multiple Deprivation[32] – which were introduced and evaluated in the ALSPAC sample in Chapter 5. As described in Chapter 5, this is an official measure of area-level deprivation in England, which considers deprivation beyond economic poverty alone, using indicators across seven domains: income, employment, education, health, crime, housing, and living environment (Table D1, Appendix D).[33,34] Based on these indicators, each residential neighbourhood in England (LSOA) is assigned a domain-specific and total deprivation rank score *relative* to all other neighbourhoods (see Appendix D for further discussion).

As discussed in Chapter 5, I had access only to participants' quintile ranks for each domain and the total deprivation index, so that at each time point (see Table 6.3), I knew the quintile of deprivation each participant's residential neighbourhood fell into relative to all other neighbourhoods in England (i.e., not just within ALSPAC). Due to the underlying exponential distribution of the rank scores (as shown in Figure 6.1), differences in deprivation are greater between the most deprived quintiles compared to the least and there is less differentiation in deprivation between the less deprived quintiles.[34] I therefore did

not expect to find a linear trend in IPV risk among the quintiles nor for this to be the most meaningful contrast of neighbourhood deprivation. Ideally, I would have analysed participants in quintile 5 (i.e., the 20% most deprived neighbourhoods in England) versus all others; however, I lacked the appropriate statistical power, with a low proportion of ALSPAC participants in quintile 5 alone dropping to <6% at later time points. As such, as in Chapter 5, I constructed a binary variable for each time point where 1=quintiles 4 and 5 (participants living in the top 40% most deprived neighbourhoods in England) and 0=otherwise. I expected this to be a more conservative test of the effect of neighbourhood deprivation.

Figure 6.1 Exponential distribution of 2010 Indices of Multiple Deprivation scores by neighbourhood (lower-layer super output area) rank. The fine dashed lines indicate quintile markers. Neighbourhoods to the right of the thick dashed line are the 40% most deprived neighbourhoods in England. Data are from the official English Indices of Multiple Deprivation 2010 (available at: https://www.gov.uk/government/statistics/english-indices-of-deprivation-2010); further details on the measure and its construction are in the English Indices of Multiple Deprivation 2010 technical report.[32]

I used inverse probability weights that predicted the probability of participants' neighbourhood deprivation exposure at each time point until age 18. To estimate the causal effect of neighbourhood-level deprivation on IPV – given 2^{10} (1,024) possible exposure

trajectories – a parametric model was necessary.[22] Based on similar longitudinal studies, I used participants' duration-weighted exposure, where I computed the mean of each participant's level of deprivation exposure (0,1) across the time points analysed to compare participants who lived in more versus less deprived neighbourhoods for longer.[23,35,36] This specification was of theoretical interest because it allowed for estimation of the effects of *sustained* exposure to neighbourhood deprivation (i.e., differences in IPV risk between women who spent more of their childhood in the most deprived neighbourhoods in England versus the least).

Covariates

To determine the appropriate covariates to account for, I drew a directed acyclic graph (DAG, or a hypothesised causal model) for the association between neighbourhood deprivation and IPV.[37] This was based on my systematic review of the longitudinal evidence (Chapter 3) and, where gaps remained, the best available cross-sectional evidence,[29] as well as prior longitudinal studies of neighbourhood disadvantage.[23,35,36,38] DAGs are an important tool, underutilised in violence research,[39] that involve explicitly drawing out assumptions regarding the causal relationships between variables and determining the appropriate variables to account for based on these.[37,40,41] In particular, they demonstrate which variables must be accounted for to estimate a causal association unbiased by confounding (i.e., variables that are common causes of both exposure and outcome, or of variable(s) that in turn cause exposure and outcome). In structural language, these are referred to as variables on the 'backdoor path' from exposure to outcome. DAGs also demonstrate which variables should *not* be adjusted for: variables that are common effects of both exposure and outcome (or of variables that cause exposure

and outcome) – adjustment for which induces a spurious association between exposure and outcome, otherwise known as *selection* or *collider-stratification bias* (see Chapter 2, Section 2.3.3 for further illustration).[42] In structural language, these are referred to as variables that open a backdoor path from exposure to outcome. This demonstrates the importance of determining covariates based on *structural* as opposed to *statistical* criteria: a variable may affect the quantitative estimate of the association between exposure and outcome, but by inducing bias (i.e., via selection bias) as opposed to correcting for it.

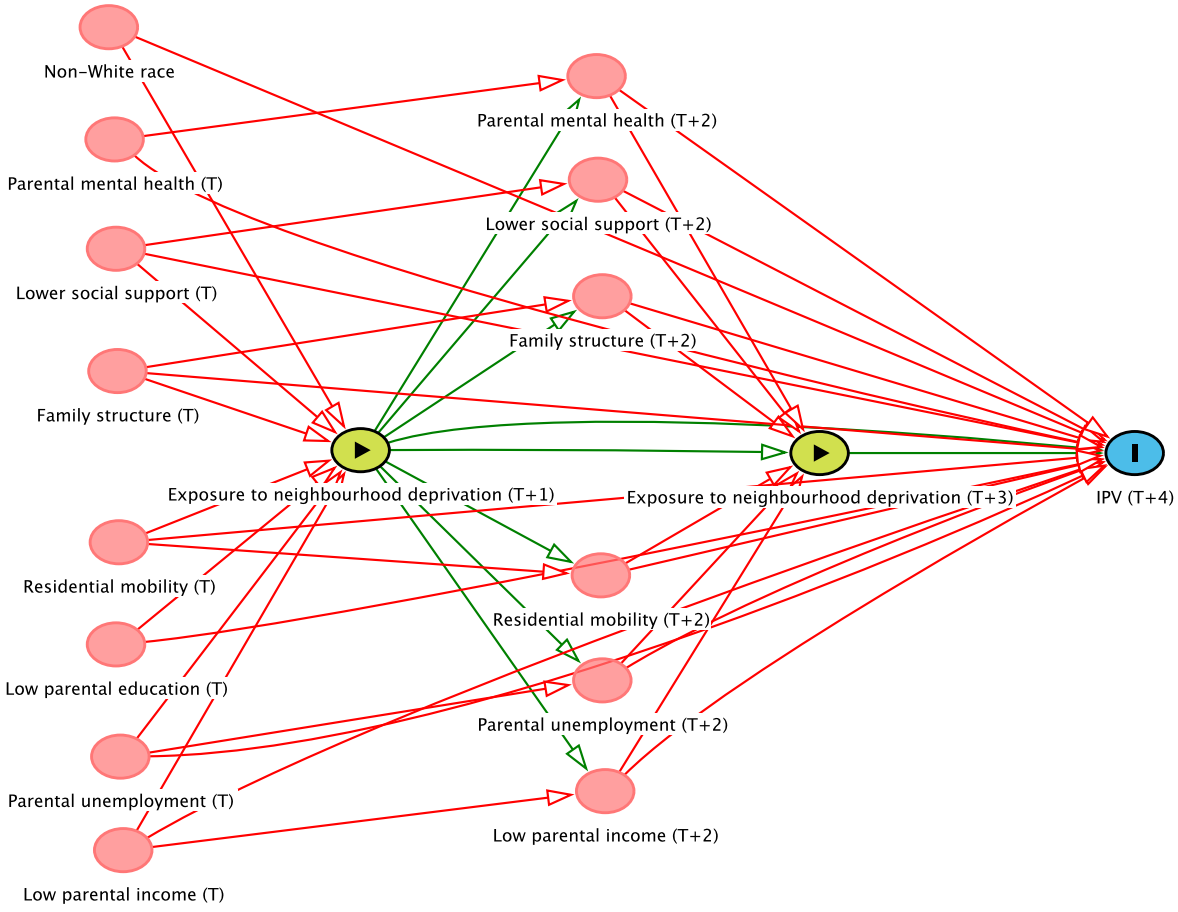

Figure 6.2 Simplified directed acyclic graph (DAG) for the effect of exposure to neighbourhood disadvantage on IPV against women over time. This DAG was drawn based on the most up-to-date synthesis of the longitudinal IPV literature (Chapter 3) and, where gaps exist in this literature, hypotheses based on available cross-sectional evidence[29] and longitudinal studies of neighbourhood disadvantage.[23,35,36,38] For simplicity, concurrent paths between confounders are not shown. Likewise, only variables that are hypothesised to confound the relationship between exposure to neighbourhood deprivation and IPV are included. To create this figure, I used DAGitty.[43]

I thus selected covariates to include in my inverse probability weights based on this DAG and the available data in ALSPAC. Full details on covariate measures are included

in Appendix D. The following variables were only available at baseline and included as *time-invariant covariates*: parental education, maternal marital status, parental occupational social class, mother's number of children, and the young person's race/ethnicity. The following *time-varying covariates* were measured throughout the study: maternal depressive symptoms, maternal social support, residential mobility, parental employment status, family structure, family financial difficulties, and family income.

6.2.2 Analysis

I used marginal structural models with inverse probability weights, which provide an unbiased estimate of the causal effect of neighbourhood deprivation on IPV under the following assumptions: exchangeability (no unmeasured confounding); consistency (unambiguously-defined exposure); positivity (non-zero probability of each possible exposure value at each possible confounder value); and correct model specification of the marginal structural model and weights.[44] I conducted my analyses in Stata 13.1 (StataCorp LP, College Station, TX). Background information on marginal structural models and inverse probability weighting is provided in Chapter 2 (Section 2.3.3). Additional details on the process of estimating the weights and marginal structural models (including computing code and equations) are in Appendix D. Results are discussed according to clinical significance (i.e., the size or precision of the estimate).[45,46]

Exposure model

As ALSPAC often consisted of multiple assessments per year measuring different variables, I combined assessments to create 11 time periods: ten time periods from

baseline (gestation) until age 18 where variations in exposure and confounders were measured and the age 21 outcome. There were two ways to compute the exposure weights: using only those confounding variables measured in all ten time periods (*consistent-covariate weights*) or using all available confounders (*available-covariate weights*). I ran analyses twice using each set of weights to assess robustness. For the available-covariate weights, when variables were not measured in a given period, the most recent measurement was used (last observation carried forward or, for family structure and income, first observation carried backwards). Table 6.3 summarises the variables used in each set of weights.

Table 6.3 Data availability for exposure and covariates used in inverse probability of exposure and censoring weights

	Time point	T_0	T_1	T_2	T_3	T_4	T_5	T_6	T_7	T_8[a]	T_9
	Start year of data collection	**1990**	**1992**	**1993**	**1994**	**1997**	**1999**	**2002**	**2006**	**2007**	**2010**
	Age (in years)	**Gestation**	**1.75**	**3**	**4-5**	**6-7.5**	**8-10**	**11-12**	**14**	**16.5**	**18**
Exposure	Neighbourhood-level deprivation	X	X	X	X	X	X	X	X	X	X
Time-invariant covariates	Parental education	X									
	Parental social class	X									
	Maternal marital status	X									
	Mother's no. of children	X									
	Young person ethnicity	X									
Time-varying covariates[a]	Residential mobility	X	X	X	X	X	X	X	[b]	-	X
	Mother employment	X	X	X	X	X	X	X	[b]	-	X
	Maternal depressive symptoms	X	X	X	X	X	X	X	[b]	-	X
Only used in available-covariate weights[a]	Maternal social support	X	X	[b]	X	X	X	X	[b]	-	X
	Family financial difficulties	X	X	X	X	X	[b]	X	[b]	-	X
	Family structure	[c]	X	X	X	X	X	[b]	[b]	-	X
	Family income	[c]	[c]	X	X	X	X	X	[b]	-	X

An 'X' indicates that the variable was measured in the corresponding time period. For T_1 through to T_8, inverse probability weights were constructed using time-varying covariates measured at T_{k-1}. As no covariates were measured at T_7 or T_8, time-varying covariates measured at T_9 were used to compute the T_9 weights for the primary analyses.

[a]As no covariates were measured at T_7 or T_8, T_8 was excluded from analyses using the consistent-covariate weights.

[b]Variable imputed with the last available measurement in the available-covariate weights.

[c]Variable imputed with the first available measurement in the available-covariate weights.

I used stabilised weights for more efficient estimates.[30] This means that rather than only using the inverse of participants' probabilities of experiencing their observed exposure histories to weight the sample, which tends to produce very variable weights that lead to unstable estimates, I constructed weights that included a numerator as well – as per standard practice in the literature.[22,23,31,35,36,38,44,47-49] In particular, to estimate the denominator of the weights, I regressed level of neighbourhood deprivation at time k (T_k) onto previous exposure and time-varying covariates at T_{k-1} and baseline covariates using binary logistic regression. The only exception was for the T_9 weights, which used time-varying covariates measured at T_9 as none were measured at T_8. I then estimated the numerator in the same way as the denominator but excluding the time-varying covariates. I checked the distribution of these stabilised weights for extreme values or high variability, which suggest violations of the assumptions of positivity and correct model specification.[44] To further confirm positivity, I checked that there were participants in each deprivation quintile at each level of the categorical confounders.

Missing data

As in most long-term studies, ALSPAC experienced significant attrition: Figure 6.3 provides a summary of permanent attrition and intermittent missingness in the sample over the study period. I accounted for these missing data in several ways. First, in analyses of participants' duration-weighted exposure, participants had to have exposure data for at least 50% of the time points analysed. Second, in my main analyses, in addition to the exposure weights, I computed stabilised weights for being permanently lost to follow-up, or censored. The only difference from the exposure weights was that the probability of being censored (rather than neighbourhood exposure) was predicted. These weights

allowed for an effect estimate independent of non-random attrition conditional on observed data. I explored additional missing data strategies in sensitivity analyses as described below.

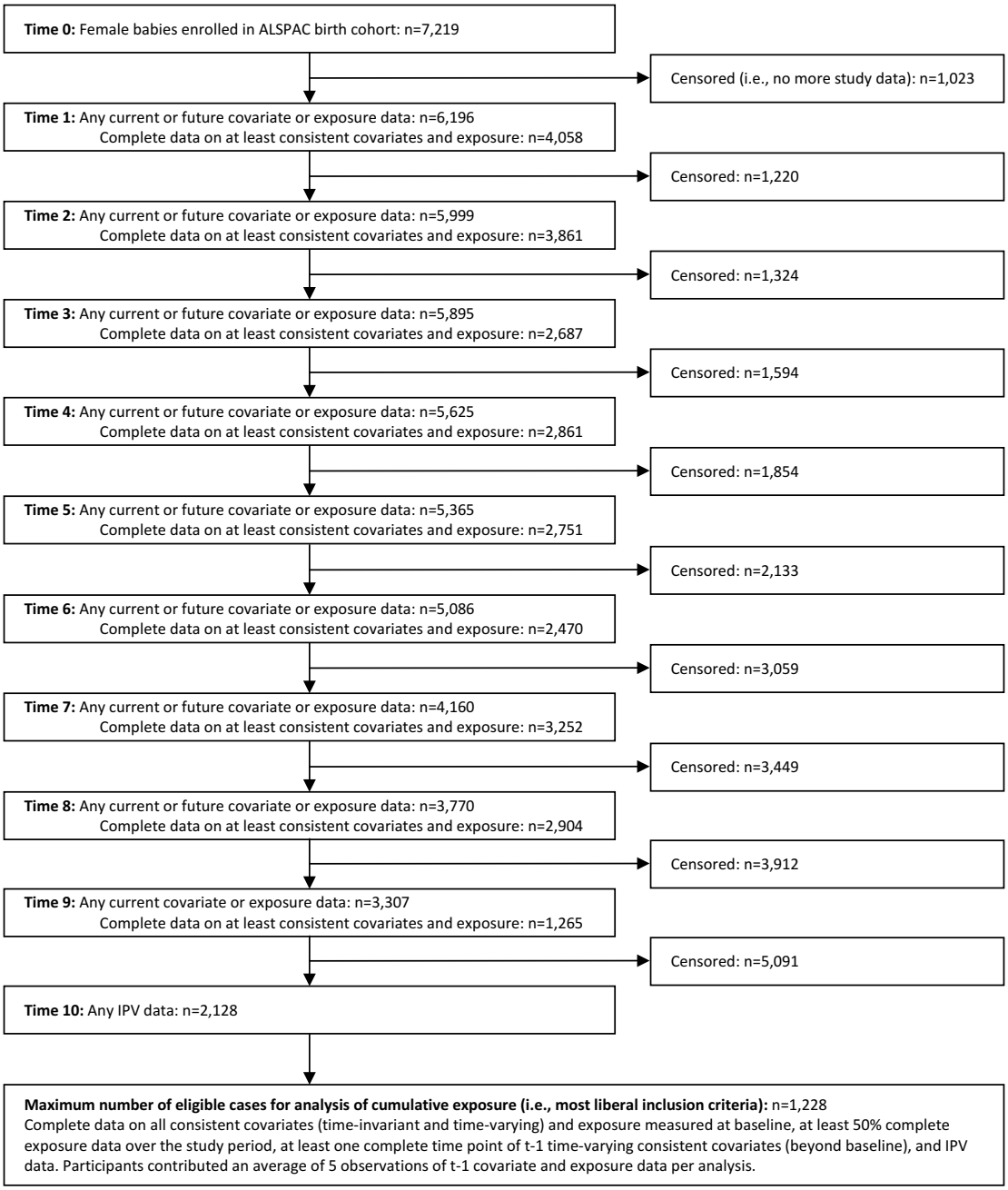

Figure 6.3 Flow chart illustrating study attrition (censoring and intermittent missingness) and the most liberal inclusion criteria used in analyses. At each time t, uncensored participants are defined as those who have data at time t or time t+k, whereas censored participants are those who were permanently lost to follow-up after time t-1. Participants with complete data at time t (i.e., without any missing data on covariates or exposure at time t) are therefore a subset of the uncensored

participants at time t. Selection bias due to non-random attrition was accounted for in multiple ways detailed in Appendix D and below, including inverse probability weights for censoring (main analysis) as well as for intermittent missingness (sensitivity analysis).

Marginal structural model

The final weights were the product of the exposure and censoring weights at each time point. The marginal structural model was either a negative binomial or log-binomial model where the IPV frequency score or risk of experiencing any IPV, respectively, was modelled as a function of cumulative exposure to neighbourhood deprivation and baseline covariates in the weighted sample. The log-binomial model allowed for estimation of risk ratios for the binary IPV outcome.[50] Negative binomial regression allowed for estimation of incidence rate ratios, which accounted for the full variation of the IPV frequency score distribution and the right-skewed, discrete, and over-dispersed (i.e., variance greater than the mean) nature of this distribution.[51] I conducted my analyses in a long-format dataset, where each participant-observation was weighted by the appropriate time-varying weight (i.e., cumulative probability of observed exposure and censoring history up until time t) with cluster-robust standard errors (i.e., standard errors that account for clustering of observations within participants over time, which are conservative).[48] This maximised my use of all available data, allowing me to include participants with at least 50% exposure data, covariate data, and complete outcome data (see Appendix D for further discussion). I explored alternative strategies in my sensitivity analyses.

I addressed the (untestable) assumption of exchangeability by using a robust set of confounders and both possible weight formulations. To address correct model specification, I ran several sensitivity analyses.

Sensitivity analyses

I ran three types of sensitivity analyses (n=38 analyses), testing: (a) stricter IPV operationalisations (e.g., any IPV with a self-reported negative impact, as computed in Chapter 4), (b) additional missing data strategies (e.g., including weights for intermittent missingness[49]), and (c) alternative model specifications (e.g., using time-varying, point-in-time exposure[36] rather than cumulative exposure). Results did not meaningfully differ from my main analyses and sensitivity analyses are thus presented in full in Appendix D. I also ran crude (adjusting for all time-invariant and time-varying covariates at baseline) and adjusted analyses (additionally adjusting for average time-varying covariates over the remaining study period) in an unweighted sample (i.e., not accounting for time-varying confounding) for more conventional estimates.

Secondary analyses

I explored alternative hypotheses in secondary analyses. First, I tested whether economic deprivation alone (income deprivation) or higher neighbourhood-level crime (crime deprivation) was associated with IPV. Second, I tested my assumption regarding the non-linear trend of IPV risk across the deprivation quintiles by re-running analyses using this ordinal variable as the exposure. Third, as my exploratory factor analysis in Chapter 4 showed evidence for either a single IPV factor or two factors of physical/psychological IPV and sexual IPV, I re-ran analyses with these as separate outcomes. Finally, extending the only prior longitudinal study of IPV against women to analyse change in neighbourhood disadvantage exposure,[19] I examined whether most

recent or past exposure to more deprived neighbourhoods was most important to IPV compared to no exposure.

6.3 Results

Most participants were White, had parent(s) with higher-level educational qualifications or occupations, or had a married mother at baseline (Table 6.4). As expected, the proportion of participants whose parents lived with them, were employed, or had recently moved decreased over time. Family financial difficulties and maternal social networks improved and maternal depressive symptoms worsened slightly over time.

At baseline, 33% lived in the most deprived neighbourhoods (quintiles 4 and 5) whereas 67% of participants lived in the least (quintiles 1-3). Over time, the proportion of participants living in the most deprived neighbourhoods decreased, influenced partly by non-random attrition (see Figure 6.3). Figures D3-D5 (Appendix D) demonstrate the within-participant mobility and variation over time in exposure to neighbourhood deprivation. For instance, 54% of participants experienced a change in deprivation quintile during the study period (Figure D4).

Between ages 18–21, 32% of women experienced any IPV (n=683); 29% experienced any IPV with at least one negative impact (n=608). The average IPV frequency score (range: 0–24) was 1.48 (SD=3.08). Among those who had experienced any IPV (n=683), the mean frequency score across all items was 4.61 (SD=3.90). Table D2 (Appendix D) provides the prevalence estimates for each IPV item, typology, and self-reported negative impact.

Table 6.4 Sample characteristics

Time-invariant variables	**Baseline (T_0)**		
Ethnicity, N (%)			
Non-White	134 (4)		
White	3,545 (96)		
At least one parent has higher than standard secondary education (i.e., O-level), N (%)			
Yes	3,224 (55)		
No	2,607 (45)		
At least one parent is part of lower occupational social class, N (%)			
Yes	1,150 (24)		
No	3,690 (76)		
Mother married, N (%)			
Yes	4,807 (75)		
No	1,577 (25)		
Number of mothers' children, M (SD)	0.74 (1.19)		
Time-varying variables	**Baseline (T_0)**	**Ages 6-7.5 (T_4)**	**Age 18 (T_9)**
Total deprivation quintiles, N (%)			
Least deprived	1,407 (26)	1,199 (34)	788 (38)
2	1,261 (23)	858 (24)	548 (26)
3	947 (18)	649 (18)	353 (17)
4	974 (18)	505 (14)	255 (12)
Most deprived	814 (15)	328 (9)	133 (6)
Lives with biological parents, N (%)			
Both parents	4,489 (90)[a]	3,287 (81)	1,455 (70)
Either biological mother or father or neither	483 (10)[a]	753 (19)	635 (30)
Mother recently moved house, N (%)			
Yes	700 (12)	990 (25)	91 (4)
No	4,978 (83)	2,914 (75)	1,999 (96)
Mother's social network index (10 items, 0-30), M (SD)	22.3 (3.9)	21.9 (4.3)	22.1 (4.5)
Family's financial difficulties (5 items, 0-15), M (SD)	2.9 (3.5)	1.5 (2.5)	1.7 (2.9)
Mother's Edinburgh Post-Natal Depression Score (10 items, 0-30), M (SD)	7.0 (4.8)	6.3 (5.1)	7.5 (5.5)
Parental employment[b], N (%)			
Yes	4,540 (87)	3,008 (95)	1,390 (66)
No	676 (13)	154 (5)	729 (34)

N is number. M is mean. SD is standard deviation. Total N=7,219 women enrolled from birth at baseline. All non-missing data are presented.
[a]Family structure was first measured at T_1 (Age 1.75).
[b]Mother's partner employment status for all time points apart from baseline, for which it is mother's employment status.

6.3.1 Main analyses

All formulations of the weights were stable (M≈1.0, SD<1.0) (Table 6.5). The distribution of deprivation quintile scores by categorical confounders further indicated the assumption of positivity was satisfied (Appendix D, Tables D3-D4).

Table 6.5 Mean, standard deviation, and range of stabilised exposure and censoring weights for main analyses

	M (SD)	1st percentile, 99th percentile
Consistent-covariate weights		
Exposure weights	1.0 (0.1)	0.8, 1.3
Censoring weights	0.9 (0.1)	0.7, 1.1
Exposure*censoring weights	0.9 (0.1)	0.4, 1.8
Available-covariate weights		
Exposure weights	1.0 (0.1)	0.7, 1.2
Censoring weights	0.9 (0.1)	0.7, 1.0
Exposure*censoring weights	0.9 (0.1)	0.6, 1.1

M is mean. SD is standard deviation.

Table 6.6 shows the effect estimates for cumulative exposure to neighbourhood deprivation on women's risk of experiencing IPV. Based on the most conservative estimates, a one-unit increase in cumulative exposure to the most versus the least deprived neighbourhoods over the study period was associated with an average 62% increase in women's IPV frequency scores (incidence rate ratio (IRR)=1.62, 95% CI 1.11–2.37) and 36% increase in their risk of experiencing any IPV (risk ratio (RR)=1.36, 95% CI 1.01–1.85) (using the consistent-covariate and available-covariate weights, respectively).

Table 6.6 Effect estimates of exposure to more severe neighbourhood deprivation on IPV among women from marginal structural models

	N_{women} ($N_{observations}$) in analysis	Relative risk	95% CI
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,228 (6,294)	1.62	1.11, 2.37
Available-covariate weights	1,041 (5,088)	1.66	1.05, 2.63
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,228 (6,294)	1.38	1.07, 1.80
Available-covariate weights	1,041 (5,088)	1.36	1.01, 1.85

CI is confidence interval. N is number. Analysis is negative binomial regression (for count outcome) or log-binomial generalised linear model (for binary outcome), weighted as indicated, conducted in a long-form data set (with participant-time as the unit of analysis) with clustering accounted for with robust (conservative) standard errors and adjusting for baseline time-invariant and time-varying covariates. Relative risk is the incidence rate ratio in the negative binomial regression and risk ratio in the log-binomial model.

6.3.2 Sensitivity analyses

Diagnostics and results for sensitivity analyses were similar to my main analyses and are in Tables D5-D6 (Appendix D). The smallest estimate observed for cumulative neighbourhood deprivation was for the effect on *any IPV with a negative impact*

accounting for the consistent covariates (RR=1.27, 95% CI 0.96–1.69). Estimates of the average effect of point-in-time exposure on IPV risk were also smaller but positive (e.g., any IPV: RR=1.23, 95% CI 0.99–1.55, using available covariates). Results from my adjusted models in the unweighted sample were further consistent with my main analyses.

6.3.3 Secondary analyses

Secondary results are in Table D7 (Appendix D). First, neither crime nor (especially) income deprivation showed consistent, clinically meaningful associations with IPV when analysed separately, with 95% confidence intervals tending to centre around the null. Second, as expected, the association between deprivation quintiles and IPV was positive but weak regardless of the weights or outcome analysed. Third, cumulative exposure to neighbourhood deprivation was positively associated with more frequent, and any experience of, physical/psychological IPV and sexual IPV. Point estimates (ranging from 1.33–2.28) and confidence intervals tended to be larger for sexual IPV, possibly due to its lower prevalence and collapsing of heterogeneous experiences (e.g., those who experienced physical or psychological but not sexual IPV would be classified as 0). Finally, I compared participants (a) living in a deprived neighbourhood at age 18 or (b) not living in a deprived neighbourhood at age 18 but with prior exposure to (c) those who had never lived in more deprived neighbourhoods. Those with prior exposure to more deprived neighbourhoods but whose neighbourhoods at age 18 were less deprived were at highest risk of experiencing IPV (RR=1.55, 95% CI 1.14–2.10).

6.4 Discussion

This chapter presents, to my knowledge, the first prospective investigation of the effect of long-term exposure to neighbourhood-level deprivation on IPV against women. Long-term exposure to more compared to less deprived neighbourhoods over the first 18 years of life was associated with more frequent IPV and 36% higher risk of any IPV in early adulthood across a variety of models. Prior prospective-longitudinal studies (mostly USA-based) of the association between neighbourhood disadvantage and IPV against women have shown mixed findings.[16-20] My positive findings may have been partly driven by uniquely considering long-term variation in neighbourhood deprivation exposure over a significant developmental period. This variation – whether a person spends one year in a more deprived neighbourhood versus several years or a decade – is important when considering the effects of neighbourhood deprivation, especially in the context of potential developmental mechanisms.[52-54] For instance, longer exposure to greater neighbourhood-level deprivation over childhood has been shown to decrease cognitive ability[35] and educational attainment[23] and increase the odds of early parenthood.[38] The results of this chapter extend this evidence base and suggest that cumulative exposure to more severe neighbourhood deprivation over childhood also increases women's eventual risk of experiencing IPV.

My findings suggest a small effect of long-term exposure to neighbourhood deprivation on IPV, which is not unusual in the neighbourhood or developmental effects literature, likely because these factors are more distal or upstream to the outcome.[23,36,55,56] Nevertheless, understanding the effects of upstream factors over time – and how they interact with more proximal factors – is necessary for a rigorous etiological understanding of IPV, and indeed most if not all complex public health problems.[57-59] This is critical to

designing interventions that can improve the wellbeing of populations rather than individuals.[60,61]

Although qualitative studies have explored the perceived role of neighbourhood deprivation in causing and exacerbating IPV (e.g., via psychological trauma or service/support barriers),[62-66] longitudinal studies that explicitly test the mechanisms underlying this effect are still needed. The current study focused on a measure of *relative* deprivation: how long-term neighbourhood inequalities affect women's IPV risk (e.g., by establishing normative standards or exacerbating psychosocial stress) and whether findings extend to *absolute* deprivation (e.g., an objective 'poverty line') should be investigated. Moreover, I found that exposure to multiple deprivation, rather than singular dimensions of income poverty or higher crime, was the most influential in increasing women's IPV risk. This supports a conceptual framework where the *accumulation* of deprivation across material and social conditions is the key construct in defining neighbourhood effects.[32,67,68] The replicability of my findings under alternative definitions of multiple deprivation should be explored. Finally, my secondary analyses suggested that recently moving to a less deprived neighbourhood after prolonged deprivation exposure was most harmful. Future research should investigate this further: including how different trajectories of neighbourhood deprivation exposure affect IPV and possible critical or sensitive periods.[11]

Neighbourhood effects research has historically focused on identifying *contextual* effects of neighbourhoods over and above *compositional* effects (i.e., due solely to the characteristics of neighbourhood residents).[69,70] However, this convention disregards the role of compositional factors in the hypothesised causal mechanisms underlying contextual effects and may artificially reduce neighbourhood effect estimates.[53,71] In other words,

contextual and compositional effects are not mutually exclusive but rather 'inextricably linked'.[72] In using marginal structural models, I sought to account for the individual socioeconomic and psychosocial factors that predict neighbourhood selection, while avoiding adjustment for later values of these variables, hypothesised as mediators of exposure. Differentiations with my conventionally adjusted estimates were small, as in prior comparisons[73] – I would expect greater differences in samples with greater variation in neighbourhood exposure over time (e.g., due to greater social mobility). Nevertheless, a primary aim of this chapter is to contribute to the continued practice of making causal assumptions explicit and avoiding over-adjustment for individual-level factors.

This chapter has several limitations. First, my estimate is biased as a causal association if there is an unobserved variable that predicts both neighbourhood deprivation exposure and IPV independent of my included covariates. My findings were robust across several model specifications using a rich set of baseline and time-varying socioeconomic and psychosocial covariates, which reduces this concern. However, it is worth noting that the interaction between individual characteristics and neighbourhood environments is a social process in itself.[11,74] The existence of residual individual-level characteristics that predict neighbourhood selection do not necessarily negate the importance of the neighbourhood; rather these interactions should be further investigated as potential effects. Additionally, it is unlikely that reverse causality underlies my findings (i.e., that experiencing IPV caused participants to move to or stay in more deprived neighbourhoods), given the timing of exposure considered (ages 0-18) and that for most participants (70%) the outcome measured was their first experiences of IPV.

Second, as discussed in Chapters 2 and 5, the Indices of Multiple Deprivation measure census-based, residential neighbourhoods,[33] which may not align with participants' conceptualisations of their neighbourhoods or daily activities, and different definitions of neighbourhoods can drastically alter results (the *modifiable areal unit problem*).[75-77] Future research should consider work, school, and social environments and triangulate across measures. Third, I used one iteration of the deprivation scores rather than multiple iterations over time. This ensured that changes in quintiles indicated changes in participants' deprivation exposure rather than measurement differences, but it means that I could not analyse changes in neighbourhoods over time. Nonetheless, as discussed in Chapter 5, I do not expect this to be biasing, given that change in relative neighbourhood deprivation in the study area (especially across quintile boundaries) was fairly minimal over the study period.[78,79]

Fourth, without access to the continuous scores, the exponential distribution of the Indices of Multiple Deprivation necessitated dichotomising my exposure. I was further under-powered to test a stricter dichotomisation of neighbourhood deprivation. Nevertheless, dichotomised measures of neighbourhood deprivation are common in the literature and central to hypotheses of threshold effects;[5,11] given the more liberal threshold I examined, I expect my results are conservative, which should be explored using alternative operationalisations in future studies. My sensitivity analyses showed that my results were at least robust to whether I estimated the effect of cumulative deprivation exposure (continuous and constant within participants) or the average effect of living in more versus less deprived neighbourhoods at each time point (time-varying and binary).

Fifth, as outlined in Chapter 4, I did not have data on women's partners nor the perpetrators and timing of IPV incidents. Finally, persons who were White and from higher socioeconomic families were over-represented compared to the national population, as first shown in Chapter 2. This further adds to the likely conservatism of my results. Future research should test generalisability, including whether the effects observed persist beyond early adulthood.

In conclusion, this study used rich data from a birth-cohort sample with substantial variation in neighbourhood deprivation exposures, a clinically meaningful measure of IPV, and robust family-level confounders over time. My novel application of marginal structural models allowed me to estimate the potentially causal effect of long-term exposure to neighbourhood deprivation on IPV, conditionally independent of differential selection into neighbourhoods and study attrition. My findings of a sustained neighbourhood effect on IPV among women over a 21-year period highlight the need for more longitudinal studies of contextual risk factors (and mechanisms) for IPV as well as the evaluation of preventive strategies that target and account for the structural determinants of these conditions.

6.5 Chapter summary

This chapter demonstrated a meaningful association between long-term exposure to objective neighbourhood-level deprivation, as measured by the Indices of Multiple Deprivation, and higher risk and intensity of experiencing IPV among women in ALSPAC. These results address an important evidence gap identified in Chapter 3, which was the lack of longitudinal studies that have investigated the risk or protective

associations between community- or structural-level factors and IPV against women – especially outside of the USA. Moreover, the investigation conducted in this chapter built off of and was informed by the measurement work presented in Chapter 4 (on the measurement and experience of IPV in ALSPAC) and Chapter 5 (on the measurement and experience of neighbourhood deprivation). In the following chapter, the contributions of this thesis as a whole are summarised, with an in-depth discussion of its strengths and limitations, directions for future research, and implications for policy and practice.

6.6 References

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Chapter 7: Discussion

This thesis aimed to advance the longitudinal evidence base and methods for understanding the etiology of IPV against women, especially regarding community and structural-level risk and protective factors. The objectives of this thesis, as originally stated in Chapter 1, were to:

1. Systematically review prospective-longitudinal evidence of risk and protective factors for IPV against women at all ecological levels (individual, relationship, community, and structural) and identify evidence gaps; and
2. Using two generations of data from the Avon Longitudinal Study of Parents and Children (ALSPAC) in the United Kingdom (UK), evaluate:
    a) The overall prevalence and gender differences in experiences and impacts of IPV in early adulthood and psychometric properties of the IPV measure,
    b) The dynamic relationship between objective neighbourhood-level deprivation and perceptions of the neighbourhood environment over time, and
    c) The effect of long-term exposure to objective neighbourhood deprivation on the risk of experiencing IPV among women in early adulthood.

This chapter begins by summarising the novel empirical contributions of this thesis in addressing each of these objectives. This is followed by a detailed discussion of the limitations of this work and directions for future research. I close with the implications of my findings for theory, policy, and practice and, finally, my overall conclusions.

7.1 Summary, strengths, and contributions to the field

This section provides an overview of the findings of each empirical chapter in relation to the stated objectives of this thesis. I discuss how each chapter in this thesis built upon the last and the ways in which this work advanced existing knowledge and methodology in the field.

7.1.1 Objective 1: systematically identifying the strongest available evidence and evidence gaps

Chapter 3 systematically summarised the current state of the evidence for prospective-longitudinal risk and protective factors for IPV against women. This was a critical step for this thesis and a novel contribution to the literature as, until this point, no systematic review of the total longitudinal evidence base for the etiology of IPV against women had ever been conducted. Rather, several reviews had been conducted on subsets of factors, including prior exposure to IPV in childhood, alcohol use, depressive symptoms, and community-level factors, which mainly combined cross-sectional and longitudinal studies – weakening the potential for causal conclusions.[1-6] The most widely cited meta-analytic review, by Stith and colleagues (2004), did not use a systematic search with fully transparent methods: identifying a total of 509 potentially relevant studies, compared to the over 10,000 studies screened in this thesis.[7] Stith and colleagues' review was critical to establishing an initial understanding of the gaps across both cross-sectional and longitudinal studies of risk and protective factors for IPV (also demonstrating an overwhelmingly individual-level focus across studies) and the potential utility of using meta-analytic methodology to summarise this evidence base. However, modern best

practices for meta-analyses of etiological factors (e.g., not combining unadjusted and adjusted effect estimates)[8] were not used, as was the case for other later meta-analytic reviews in the IPV field.[3,4] Following completion of my systematic review, several additional reviews were conducted, some of which built upon the 2004 Stith et al. review and only conducted systematic searches from this time point.[9-13] Again, these updated reviews and other recent reviews[2,14,15] have only focused on subsets of factors or countries and typically combined cross-sectional and longitudinal studies.

Reflecting its comprehensive and rigorous search, the systematic review presented in Chapter 3 identified more prospective-longitudinal studies of etiological factors for IPV against women than had ever been identified in prior (or later) reviews (N=60).[1-4,6,7,9-15] Using robust meta-analytic methodology, which included the widest possible standardisation of all available effect estimates while maintaining best analytic practices (e.g., only combining conceptually and quantitatively similar effect estimates), I was able to quantitatively synthesise the associations between 20 risk and protective factors and IPV victimisation among women. Through these analyses, I identified that the strongest evidence available for modifiable risk factors for IPV against women was for having had an unplanned pregnancy or parents with less than a high school education, while women who were older or married had lower odds of experiencing recent IPV.

Arguably the most important contribution of this systematic review was in specifying the evidence that was *not* available (i.e., the evidence gaps). First, 80% of the 60 longitudinal studies identified were from the USA, with only seven other countries represented. Of relevance to this thesis, only one of these studies was from the UK, which was of a cohort born in the 1950s (N=212).[16] Second, most studies (57%) did not begin

following participants until adulthood and used basic regression (e.g., linear or logistic) without accounting for repeated measures or the duration of exposures (67%). This demonstrated the lack of longitudinal research on whether and how developmental, sustained, or time-varying exposures affect the risk of IPV against women. Third, and most notably, of the 71 risk and protective factors investigated by the included studies, only seven were community-level factors (e.g., neighbourhood disadvantage) and none were structural (including, for instance, policies or societal norms). This illustrated the overwhelmingly individual-level focus within the longitudinal evidence base for IPV and the dearth of studies that had investigated contextual risk and protective factors. All of these evidence gaps were subsequently addressed in the later investigate work of this thesis (Chapters 4-6).

Chapter 3 demonstrated that many of the commonly-held hypotheses on the contributing factors to IPV were under-evidenced in the longitudinal literature (e.g., the intergenerational transmission of violence).[17,18] In the context of this minimal evidence, the meta-analytic finding of a protective association between neighbourhood disadvantage and IPV was further surprising, sparsely evidenced, and disparate from the bulk of the cross-sectional evidence base and researcher assumptions. That is, cross-sectional studies have largely found that neighbourhood disadvantage is positively associated with a higher prevalence of IPV among women.[1,6,19-24] Without more rigorous evidence, causal conclusions were uncertain. There was thus a clear need, which this thesis contributed to, to further interrogate the available longitudinal studies of this association and begin addressing their conceptual and methodological limitations (Chapter 6).

7.1.2 Objectives 2 and 3: interrogating and summarising measurement of IPV and community-level exposures in the ALSPAC sample

Chapter 3 highlighted that only 32% of 25 cohorts from which data could be meta-analysed were designed to measure women's intimate relationships or experience of violence, all of which were from the USA (n=7) or Canada (n=1). Therefore, this thesis not only built upon an evidence base with few existing studies on the relationship between neighbourhood deprivation and IPV among women, but operated in an empirical space where little longitudinal data were available from studies designed for the purpose of investigating this relationship. This is all the more critical to consider within a research context where the need for robust and sensitive measurement has been amplified by the complexities of IPV disclosure and politics around defining outcome cases or severity, as outlined in Chapter 1. Prior to undertaking my own secondary data analysis of this association, it was thus of an even higher imperative to first consider the robustness and characteristics of the available measures of both IPV and neighbourhood disadvantage in ALSPAC, which offered additional avenues for this thesis to advance methodologies in the field.

Chapter 4 evaluated the psychometric properties of the measure for IPV available in ALSPAC. This was a unique measure developed for ALSPAC by a team of IPV researchers that, in its current form, had never been validated or used with an adult sample.[25,26] Unlike the most commonly used measure in the IPV literature, the Conflict Tactics Scale, the ALSPAC measure *minimised* priming bias towards less coercive, controlling, or severely patterned forms of IPV by not referencing the context of 'conflict resolution' when asking about experiences of violence.[27-30] Moreover, the scale was short-

form, minimising response fatigue (especially in the context of long social surveys like those used in ALSPAC)[31] and, unlike many existing scales,[32-34] captured acts of physical, psychological, and sexual IPV. Additionally, the measure of victimisation in ALSPAC was included alongside a measure of (primarily psychosocial) self-reported impacts of violence, allowing for consideration of severity and context (essential to understanding the clinical burden of IPV and gender differences, as discussed in Chapter 1). Using a robust analytic strategy, I demonstrated that the scale could be reliably analysed as a single factor (or as two: physical/psychological IPV and sexual IPV), was strongly, positively associated with more negative self-reported impacts (providing initial evidence of convergent validity), and items were highly consistent with each other, an indicator of reliability. These results thus provided direction for the appropriate use of the ALSPAC IPV scale in this thesis and demonstrated the suitability of the measure for future aetiological investigations of IPV that use ALSPAC data.

On top of evaluating the psychometric properties of the ALSPAC IPV measure, Chapter 4 summarised, for the first time, the extent of IPV in the birth cohort sample (i.e., the *Children of the Nineties* cohort) and gender differences in prevalence and impact. IPV prevalence was high in the sample: 37% for overall lifetime prevalence and 29% for current prevalence (in the last three years). I found evidence for significant gender differences, with women experiencing more frequent and severe IPV by any available measure. For instance, the lifetime prevalence of IPV for women was 42% versus 29% for men and the current prevalence of IPV for women was 32% versus 24% for men. These prevalence estimates were considerably higher than those estimated in the national population from the Crime Survey for England and Wales (e.g., lifetime prevalence: women=23%, men=11%)[35,36] and among women sampled from high-income countries in

the most recent global systematic review of IPV prevalence estimates (lifetime prevalence=23%) – which synthesised similar national surveys.[37] This is likely reflective of several factors: the younger age of the ALSPAC population, a risk factor for IPV identified in Chapter 3; differing definitions and priming contexts between ALSPAC and national surveys (e.g., the Crime Survey for England and Wales may have primed participants to report acts of IPV perceived as criminal, as discussed in Chapter 1); and the trust built among ALSPAC participants through over 20 years of engaging with the study, which likely increased the propensity for disclosure. It is worth noting, however, that given the higher, overall socioeconomic status of the ALSPAC sample as a whole,[38] and especially those most likely to remain in the sample at later time points (as highlighted in Chapters 2-6), the high prevalence of IPV observed is likely an underestimate for both the regional and national populations.

The results of Chapter 4 served to extend a large body of evidence (described in depth in Chapter 1) which shows that women experience the majority of negative consequences from IPV and, relatedly, are more likely to experience more severe forms of IPV.[39,40] For instance, I found that more women than men experienced every negative impact from IPV (apart from increased substance use), with the most dramatic difference observed in feeling scared of one's partner (56% women versus 14% men who had experienced IPV). These findings bolstered the empirical focus of this thesis on women's experiences of IPV as a distinct clinical outcome from that of men's whose etiological factors should be considered separately (as initially outlined in Chapter 1 and reflected in Chapters 3 and 6), while evidencing the substantial experience of IPV among men worthy of research and service resources.

In addition to evaluating the measure of IPV available in ALSPAC, this thesis interrogated the community-level exposures available in the study to determine how these varied over time and dynamically related to each other to assess the overlap of constructs measured (Chapter 5). This was beneficial to understanding the role of neighbourhood disadvantage in predicting the risk of IPV (Chapter 6) for two reasons. First, from a practical perspective, knowing the level of variation in the measures over time indicated the importance of using analytic methods that could account for time variation in exposure and confounding – and relatedly, inform the level of bias likely in studies that adopt the common practice of using point-in-time exposure.[1,6] Second, from a conceptual and methodological perspective, while the Indices of Multiple Deprivation were the most robust and widely-used measure available of neighbourhood disadvantage in ALSPAC, understanding how this measure related to self-report measures provided important insight into the potential mechanisms of the effects of this exposure on IPV.

In regard to the first point, Chapter 5 demonstrated distinguishable differences in exposure to objective neighbourhood-level deprivation (as measured by the Indices of Multiple Deprivation) among ALSPAC participants over time, with subgroups experiencing increasing (5%), decreasing (11%), chronically high (20%), or stably low (64%) neighbourhood deprivation over time. Trajectories of the perceived neighbourhood measures (opinion of the neighbourhood as a place to live, social cohesion, and neighbourhood disorder) likewise suggested variations in neighbourhood perceptions over time but tended to be more stable or less meaningfully differentiable over time. For instance, apart from one trajectory group (for neighbourhood opinion), the relative differences between participants' perceptions of their neighbourhood environments remained mostly constant over time (e.g., the same subgroups of participants had the most

or the least negative perceptions of their neighbourhoods at all time points). This suggested the utility of exploring longitudinal exposure to measures of the neighbourhood environment, using methods that accounted for time-varying exposure, and perhaps especially so for *objective* neighbourhood deprivation.

In turn, these differences in the trajectories of the perceived and objective neighbourhood exposures demonstrated important differences in their relationships over time. In Chapter 5, I used dual trajectory analysis to explore these, which represented the first investigation, to my knowledge, of the dynamic nature of the relationship between repeated perceived and objective measures of several neighbourhood social variables over time.[41,42] My findings are thus not only relevant to investigations of neighbourhood disadvantage and IPV, but also have implications for the broader neighbourhood effects field. For one, the weakest relationship observed was between perceived social cohesion and objective neighbourhood deprivation. This has implications for the hypothesised mechanisms of the effects of neighbourhood-level deprivation, which is often expected to affect health and behavioural outcomes via poorer social cohesion and informal social control.[1,43-46] In concert with prior research,[41] my results demonstrate that the self-reported and objective measures of the neighbourhood environment measure divergent constructs. Moreover, the amount of variation in objective neighbourhood-level deprivation that is not explained by variables such as perceived social cohesion suggests that while this may be one mediating mechanism of its potential effects on IPV (and other outcomes) there are certainly others (e.g., normalising aggression) that require explicit testing. In examining these relationships over time, Chapter 5 further provided a more accurate assessment: focusing on a single time point may have masked the extent of differences and similarities

between these measures and failed to illustrate the potential influence of the life course on measurement.

Chapter 5 provided additional evidence of the individual-level sources of variation in participants' perceptions of their neighbourhood environments. Using multinomial logistic regression analyses, I found that participants with markers of lower socioeconomic status and poorer mental health had more negative perceptions of their neighbourhoods over time, controlling for objective neighbourhood-level deprivation. These findings extend prior cross-sectional evidence and demonstrate the intertwinement of the individual and the neighbourhood in self-reported measures on neighbourhoods and communities.[47-51] That is, using self-reported measures of the neighbourhood environment, it becomes impossible to parse out individual characteristics from neighbourhood characteristics – which, in this thesis, would have posed problems related to same-source bias given that the outcome (IPV) was also participant reported (i.e., there is likely correlation between mother and child characteristics).[52,53] These findings have important implications for considering how the neighbourhood environment exerts an impact on individuals, specifically the potential importance of the interaction between individual- and neighbourhood-level characteristics: this is discussed further in Sections 7.2.1 and 7.2.2. However, given the inconsistency of the constructs underlying the perceived neighbourhood measures,[54] objective neighbourhood-level deprivation was clearly the more conceptually robust and analytically suitable measure for an initial investigation of the effect of exposure to neighbourhood disadvantage over time on the risk of IPV. This is in addition to some of the added methodological challenges involved in using continuous (rather than categorical) exposures in longitudinal analyses accounting for time-varying confounding, described in Section 7.2.1.

7.1.3 Objective 4: estimating the effect of long-term exposure to neighbourhood deprivation on the risk of experiencing IPV among women over time

Guided by the results of Chapters 3-5, Chapter 6 presented a novel analysis of the effect of long-term exposure to more severe neighbourhood-level deprivation on the risk of experiencing IPV among women. To my knowledge, this is the first study of its kind, which built upon the mixed findings and limitations of the available longitudinal studies of this association identified in Chapter 3.[55-59] This study applied causal inference methods that accounted for time-varying confounding affected by time-varying exposures (marginal structural models using inverse probability weighting),[60] which had never been used before in the IPV field. Using these methods, I estimated that a one-unit increase in cumulative exposure to more severe neighbourhood deprivation over the first 18 years of life was associated with a 62% increase in the frequency of participants' IPV experiences and 36% increase in their risk of experiencing any IPV in early adulthood.

In the end, the results of analyses that did not account for time-varying confounding (i.e., conventionally adjusted analyses) did not dramatically differ from those that did. This is potentially because, as demonstrated in the findings of Chapter 5, although there was notable variation in participants' exposure to neighbourhood deprivation over time, ultimately this was only among 16% of the total sample. If the ALSPAC sample had been more mobile in terms of the level of neighbourhood (or household) deprivation they were exposed to over the study period, I likely would have observed larger differences between the conventional and marginal structural model estimates.

Nonetheless, applying these causal inference methods involved using robust, transparent, and detailed analytic practices that greatly strengthened this investigation. Based on my systematic review (Chapter 3), and where necessary the best available cross-sectional evidence,[15] I developed a directed acyclic graph to determine (and make explicit) the confounding variables most important to account for in my analyses. Drawing out my causal assumptions, in concert with the rich data on family-level psychosocial and socioeconomic variables available through ALSPAC, ensured that I both accounted for confounding as best as possible and that I did not over-adjust my analyses with variables on the causal pathway, as done in prior studies.[55-59] The operationalisation of the primary exposure (cumulative exposure to neighbourhood deprivation) offered a methodologically and conceptually coherent construct that built on prior investigations demonstrating the importance of cumulative exposure over childhood to neighbourhood-deprivation on negative behaviour and wellbeing.[61-63] Additionally, and perhaps most uniquely of all, I tested the robustness of my findings through 38 rigorous sensitivity analyses that included testing a variety of missing data strategies, alternative exposure and outcome definitions, and other possible model specifications. Across the primary and sensitivity analyses, results remained consistent, bolstering study conclusions of the importance of cumulative exposure to neighbourhood deprivation in increasing women's risk of experiencing IPV.

Chapter 1 outlined the importance of considering the spectrum of IPV severity (rather than only dichotomised experiences of any IPV versus none) while Chapter 4 demonstrated the suitability of the ALSPAC measure to this task (e.g., in finding that higher scores on the IPV measure were strongly correlated with experiencing more negative impacts from this violence). Chapter 6 put this into practice by analysing both *IPV frequency* as well as *any IPV* as primary outcomes. Likewise, my sensitivity analyses

used *any IPV with at least one negative impact*, as analysed in Chapter 4, as well as several 'stricter' definitions of IPV, based on a recent measurement study.[64] Chapter 4 showed that a single factor solution for the ALSPAC IPV measure was strongest; however, I also found evidence that a two-factor solution of physical/psychological IPV and sexual IPV could be reliably analysed. Therefore, in Chapter 6, in addition to my sensitivity analyses, I ran secondary analyses where each of these factors was defined as a separate outcome. Results were largely consistent (although more precise for physical/psychological IPV), demonstrating that long-term exposure to neighbourhood deprivation increased the risk of all of physical, psychological, and sexual IPV. This was an additional contribution to the existing longitudinal evidence base, which had largely focused only on physical IPV as demonstrated in Chapter 3.

Finally, the results of Chapters 5 and 6 taken together provide interesting insights into the potential mechanisms underlying the observed effect of cumulative exposure to neighbourhood-level deprivation on IPV among women. For one, the divergence in the trajectories between neighbourhood-level deprivation (as defined by the Indices of Multiple Deprivation) and perceived social cohesion (Chapter 5) suggest that cumulative exposure to neighbourhood deprivation affects women's risk of experiencing IPV (Chapter 6) beyond the social resources or informal social control hypothesised as mediators by social disorganisation or collective efficacy theories.[1,43,65] The idea that there are other influential indirect pathways of this exposure is further compelling when considering the timing of the exposure period investigated – from gestation to age 18 – and the secondary analysis findings which showed that prior cumulative exposure was a stronger predictor of IPV risk than the most recent exposure. This supports developmental theories of the effects of cumulative exposure to neighbourhood deprivation over childhood and adolescence.

Such potential mechanisms include: the normalisation of aggression (either by increased exposure to neighbourhood violence or family violence, the probability of which may be increased by stressful neighbourhood conditions); increased propensity of other family-level risks (e.g., lessened parental involvement or fewer socioeconomic resources); learned unreliability or distrust in neighbourhood/community services (e.g., due to unavailability or under-resourcing); or increased psychological stress or trauma from marginalising or stressful neighbourhood living conditions.[1,6,19,20,66,67] These potential mechanisms are further congruent with findings in Chapter 6 that suggested that the accumulation of deprivation across social and material conditions was more influential than singular dimensions of deprivation (i.e., low neighbourhood income or high violent and non-violent crime). This is in line with perspectives that consider *compounding* or *concentrated* disadvantage to be the most meaningful construct in neighbourhood effects on health, wellbeing, and behaviour.[20,43,68,69] Limitations of these inferences and resulting directions for future research are discussed below.

7.1.4 Overall contribution to knowledge

Summarising across its objectives, this thesis systematically determined the current evidence base for a longitudinal model of the risk and protective factors for IPV against women, identified where gaps exist in this model, and demonstrated the relevance and applicability of rigorous epidemiologic methods for addressing these gaps. In doing so, this thesis further established the reliability and validity of a novel IPV scale, time-variation and social patterning in the measurement of the neighbourhood environment, and a consistent and clinically meaningful association between long-term exposure to neighbourhood-level deprivation and higher risk of experiencing IPV among women – all

using robust longitudinal data. There are thus several overall methodological strengths of this thesis. First, I only used prospective-longitudinal data, which was best suited to establishing the temporality between exposures and IPV, producing stronger evidence to inform effective intervention targets.[70] Second, I used comprehensive and transparent methods, including best practices for systematic reviews and meta-analyses[8,71] and extensive sensitivity analyses to test the robustness of conclusions.[72] Third, the empirical analyses in this thesis used repeated measurement of neighbourhood exposures and methods that accounted for variation in exposure over time (e.g., trajectory modelling and marginal structural models), providing richer and more accurate conclusions than studies that assume time-invariance of exposures (e.g., studies that use point-in-time exposures).[60,63,73-75] Finally, this thesis used causal inference methods, which entailed designing analytic strategies based on explicit causal assumptions to produce the most robust causal estimates given the available data.[60,75-80]

7.2 Limitations and directions for future research

The limitations specific to each study conducted in this thesis are discussed in the corresponding results chapter (Chapters 3-6). In what follows I detail the overarching limitations of this work and directions for future research based on both these limitations and my findings.

7.2.1 Neighbourhoods and other risk factors as black boxes

Risk factor research, and especially studies of neighbourhoods and other contextual effects, are often criticised for failing to meaningfully uncover the *black box*.[43,81-84] That is,

these studies often entail investigating associations between various factors and outcomes of interest, where little attention is paid to actually considering and testing *how* (i.e., via what mechanisms or processes) the factor affects the outcome. Concentrating only on factors rather than pathways stunts etiological understanding of complex outcomes such as IPV and inevitably weakens the implications for strong intervention and policy design.[85]

A priority and strength of this thesis was the focus on moving beyond correlational evidence, which thus far has dominated the literature, and using the best available observational designs (prospective-longitudinal studies) and methods to infer potentially causal effects. This is necessary for informing etiological understanding and preventive interventions for IPV but on its own it is not sufficient. Given the breadth and complexity of the available longitudinal literature summarised in Chapter 3 (i.e., 71 risk and protective associations across 60 studies), it was beyond the scope of this work to summarise all mediators investigated in the available studies. Moreover, while the interrogation of participants' trajectories of exposure to neighbourhood-level deprivation clarified the scope of variation over time and provided insight into how this exposure con/diverged with perceptions and experiences of the neighbourhood environment (Chapter 5), this thesis did not formally test mediated pathways via which cumulative exposure to neighbourhood-level deprivation affected the risk of experiencing IPV. A critical direction for future research is thus to both systematically summarise the available evidence for the risk and protective *pathways* to IPV over time and, where evidence is limited, including for neighbourhood disadvantage, to directly test for potential mediators. For neighbourhood disadvantage, as discussed above, this may include, for instance: family-level socioeconomic resources, psychological stress and trauma, attitudes towards IPV, and availability and/or interaction with social services.

Relatedly, this thesis raises a number of interesting questions regarding how neighbourhoods may affect the risk of IPV over time. First, given the many possible ways of defining neighbourhood disadvantage[86,87] and the fact that objective and perceived measures did not perfectly converge, it would be valuable for future research to explore whether people's perceptions and experiences of their neighbourhoods and communities are as influential in predicting their experience of IPV as compared to objective neighbourhood deprivation. This would in part address issues of convenient neighbourhood boundaries used in objective measures (e.g., census tracts) and participants' neighbourhood or community exposures involving more than just their residential neighbourhood.[88-91] However, it raises additional methodological complexities (e.g., addressing time-varying confounding with continuous exposures is often problematic due to issues of model misspecification, instability, and inefficiency[73]) and practical challenges (e.g., what are the intervention implications when there is no consistent definition of the area of interest beyond participants' personal interpretations of the scale items?). There is thus ample room for additional methodological advancement and triangulation in the field to address these issues, which as demonstrated by my systematic review (Chapter 3), would greatly contribute to the limited longitudinal evidence base on community-level risk and protective factors for IPV. As with objective neighbourhood disadvantage, the available longitudinal studies (identified in my systematic review or published thereafter) of the relationship between perceived neighbourhood factors (e.g., collective efficacy, social support) and IPV against women have shown mixed findings.[55,58,59,92-94]

An additional but related empirical question arising from this thesis is whether there is a meaningful interaction between perceived and objective neighbourhood

disadvantage in predicting the risk of IPV against women. That is, does living in more or less deprived neighbourhoods exacerbate or weaken the expected relationship between women's negative perceptions of the environment (e.g., perceived neighbourhood disorder) or poor relational experiences (e.g., low social cohesion) and their experience of IPV? Considering this question in ALSPAC is limited at present, as the birth cohort sample is only in young adulthood with limited self-report data on their neighbourhoods – both reasons why this thesis relied on mothers' longitudinal perceptions of their neighbourhood environments in Chapter 5. However, at least one existing longitudinal study, identified in my systematic review, has considered this interaction: Jain et al. found that in low-to-moderate poverty neighbourhoods, collective efficacy (based on the aggregate reports of residents) was negatively associated with the odds of young adult men perpetrating IPV, whereas in high poverty neighbourhoods, collective efficacy was surprisingly positively associated with IPV perpetration.[58] These findings suggest that collective efficacy was a protective factor for IPV only when neighbourhood deprivation was low; when neighbourhood deprivation was more severe, it served as a risk factor. However, potential mechanisms and, as with other prior neighbourhood-IPV studies, longitudinal exposures were not considered, demonstrating the need for future prospective research using repeated measures in this area.

Likewise, future research is needed to parse out the contextual and methodological factors associated with positive (as found in Chapter 6 and one previous longitudinal study[55]) versus negative associations found between neighbourhood disadvantage and IPV in other prior longitudinal studies (Chapter 3).[56,58,59,94] There are a number of possible reasons for differential findings, including sociodemographic context, variation in exposure being considered or not, as well as the analytic issues described in Chapter 6

(e.g., selection or collider-stratification bias). However, given the unique duration and timing of neighbourhood deprivation exposure considered in this thesis (the first 18 years of life), further attention should be paid to the potential developmental mechanisms underlying this association, as discussed above. Given my findings of unique trajectories of exposure to neighbourhood-level deprivation (Chapter 5), and that more recent exposure to less deprived neighbourhoods did not nullify the cumulative effect of prior deprivation exposure (Chapter 6), it would be valuable for future research to consider whether these different trajectories are associated with differential odds of IPV and possible critical or sensitive periods for exposure – and, if so, *why* (e.g., interactions with cognitive, emotional, or social development).[95]

7.2.2 Interactions between ecological levels

In addition to considering interactions *within* ecological levels (i.e., between perceived and objective neighbourhood measures) it is also worthwhile for future research to consider interactions *between* ecological levels. For instance, one criticism of solely looking at the effects of negative neighbourhood-level exposures is that the most deprived people do not necessarily live in the most deprived communities.[96] Individual deprivation may be the result of the broader structural context (e.g., national or regional laws and policies) that produces socioeconomic inequities between people, regardless of the neighbourhoods they reside in. Individual-level deprivation may operate cumulatively with neighbourhood-level deprivation, increasing the risk of IPV to a greater extent than either factor on its own (cumulative disadvantage). However, it may also be the case that people with lower socioeconomic status are at greater risk for experiencing IPV when living in less deprived neighbourhoods (relative disadvantage).

Indeed, this latter hypothesis has found empirical support. For instance Odgers et al. have shown that lower-income boys living in more as opposed to less affluent neighbourhoods engage in more antisocial behaviour,[97] while Nieuwenhuis et al. found that moving to a more affluent neighbourhood was associated with greater depression, social phobia, aggression, and conflict with parents among adolescents.[98] These studies further bolster the hypothesised importance of *relative* deprivation, as investigated in this thesis (Chapters 5 and 6). That is, greater (real or perceived) disadvantage compared to one's neighbours or people in other neighbourhoods may be an important mechanism as to how deprivation increases psychological strain, relational conflict, marginalisation, and barriers to accessing social resources – all of which may then predict negative behavioural and wellbeing outcomes, such as IPV.[68,69,97-100] This may further explain the finding in Chapter 6 that participants with prior exposure to deprived neighbourhoods but living in less deprived neighbourhoods at age 18 were at highest risk for experiencing IPV: these women may have experienced greater socioeconomic disadvantage relative to their new neighbours. Future research should thus consider whether the effect of neighbourhood-level deprivation is constant (or varies) across levels of individual-level socioeconomic status (along with considering potential interactions between absolute and relative deprivation[100]). Likewise, a systematic review of interaction effects within and across ecological levels of risk and protective factors for IPV against women would be an important extension of the work undertaken in this thesis (Chapter 3), which would strengthen our understanding of the etiology of IPV and targets for intervention strategies.

7.2.3 The broader role of the structural context and the true 'causes of causes'

This thesis identified an important and detrimental lack of longitudinal studies investigating the role of structural-level factors such as societal norms or policies on the risk of IPV against women (Chapter 3). In some ways, the work presented in this thesis contributes to addressing this gap, by investigating the measurement properties of IPV (Chapter 4) and structural deprivation (the Indices of Multiple Deprivation, Chapter 5), and the relationship between these (Chapter 6), in a cohort study. However, there are also many ways in which a wider conception of structural factors is left unaddressed in this thesis and more broadly the current evidence base, which requires further rigorous research.

First, this thesis does not address what determines neighbourhood-level deprivation in the first place. That is, inequalities in neighbourhoods (and by extension *who* lives in more disadvantaged neighbourhoods) are the result of social, economic, and political structures that are not amenable to individual-level interventions.[101-103] These structural factors are otherwise known as the 'fundamental causes', or 'causes of causes', of poor health and wellbeing and they are critical to address for meaningful and sustained improvements to health equity.[104-106] Therefore, future longitudinal research should investigate the potential indirect effects of structural determinants, such as social and economic policies, on IPV via neighbourhood-level deprivation – and possible interactions with individual-level factors such as socioeconomic status and race[107] – to develop a more robust evidence base that can inform policy strategies for preventing IPV in the first instance.

Second, addressing an important evidence gap in the field, this thesis focused in large part on understanding the variation in exposure to neighbourhood-level deprivation

over time and investigating how this related to IPV risk. This was critical to properly addressing mobility between neighbourhoods (and deprivation levels) over time (and the time-varying confounding associated with this), yet these methods do not discount the experience of sustained deprivation exposure over time. Indeed, the accumulation of deprivation both in terms of domains (i.e., across social and economic conditions) and time (i.e., over the first 18 years of life) was of prime importance in the operationalisation of exposure. Nonetheless, an important limitation of this thesis is that I was not able to address the so-called 'stickiness' of deprivation (or conversely, wealth) across generations.[108] The past neighbourhood environments of caregivers are predictive of their future neighbourhood environments (i.e., where their children grow up), and exposure to more deprived neighbourhoods in either generation has been shown to uniquely – and when present in both generations, cumulatively – affect child outcomes (e.g., cognitive ability).[61]

This adds important complexity to investigating and contextualising the role of neighbourhood deprivation in increasing IPV risk. For one, it means that earlier generational exposure to neighbourhood deprivation may be a confounder of the relationship between exposure to neighbourhood-level deprivation and risk of IPV among young women. It also means that addressing the current state of inequalities among neighbourhoods may not be sufficient to protect against the intergenerational transmission of trauma and marginalisation.[109,110] This adds to the call made in this chapter for further consideration of the developmental mechanisms underlying the effect of exposure to neighbourhood-level deprivation on IPV risk, but extends this to also consider mechanisms related to the *multi-generational* development of the family unit.

Third, in addition to neighbourhood-level deprivation and its structural determinants, there are also an array of structural factors not considered in this thesis and understudied in the existing longitudinal literature which may have important interactions with the former. For instance, gender inequality (unequal access to and distribution of resources among women at a societal level) and societal norms (including around gender role expectations and the acceptability of violence) have been hypothesised as important risk factors for IPV with cross-sectional evidence demonstrating positive correlations.[111-118] Gender inequality, societal norms, and neighbourhood deprivation may interact in unique ways to affect the risk of IPV among women that should be explored in future research: for instance, women with fewer socioeconomic resources, facing societal, family, and/or partner pressure to not participate in the labour market or advance her education, and with fewer opportunities available to do so may be at an even greater risk of experiencing IPV.

It is worth noting, however, that the relationship between women's economic empowerment – including poverty alleviation directed specifically to women (e.g., through cash transfer, savings, or microfinance programmes) – and IPV risk has shown mixed findings in the intervention literature.[119-122] A number of contextual factors have been proposed as intervention effect modifiers therein, including: method of poverty reduction, whether gender norms are also addressed, socio-cultural context, and socio-demographics of participating individuals and households. This has implications for the intervention and policy recommendations that can be derived from this thesis, discussed in Section 7.4, including the need for further intervention and policy strategies that target community and structural factors at the *structural* level rather than to individuals or households.[123] However, it also demonstrates that further longitudinal research is needed that tests the

interactive effects of structural and community-level factors, particularly with a gendered-lens, to understand the complex ways that multiple deprivation may uniquely affect the risk (and consequences) of IPV against women.

7.2.4 Issues of generalisability: perpetration, heteronormativity and gender, and socio-demographic context

The empirical work of this thesis (Chapters 3-6) largely relied on women's self-reports of victimisation. This was justified as an issue of practicality and validity: most studies, especially long-term cohort studies, are not able to sample couples and, in the case of a single participant, self-reports of victimisation tend to be more sensitive than self-reports of perpetration.[124-126] Sampling couples can further pose ethical and methodological problems in IPV research, considering that the most severe IPV experienced by women often occurs in the context of separation and barriers to disclosure may increase if a violent partner is also participating.[127-130] Nonetheless, the implications of focusing on women's self-reports are that many of the conclusions of this thesis apply to women's risk of *experiencing* violence (and the conditions that affect this) rather than men's (or women's) risk of *perpetrating* violence. In the context of a literature that has predominantly focused on individual-level factors as potential causes of IPV, this may have the unintended consequence of contributing to a victim-blaming perspective where women are conceived of as 'risk avoiders', vulnerable victims, or unitary in their experiences.[131,132] Relatedly, this perspective can be seen as failing to place accountability and responsibility upon perpetrators, or perhaps even more importantly, structural and societal contexts.

It has been an important aim of this thesis to reject this perspective by explicitly drawing critical attention to the individual-level focus of the longitudinal IPV literature (Chapter 3) and developing the methodology and empirical knowledge for longitudinal community- and structural-level risk factors for IPV against women (Chapters 4-6). This thesis inevitably represents one piece of the puzzle, oriented around victimisation – and future research should investigate whether findings (particularly in Chapters 3 and 6) replicate for IPV perpetration. Nonetheless, it is important to note that the former is a valuable approach that is not simply a proxy for the latter nor one that necessarily misattributes accountability.

A victim-oriented approach is critical, for one, because it considers acts of violence beyond only those where the perpetrator *intended* the harm that occurred (based on motivations) to also include acts where harm was *foreseeable* but not necessarily intended as such (based on consequences).[133] That is, in defining violence, all of actions, intentions, and harms should be considered and these will not necessarily always align.[134] This is not unique to IPV but rather a central tenet of modern criminal justice systems: for instance, acts that were not intended to cause harm, but where harm was foreseeable, are still classified as crimes as opposed to mere accidents. Likewise, threatened action (where the action did not ever occur) can still cause significant harm to the target and be an important type of violence. In only focusing on self-reports of IPV perpetration, we may fail to capture those acts of IPV which were not intended or interpreted as such by the perpetrator, but were still harmful or impactful to the victim.[133] The victim-oriented approach thus has clear import beyond the criminal justice system to public health priorities as well, where minimising the clinical burden of IPV is of prime concern.[111,135] In other words, investigating risk and protective factors based on an understanding of

violence from the perspective of victims, here women, provides a critical piece to the prevention puzzle both from a criminological and public health standpoint. Furthermore, this woman-centred approach explicitly offers directions for designing stronger support services and intervention strategies for women who are experiencing, or most at risk for experiencing, IPV.[132]

A 'two-pronged' approach to understanding IPV, in terms of considering both victimisation and perpetration,[133] is thus useful to developing a robust etiology of this violence and preventive strategies. Greater exploration therein of risk factors beyond women's characteristics to those of their partners, families, communities, and structural contexts is critical. This includes dyadic factors within the relational context, which may be developed from separate studies on each of victimisation and perpetration as well as studies of couples, from situational determinants to more complex, developmental considerations (e.g., the consequences of cumulative histories of trauma and disadvantage among both partners).[136] A dyadic understanding, in turn, raises further complicated questions regarding IPV typologies in relationships (e.g., violence and/or controlling behaviour among one or both partners) as raised in Chapter 1, that should be explored (and validated) in future research in terms of the exposure histories (including community and structural risk factors) that determine them.[29,137-139]

In adopting a women-centred approach, this thesis did not address risk factors related to men's experiences of IPV. As demonstrated in Chapter 4, male participants of ALSPAC experienced a significant amount of IPV, which should be addressed in future research. However, given the significantly greater clinical burden of IPV experienced by women compared to men, found both in ALSPAC (Chapter 4) and the broader literature as

well as the gendered contexts and etiologies of this violence (as outlined in Chapter 1), this thesis focused on women's risk factors for IPV. It would be valuable for future research to test for gender differences in the findings of both my systematic review (Chapter 3) and investigation of the role of neighbourhood deprivation (Chapter 6), and to continue considering possible gender differences in the measurement of IPV (as discussed in Chapter 4).

In relying on secondary data, this thesis was also limited in its ability to address heteronormativity and the experience of IPV among sexual and gender minority populations. Available longitudinal studies on IPV against women identified in my systematic review (Chapter 3) did not consider IPV risk based on gender identity (e.g., cis or trans) or sexual orientation (same-sex or heterosexual partner). Likewise, there were no available data in ALSPAC on gender identity and the one item on sexual orientation at age 21 did not allow for interpretation of the gender identity of the partners who participants reported perpetrated IPV. Future research should test the replicability of the findings from this thesis to sexual and gender minority populations, especially considering that sexual and gender minority populations are over-represented in IPV cases, face significant barriers to service access, and may require unique or targeted programming.[140-142]

Finally, this thesis identified a lack of longitudinal studies of risk and protective factors for IPV against women in LMIC (Chapter 3). Whether the risk and protective associations identified in my systematic review or empirical findings on IPV, neighbourhood disadvantage, and their association apply to LMIC contexts is an empirical question that should be investigated in future research. Notably, however, questions of contextual generalisability are not limited to LMIC versus HIC (either in this thesis or

beyond): rather, there is substantial variability in the prevalence (and potentially the determinants) of IPV *within* as well as between countries, regions, and communities.[37] Therefore, future replication analyses are not only required across countries but within countries, considering variation in sociodemographic characteristics at the individual level – including socioeconomic status, race/ethnicity, employment, residential mobility, and family structure – and community and structural levels – including neighbourhood/community compositions, physical environment and resources, service provision, and policy contexts.

7.2.5 Qualitative research

The focus of this thesis has been on investigating and building the quantitative evidence base for risk and protective factors for IPV against women. As outlined in Chapter 2, rigorous quantitative evidence is critical to establishing the potential causes of IPV, a central aim of this thesis. However, robust etiological theory – as well as eventual intervention development and evaluation –requires qualitative evidence; findings are perhaps most useful when a mixed-methods approach to research is used (i.e., when quantitative and qualitative research findings are used to complement and inform each other).[143-145] For instance, qualitative research is critical to explaining potential mechanisms underlying effects (whether direct or indirect) identified in quantitative research as well as generating hypotheses beyond those that have been tested (or are most prolific) within the quantitative evidence base.[146] Participatory and qualitative research is further all the more important in understanding and intervening in complex health and social problems like IPV to ensure ethical and sensitive methods that are acceptable and just to stakeholders and knowledge users, with findings that address and are enriched by

their direct perspectives.[127,147-149] While beyond the scope of this thesis to review in depth, qualitative and mixed-methods research has played a critical role in our understanding of IPV: for instance, in understanding barriers to disclosure and engagement,[150] the potential role of neighbourhoods and structural contexts,[109,151-155] and effect mechanisms of interventions and policies.[156-159]

Directions for future research based on the findings of this thesis are thus not limited to quantitative research. A systematic meta-synthesis of qualitative studies on the risk and protective factors for IPV against women would be useful to identify areas of overlap or divergence with the quantitative evidence and where factors have only been investigated quantitatively or qualitatively. Indeed, as in the quantitative literature, existing qualitative meta-syntheses on IPV have focused on subsets of factors or subpopulations.[160-164] Moreover, investigating the perspectives of women who have experienced IPV, service providers, policymakers, and other relevant knowledge users and stakeholders on how *sustained* exposure to neighbourhood deprivation affects IPV may illuminate additional effect mechanisms and contextual differences beyond existing evidence.[151,152,155] Likewise, exploring acceptable and feasible intervention strategies that target and account for community- and structural-level factors among stakeholders remains an important strategy for maximising the odds of effectively preventing IPV.[148]

7.3 Contribution to theory

As demonstrated in the above discussion (both Sections 7.1 and 7.2), a key contribution of this thesis is its illustration of the many gaps that remain in our theoretical understanding of IPV against women. This is especially true regarding how (i.e., by what

mechanisms) factors at the more distal levels of the so-called ecological model (first discussed in Section 1.2.2) affect IPV (or other more proximal causal factors, as suggested in Section 7.2.2). These evidence gaps were systematically summarised in Chapter 3, which illustrated the lack of available longitudinal studies on the relationship between any community- or structural-level risk or protective factors and IPV against women. Despite these gaps, several implications for etiological theories of IPV can still be drawn from the findings in this thesis. First, this thesis, and in particular Chapter 6, demonstrates that, at least in some contexts, exposure to neighbourhood-level deprivation is a potentially causal risk factor for IPV against women. This supports the appropriateness of an ecological approach to understanding the etiology of IPV, where contextual-level factors, like neighbourhood deprivation, are integrated with individual-level factors (e.g., mental health, substance use, and socioeconomic status) in conceptions of risk.[165] In other words, this thesis illustrates that individual-level explanations – which currently dominate the longitudinal evidence base for IPV risk and protective factors – are, on their own, insufficient for understanding the mechanisms underlying the long-term risk of experiencing IPV.

Nonetheless, as was discussed in Chapter 1, the ecological model alone is uninformative regarding the theoretical mechanisms that link community- and structural-level environments to IPV. In light of this, the ecological model is perhaps best viewed as a conceptual approach that explicitly recognises the importance of both individual- and structural-level factors to health and behavioural outcomes, but requires further theoretical explication to establish the causal mechanisms at play.[166] As discussed throughout this thesis, to date, the community and structural mechanisms hypothesised in the context of IPV have largely been grounded in social disorganisation or collective efficacy theories,

where informal social control and social cohesion are hypothesised as mediators.[1,6] A second contribution of this thesis to IPV theory is thus my conclusion that there are likely other indirect mechanisms of community- and structural-level effects on IPV against women (where these exist, e.g., for neighbourhood deprivation) beyond social cohesion and control.[1,43,65] As outlined in Section 7.1.3, this is evidenced by the substantial divergence observed between objective neighbourhood-level deprivation and perceived social cohesion in Chapter 5 and consideration of the exposure relevant to IPV risk in Chapter 6 (i.e., cumulative exposure to neighbourhood deprivation from gestation to age 18). Potential theoretical mechanisms that are congruent with the empirical work in this thesis were discussed above and include: the normalisation of aggression, increased propensity of family- or individual-level risks, learned distrust in neighbourhood or community services, or increased psychological stress or trauma.[1,6,19,20,66,67] These should be further explored in both quantitative and qualitative research.

Likewise, as was discussed in Section 7.1.3, the results of Chapter 6 suggest the importance of cumulative and relative neighbourhood-level deprivation to the risk of experiencing IPV. This bolsters theoretical perspectives that view concentrated disadvantage across social and material conditions as more influential to health and behavioural outcomes than more narrow operationalisations of economic poverty[20,43,68,69] and extends this principle to the etiology of IPV. These multiple dimensions of deprivation may operate in multiple pathways that cumulatively increase the risk of IPV – for instance, normalisation of violence via neighbourhood crime compounded by increased stress and isolation from disordered living conditions, service deficiencies, and a lack of economic opportunity.[20,43,69,99] Likewise, psychological strain in particular (and the resultant propensity towards individual-level risk behaviours and conditions) may cumulatively

increase with each deprivation domain, indirectly exacerbating IPV risk. Relatedly, this thesis suggests that living in *relatively* more deprived neighbourhoods is impactful in increasing women's risk of experiencing violence, which may be important to these mechanisms by increasing risk-promoting conditions for IPV, such as psychological strain, barriers to service access, and relational conflict.[68,69,97-100]

Finally, as discussed in Section 7.1.2, this thesis (and in particular Chapter 4) extends the cumulative body of evidence that demonstrates the importance of gender to our understanding of IPV and its associated clinical burdens (introduced in Chapter 1).[30,39,40] I observed gender differences, which were often dramatic, in the experiences of acts of IPV (particularly sexual violence), frequency of experiences, and consequences of this violence (especially fear of partner). These differences – in conjunction with prior evidence – bolster the need for a theory of IPV that fully accounts for (a) potential etiological differences in IPV by gender and, similarly, gender-related causal factors (e.g., gender inequality and gender norms and attitudes) and (b) the gendered social context in which IPV occurs (e.g., the greater social and economic power of men versus women in society).[167-169] As discussed in Section 7.2.4, appropriate theoretical development of the role of gender in IPV requires future research to consider the full spectrum of gender and sexual identities and potential etiological differences therein.

7.4 Implications for policy and practice

This thesis demonstrated that, even amongst a cohort of participants engaged in a study in the UK for over 20 years and with relatively higher socioeconomic status than the national population, IPV is a highly prevalent and clinically significant problem –

especially among women (Chapter 4). These findings extend national prevalence estimates[35] and illustrate the need for IPV to remain a public health priority in the UK. The risk factors for IPV against women identified in this thesis further suggest several potential policy and intervention targets. For instance, based on predominantly USA-based evidence, programs and policies that aim to improve the socioeconomic status (namely, via education) and reproductive health and safety among women and their families may maximise prevention effectiveness (Chapter 3). Moreover, targeting the structural determinants of neighbourhood-level deprivation, as well as early and/or long-term exposure to more disadvantaged conditions, may be an important avenue for preventing IPV against women (Chapter 6). Finally, given the higher prevalence of IPV among women living in more deprived neighbourhoods, support services for women experiencing or fleeing from violence should consider and account for the contexts in which their clients may be currently or historically situated in and the psychosocial trauma, stress, and isolation that may be associated with or exacerbated by these.

As noted in Section 7.2.3, these potential directions for policy and practice must be considered in light of the mixed evidence base for the effectiveness of economic interventions for IPV against women.[119-122] Although, as outlined in Section 1.4, the evidence base for the primary prevention of IPV is limited overall, with most effective interventions (at least in LMIC) involving some structural component (e.g., microfinance, cash transfers).[157,170,171] Further research is undoubtedly needed on the indirect (and potentially interactive) effects of structural factors, such as social policies, via neighbourhood- and individual-level deprivation on IPV across contexts before policy and programmatic conclusions can be drawn. Nonetheless, this thesis draws attention to the relevant intersections of multiple policy and service sectors that affect (and could

potentially address) IPV against women. Specifically, the criminal justice sector has been historically dominant in leading response strategies for IPV, with a focus on perpetrators and recidivism (e.g., arrest policies, 'batterer intervention programmes', and risk assessments for severe violence or re-victimisation).[158,172-175] However, alone this narrow focus is both inadequate for the primary prevention of IPV (i.e., preventing IPV in the first instance) and unable to target the broader risk and protective factors that this thesis identified as important to women's experiences of violence (e.g., socioeconomic conditions, reproductive safety). Likewise, while Chapter 1 illustrated the many health consequences of IPV against women, demonstrating the import of this violence to the public health agenda, my findings further illustrate how preventive strategies for IPV also require policies from beyond the health sector alone.

This is well reflected, for instance, in the finding of Chapter 6 that long-term exposure to neighbourhood deprivation increased women's risk of experiencing IPV: neither a narrow criminological nor healthcare approach would be sufficient to target or account for such an effect in preventive efforts. Instead, the findings of this thesis lend support to a *health in all policies* approach, which explicitly recognises that complex health outcomes (including both IPV and its health consequences) are affected by policies in all sectors, not just health.[176-180] Intra-sectoral initiatives – involving, for instance, economic and social service sectors as well as criminal justice and public health – are likely necessary for any complete IPV prevention strategy to address the full array of risk and protective factors considered in the preceding chapters.[135,181,182] This also means that interventions not necessarily designed to target IPV (e.g., poverty reduction strategies) may have the unintended benefit of reducing IPV – or the potential to do so. Therefore, the suite of interventions considered relevant to IPV should not necessarily be only those

specifically designed to prevent IPV perpetration: rather this thesis suggests that any number of general public health, economic, or social care interventions and policies could have a potentially positive effect in reducing IPV (e.g., by targeting an evidenced risk factor such as neighbourhood deprivation). These would further have the benefit of affecting positive change in other health and wellbeing outcomes beyond IPV alone.[178,180] The most effective design of such strategies – and how targeted IPV or gender components (e.g., gender training) may amplify (or modify) intervention effects – remains a critical question for future research.

7.5 Conclusions

IPV against women has been recognised globally as a public health priority and violation of women's rights. This thesis has demonstrated that the longitudinal evidence base for the conditions that increase or decrease the risk of this violence is limited in terms of research methodology, contextual diversity, and conceptual focus on individual-level factors. In order to maximise the effectiveness of preventive interventions and policies for IPV, further longitudinal research is needed to investigate the community- and structural-level determinants of this violence, with attention to measurement validity, the timing and duration of exposures, theoretical mechanisms (including interactive and mediating pathways), and generalisability across populations and regions. This thesis demonstrated the applicability of rigorous psychometric, longitudinal measurement, and causal inference methods to improving etiological understanding of IPV and identified an important association between long-term exposure to neighbourhood deprivation and greater IPV against women. The prevalence of IPV against women remains high worldwide and in the UK. However, the results of this work and the prioritised areas for future research offer

clear directions for strengthening the evidence base to inform policy and intervention strategies that can more effectively prevent IPV and support women experiencing this violence.

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Volume 2: Appendices

Building a systematic model of risk and protective factors for intimate partner violence against women: the role of long-term community and structural disadvantages

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Trinity Term 2019

Appendix A

Figure A1: **Ecological model of risk and protective factors for IPV against women[1,2]**

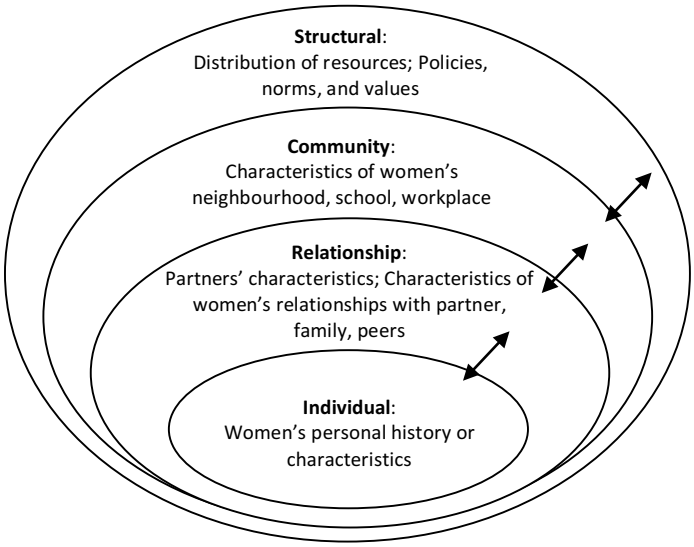


Table A1: **Search strategy**

Step	Strategy
1	I searched the following electronic databases from inception to June 2016: MEDLINE, Embase, Global Health, CINAHL, PsycINFO, and Web of Science. I used a standardised set of free-text terms as well as database-specific controlled vocabulary. Free-text terms included those for: violence ("violence", "abuse*", "maltreat*", "aggress*", "batter*", "assault*", "beat*", "victim*"); partner ("partner*", "domestic", "marriage*", "marital", "spouse", "intimate", "husband*", "wife", "wives", "dating"); intimate partner violence ("IPV", "intimate partner violence", "relationship violence", "spousal abuse", "battered women"); women ("wom?n"); adults ("adult*", "adolescen*", "teen*", "age*"); longitudinal design ("longitudinal", "cohort*", "retrospective", "prospective"); and other designs (e.g., "cross section*", "trial", "evaluation*"). All search terms were first piloted in MEDLINE to balance sensitivity-specificity and ensure known studies were retrieved. Through this piloting, I developed a more sensitive search strategy for retrieving longitudinal studies. This involved leaving out the primary population of interest (women) from the search to retrieve longitudinal studies that may have both male and female participants but provide gender disaggregated results.
2	I searched the following smaller and/or less systematic databases: Google Scholar, ASSIA, ProQuest Dissertations and Abstracts, OpenGrey, National Criminal Justice Reference Service Abstracts Database, Cochrane Library, and Sociological Abstracts. All free-text search terms from step 1 were used except those for study design. I also searched for intimate partner or domestic violence in the World Bank Open Knowledge Repository, WHO Institutional Repository for Information Sharing, and WHO Prevent Violence Evidence Base and Resources.
3	Reference lists of all systematic reviews identified were screened for eligible studies.[3-18]
4	Authors of relevant studies were contacted by email wherever their manuscripts mentioned but did not present eligible data (e.g., gender-disaggregated analyses).

Table A2: **Search strategy as implemented in MEDLINE (inception until May Week 2 2016)**

String	Search term	Results	Question component
1	violence.ti	16 170	
2	abuse*.ti	37 800	
3	maltreat*.ti	2 067	
4	aggress*.ti	21 796	
5	batter*.ti	5 397	
6	assault*.ti	3 041	
7	beat*.ti	6 742	
8	victimi*.ti	1 774	
9	domestic.ti	16 170	
10	partner.ti	7 752	
11	partners.ti	5 992	
12	marriage*.ti	4 191	
13	marital.ti	3 385	
14	spouse*.ti	2 086	
15	spousal.ti	656	
16	intimate.ti	3 503	
17	husband*.ti	2 072	
18	wife.ti	925	
19	wives.ti	783	
20	dating.ti	1 586	
21	or/1-9	92 621	Violence
22	or/12-20	44 925	Intimate partner
23	21 and 22	6 658	Intimate partner violence
24	IPV.ti	306	
25	relationship violence.tw	115	
26	battered women/	2 425	
27	spouse abuse/	6 761	
28	exp intimate partner violence/	6 926	
29	or/24-28	8 293	IPV
30	23 or 29	10 862	Final IPV
31	wom?n.tw	825 906	
32	women/	13 949	
33	31 or 32	830 822	Women
34	longitudinal.tw	149 165	
35	cohort*.tw	314 833	
36	retrospective.tw	300 827	
37	prospective.tw	376 251	
38	cross section*.tw	204 973	
39	regression*.tw	444 303	
40	case control*.tw	86 765	
41	intervention*.tw	588 344	
42	program*.tw	583 552	
43	trial*.tw	662 201	
44	experiment*.tw	1 389 882	
45	evaluation*.tw	864 634	
46	associat*.tw	2 956 770	
47	risk*.tw	1 409 214	
48	protect*.tw	549 617	
49	predict*.tw	968 251	
50	correlat*.tw	1 292 100	
51	pathway*.tw	720 003	
52	exp epidemiologic studies/	1 886 303	
53	exp regression analysis/	336 304	
54	exp risk/	943 034	
55	or/34-37	977 782	Longitudinal studies
56	or/38-54	9 089 840	Other study designs
57	adult*.tw.	1 179 127	
58	adolescen*.tw.	260 177	
59	teen*.tw.	31 232	
60	age*.tw.	3 920 755	
61	exp adult/	5 120 731	
62	adolescent/	135 471	
63	or/57-62	8 856 615	Age
64	30 and 55 and 63	796	Sensitive longitudinal strategy
65	30 and 33 and 56 and 63	3 753	More specific strategy for other study designs
66	64 or 65	4 075	ALL

Table A3: **Final selection criteria and rationale**

Inclusion criteria	Exclusion criteria	Rationale
Published or unpublished studies	-	To minimise publication bias.
Any location	-	To identify regional evidence gaps and potential contextual differences.
Available in the English language	Not available in English	Due to limited resources.
Quantitatively analysed at least one risk or protective factor	Intervention studies; trends analyses; qualitative studies; reviews	Outside the scope of this review.
Physical, sexual, or psychological IPV	Any non-IPV outcome	According to internationally-recognised definition of IPV.[19]
Participants aged 19 years or older at outcome measurement	Participants aged less than 19 years at outcome measurement	Based on the WHO definition of adulthood.[20]
Studies of women's self-reported victimisation, including studies that measured IPV victimization using both partner- (perpetrator) and self- (victim) report (e.g., by selecting the maximum or more severe count) and studies with male victims so long as they provided disaggregated data for female victims	Studies with male-only samples; studies that only measured self-reported perpetration; studies of male and female victims that did not provide gender-disaggregated data	A previous meta-analysis[21] and later reliability studies[22] have shown that in the same relationship, people tend to self-report being victimized by their partners more than their partners self-report perpetrating IPV. This suggests a potential reporting bias, with one possible explanation being that self-reporting violent behaviours is not socially desirable, which leads to underestimation.
Prospective design: At least two assessment waves, where the study measured exposure to the risk/protective factor before the outcome	Studies that measured the risk/protective factor(s) at the same time-point as the outcome, without analyzing change; Risk/protective factors measured retrospectively (e.g., retrospective history of child abuse); Studies that only measured lifetime as opposed to current prevalence of IPV	Prospective, longitudinal designs allow for stronger evidence as to whether the risk or protective factor precedes the outcome, a necessary criterion for causal relationships.[23]
Population, community, or birth cohort sample	Studies of clinical samples or special subpopulations (e.g., pregnant or postpartum women, migrant or refugee women, women who use drugs, women living with disabilities or specific diseases, university or college students, women accessing clinical services); Studies that only included victims of IPV without a control group	Similar to previous IPV reviews,[6,18] this is because (a) the risk and protective factors for IPV are expected to vary based on special characteristics among these subgroups, and the scope of the current review is to inform universal primary prevention of IPV among women, and (b) many subpopulation-studies are defined subjectively making the practical value of comparisons and meta-analysis uncertain.
Studies that controlled for (either by matching or in analysis) any other variables when analyzing the association between the risk/protective factor and IPV	Studies with only unadjusted effect estimates; Analyses of IPV trends or stability	Similar to prior reviews,[18] this is because any simple bivariable association between IPV and a risk or protective factor is likely to be confounded by a number of other variables and may bias results.[24]

Table A4: **Cambridge Quality Checklists[23]**

Correlate score (out of 5): Quality of evidence for variables associated with IPV	
Sampling	
1	Total population or random sampling
0	Convenience, case–control, or representative sampling
Participation rates	
1	Response and retention rates ≥70%
0	Response rate <70% or retention rate <70%, *or* not known
Sample size in analysis	
1	Sample size ≥400*
0	Sample size <400
Measure of risk or protective factor	
1	Reliability coefficient ≥.7 and reasonable face validity *or* criterion or convergent validity coefficient ≥.3 *or* more than one instrument or information source used to assess correlate
0	None of the above or not known
Measure of outcome	
2	Validated scale *or* reliability coefficient ≥.7 and reasonable face validity *or* criterion or convergent validity coefficient ≥.3
1	Non-validated inventory of specific violent behaviours *or* more than one instrument or information source used to assess outcome
0	None of the above or not known Including general experiences of violence
Risk/protective factor score (out of 3): Quality of evidence for variables associated with IPV that precede it	
1	Cross-sectional data
2	Retrospective data
3	Prospective data (or study of fixed risk factor)
Causal risk/protective factor score (out of 7): Quality of evidence for variables that, when changed, alter the risk of IPV	
1	Study without variation in the risk or protective factor No analysis of change†
2	Study with variation in the risk/protective factor but confounding is unaccounted for No analysis of change
3	Study without variation in the risk/protective factor With analysis of change
4	Study with variation in the risk/protective factor but confounding is unaccounted for With analysis of change
5	Study with variation in the risk/protective factor and confounding accounted for No analysis of change
6	Study with variation in the risk/protective factor and confounding accounted for With analysis of change
7	Randomised experiment Targeting a risk/protective factor

Adaptations were made as follows: (a) representative sampling was added as a sampling method due to its preponderance in the literature; (b) differential attrition was removed from the participation rates criterion, as this was not useful for a multi-risk and protective factor review; (c) validated scale was added as an option for outcome measures, as most studies use a common validated scale for IPV (the Conflict Tactics Scale) and therefore few studies report validity or reliability checks; (d) Moved 'more than one instrument' to one point below for outcome measure to differentiate between measures of IPV with better psychometric properties and poorer measures that had multiple informants.

*Where analyses were conducted on paired data, the number of couples was used to judge this criterion.

†Analysis of change is defined as balancing on a previous (e.g., baseline) measure of IPV, analysing change scores or lagged associations, or conducting fixed effects analysis. Ideally, change in IPV would be measured before and after exposure to the risk or protective factor. However, this was not the case for most included studies and therefore any change in IPV analysed was counted as an analysis of change.

Table A5: **Conventions applied for data transformations into log odds**

Effect estimate	Transformation of point estimate to B	Transformation of SE or 95% CI to SE(B)	Transformation of p-value to SE(B)	Source
Log odds (unstandardised B)	-	$\frac{B_{upper} - B_{lower}}{(1.96 \times 2)}$	$\frac{B}{ABS(NORMSINV\left(\frac{p-value}{2}\right))}$	Higgins (Section 7.7.7.2)[25]
Odds ratio (OR)	$ln(OR)$	$\frac{SE(OR)}{OR}$ $\frac{ln(OR_{upper}) - ln(OR_{lower})}{(1.96 \times 2)}$	N/A	Sribney[26]
β (standardised)	Online calculator Data: β, SD(outcome), exposed and unexposed group sizes	Online calculator	N/A	Higgins (Section 7.7.7.2), Wilson[25,27]
Risk ratio (RR)	$\frac{ln(RR \times (1 - ACR))}{(1 - (RR \times ACR))}$	$\frac{ln(RR_{upper} \times (1 - ACR))}{(1 - (RR_{upper} \times ACR))} - \frac{ln(RR_{lower} \times (1 - ACR))}{(1 - (RR_{lower} \times ACR))}$ (1.96×2)	N/A	Higgins (Section 12.5.5.4)[25]
Hazard ratio	Treated as RR	Treated as RR	N/A	Herbison[28]
Probit	$probit \times 1.6$	$SE(probit) \times 1.6$	N/A	Train (p. 28)[29]
F-score	Online calculator Data: F-score, exposed and unexposed group sizes	Online calculator	N/A	Wilson[27]

All transformations were entered as shown into Microsoft Excel, except where otherwise noted. SE is standard error. 95% CI is 95% confidence interval. SD is standard deviation. ACR is assumed control risk (i.e., prevalence of IPV events in the referent group). N/A is 'not applicable' and indicates where the transformation was not required for any included study.

Table A6: **Quality assessment of 60 included studies**

First author, year	Sampling	Participation rate	Sample size	Measure of factor*	Measure of outcome	Analysis of change	Correlate score /6	Risk/protective factor score /3	Causal score /7	Total score /16	% Total score
Add Health (USA)											
Brown (2008)[30]	0	0	1	0.7	2	0	3.7	3	5	11.7	72.9%
Exner-Cortens (2013)[31]	0	0	1	0.4	1	1	2.4	3	6	11.4	71.3%
Fang (2007)[32]	0	0	1	0.7	1	0	2.7	3	5	10.7	66.7%
Kuhl (2015)[33]	0	0	0†	0.0	2	0	2.0	3	5	10.0	62.5%
Lehrer (2006)[34]	0	0	1	1.0	1	1	3.0	3	6	12.0	75.0%
Nowotny (2013)[35]	0	0	1	0.0	1	1	2.0	3	6	11.0	68.8%
Turay (2014)[36]	0	0	1	0.8	1	0	2.8	3	5	10.8	67.2%
Renner (2012)[37]	0	0	1	0.5	1	0	2.5	3	5	10.5	65.6%
Add Health and 1990 Census (USA)											
Gomez (2011)[38]	0	0	1	0.8	1	1	2.8	3	6	11.8	73.8%
Adult Development Study (USA)											
Smith (2014)[39]	0	1	0†	0.7	2	1	3.7	3	6	12.7	79.5%
Schumacher (2008)[40]	0	0	1	0.7	2	1	3.7	3	6	12.7	79.2%
Buffalo Newlywed Study (USA)											
Leonard (1996)[41]	0	0	1	0.5	2	1	3.5	3	6	12.5	77.9%
Leonard (1998)[42]	0	0	1	0.6	2	0	3.6	3	5	11.6	72.7%
Quigley (2000)[43]	0	0	1	1.0	2	1	4.0	3	6	13.0	81.3%
Cambridge Study in Delinquent Development (UK)											
Theobald (2012)[44]	1	0	0	0.0	2	0	3.0	3	5	11.0	68.8%
Child Development Project (USA)											
Lansford (2007)[45]	1	0	0	0.5	1	0	2.5	3	5	10.5	65.6%
Christchurch Health and Development Study (New Zealand)											

First author, year	Sampling	Participation rate	Sample size	Measure of factor*	Measure of outcome	Analysis of change	Correlate score /6	Risk/protective factor score /3	Causal score /7	Total score /16	% Total score
Fergusson (2008)[46]	1	0	1	1.0	2	0	5.0	3	5	13.0	81.3%
Fergusson (1986)[47]	1	0	1	0.7	0	0	2.7	3	5	10.7	66.7%
McLeod (2014)[48]	1	0	1	0.8	2	0	4.8	3	5	12.8	79.7%
Colorado Behavioural Risk Factor Surveillance System (USA)											
Koziol-McLain (2001)[49]	0	0	0	1.0	2	1	3.0	3	6	12.0	75.0%
Dunedin Multidisciplinary Health and Development Study (New Zealand)											
Magdol (1998)[50]	1	1	0	1.0	2	0	5.0	3	5	13.0	81.3%
Fragile Families and Child Wellbeing Study (USA)											
Huang (2010)[51]	1	0	1	0.5	1	1	3.5	3	6	12.5	78.1%
Illinois Families Study (USA)											
Staggs (2007)[52]	1	0	1	0.0	2	1	4.0	3	6	13.0	81.3%
Iowa Youth and Families Project and Family Transitions Project (USA)											
Cui (2010)[53]	0	1	0	1.0	2	0	4.0	3	5	12.0	75.0%
Korean Welfare Panel Study (South Korea)											
Kim (2013)[54]	1	0	1	1.0	0	1	3.0	3	6	12.0	75.0%
National Crime Victimisation Survey (USA)											
Waltermaurer (2007)[55]	1	0	1	0.0	0	0	2.0	3	5	10.0	62.5%
National Epidemiologic Survey on Alcohol and Related Conditions (USA)											
Hahn (2014)[56]	0	1	1	0.7	2	0	4.7	3	5	12.7	79.5%
National Family Health Survey (NFHS-2) and follow up rural survey (India)											
Bourey (2013)[57]	1	1	1	0.3	1	1	4.3	3	6	13.3	82.9%
Sabarwal (2014)[58]	1	1	1	0.0	1	1	4.0	3	6	13.0	81.3%
National Survey of Families and Households											
Jasinski (2001)[59]	1	0	1	1.0	1	1	4.0	3	6	13.0	81.3%
National Survey of Families and Households and 1990 Census (USA)											
Benson (2003)[60]	1	0	1	0.5	1	1	3.5	3	6	12.5	78.1%
Fox (2002)[61]	1	0	1	0.0	1	1	3.0	3	6	12.0	75.0%
Lanier (2009)[62]	1	0	1	1.0	1	1	4.0	3	6	13.0	81.3%
National Women's Study (USA)											
Cougle (2009)[63]	1	1	1	0.7	1	1	4.7	3	6	13.7	85.4%
National Youth Survey Family Study (USA)											
Menard (2014)[64]	1	1	0	0.3	2	0	4.3	3	5	12.3	77.1%
Mihalic (1997)[65]	1	1	0	1.0	2	0	5.0	3	5	13.0	81.3%
Oregon Youth Study (USA)											
Capaldi (2001)[66]	1	0	0	1.0	2	0	4.0	3	5	12.0	75.0%
Kerr (2011)[67]	1	0	0	0.7	2	1	3.7	3	6	12.7	79.5%
Kim (2008)[68]	1	1	0	1.0	2	0	5.0	3	5	13.0	81.3%
Project HOW: Health Outcomes of Women (USA)											
Temple (2008)[69]	0	0	1	0.0	2	1	3.0	3	6	12.0	75.0%
Project on Human Development in Chicago Neighborhoods and 1990 Census (USA)											
Jain (2010)[70]	1	0	0	0.8	2	0	3.8	3	5	11.8	74.0%
Rakai Community Cohort Study (Uganda)											
Kouyoumdjian (2013)[71]	0	0	1	0.4	1	0	2.4	3	5	10.4	65.2%
RAND Adolescent/Young Adult Panel Study (USA)											
Martino (2005)[72]	0	1	1	0.0	0	1	2.0	3	6	11.0	68.8%
Reach for Health Study (USA)											

First author, year	Sampling	Participation rate	Sample size	Measure of factor*	Measure of outcome	Analysis of change	Correlate score /6	Risk/protective factor score /3	Causal score /7	Total score /16	% Total score
O'Donnell (2009)[73]	1	1	1	0.6	2	0	5.6	3	5	13.6	85.0%
Rutgers Health and Human Development Project (USA)											
Chen (2004)[74]	1	0	0	0.8	1	0	2.8	3	5	10.8	67.5%
Samata Health Study (India)											
Krishnan (2010)[75]	0	0	1	0.5	1	0	2.5	3	5	10.5	65.6%
Stepping Stones (South Africa)											
Nduna (2010)[76]	0	0	1	1.0	2	1	4.0	3	6	13.0	81.3%
Toledo Adolescent Relationships Study (USA)											
Giordano (2016)[77]	1	0	1	0.7	2	0	4.7	3	5	12.7	79.5%
Kaufman (2015)[78]	1	0	1	0.9	1	1	3.9	3	6	12.9	80.5%
Women 2000 (USA)											
Testa (2003)[79]	0	0	1	0.7	2	1	3.7	3	6	12.7	79.2%
Testa (2007)[80]	1	1	1	0.8	2	1	5.8	3	6	14.8	92.4%
Unnamed: New York study of married couples (USA)											
Heyman (1995)[81]	0	0	0	0.0	2	0	2.0	3	5	10.0	62.5%
MacEwan (1988)[82]	0	0	0	0.3	2	1	2.3	3	6	11.3	70.8%
O'Leary (1994)[83]	0	0	0	0.0	2	0	2.0	3	5	10.0	62.5%
Unnamed: National study of married and cohabiting couples (USA)											
Caetano (2008)[84]	1	1	0	0.0	2	1	4.0	3	6	13.0	81.3%
Caetano (2005)[85]	1	1	1	0.4	2	1	5.4	3	6	14.4	90.2%
Field (2003)[86]	1	1	0	1.0	2	1	5.0	3	6	14.0	87.5%
Unnamed: 56-day diary study of married and cohabiting couples (USA)											
Testa (2014)[87]	0	0	0	0.2	1	1	1.2	3	6	10.2	63.8%
Unnamed: Quebecois study of women beginning in adolescence (Canada)											
Vezina (2015)[88]	1	0	1	0.5	2	1	4.5	3	6	13.5	84.4%
Unnamed: Prospective case-control study of abused and non-abused children (USA)											
Widom (2014)[89]	0	0	0	1.0	2	0	3.0	3	5	11.0	68.8%

*The measure used for each risk/protective factor investigated was appraised separately within each study. The ratings given here are based on the average of these ratings, when applicable.
†Sample size for women not reported.

Table A7: **Outcomes measured in 60 included studies**

First Author, Year	IPV Type	Informant	Measure	Percentage or Mean (SD)	Outcome type*	Time frame†	Follow-up age: Range or M (SD)
Add Health (USA)							
Brown (2008)[30]	Physical	Self-report	Partner threatened violence, pushed or shoved, or threw something that could hurt; or slapped, hit, or kicked	Dating: 11%; Cohabiting: 27%; Married: 18%	Prevalence	Last year	18-28
Exner-Cortens (2013)[31]	Physical	Self-report	Partner threatened violence, pushed or shoved, or threw something that could hurt; or slapped, hit, or kicked	Not reported	Prevalence	Last year	18-25
Fang (2007)[32]	Physical and/or sexual	Self-report	Partner threatened violence, pushed or shoved, or threw something that could hurt; or slapped, hit, or kicked	27.8%	Prevalence	1-3 years	18-26
Kuhl (2015)[33]	Physical	Self-report	Revised CTS	Victim only: 11.1% Bidirectional: 19.3%	Prevalence	Last year at Waves 3 and 4	24-32
Lehrer (2006)[34]	Physical	Self-report	Partner slapped, hit, or kicked; or injury because of fight with partner	18.6%	Prevalence	Last year	18-27
Nowotny (2013)[35]	Physical	Self-report	Partner threatened violence, pushed or shoved, threw something that could hurt	18.1%	Prevalence	Last year	28.9 (1.7)‡
	Physical	Self-report	Partner slapped, hit, or kicked	8.8%	Prevalence	Last year	
	Sexual	Self-report	Partner forced sexual relations	5.9%	Prevalence	Last year	
	Physical	Self-report	Injury because of fight with partner	4.9%	Prevalence	Last year	
Turay (2014)[36]	Physical and/or sexual	Self-report	Partner threatened violence, pushed or shoved, threw something that could hurt; or slapped, hit, or kicked; or forced sexual relations	20.0%	Prevalence	Last year	18-26
Renner (2012)[37]	Physical and/or sexual	Self-report	Partner threatened violence, pushed or shoved, threw something that could hurt; or slapped, hit, or kicked; or forced sexual relations; or caused injury	Victim only: 12.1%; Bidirectional: 27.4%	Prevalence	Last year	18-27
Add Health and 1990 Census (USA)							
Gomez (2011)[38]	Physical and/or sexual	Self-report	Partner threatened violence, pushed or shoved, threw something that could hurt; or slapped, hit, or kicked; or forced sexual relations; or caused injury	27.4%	Prevalence	Last 4 years	22-25+
Adult Development Study (USA)							
Smith (2014)[39]	Physical	Partner and self-report	Revised CTS	5.1 (14.4)	Prevalence	Last year at Waves 1-6	30.8 (5.8)‡§
Schumacher (2008)[40]	Physical	Partner and self-report	Revised CTS	25.5%-38.2% (over 4 waves)	Prevalence	Last year at Waves 1-4	35.8 (5.8)‡§
Buffalo Newlywed Study (USA)							
Leonard (1996)[41]	Physical	Partner and self-report	1=Any IPV, 0=none: Modified version of CTS, which included moderate (e.g., 'push, grab, or shove' 'slap') to severe items (e.g., 'hit with fist,' 'beat up') but not the very severe items (e.g., use of weapons)	29.0%	Prevalence	Last year	24.3 (3.1)‡§
Leonard (1998)[42]	Physical	Partner and self-report	1=Any IPV, 0=none: Modified version of CTS, which included moderate to severe items but not the very severe items	28.0%	Prevalence	Last year	24.3 (3.1)‡§
Quigley (2000)[43]	Physical	Partner and self-report	1=Any IPV, 0=none: Modified version of CTS, which included moderate to severe items but not the very severe items	45.2%	Prevalence	Last 2 years	26.3 (3.3)‡§
Cambridge Study in Delinquent Development (UK)							
Theobald (2012)[44]	Physical	Self-report	CTS, 1=at least one of: slapping, shaking, throwing an object at, kicking, biting or hitting with a fist, hitting with an object, twisting arms, throwing bodily, beating up (multiple blows), choking or strangling, or threatening with a knife or gun; 0=none	17.5%	Prevalence	Last 5 years	48¶
Child Development Project (USA)							

First Author, Year	IPV Type	Informant	Measure	Percentage or Mean (SD)	Outcome type*	Time frame†	Follow-up age: Range or M (SD)
Lansford (2007)[45]	Physical	Self-report	Shortened CTS: Average of 3 items: whether partner had threatened to throw something; pushed, shoved, or grabbed; or hit respondent	0.1 (SD not reported)	Prevalence	Last year	21
Christchurch Health and Development Study (New Zealand)							
Fergusson (2008)[46]	Physical, sexual, and/or psychological	Self-report	Revised CTS: Scored > 1 on victimization scale	66.0%	Prevalence	Last year	<26-41+‡
Fergusson (1986)[47]	Physical	Self-report	Self-reported assault by husband in the 6-year periodx	8.5%	Prevalence	Last year, at Waves 4-9	24-25
McLeod (2014)[48]	Physical	Self-report	Revised CTS: Exposure to any of 18 acts of physical violence or threats.	For each item: 0.1-0.8 (SD not reported)	Prevalence	Last year	30
Colorado Behavioural Risk Factor Surveillance System (USA)							
Koziol-McLain (2001)[49]	Physical	Self-report	CTS: 1=1 or more episodes of physical or severe physical violence by a current or former partner, 0=none	4.0%	Prevalence	Last 3-5 months	18-93
Dunedin Multidisciplinary Health and Development Study (New Zealand)							
Magdol (1998)[50]	Physical	Self-report	Sum of all 9 physical violence items in the CTS plus 4 additional items that capture other physically abusive behaviors	Not reported	Prevalence	Last year	21
	Psychological	Self-report	Sum of 2 items from the CTS and 18 additional items that capture controlling, terrorizing, demeaning, and other psychologically abusive behaviors items	Not reported	Prevalence	Last year	
Fragile Families and Child Wellbeing Study (USA)							
Huang (2010)[51]	Physical, sexual, and/or psychological	Self-report	1=Any of the following, 0=none: Partner slapped, kicked, or hit respondent; hit her with his fist or a dangerous object; tried to isolate her from family and friends; tried to prevent her from going to work or school; withheld money; made her ask for money; took her money; or tried to make her have sex or do sexual things she didn't want	20.2%	Prevalence	Current frequency or last month if no longer with study child's father	<26-32+‡
Illinois Families Study (USA)							
Staggs (2007)[52]	Physical, sexual, and/or psychological	Self-report	Continuous index of items from the Mother's Survey (Allard, Albelda, Colten, & Cosenza, 1997)	0.3 (SD not reported)	Prevalence	Last year	21-61‡
Iowa Youth and Families Project and Family Transitions Project (USA)							
Cui (2010)[53]	Physical	Self-report and observer	Latent variable based on: observer report of spouse/partner physical aggression and respondent's report of how often partner got into a fight with respondent, or hit, pushed, grabbed, or shoved her	Not reported	Prevalence	Last month at Waves 6-8	32
	Psychological	Self-report and observer	Latent variable based on: observer report of spouse/partner verbal aggression and respondent's report of how often partner shouted, yelled, insulted, or swore at respondent, called her bad names, or threatened to hurt her	Not reported	Prevalence	Last month at Waves 6-8	
Korean Welfare Panel Study (South Korea)							
Kim (2013)[54]	Physical	Self-report	Experienced the act or threat of 'physical violence'	4.9%	Prevalence	Last year	51.5 (14.8) ‡
National Crime Victimization Survey (USA)							
Waltermaurer (2007)[55]	Physical and/or sexual	Self-report	Any reported violent crime incidents, burglaries (completed or attempted), forcible entries (completed or attempted), or threatened, attempted, or completed physical or sexual assault perpetrated by a spouse, ex-spouse or boyfriend, or ex-boyfriend.	1.5%	Prevalence	Last 6 months at Waves 1 and 2 (Last 12 months)	18-44‡
National Epidemiologic Survey on Alcohol and Related Conditions (USA)							

First Author, Year	IPV Type	Informant	Measure	Percentage or Mean (SD)	Outcome type*	Time frame†	Follow-up age: Range or M (SD)
Hahn (2014)[56]	Physical and/or sexual	Self-report	CTS: Any experience of IPV	4.4%	Prevalence	Last year	22-69+‡§
National Family Health Survey (NFHS-2) and follow up rural survey (India)							
Bourey (2013)[57]	Physical	Self-report	Husband perpetrated any of the following: pushed, pulled or held her down, hit her with his fist or did something that could cause injury, kicked or dragged her, tried to strangle or burn her, or attacked her with a knife, gun, or other weapon	16.4%	Incidence	Last year	20-43‡
Sabarwal (2014)[58]	Physical	Self-report	Husband perpetrated any of the following: pushed, pulled or held her down, hit her with his fist or did something that could cause injury, kicked or dragged her, tried to strangle or burn her, or attacked her with a knife, gun, or other weapon	24.0%	Prevalence	Last year	19-43
National Survey of Families and Households							
Jasinski (2001)[59]	Physical	Partner and self-report	Disagreement or argument ended with the husband hitting, shoving, or throwing things at the wife	5.1%	Incidence	Last year	25-56‡§
National Survey of Families and Households and 1990 Census (USA)							
Benson (2003)[60]	Physical	Partner and self-report	Disagreement or argument ended with the husband hitting, shoving, or throwing things at the wife	4.2%	Prevalence	Last year	47.5 (14.2)‡
Fox (2002)[61]	Physical	Partner and self-report	Disagreement or argument ended with the husband hitting, shoving, or throwing things at the wife	4.2%	Prevalence	Last year	41.2 (SD not reported)
Lanier (2009)[62]	Physical	Partner and self-report	Disagreement or argument ended with the husband hitting, shoving, or throwing things at the wife	5.1-5.9%	Prevalence	Last year	Urban: 45.5 (14.0)‡ Rural 47.4 (16.7)‡
National Women's Study (USA)							
Cougle (2009)[63]	Physical and/or sexual	Self-report	1=Any of the following; 0=none. Respondent was forced or threatened into having sex; forced to have oral or anal sex or had objects put in her vagina or anus against her will; molested or forced to touch penis; in a situation where someone attempted to force her into unwanted sexual contact; was attacked with or without a weapon with the intent to kill or seriously injure her. Assaults were coded by perpetrator, with intimate partner defined as husband/ex-husband or boyfriend/ex-boyfriend.	1.7%	Incidence	Last year at Wave 2 or 3	20-91‡
National Youth Survey Family Study (USA)							
Menard (2014)[64]	Physical and/or psychological	Self-report	CTS: Sum of items for general victimization (minor and severe)	0.9 (4.2)	Prevalence	Last year	37-43
Mihalic (1997)[65]	Physical and/or psychological	Self-report	CTS: Sum of 4 items for severe victimization	White: 0.5, Non-white: 0.8 (SD not reported)	Prevalence	Last year at Waves 6-8	24-28‡
Oregon Youth Study (USA)							
Capaldi (2001)[66]	Physical and/or psychological	Partner and self-report, observation	Mean of six standardized indicators of aggression at the three waves: Adjustment with Partner Scale (Kessler, 1990), Conflict Tactics Scale, Dyadic Social Skills Questionnaire (Capaldi, 1994), Partner Interaction Questionnaire (Capaldi, 1991), Couples Interviews, and observational measures of men's Physical and/or psychological aggression toward their partner during discussion tasks (coded by trained, professional research assistants).	Not reported	Prevalence	Current partner at Waves 4-6	16-42§ (M=20.8)

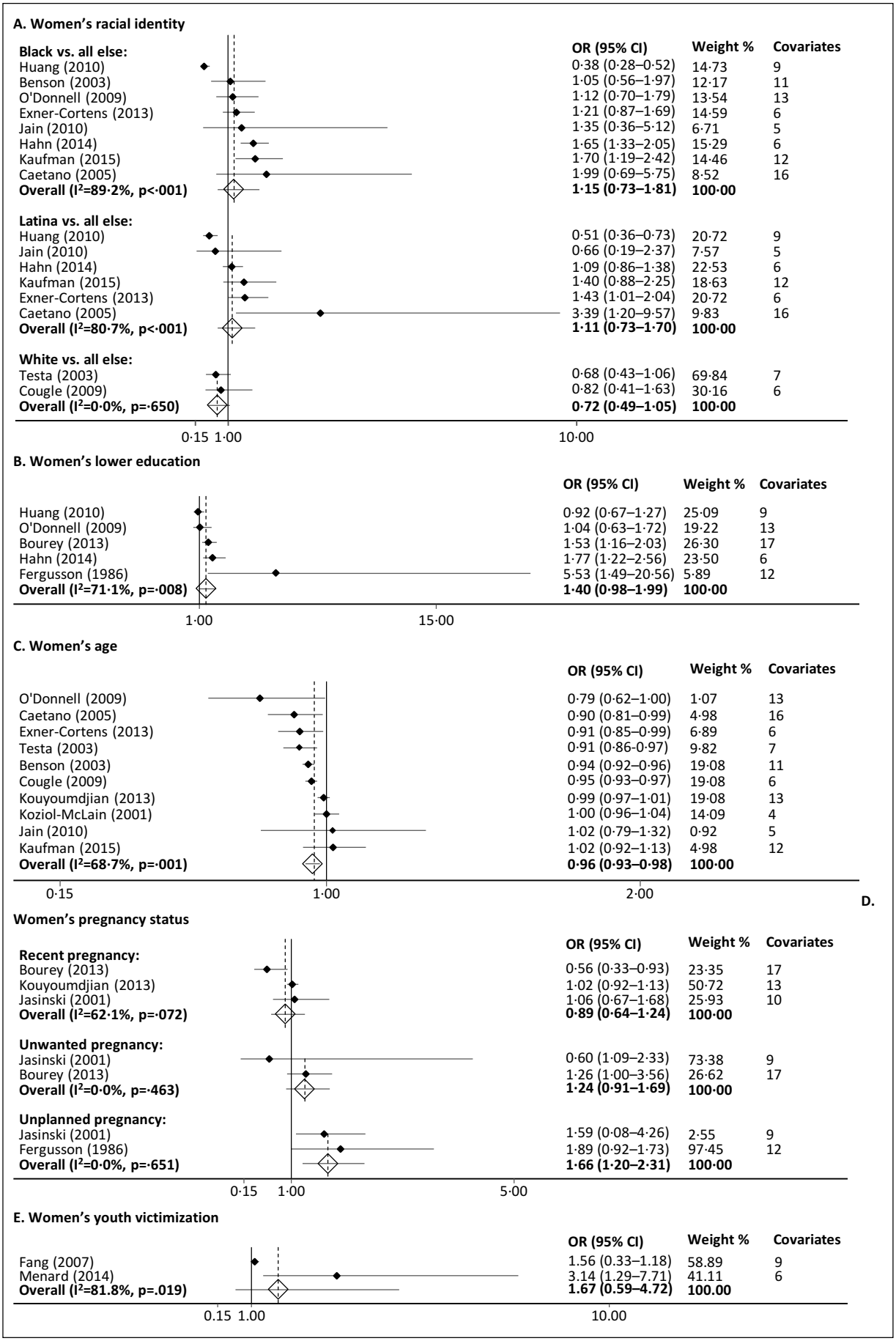
First Author, Year	IPV Type	Informant	Measure	Percentage or Mean (SD)	Outcome type*	Time frame†	Follow-up age: Range or M (SD)
Kerr (2011)[67]	Physical and/or psychological	Partner and self-report	Mean of six standardized indicators of aggression at the three waves: Adjustment with Partner Scale (Kessler, 1990), CTS, Dyadic Social Skills Questionnaire (Capaldi, 1994), Partner Interaction Questionnaire (Capaldi, 1991), Couples Interviews, and observational measures of men's Physical and/or psychological aggression toward their partner during discussion tasks.	Not reported	Prevalence	Current partner at Waves 14-16	23.1 (SD not reported)§
Kim (2008)[68]	Physical	Partner and self-report, observation	Mean of standardized indicators: Adjustment with Partner (Kessler, 1990); Dyadic Social Skills Questionnaire (Capaldi, 1994); CTS (Straus, 1979); observational measures of men's physical aggression toward their partner during discussion tasks.	7.0%	Prevalence	Current partner at Waves 1-5	28.0 (4.4)§
	Psychological	Partner and self-report, observation	Mean of standardized indicators: Dyadic Social Skills Questionnaire (Capaldi, 1994); CTS (Straus, 1979); observational measures of men's psychological aggression toward their partner during discussion tasks.	76.0%	Prevalence	Current partner at Waves 1-5	
Project HOW: Health Outcomes of Women (USA)							
Temple (2008)[69]	Physical and/or sexual	Self-report	The Severity of Violence Against Women Scale (SVAWS; Marshall, 1992) measured physical violence (20 items) and sexual aggression (6 items).	Not reported	Prevalence	Since start of relationship at Wave 1; Since last interview at Waves 2, 3, 5, and 6	28-57‡
Project on Human Development in Chicago Neighborhoods and 1990 Census (USA)							
Jain (2010)[70]	Physical	Self-report	Revised CTS: 1 or more acts of abuse constituted victimization	24.2%	Prevalence	Last year	18-25
Rakai Community Cohort Study (Uganda)							
Kouyoumdjian (2013)[71]	Physical, sexual, and/or psychological	Self-report	Any of the following, modified from the revised CTS: husband or partner 'pushed you, pulled you, slapped you or held you down,' 'punched you with a fist or with something that could hurt you,' 'kicked you or dragged you,' 'tried to strangle you or burn you,' 'attacked you with a knife, gun or other weapon,' 'verbally abused or shouted at you', 'threatened you with a knife, gun, or other weapon,' 'used verbal threats to force you to have sex when you did not want to,' 'physically forced you to have sex when you did not want to,' or 'forced you to perform other sexual acts you did not want to do'.	29.0%	Prevalence	Current year at Waves 1-7	24-58‡
RAND Adolescent/Young Adult Panel Study (USA)							
Martino (2005)[72]	Physical	Self-report	1=spouse or live-in partner hit or beat up respondent, 0=otherwise	18.0%	Prevalence	Last 3 years	29.4 (SD not reported)
Reach for Health Study (USA)							
O'Donnell (2009)[73]	Physical, sexual, and/or psychological	Self-report	Any of the following occurrences of violence=1, none=0. Sexual or dating partner hit, punched or slapped respondent; threw something at her or hit her with an object; pushed, grabbed or shoved her; pulled her hair; scratched or bit her; kicked or choked her; threatened her with a knife; threatened her with a gun; used a knife against her; used a gun against her; forced or threatened to hurt her to have oral, anal, or vaginal sex; made her family or friends worry about her; threatened to hurt her or someone she cared about; threatened to hurt himself or the respondent if she tried to leave him or break up with	28.5%	Prevalence	Last year	22-25

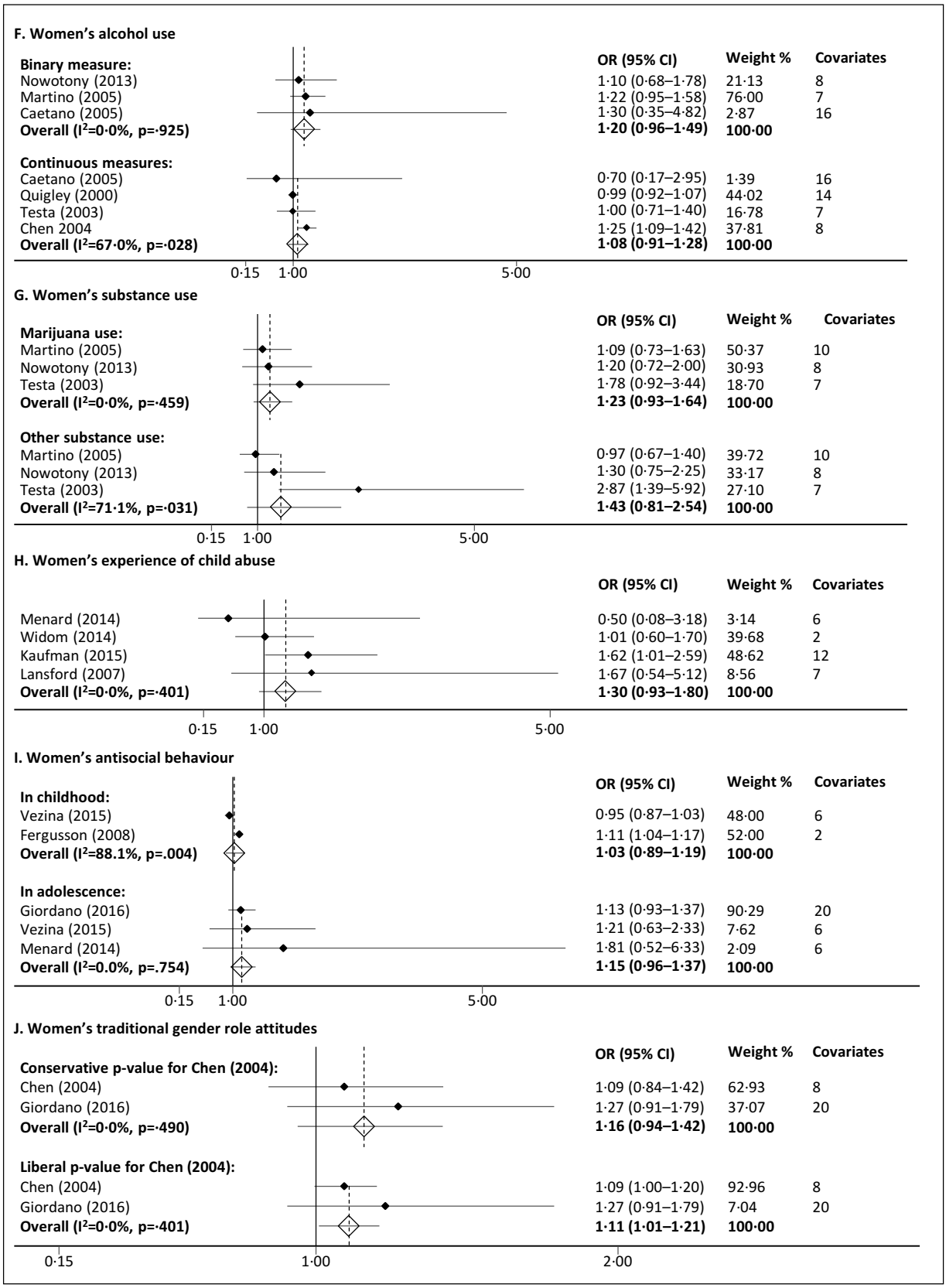
First Author, Year	IPV Type	Informant	Measure	Percentage or Mean (SD)	Outcome type*	Time frame†	Follow-up age: Range or M (SD)
			him; or had been jealous or possessive of the respondent, had checked up on her, or had refused to let her go out with friends.				
Rutgers Health and Human Development Project (USA)							
Chen (2004)[74]	Physical	Self-report	Interval scale of the total number of times that partner hit, shoved, or threw objects at respondent; cut, bruised, or seriously injured respondent; or threatened respondent with a weapon.	Being hit, shoved, thrown at: 0.3 (0.9) Being cut, bruised, injured: 0.1 (0.3) Being threatened with weapon: 0.02 (0.2)	Prevalence	Last year	28-31
Samata Health Study (India)							
Krishnan (2010)[75]	Physical	Self-report	1=partner had hit, kicked, or beaten respondent; 0=otherwise		Prevalence	Last 6 months at Waves 2 and 3	18-27‡
Stepping Stones (South Africa)							
Nduna (2010)[76]	Physical and/or sexual	Self-report	WHO Multi-Country Study on Women's Health and Domestic Violence scale: 0=0 or 1 episodes of physical or sexual abuse; 1=2 or more episodes	21.0%	Prevalence	Last year	16-27‡
Toledo Adolescent Relationships Study (USA)							
Giordano (2016)[77]	Physical	Self-report	Revised CTS: 1=Any IPV victimization, 0=none	Not reported	Prevalence	Current or latest relationship	22-29
Kaufman (2015)[78]	Physical	Self-report	During this relationship, how many times has partner: thrown something at you; pushed, shoved, or grabbed you; slapped you in the face or head with an open hand; or hit you?' 1=Any IPV victimization; 0=none	18.2%	Prevalence	Last year	22-29
Women 2000 (USA)							
Testa (2003)[79]	Physical and/or psychological	Self-report	Revised CTS: 1=experienced severe violence from Wave 1 current partner; 0=did not	12.2%	Prevalence	Last year	21-33‡
Testa (2007)[80]	Sexual	Self-report	1=Any IPV; 0=none: 11-item modified version of the Sexual Experiences Survey (SES) (Testa, VanZile-Tamsen, Livingston, & Koss, 2004). Intimate partners were defined as a boyfriend/dating partner, husband, ex-boyfriend, or ex-husband.	12.1%	Prevalence	Last 2 years	20-32‡
Unnamed: New York study of married couples (USA)							
Heyman (1995)[81]	Physical	Partner and self-report	CTS: 1=Couples reported two or more acts of moderate physical aggression or one or more severe aggression; 0=otherwise	32.4%	Prevalence	Last year	26.1 (SD not reported)‡§
MacEwan (1988)[82]	Physical	Partner and self-report	CTS: Average of couples' reports of 3-point ratings (nonoccurrence, single occurrence, and multiple occurrence) on each physical aggression item. Item scores were standardized to weight items for severity (so that more severe behaviors that occur less frequently received a greater weighting than less severe behaviors that occur more frequently) and then summed.	Male report of aggression: 0.9%-31.6%)	Prevalence	Last year	19.5-37.5‡§
O'Leary (1994)[83]	Physical	Partner and self-report	CTS: Average of couples' reports on each physical aggression item. Item scores were standardized and then summed.	Not reported	Prevalence	Last year	26.1 (SD not reported)‡§
	Psychological	Partner and self-report	Spouse-Specific Aggression Scale (O'Leary & Curley, 1986)	Not reported	Prevalence	Last year	
Unnamed: National study of married and cohabiting couples (USA)							

First Author, Year	IPV Type	Informant	Measure	Percentage or Mean (SD)	Outcome type*	Time frame†	Follow-up age: Range or M (SD)
Caetano (2008)[84]	Physical and/or sexual	Partner and self-report	Revised CTS: Rates for each of 11 items	0-7%	Prevalence	Last year	49.6 (0.7)‡§
Caetano (2005)[85]	Physical and/or sexual	Partner and self-report	Revised CTS: IPV at Wave 2 (2000) but not Wave 1 (1995) vs. No IPV at either wave	5.7%	Incidence	Last year	51.1 (14.1)‡§
Field (2003)[86]	Physical and/or sexual	Partner and self-report	Revised CTS: 1=IPV reported at Wave 2, 0=otherwise	Black couples: 19.9%; White couples: 20.6%	Prevalence	Last year	49.6 (0.7)‡§
Unnamed: 56-day diary study of married and cohabiting couples (USA)							
Testa (2014)[87]	Physical	Partner and self-report	1=either partner reported IPV against woman; 0=no. 'At any time yesterday, did you and your partner have a conflict, argument, or disagreement, whether major or minor?' Participants who responded positively recorded the hour the conflict began and its duration. In addition, they were asked whether or not the conflict involved: throwing things, kicking, or hitting something; being pushed, grabbed, or hit.	18.6%	Prevalence	In a given hour, recorded daily during study period (56 days)	21-46‡§
	Psychological	Partner and self-report	1=either partner reported IPV against woman; 0=no. 'At any time yesterday, did you and your partner have a conflict, argument, or disagreement, whether major or minor?' Participants who responded positively recorded the hour the conflict began and its duration. In addition, they were asked whether or not the conflict involved: yelling; making threats; or being insulted or called names.	Reported for 50.4% of conflicts	Prevalence	In a given hour, recorded daily during study period (56 days)	
Unnamed: Quebecois study of women beginning in adolescence (Canada)							
Vezina (2015)[88]	Psychological	Self-report	IPV reported only in early adulthood only vs. not: Two items from the Violence faite aux Filles dans les Fréquentations à l'Adolescence questinnaire (Lavoie & Vézina, 2001) and two items from the Revised CTS	20.3%	Incidence	Last year	21.2 (0.7)
	Physical and/or sexual	Self-report	IPV reported only in early adulthood only vs. not: Seven items evaluating physical violence and two items referring to sexual violence from the Revised CTS	10.8%	Incidence	Last year	
Unnamed: Prospective case-control study of abused and non-abused children (USA)							
Widom (2014)[89]	Physical, sexual, and/or psychological	Self-report	1=Any IPV, 0=none: 41-item scale for IPV composed of items from the Partner Conflict Tactics Scale (Moffitt et al., 1997), CTS (Straus, 1990), Psychological Maltreatment of Women Inventory (Tolman, 1989), and National Violence against Women survey (Tjaden & Thoennes, 2000).	79.%-80.0%	Prevalence	Last year	20-27

*Incidence is assigned when the outcome measured was the first time IPV was reported in the study period. †Time frame is from last follow-up, unless otherwise noted. ‡Age estimated from baseline report (age at follow-up not reported). §Age reported for women only. ¶Age reported for male partners only.

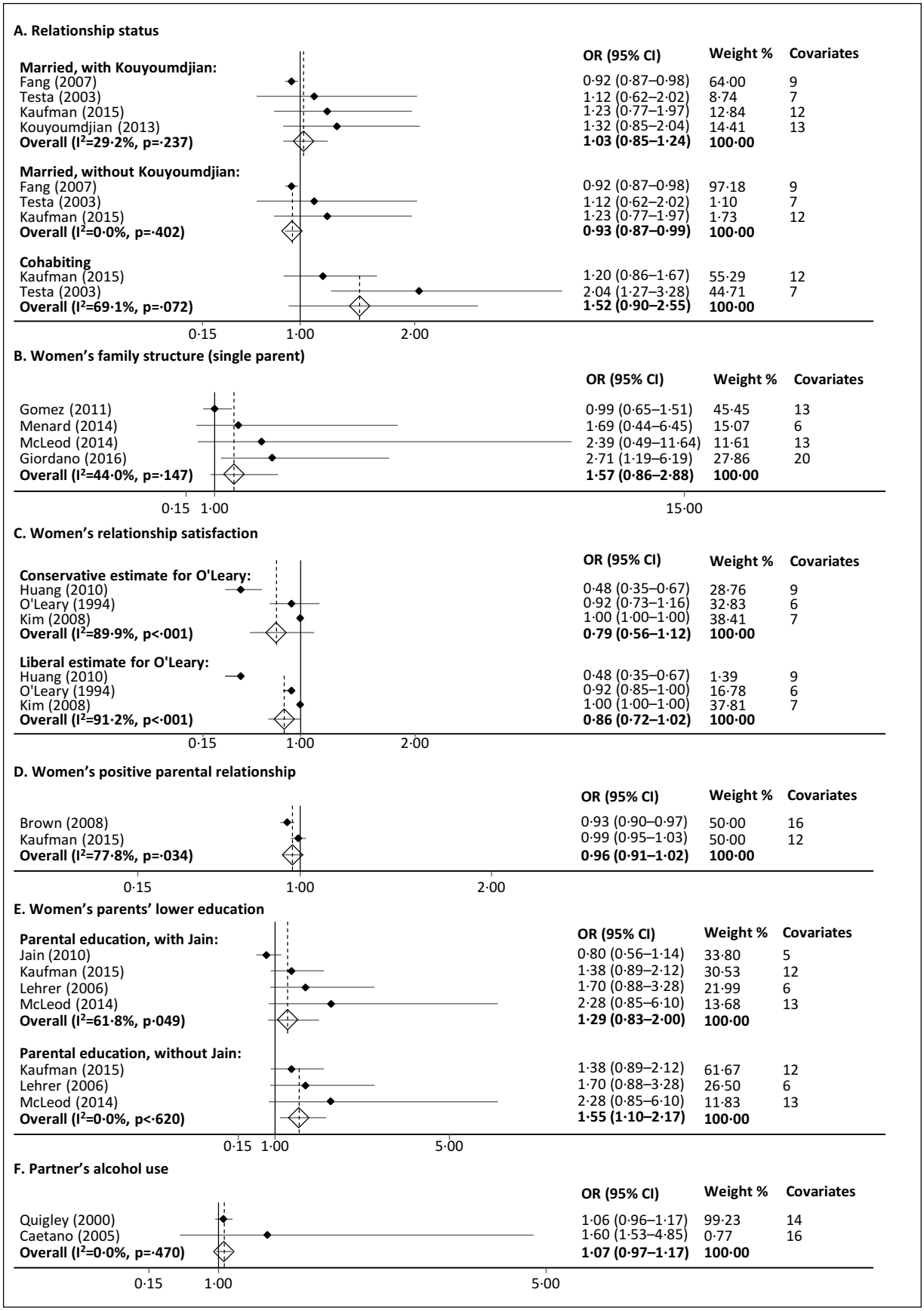
Figure A2: **Meta-analyses of individual factors**

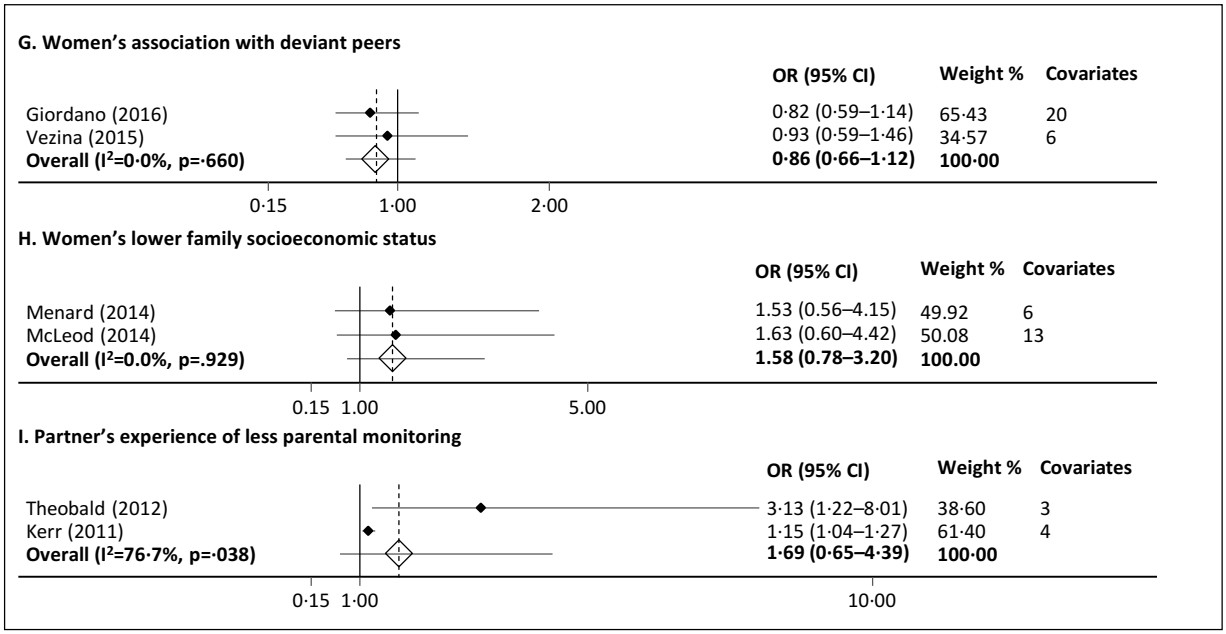




(A) Women's racial identity. (B) Women's lower education. (C) Women's age. (D) Women's pregnancy status. (E) Women's youth victimisation. (F) Women's alcohol use. (G) Women's substance use. (H) Women's experience of child abuse. (I) Women's antisocial behaviour. (J) Women's traditional gender role attitudes, with both a liberal and conservative p-value assumed for Chen et al.,[74] which did not provide exact p-values or precision estimates. OR=odds ratio. 95% CI=95% confidence interval.

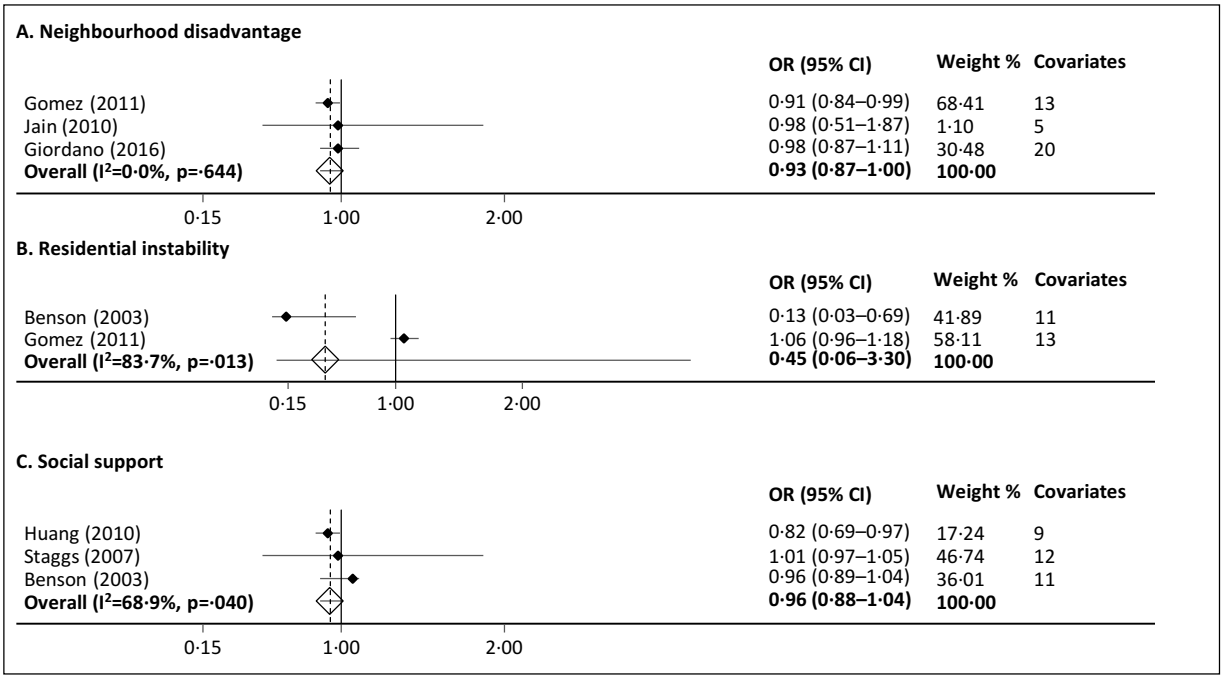
Figure A3: **Meta-analyses of relational factors**





(A) Relationship status, including and excluding Kouyoumdjian et al.,[71] which used a heterogeneous variable (monogamous marriage versus all el se). (B) Women's family structure (single parent household). (C) Women's relationship satisfaction, with both a liberal and conservative p-value assumed for O'Leary et al.,[83] which did not provide exact p-values or precision estimates. (D) Women's positive parental relationship. (E) Women's parents' lower education – all studies examined parents having less than a high school education except Jain et al.,[70] which examined having less than a college education. (F) Partner's alcohol use. (G) Women's association with deviant peers. (H) Women's lower family socioeconomic status. (I) Partner's experience of less parental monitoring. OR=odds ratio. 95% CI=95% confidence interval.

Figure A4: Meta-analyses of community factors



(A) Neighbourhood disadvantage. (B) Residential instability. (C) Social support. OR=odds ratio. 95% CI=95% confidence interval.

The algorithm, based on prior reviews,[90] used to select among multiple effect estimates from a single cohort is shown below. For the harvest plots, when multiple effect estimates remained after applying the algorithm, and at least one of these was statistically significant, the study was depicted as providing evidence for a positive or negative effect as appropriate. As shown, the additional algorithm for the meta-analyses preferentially selected the effect estimate for the most common outcome and/or exposure measure for the sake of comparability.

Figure A5: **Algorithm for selecting among multiple effect estimates from a single cohort**

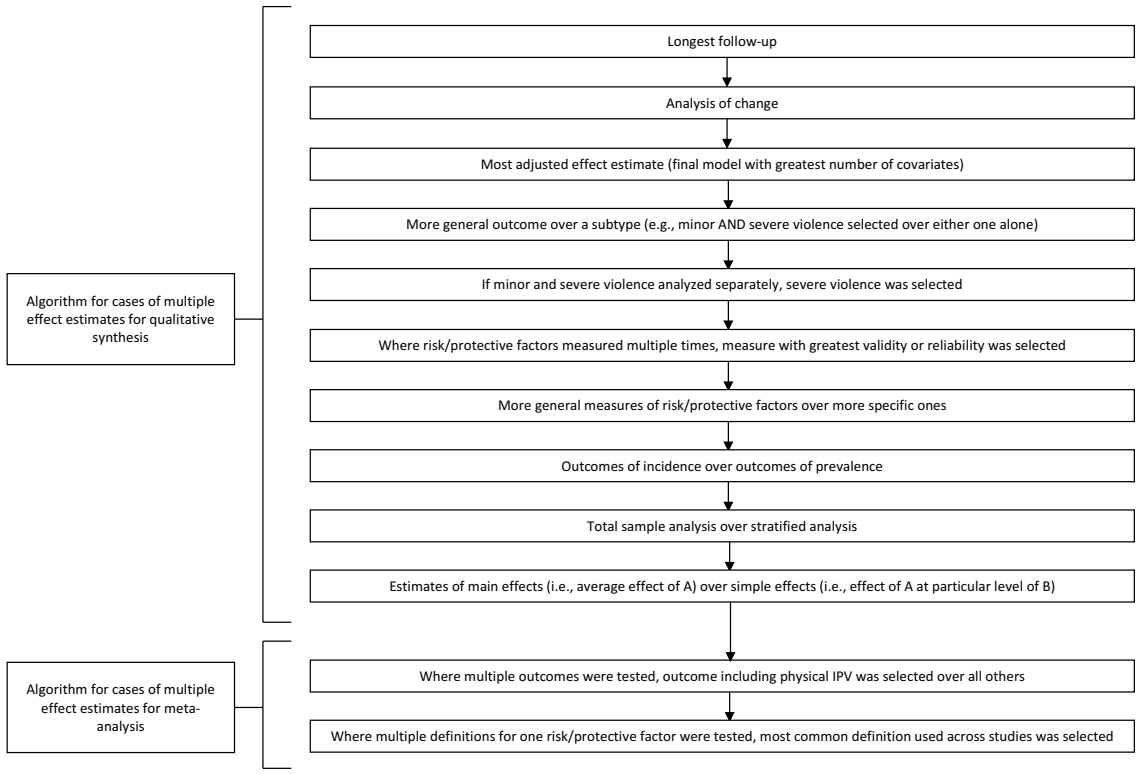
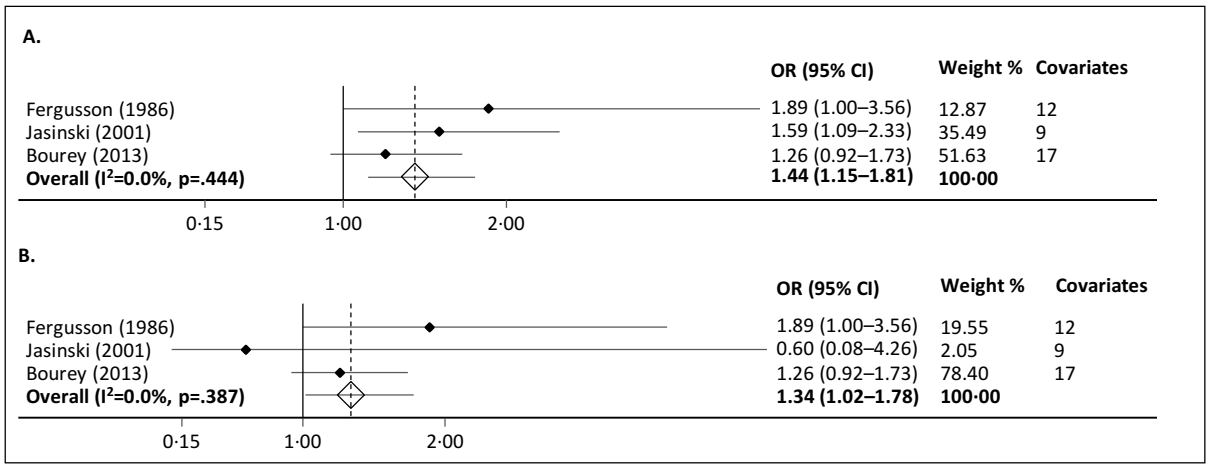


Figure A6: **Sensitivity analyses for unplanned or unwanted pregnancy**



(A) Unplanned pregnancy meta-analysis, with Bourey et al. (2013) effect estimate for 'unwanted pregnancy' included. (B) Unplanned pregnancy meta-analysis, with Bourey et al. (2013) effect estimate for 'unwanted pregnancy' included and Jasinski et al. (2001) effect estimate for 'unplanned pregnancy' substituted with their effect estimate for 'unwanted pregnancy
'. OR=odds ratio. 95% CI=95% confidence interval.

Full results

Results eligible for qualitative analysis from all included studies are included in the following tables, organised by ecological level. For the sake of space, cohorts are identified in the results tables as follows:

Identifier	Cohort sample (Country of origin)
1	Add Health (USA)
2	Add Health and 1990 Census (USA)
3	Adult Development Study (USA)
4	Buffalo Newlywed Study (USA)
5	Cambridge Study in Delinquent Development (UK)
6	Child Development Project (USA)
7	Christchurch Health and Development Study (New Zealand)
8	Colorado Behavioural Risk Factor Surveillance System (USA)
9	Dunedin Multidisciplinary Health and Development Study (New Zealand)
10	Fragile Families and Child Wellbeing Study (USA)
11	Illinois Families Study (USA)
12	Iowa Youth and Families Project and Family Transitions Project (USA)
13	Korean Welfare Panel Study (South Korea)
14	National Crime Victimisation Survey (USA)
15	National Epidemiologic Survey on Alcohol and Related Conditions (USA)
16	National Family Health Survey (NFHS-2) and follow up rural survey (India)
17	National Survey of Families and Households
18	National Women's Study (USA)
19	National Youth Survey Family Study (USA)
20	Oregon Youth Study (USA)
21	Project HOW: Health Outcomes of Women (USA)
22	Project on Human Development in Chicago Neighborhoods and 1990 Census (USA)
23	Rakai Community Cohort Study (Uganda)
24	RAND Adolescent/Young Adult Panel Study (USA)
25	Reach for Health Study (USA)
26	Rutgers Health and Human Development Project (USA)
27	Samata Health Study (India)
28	Stepping Stones (South Africa)
29	Toledo Adolescent Relationships Study (USA)
30	Women 2000 (USA)
31	Unnamed: New York study of married couples (USA)
32	Unnamed: National study of married and cohabiting couples (USA)
33	Unnamed: 56-day diary study of married and cohabiting couples (USA)
34	Unnamed: Quebecois study of women beginning in adolescence (Canada)
35	Unnamed: Prospective case-control study of abused and non-abused children (USA)

Individual factors

Table A8: **Women's race**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Exner-Cortens (2013)	2 816	Ref: White	Self-report	Fixed (Wave 1)	1					6
			Black				OR: 1.21	0.87	1.68	-	
			Hispanic				**OR: 1.44**	**1.01**	**2.06**	-	
			Other				OR: 0.71	0.45	1.13	-	
4	Leonard (1996)	541 couples	**Non-white (ref: White)**	**Self-report**	**Fixed (Wave 1)**	**1**	**β: 0.17**	**-**	**-**	**<.001**	**16**
10	Huang (2010)	2 237	Race (ref white)	Self-report	Fixed (Wave 1)	1					9
			Black				**OR: 0.38**	**0.06**		**<.001**	
			Hispanic				**OR: 0.51**	**0.09**	**-**	**<.001**	
			Other				OR: 0.71	0.27	-	NS	
15	Hahn (2014)	20 089	Ref: White	Self-report	Fixed (Wave 1)	1					6
			Black				**OR: 1.65**	**1.33**	**2.04**	-	
			Latino				OR: 1.09	0.87	1.37	-	
			Asian/Pacific Islander				OR: 0.63	0.35	1.12	-	
			Native American				**OR: 1.69**	**1.05**	**2.73**	-	
17	Benson (2003)	3 006 couples	Ref: White	Self-report	Fixed (Unclear)	1					11
			Black				B: 0.05	0.32	-	NS	
			Other non-White				B: 0.24	0.44	-	NS	
18	Cougle (2009)	2 875	White (ref: non-White)	Self-report	Fixed (Wave 1)	1	OR: 0.82	0.41	1.63	-	6
22	Jain (2010)	352	Ref: White/other	Self-report	Fixed (Wave 1)	1					5
			Non-Hispanic Black				B: 0.30	0.68	-	NS	
			Hispanic				B: -0.41	0.65	-	NS	
25	O'Donnell (2009)	526	Black (ref: other)	Self-report	Fixed (Wave 3)	1	OR: 1.12	0.69	1.79	-	13
29	Kaufman (2015)	507	Ref: White	Self-report	Fixed (Wave 1)	1					12
			Black				**GEE (B): 0.53**	**0.18**	**-**	**<.01**	
			Hispanic				GEE (B): 0.34	0.24	-	NS	
			Other				GEE (B): -1.16	0.70	-	NS	
32	Caetano (2005)	1 136 couples	Ref: White	Self-report	Fixed (Wave 1)	1					16
			Black				OR: 2.00	0.70	5.72	-	
			Hispanic				**OR: 3.40**	**1.18**	**9.54**	-	
			Mixed/other				OR: 0.90	0.33	2.72	-	
30	Testa (2003)	724	White (ref: non-white)	Self-report	Fixed (Wave 1)	1	OR: 0.68	0.44	1.07	-	7
30	Testa (2007)	877	White (ref: other)	Self-report	Fixed (Wave 1)	1	*Multinomial (Ref: none):* IPV only OR: 0.94 IPV + other violence OR: 0.71	IPV only: 0.54 IPV + other violence: 0.24	IPV only: 1.65 IPV + other violence: 2.10	-	10

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A9: **Women's age**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Exner-Cortens (2013)	2 816	**Age**	**Self-report**	**Fixed (Wave 2)**	**1**	**OR: 0.91**	**0.83**	**0.99**	**-**	**6**
8	Koziol-McLain (2001)	405	Age	Self-report	Fixed (Wave 1)	1	OR: 1.00	0.94	1.00	-	4
10	Huang (2010)	2 237	Age (ref: ≥25)	Self-report	Fixed (Wave 1)	1					9
			<20				OR: 1.07	0.18	-	NS	
			20-24 years				OR: 1.01	0.14	-	NS	
15	Hahn (2014)	20 089	Age (ref: 65+)	Self-report	Fixed (Wave 1)	1					6
			18-35				**OR: 3.55**	**2.38**	**5.29**	-	
			35-49				**OR: 3.26**	**2.16**	**4.94**	-	
			50-64				OR: 1.27	0.79	2.02	-	
17	Benson (2003)	3 006 couples	**Age**	**Self-report**	**Fixed (Unclear)**	**1**	**B: -0.06**	**0.01**	**-**	**<.01**	**11**
18	Cougle (2009)	2 875	**Age**	**Self-report**	**Fixed (Wave 1)**	**1**	**OR: 0.95**	**0.92**	**0.97**	**-**	**6**
22	Jain (2010)	352	Age	Self-report	Fixed (Wave 3)	1	B: 0.02	0.13	-	NS	5
23	Kouyoumdjian (2013)	6 916	Age	Self-report	Fixed (Wave 1)	1	OR: 0.99	0.98	1.00	-	13
25	O'Donnell (2009)	526	Age	Self-report	Fixed (Wave 3)	1	OR: 0.79	0.63	1.00	-	13
26	Chen (2004)	359	Age (12, 15, or 18 years)	Self-report	Fixed (Wave 1)	1	B: -0.02	-bet	-	NS	8
29	Kaufman (2015)	507	Age	Self-report	Fixed (Wave 1)	1	GEE (B): 0.02	0.05	-	NS	12
31	MacEwan (1988)	275 couples	Age	Self-report	Fixed (Time 1)	1	β: 0.00	-	-	NS	5
32	Caetano (2005)	1 136 couples	Age	Self-report	Fixed (Wave 1)	1	OR: 0.90	0.88	1.06	-	16
30	Testa (2003)	724	**Age**	**Self-report**	**Fixed (Wave 1)**	**1**	**OR: 0.91**	**0.86**	**0.97**	**-**	**7**
30	Testa (2007)	877	Age	Self-report	Fixed (Wave 1)	1	*Multinomial (Ref: none):* IPV only OR: 0.98 IPV + other violence OR: 0.86	IPV only: 0.91 IPV + other violence: 0.74	IPV only: 1.05 IPV + other violence: 1.00	-	10

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A10: **Women's alcohol use**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Nowotny (2013)	2 959	Binge drinking at least 2 times per month	Self-report	Last year (Wave 1)	0	Physical 1 OR: 1.00 Physical 2 OR: 1.10 Rape OR: 0.70 Injury OR: 1.00	Physical 1: 0.67 Physical 2: 0.70 Rape: 0.30 Injury: 0.53	Physical 1: 1.44 Physical 2: 1.84 Rape: 1.47 Injury: 1.90	-	8
1	Renner (2012)	5 237	**Adolescent problems with alcohol use**	**Self-report**	**Last year (Wave 1)**	**0**	*Multinomial (Ref: none):* Victim only OR: 1.15 **Bidirection OR: 1.30**	Victim only: 0.87 **Bidirection: 1.06**	Victim only: 1.50 **Bidirection: 1.59**	**-**	**10**
3	Schumacher (2008)	634 couples	Alcohol Dependence Scale	Self-report	Unclear (Waves 1-4, lagged with outcome)	1	RRR: 1.02	0.98	1.05	-	12

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
4	Quigley (2000)	414 couples	Standardized sum of Alcohol Dependence Scale and average daily volume of alcohol	Self-report	Last year (Wave 2)	1	B: -0.01	0.04	-	NS	14
21	Temple (2008)	509	**Frequency of alcohol use (categorical)**	**Self-report**	**Unclear (Wave 1)**	**0**	**Physical F: 4.66** Sexual F: 1.34	**-**	**-**	**Physical: <.05** Sexual: NS	**5**
24	Martino (2005)	509	Binge drinking last month	Self-report	Last month (Wave 1)	0	B: 0.20	0.13	-	NS	7
26	Chen (2004)	359	**Rutgers Alcohol Problem Index**	**Self-report**	**Last 3 years (Wave 2)**	**1**	**B: 0.22**	**-**	**-**	**<.001**	**8**
32	Caetano (2005)	1 136 couples	Any one of 14 specific alcohol-related problems	Self-report	Last year (Wave 1)	0	OR: 1.40	0.27	7.27	-	16
			Binge drinking last year	Self-report	Last year (Wave 1)	0	OR: 1.30	0.35	4.81	-	16
			Average alcohol volume per week	Self-report	Last year (Wave 1)	0	OR: 0.70	0.08	1.42	-	16
33	Testa (2014)	118 couples	Total drinking days in the study period	Self-report	56 days	0	Phys OR: 1.00 Psych OR: 0.97	Phys OR: 0.98 Psych OR: 0.92	Phys OR: 1.03 Psych OR: 1.02	-	4
			Alcohol use in a given 4-hour interval	**Self-report**	**56 days**	**0**	**Phys OR: 1.60 Psych OR: 3.27**	**Phys OR: 1.15 Psych OR: 1.16**	**Phys OR: 2.22 Psych OR: 9.27**	**-**	**4**
30	Testa (2003)	724	Mean frequency of binge drinking or becoming drunk or intoxicated	Self-report	Last year (Wave 1)	1	OR: 1.00	0.71	1.40	-	7
30	Testa (2007)	877	Mean frequency of binge drinking or becoming drunk or intoxicated	Self-report	Last year (Wave 1)	1	*Multinomial (Ref: none):* IPV only OR: 0.84 IPV + other OR: 0.90	IPV only: 0.61 IPV + other violence: 0.48	IPV only: 1.16 IPV + other violence: 1.67	-	10

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A11: **Women's marijuana use**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Nowotny (2013)	2 959	Any marijuana use	Self-report	Last year (Wave 1)	0	Physical 1 OR: 0.90 Physical 2 OR: 1.20 Rape OR: 0.70 Injury OR: 0.90	Physical 1: 0.57 Physical 2: 0.71 Rape: 0.32 Injury: 0.43	Physical 1: 1.29 Physical 2: 1.98 Rape: 1.31 Injury: 1.75	-	8
3	Smith (2014)	Not reported	**Frequency of marijuana use per month**	**Self-report**	**Last year (Waves 1-6)**	**0**	**IRR: 0.94**	**0.90**	**0.99**	**-**	**10**
24	Martino (2005)	509	Any marijuana use	Self-report	Last month (Wave 1)	0	B: 0.087	0.205	-	NS	7
30	Testa (2003)	724	Marijuana but not 'hard drug' use	Self-report	Last year (Wave 1)	0	OR: 1.78	0.92	3.44	-	7

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. IRR is incidence rate ratio. Variables are bolded where p<.05.

Table A12: **Women's substance use**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Nowotny (2013)	2 959	Any illicit drug use	Self-report	Last year (Wave 1)	0	Physical 1 OR: 0.90 Physical 2 OR: 1.30 Rape OR: 0.70 Injury OR: 1.10	Physical 1: 0.59 Physical 2: 0.76 Rape: 0.30 Injury: 0.53	Physical 1: 1.43 Physical 2: 2.28 Rape: 1.61 Injury: 2.30	-	8
10	Huang (2010)	2 237	Alcohol or drug use interfered with work or relationships	Self-report	Last year (Wave 1)	0	OR: 1.27	0.40	-	NS	9
24	Martino (2005)	509	Any illicit substance use other than marijuana	Self-report	Last year (Wave 1)	0	B: -0.035	0.189	-	NS	7
30	Testa (2003)	724	**Any illicit drug use**	**Self-report**	**Last year (Wave 1)**	**0**	**OR: 2.87**	**1.39**	**5.92**	**-**	**7**
30	Testa (2007)	877	**Frequency of use for most-used drug**	**Self-report**	**Last year (Wave 1)**	**0**	*Multinomial (Ref: none):* **IPV only OR: 1.18** IPV + other violence OR: 1.13	**IPV only: 1.05** IPV + other violence: 0.89	**IPV only: 1.33** IPV + other violence: 1.44	-	**10**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A13: **Women's depressive symptoms**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Renner (2012)	5 237	Depressive symptoms	Self-report	Last week (Wave 1)	1	*Multinomial (Ref: none):* Victim only OR: 0.98 Bidirection OR: 1.02	Victim only: 0.97 Bidirection: 1.00	Victim only: 1.00 Bidirection: 1.03	-	10
13	Kim (2013)	3 153	**Depressive symptoms (CESD-11)**	**Self-report**	**Last week (Waves 1-4)**	**1**	**Level, B: 0.97** **Linear trend, B: 3.34**	**Level: 0.13** **Linear trend: 0.61**	**-**	**Level: <.001** **Linear trend: <.001**	**6**
18	Cougle (2009)	2 875	Major depression (DSM–IV)	Self-report	Last year (Wave 1)	0	OR: 1.69	0.86	3.31	-	6
20	Kim (2008)	194 couples	**Depressive symptoms (CES-D)**	**Self-report**	**Last week (Waves 1-5)**	**1**	**Physical MLM: 0.003** **Psych MLM: 0.01**	**Physical: 0.001** **Psych: 0.002**	**-**	**Physical: .003** **Psych: <.001**	**7**
28	Nduna (2010)	995	**Depressive symptoms (CES-D)**	**Self-report**	**Last week (Wave 1)**	**1**	**OR: 1.67**	**1.18**	**2.36**	**-**	**3**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. MLM is multi-level modelling coefficient. Variables are bolded where p<.05.

Table A14: **Women's mental health (general)**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Renner (2012)	5 237	**Any suicide attempt**	**Self-report**	**Last year (Wave 1)**	**0**	*Multinomial (Ref: none):* **Victim only OR: 1.66 Bidirection OR: 1.58**	**Victim only: 1.03 Bidirection: 1.07**	**Victim only: 2.70 Bidirection: 2.33**	-	**10**
15	Hahn (2014)	20 089	**Mental health impairment below sample mean score**	**Self-report**	**Current (Wave 1)**	**1**	**OR: 1.88**	**1.58**	**2.23**	**-**	**6**
18	Cougle (2009)	2 875	# PTSD-related avoidance/numbing symptoms reported (DSM-4)	Self-report	Last year (Wave 1)	1	OR: 1.14	0.91	1.44	-	6
			# PTSD-related re-experiencing symptoms (DSM-4)	Self-report	Last year (Wave 1)	0	OR: 0.79	0.55	1.12	-	6
			# PTSD-related arousal symptoms (DSM-4)	Self-report	Last year (Wave 1)	1	OR: 1.31	0.99	1.72	-	6
26	Chen (2004)	359	Mean negative affectivity score	Self-report	Last month (Wave 2)	1	B: 0.09	-	-	NS	8

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A15: **Women's physical health**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Turay (2014)	1 680	**Overweight BMI**	**Self-report**	**None (Wave 2)**	**1**	**OR: 1.51**	**1.01**	**2.25**	**-**	**10**
15	Hahn (2014)	20 089	**Physical health impairments**	**Self-report**	**Current (Wave 1)**	**1**	**OR: 1.24**	**1.01**	**1.51**	**-**	**6**
23	Kouyoumdjian (2013)	6 916	Positive HIV status	Self-report	Last year (Waves 1-7, year prior to IPV)	0	OR: 1.03	0.90	1.18	-	13

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A16: **Women's attitudes**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
23	Kouyoumdjian (2013)	6 916	**Man is justified in beating his wife (any 1 of 9 situations)**	**Self-report**	**Last year (Waves 1-7, year prior to IPV)**	**0**	**OR: 1.43**	**1.28**	**1.59**	**-**	**13**
26	Chen (2004)	359	Sum of 18 items assessing attitudes towards traditional gender roles	Self-report	None (Wave 2)	1	B: 0.09	-	-	NS	8
29	Giordano (2016)	506	Level of agreement with 'In most relationships the guy should be in charge'	Self-report	Unclear (Wave 1)	0	OR: 1.27	0.91	1.79	-	20

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A17: **Women's sexual risk**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Exner-Cortens (2013)	2 816	Physically forced to have sex against her will by any person in adolescence	Self-report	Lifetime (Waves 1-2)	0	OR: 1.44	0.93	2.24	-	6

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
23	Kouyoumdjian (2013)	6 916	**Woman used alcohol before sex**	**Self-report**	**Last year (Waves 1-7, year prior to IPV)**	**0**	**OR: 1.21**	**1.10**	**1.32**	**-**	**13**
			More than 1 partner in past year	Self-report	Last year (Waves 1-7, year prior to IPV)	0	OR: 1.27	0.99	1.62	-	13
25	O'Donnell (2009)	526	Early sexual initiation	Self-report	Lifetime (Wave 1: Grade 8)	0	OR: 0.77	0.47	1.29	-	13
30	Testa (2007)	877	Sexually victimization by non-partner	Self-report	Since age 14 (Wave 1)	1	*Multinomial (Ref: none):* IPV only OR: 0.79 IPV + other violence OR: 0.85	IPV only: 0.38 IPV + other violence: 0.17	IPV only: 1.67 IPV + other violence: 4.34	-	9
			# of sexual partners	Self-report	Last year (Wave 1)	0	*Multinomial (Ref: none):* IPV only OR: 2.04 IPV + other violence OR: 1.20	IPV only: 0.56 IPV + other violence: 0.10	IPV only: 7.47 IPV + other violence: 13.78	-	
			Sexual Assertiveness Scale	**Self-report**	**Unclear (Wave 1)**	**1**	*Multinomial (Ref: none):* **IPV only OR: 0.56** IPV + other violence OR: 0.68	**IPV only: 0.40** IPV + other violence: 0.34	**IPV only: 0.77** IPV + other violence: 1.35	-	**6**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. Variables are bolded where p<.05.

Table A18: Women's pregnancy status

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
16	Bourey (2013)	4 749	**Had child (vs. no change)**	**Self-report**	**Last 5 years (Waves 1-2)**	**0**	**RRR: 0.60**	**0.38**	**0.95**	**-**	**17**
17	Jasinski (2001)	1 741 couples	Pregnant or had baby	Partner and self-report	Last year (Wave 1)	1	OR: 1.06	0.25	-	NS	10
		2 125 couples	Had first baby	Partner and self-report	Last year (Wave 1)	1	OR: 0.98	0.03	-	NS	6
23	Kouyoumdjian (2013)	6 916	Pregnant (ref: not pregnant)	Self-report	Last year (Waves 1-7, year prior to IPV)	1	OR: 1.02	0.92	1.12	-	13

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A19: Women's unwanted pregnancy

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
16	Bourey (2013)	4 749	Indicated she or her husband did not want more children but subsequently had child	Self-report	Last 5 years (Waves 1-2)	0	RRR: 1.21	0.94	1.56	-	17
17	Jasinski (2001)	2 125 couples	Indicated they had a child after they intended not to	Partner and self-report	Last year (Wave 1)	1	OR: 0.60	0.60	-	NS	9

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A20: **Women's unplanned pregnancy**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
7	Fergusson (1986)	960	**Either parent was using contraception without hope or expectation of conceiving**	**Self-report**	**Last year (Wave 1)**	**0**	**Hazards: 1.80**	**-**	**-**	**<.05**	**12**
17	Jasinski (2001)	2 125 couples	Became pregnant with child sooner than intended	Partner and self-report	Last year (Wave 1)	1	OR: 1.59	0.31	-	NS	9

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A21: **Women's religiosity**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Brown (2008)	1 967	Personal importance of religion (1=not at all, 4=very)	Self-report	None (Wave 1)	0	B: 0.09	0.09	-	NS	16
7	Fergusson (1986)	960	**Church attendance (ref: at least once per month)**	**Partner-report**	**None (Wave 1)**	**0**				**<.05**	**12**
			Less than once per month				Hazards: 1.39	-	-		
			Never attends				Hazards: 1.94	-	-		
23	Kouyoumdjian (2013)	6 916	Religion (ref: Catholic)	Self-report	None (Waves 1-7, year prior to IPV)	1					13
			Protestant				OR: 1.02	0.92	1.14	-	
			Muslim				OR: 0.88	0.77	1.02	-	
			Other				OR: 1.00	0.79	1.28	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. Variables are bolded where p<.05.

Table A22: **Women's aggressive tendencies**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Schumacher (2008)	634 couples	**Hostility: 10 items (e.g., 'I was argumentative with people')**	**Self-report**	**Unclear (Waves 1-4, lagged with outcome)**	**1**	**RRR: 1.76**	**1.31**	**2.35**	**-**	**12**
4	Leonard (1996)	541 couples	**Physically aggressive behavioural tendencies: 10-item Assault subscale of Buss-Durkee Hostility Inventory**	**Self-report**	**None (Wave 1)**	**1**	**β: 0.11**	**-**	**-**	**<.01**	**16**
1	O'Leary (1994)	272 couples	**Aggressive personality: Jackson Personality Research Form**	**Self-report**	**None (Wave 1)**	**0**	Physical β: 0.072 **Psych β: 0.196**	**-**	**-**	Physical: NS **Psych: <.01**	Physical: 6* **Psych: 7***

Note. Score is reliability score based on adapted Cambridge Quality Checklist. RRR is relative risk ratio. Variables are bolded where p<.05. *Variables not paths.

Table A23: **Women's personality (general)**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
4	Leonard (1996)	541 couples	Problem solving	Self-report	None (Wave 1)	0	β: -0.06	-	-	NS	16
			Withdrawal	Self-report	None (Wave 1)	0	β: -0.06	-	-	NS	16
			The Spielberger Trait Anger Scale	Self-report	None (Wave 1)	1	β: 0.02	-	-	NS	16
4	Leonard (1998)	530 couples	Neuroticism	Self-report	None (Wave 1)	0	β: 0.08	-	-	NS	20
			Masculinity	Self-report	None (Wave 1)	0	β: -0.01	-	-	NS	20

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
			Femininity	Self-report	None (Wave 1)	1	β: -0.01	-	-	NS	20
31	O'Leary (1994)	272 couples	Impulsivity	Self-report	None (Wave 1)	0	Physical B: -0.03 Psych B: 0.09	-	-	Physical: NS Psych: NS	Physical: 6* Psych: 7*
			Defendence (readiness to defend oneself, suspicion of others, tendency to be offended easily)	**Self-report**	**None (Wave 1)**	**0**	Physical B: -0.06 **Psych B: 0.21**	**-**	**-**	Physical: NS **Psych: <.01**	Physical: 6* **Psych: 7***
32	Caetano (2005)	1 136 couples	Impulsive (vs. not)	Self-report	None (Wave 1)	0	OR: 1.2	0.54	2.77	-	16

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05. *Variables not paths.

Table A24: **Women's antisocial behaviour or delinquency**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Renner (2012)	5 237	**Any youth violence perpetration in adolescence**	**Self-report**	**Last year (Wave 1)**	**0**	*Multinomial (Ref: none):* **Victim only OR: 2.49** **Bidirection OR: 1.98**	**Victim only: 1.92** **Bidirection: 1.61**	**Victim only: 3.23** **Bidirection: 2.43**	**-**	**10**
7	Fergusson (2008)	437	**Conduct problems in childhood**	**Parent and teacher report**	**Current (Waves 8-15: Ages 7-13)**	**1**	**B: 0.10**	**0.03**	**-**	**<.05**	**2**
9	Magdol (1998)	317	**Childhood and adolescent problem behaviours**	**Observation, parent report, teacher report, self-report, official records**	**None (Waves 2-5, 8: Ages 3-5, 7-9, and 15)**	**1**	**Physical β: 0.30** **Psych: β: 0.22**	**-**	**-**	**Physical: <.01** **Psych: <.01**	**3**
19	Menard (2014)	393	Adolescent felony assault perpetration	Self-report	Lifetime (Waves 1-5)	0	B: 0.595	-	-	NS	6
20	Kim (2008)	194 couples	**Antisocial behaviour**	**Self-report, observation, official records**	**Last year (Waves 1-5)**	**1**	**Physical MLM: 0.04** **Psych MLM: 0.22**	**Physical: 0.02** **Psych: 0.03**	**-**	**Physical : .01** **Psych: <.001**	**7**
25	O'Donnell (2009)	526	**Any middle school aggression**	**Self-report**	**Last 1.5 months (Wave 1: Grade 8)**	**0**	**OR: 1.88**	**1.23**	**2.87**	**-**	**13**
29	Giordano (2016)	506	Delinquency in adolescence	Self-report	Unclear (Wave 1)	1	OR: 1.13	0.93	1.36	-	20
35	Vezina (2015)	443	High risk behaviours in adolescence	Self-report	Lifetime (Wave 2: Age 15)	1	Psych OR: 1.06 Phys/sex OR: 1.21	Psych OR: 0.61 Phys/sex OR: 0.63	Psych OR: 1.84 Phys/sex OR: 2.33	-	6
			Childhood behaviour problems	**Parent report**	**None (Wave 1: Age 6)**	**1**	**Psych OR: 1.09** Phys/sex OR: 0.95	**Psych OR: 1.01** Phys/sex OR: 0.88	**Psych OR: 1.17** Phys/sex OR: 1.04	**-**	**6**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. MLM is multi-level modelling coefficient. Variables are bolded where p<.05.

Table A25: **Women's pubertal status**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Exner-Cortens (2013)	2 816	More advanced pubertal status	Self-report	None (Wave 2)	0	OR: 1.17	0.97	1.40	-	6

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A26: **Women's youth victimization**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Fang (2007)	5 179	Any youth violence victimization	Self-report	Last year (Wave 1)	0	Probit: 0.044	0.031	-	NS	9
19	Menard (2014)	393	**Any adolescent general violent victimization**	**Self-report**	**Lifetime (Waves 1-5)**	**0**	**B: 1.147**	**-**	**-**	**≤0.01**	**6**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A27: **Women's experience of child abuse**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
6	Lansford (2007)	131	Child was hit severely enough by any adult to be hurt or to require medical attention	Parent report	Last 5 years (Wave 1)	1	F: 0.80	-	-	NS	7
12	Cui (2010)	119	**Observer report during parent-child discussion task; Adolescent report of mother and father abuse (e.g., mother/father hit, pushed, grabbed, or shoved her)**	**Self-report and observation**	**Last month (Waves 4-5)**	**1**	**Physical β: 0.33 Psych β: 0.47**	**-**	**-**	**Physical: <.05 Psych: <.05**	**5**
19	Menard (2014)	393	Child (female partner) was beaten up by a parent	Self-report	Last year (Waves 1-5)	0	B: -0.69	-	-	NS	6
29	Kaufman (2015)	507	**Positive response to any of 3 items (e.g., When you and your parents disagree about things, how often do they push, slap, or hit you?)**	**Self-report**	**None (Waves 1-5)**	**0**	**GEE (B): 0.48**	**0.24**	**-**	**<.05**	**12**
35	Widom (2014)	366	Official records of physical abuse, sexual abuse, or neglect from county juvenile and adult criminal courts	Official records	4-year period in Wave 1	1	OR: 1.01	0.60	1.70	-	2

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A28: **Women's exposure to inter-parental IPV**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
6	Lansford (2007)	131	Type of violence exposed to between parents, between others in the home, and outside the home (1=none, 2=mild verbal, 3=major verbal, 4=mild physical/major verbal, 5=physical more than once).	Parent report	Last 5 years (Wave 1)	0	F: 1.45	-	-	NS	7
12	Cui (2010)	119	Degree to which each of mother and father engaged in physical attacks during interaction tasks; Mother report of father's physical aggression; Father report of mother's physical aggression	Self-report and observation	Last month (Waves 1-3)	1	Phys β: 0.24 Psych β: 0.03	-	-	Phys: NS Psych: NS	5
26	Chen (2004)	359	Parents fight or argue with each other	Self-report	None (Wave 1)	0	B: <0.01	-	-	NS	8

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A29: **Women's employment**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informat	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Fang (2007)	5 179	Employed	Self-report	None (Wave 1)	1	Probit: 0.0001	0.02	-	NS	9
11	Staggs (2007)	825	Proportion of time employed vs. not	Self-report	Last year (Wave 1)	0	B: -0.002	0.001	-	NS	12
			Average # jobs held per month	Self-report	Last year (Wave 1)	0	B: -0.29	0.44	-	NS	12
16	Bourey (2013)	4 749	Employment status (ref: None)	Self-report	Current (Waves 1-2)	0					17
			Decreased from T1 to T2				RRR: 0.75	0.51	1.09	-	
			Increased from T1 to T2				RRR: 1.05	0.83	1.33	-	
			Continued from T1 to T2				RRR: 1.09	0.85	1.40	-	
17	Fox (2002)	3 006 couples	# of job spells of at least 6 months duration	Self-report	Last 6 years (Waves 1-2)	0	OR: 0.90	-	-	NS	5
27	Krishnan (2010)	653	Employment status (ref: did not change)	Self-report	Last 6 months Waves 1-2 and Waves 2-3)	1					
			Employment to no employment				OR: 0.62	0.30	1.26	-	
			No employment to employment				OR: 1.86	1.38	2.50	-	
29	Kaufman (2015)	507	**Gainful activity (Attending school or employed)**	**Self-report**	**None (Waves 1-5)**	**1**	**GEE (B): -0.37**	**0.13**	**-**	**<.01**	**12**
32	Caetano (2005)	1 136 couples	Employment (ref: employed)	Self-report	Current (Wave 1)	1					16
			Unemployed				OR: 0.40	0.13	1.49	-	
			Homemaker				OR: 1.60	0.69	3.67	-	
			Retired/other				OR: 1.50	0.31	7.05	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A30: **Women's education**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
4	Leonard (1996)	541 couples	Lower education	Self-report	Lifetime (Wave 1)	0	β: -0.07	-	-	NS	16
7	Fergusson (1986)	960	**Wife's education (ref: Tertiary)**	**Self-report**	**None (wave 1)**	**1**				**<.01**	**12**
			Secondary				**Hazards: 2.04**	-	-		
			No formal education				**Hazards: 4.15**	-	-		
10	Huang (2010)	2 237	Education (ref: ≥high school)	Self-report	Lifetime (Wave 1)	1					9
			Less than high school diploma				OR: 0.92	0.15	-	NS	
			High school diploma				OR: 1.02	0.16	-	NS	
15	Hahn (2014)	20 089	**Education (ref: College grad or more)**	**Self-report**	**Lifetime (Wave 1)**	**1**					**6**
			Less than high school diploma				**OR: 1.77**	**1.22**	**2.55**	**-**	
			High school graduate/GED				**OR: 1.83**	**1.34**	**2.50**	**-**	
			Some college or associates				**OR: 1.70**	**1.26**	**2.29**	**-**	
16	Bourey (2013)	4 749	Education (ref: none)	Self-report	Lifetime (Wave 1)	1					17
			Primary				RRR: 0.86	0.66	1.12	-	
			≥Secondary				**RRR: 0.69**	**0.52**	**0.91**	**-**	
25	O'Donnell (2009)	526	≥high school education	Self-report	Lifetime (Wave 3)	1	OR: 0.96	0.58	1.59	-	13
31	MacEwan (1988)	275 couples	Education (years)	Self-report	Lifetime (Wave 1)	0	β: -0.06	-	-	NS	5

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A31: **Woman's education achievements**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE	p-value (if no interval)	# Co-variates
9	Magdol (1998)	317	Educational achievements: IQ, reading achievement, age at leaving secondary school	Self-report	None (Waves 4, 5, 8: Ages 7-9, and 15)	1	Physical β: 0.02 Psych: β: 0.06	- -	Physical: NS Psych: NS	3

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A32: **Women's school enrolment**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE	p-value (if no interval)	# Co-variates
1	Fang (2007)	5 179	**School enrollment in adolescence**	**Self-report**	**None (Wave 1)**	**1**	**Probit: -0.06**	**0.02 -**	**<.01**	**9**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A33: **Woman's socioeconomic status (SES)**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE	p-value (if no interval)	# Co-variates
4	Leonard (1998)	530 couples	SES (Hollingshead Index)	Self-report	None (Wave 1)	1	β: -0.08	- -	NS	20
16	Bourey (2013)	4 749	Caste (ref: General caste)	Self-report	Fixed (Wave 1)	0				17
			Scheduled caste/tribe				**RRR: 1.53**	**1.11 2.11**	-	
			Other backward caste				**RRR: 1.43**	**1.06 1.94**	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A34: **Women's occupation**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE	p-value (if no interval)	# Co-variates
23	Kouyoumdjian (2013)	6 916	Occupation (ref: Agriculture)	Self-report	Last year (Waves 1-7, year prior to IPV)	0				13
			Shopkeeper/trading/vending				OR: 0.92	0.80 1.06	-	
			Housework				OR: 0.99	0.80 1.21	-	
			Professional				OR: 0.88	0.73 1.07	-	
			Student				OR: 1.04	0.34 3.15	-	
			Home brewing/bar worker/owner				OR: 1.12	0.84 1.50	-	
			Other				OR: 1.01	0.87 1.17	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. Variables are bolded where p<.05.

Relational factors

Table A35: **Partner's race**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE	p-value (if no interval)	# Co-variates
3	Smith (2014)	Not known	Ref: European American	Partner report	Fixed (Wave 1)	1				10

			Black				**IRR: 1.32**	**1.09**	**1.60**	-	
			Other				IRR: 0.92	0.74	1.15	-	
4	Leonard (1998)	530 couples	**At least one partner is minority (ref: neither)**	**Partner and self-report**	**Fixed (Wave 1)**	**1**	**β: 0.22**	**-**	**-**	**<.01**	**20**
17	Jasinski (2001)	1 741 couples	Ref: White	Partner report	Fixed (Wave 1)	1					10
			Black				OR: 1.29	0.49	-	NS	
			Hispanic				OR: 1.50	0.57	-	NS	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. IRR is incidence rate ratio. Variables are bolded where p<.05.

Table A36: **Partner's age**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Smith (2014)	Not reported	Age	Partner report	Fixed (Wave 1)	1	IRR: 0.99	0.98	1.00	-	10
4	Leonard (1996)	541 couples	Age	Partner report	Fixed (Wave 1)	1	β: -0.07	-	-	NS	16
6	Fergusson (1986)	960	**Age (ref: 25+ years)**	**Self-report**	**Fixed (Wave 1)**	**1**				**<.05**	**12**
			20-24 years				Hazards: 1.37	-	-		
			<20 years				Hazards: 1.87	-	-		
32	Caetano (2005)	1 136 couples	Age	Partner report	Fixed (Wave 1)	1	OR: 0.9	0.88	1.04	-	16

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. IRR is incidence rate ratio. Variables are bolded where p<.05.

Table A37: **Age difference between partners**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Turay (2014)	1 680	Partner age difference (ref: same age)	Self-report	None (Wave 3)	1					10
			Older Than				OR: 1.39	0.87	2.23	-	
			Younger Than				**OR: 2.10**	**1.11**	**4.00**	-	
4	Leonard (1998)	530 couples	**Mean age of couple**	**Partner and self-report**	**None (Wave 1)**	**1**	**β: -0.14**	**-**	**-**	**<.01**	**20**
16	Bourey (2013)	4 749	Spousal age difference (ref: Wife older or husband 0-4 years older)	Self-report	None (Wave 1)	1					17
			Husband 5-9 years older				RRR: 1.09	0.91	1.31	-	
			Husband 10 or more years older				RRR: 0.88	0.70	1.12	-	
23	Kouyoumdjian (2013)	6 916	Partner age difference (Ref: same age)	Self-report	Last year (Waves 1-7, year prior to IPV)	1					13
			≥10 years older				OR: 0.83	0.69	1.00	-	
			5-9 years older				**OR: 0.83**	**0.69**	**0.99**	-	
			<5 years older				**OR: 0.83**	**0.70**	**0.99**	-	
			<5 years younger				OR: 1.02	0.79	1.32	-	
			≥5 years younger				OR: 1.21	0.76	1.91	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A38: **Partner's alcohol use**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Schumacher (2008)	634 couples	Alcohol Dependence Scale	Partner report	Unclear (Waves 1-4, lagged with outcome)	1	RRR: 0.98	0.95	1.02	-	12
4	Quigley (2000)	414 couples	Standardized sum of Alcohol Dependence Scale and average daily alcohol consumption	Partner report	Last year (Wave 2)	1	B: 0.06	0.05	-	NS	14
21	Temple (2008)	509	**Alcohol use: Negligibly, rarely, occasionally, or often**	**Self-report**	**Unclear (Wave 1)**	**0**	**Physical F: 6.45 Sexual F: 3.93**	**-**	**-**	**Physical: <.001 Sexual: <.01**	**5**
32	Caetano (2005)	1 136 couples	Sum of 14 alcohol problem behaviours	Partner report	Last year (Wave 1)	0	OR: 1.60	0.53	4.87	-	16
			Partner had 5 or more drinks per occasion	Partner report	Last year (Wave 1)	0	OR: 0.80	0.33	1.90	-	16
			Partner's average alcohol volume per week	Partner report	Last year (Wave 1)	0	OR: 0.90	0.81	1.09	-	16
33	Testa (2014)	118 couples	Partner's total drinking days in the study period	Partner report	Last 56 days	0	Phys OR: 0.99 Psych OR: 1.00	Phys OR: 0.97 Psych OR: 0.96	Phys OR: 1.01 Psych OR: 1.04	-	4
			Partner's alcohol use in a given 4-hour interval	**Partner report**	Last 56 days	**0**	**Phys OR: 1.39** Psych OR: 2.53	**Phys OR: 1.01** Psych OR: 0.91	**Phys OR: 1.92** Psych OR: 7.02	-	4

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A39: Partner's substance use

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Smith (2014)	Not reported	**Frequency of marijuana use per month**	**Partner report**	**Last year (Waves 1-6)**	**0**	**IRR: 0.95**	**0.91**	**0.99**	**-**	**10**
17	Benson (2003)	3 006 couples	**Either male or female indicated that the male had a problem with drinking or drugs**	**Partner and self-report**	**Unclear (Wave 1)**	**1**	**B: 0.34**	**0.32**	**-**	**<.05**	**11**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. IRR is incidence rate ratio. Variables are bolded where p<.05.

Table A40: Partner's depressive symptoms

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
20	Kim (2008)	194 couples	**Depressive symptoms (CES-D)**	**Partner report**	**Last week (Waves 1-5)**	**1**	Physical MLM: 0.001 **Psych MLM: 0.006**	Physical: 0.001 **Psych: 0.003**	-	Physical: NS **Psych: .02**	**7**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. MLM is multi-level modelling coefficient. Variables are bolded where p<.05.

Table A41: Partner's attitudes

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
32	Field (2003)	232 Black couples; 387 Hispanic couples	Approval of marital aggression (4 items)	Partner and self-report	None (Wave 1)	1	Black couples OR: 2.4 Hispanic couples OR: 1.6	Black couples: 0.4 Hispanic couples: 0.4	Black couples: 13.0 Hispanic couples: 5.6	-	Black couples: 14 Hispanic couples: 16
16	Bourey (2013)	4 749	**Husband's reaction to marriage dowry (ref: unsatisfied)**	**Self-report**	**None (Wave 1)**	**0**					**17**
			Neutral/ unsure				**RRR: 0.39**	**0.25**	**0.60**	-	
			Satisfied				**RRR: 0.37**	**0.25**	**0.53**	-	
			No dowry				**RRR: 0.47**	**0.29**	**0.75**	-	
20	Capaldi (2001)	206 couples	**Hostile talk about women during discussion task**	**Observation**	**Wave 5: Partners aged 17-18**	**1**	**β: 0.15**	**-**	**-**	**<.05**	**4***

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05. *Variables not paths.

Table A42: Partner's sexual risk

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
23	Kouyoumdjian (2013)	6 916	**Used alcohol before sex**	**Self-report**	**Last year (Waves 1-7, year prior to IPV)**	**0**	**OR: 1.46**	**1.34**	**1.60**	**-**	**13**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A43: Partner's aggressive tendencies

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Schumacher (2008)	634 couples	**Hostility (e.g., 'I was argumentative with people')**	**Partner report**	**None (Waves 1-4, lagged with outcome)**	**1**	**RRR: 1.33**	**1.01**	**1.77**	**-**	**12**
31	Heyman (1995)	272 couples	Aggressive personality (Jackson Personality Research Form)	Partner report	None (Wave 1: Pre-marriage)	0	β: 0.15	0.09	-	NS	2

Note. Score is reliability score based on adapted Cambridge Quality Checklist. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A44: Partner's personality

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
4	Leonard (1996)	541 couples	**Problem solving**	**Self-report**	**None (Wave 1)**	**0**	**β: 0.10**	**-**	**-**	**<.05**	**16**
			Withdrawal	**Self-report**	**None (Wave 1)**	**0**	**β: -0.08**	**-**	**-**	**<.05**	**16**
			Masculinity	Partner report	None (Wave 1)	0	β: 0.03	-	-	NS	16
			Femininity	**Partner report**	**None (Wave 1)**	**1**	**β: -0.08**	**-**	**-**	**<.05**	**16**
4	Leonard (1998)	530 couples	Neuroticism	Partner report	None (Wave 1)	0	β: 0.00	-	-	NS	20
5	Theobald (2012)	212 couples	Impulsivity	Partner report	(Age 10)	0	B: 0.75	0.49	-	NS	3
32	Caetano (2005)	1 136 couples	**Impulsive**	**Partner report**	**None (Wave 1)**	**0**	**OR: 2.20**	**1.08**	**4.47**	**-**	**16**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A45: Partner's antisocial behaviour

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
20	Kerr (2011)	153 couples	**Adolescent antisocial behaviour**	**Partner, partner's parent, partner's teacher report**	**Last year (Waves 1-8: Ages 10-17)**	**1**	**B: 0.11**	**0.05**	**-**	**<.05**	**4**
20	Kim (2008)	194 couples	**Antisocial behaviour**	**Partner report and observation**	**Last year (Waves 1-5)**	**1**	Physical MLM: 0.03 **Psych MLM: 0.16**	Physical: 0.02 **Psych: 0.04**	-	Physical: NS **Psych: <.001**	**7**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. MLM is multi-level modelling coefficient. Variables are bolded where p<.05.

Table A46: Women's life stress

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
6	Lansford (2007)	131	Family stress (e.g., death of a family member)	Parent report	Last 5 years (Wave 1)	0	F: 0.03	-	-	NS	7
16	Bourey (2013)	4 749	**Child(ren) died**	**Self-report**	**Last five years (Waves 1-2)**	**0**	**RRR: 1.43**	**1.13**	**1.79**	**-**	**17**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A47: Partner's life stress

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Schumacher (2008)	634 couples	Husband daily hassles	Partner report	None (Waves 1-4, lagged with outcome)	1	RRR: 1.15	0.70	1.89	-	12
			Husband avoidance coping	**Partner report**	**None (Waves 1-4, lagged with outcome)**	**1**	**RRR: 1.82**	**1.27**	**2.62**	**-**	**12**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A48: Parenting status

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Schumacher (2008)	634 couples	Partner had child(ren) prior to current marriage	Partner report	Lifetime (Wave 1)	1	RRR: 1.50	0.99	2.16	-	12
4	Leonard (1998)	530 couples	Have at least one child	Self-report	None (Wave 1)	1	β: 0.07	-	-	NS	20
16	Bourey (2013)	4 749	Baseline parity (i.e., # of still or live births) (ref: 0)	Self-report	Lifetime (Wave 1)	0					17
			1				RRR: 0.80	0.52	1.22	-	
			2				**RRR: 0.59**	**0.38**	**0.90**	-	
			3				**RRR: 0.52**	**0.34**	**0.80**	-	
			4 or more				**RRR: 0.42**	**0.27**	**0.64**	-	
17	Jasinski (2001)	1 741 couples	# of children	Partner and self-report	Lifetime (Wave 1)	1	OR: 1.00	0.05	-	NS	10
29	Giordano (2016)	506	Woman has children	Self-report	Lifetime (Wave 4)	1	OR: 0.99	0.49	2.01	-	20

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A49: Early union

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
16	Bourey (2013)	4 749	Age at start of current union (ref: 12-14)	Self-report	None (Wave 1)	1					17

			15-17				RRR: 0.93	0.75	1.15	-	
			18 or older				RRR: 0.85	0.67	1.09	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A50: **Relationship status**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Fang (2007)	5 179	**Married**	**Self-report**	**None (Wave 1)**	**1**	**Probit: -0.05**	**0.02**	**-**	**<.05**	**9**
8	Koziol-McLain (2001)	405	**Separated (ref: married, divorced, widowed, never married or unmarried couple)**	**Self-report**	**Current (Wave 1)**	**1**	**OR: 6.20**	**1.10**	**33.80**	**-**	**4**
20	Kim (2008)	194 couples	1=dating, 2=living together, 3=married	Partner report	None (Waves 1-5)	1	Physical B (MLM): 0.04 **Psych B (MLM): 0.22**	Physical: 0.01 **Psych: 0.03**	-	Physical: <.01 **Psych: <.001**	7
23	Kouyoumdjian (2013)	6 916	Marital status (ref: never married)	Self-report	Last year (Waves 1-7, year prior to IPV)	1					13
			Previously married				OR: 1.08	0.85	1.38	-	
			Married: polygamous				OR: 1.44	0.92	2.25	-	
			Married: monogamous				OR: 1.32	0.85	2.04	-	
29	Kaufman (2015)	507	Relationship status (ref: dating)	Self-report	Current (Waves 1-5)	1					
			Cohabiting				GEE (B): 0.18	0.17	-	NS	12
			Married				GEE (B): 0.21	0.24	-	NS	12
30	Testa (2007)	877	Married or cohabiting (ref: single)	Self-report	None (Wave 1)	1	*Multinomial (Ref: none):* **IPV only OR: 1.95** IPV + other violence OR: 1.06	**IPV only: 1.17** IPV + other violence: 0.36	**IPV only: 3.23** IPV + other violence: 3.09	-	10
30	Testa (2003)	724	Relationship status (ref: single)	Self-report	Current (Wave 1)	1				-	7
			Cohabiting				**OR: 2.04**	**1.27**	**3.29**	-	
			Married				OR: 1.12	0.62	2.02	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. MLM is multi-level modelling. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A51: **Relationship duration**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Turay (2014)	1 680	**Relationship duration**	**Self-report**	**Fixed (Wave 3)**	**1**	**OR: 1.18**	**1.09**	**1.28**	**-**	**10**
3	Schumacher (2008)	634 couples	Months cohabitating prior to marriage	Partner report	Lifetime (Wave 1)	0	RRR: 1.00	1.00	1.01	-	12
4	Leonard (1998)	530 couples	Relationship length (prior to marriage)	Self-report	Fixed (Wave 1)	1	β: -0.04	-	-	NS	20
7	Fergusson (1986)	960	**Length of marriage (ref: 5+ years)**	**Self-report**	**None (Wave 1)**	**1**				**<.01**	**12**
			1-4 years				Hazards: 1.77	-	-		
			<1 year				Hazards: 3.15	-	-		

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
21	Temple (2008)	509	**Remained with partner from Wave 1**	**Self-report**	**None (Waves 1-5)**	**0**	**Physical F: 18.86 Sexual F: 17.03**	**-**	**-**	**Physical: <.001 Sexual: <.001**	**5**
23	Kouyoumdjian (2013)	6916	Length of relationship (ref: <3 years)	Self-report	Last year (Waves 1-7, year prior to IPV)	1					13
			4-6 years				**OR: 0.84**	**0.75**	**0.94**	-	
			>6 years				**OR: 0.79**	**0.70**	**0.90**	-	
29	Kaufman (2015)	507	**Relationship length, from 1=less than a week to 8=a year or more**	**Self-report**	**Current (Waves 1-5)**	**1**	**GEE (B): 0.15**	**0.04**	**-**	**<.001**	**12**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A52: Woman's relationship satisfaction

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
10	Huang (2010)	2 237	**Positive relationship quality index with father of woman's child (4 items)**	**Self-report**	**Unclear (Wave 1)**	**0**	**OR: 0.48**	**0.08**	**-**	**<.001**	**9**
20	Kim (2008)	194 couples	**32-item Dyadic Adjustment Scale**	**Self-report**	**None (Waves 1-5)**	**0**	**Physical B (MLM): -0.002 Psych B (MLM): -0.01**	**Physical: 0.001 Psych: 0.001**	**-**	**Physical: .002 Psych: <.001**	7
31	O'Leary (1994)	272 couples	Marital discord: Locke-Wallace Short Marital Adjustment Test	Self-report	None (Wave 2)	0	B: 0.08	-	-	NS	6*

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. MLM is multi-levelling model coefficient. Variables are bolded where p<.05. *Variables not paths.

Table A53: Partner's relationship satisfaction

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
20	Kim (2008)	194 couples	32-item Dyadic Adjustment Scale	Partner report	None (Waves 1-5)	0	**Psych B (MLM): -0.01**	**0.002**	**-**	**<.001**	7

Note. Score is reliability score based on adapted Cambridge Quality Checklist. MLM is multi-levelling model coefficient. Variables are bolded where p<.05.

Table A54: Women's family structure

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
2	Gomez (2011)	1 949	Family structure (ref: 2 biological parents)	Self-report	Fixed (Wave 1)	1					13
			Two other parents				OR: 0.97	0.65	1.45	-	
			Single mother				OR: 0.99	0.65	1.51	-	
			Single father				OR: 0.62	0.29	1.32	-	
			Other				OR: 0.76	0.42	1.38	-	
6	Lansford (2007)	131	Has single parent (or not)	Parent report	Current (Wave 1)	1	F: 1.49	-	-	NS	7
7	McLeod (2014)	420	Born into 1-parent family (ref: 2-parent family)	Parent report	Fixed (Birth)	1	OR: 2.39	0.49	11.63	-	13
			Any change of parents (0-16 years)	Parent report	Last year (Ages 0-16)	1	OR: 0.81	0.28	2.38	-	13
19	Menard (2014)	393	Two-parent family structure	Parent report	Fixed (Wave 1)	1	B: -0.53	-	-	NS	6

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
29	Giordano (2016)	506	Family structure (ref: 2 biological parents)	Self-report	Lifetime (Wave 1)	1					20
			Step family				OR: 1.47	0.53	4.06	-	
			Single parent				**OR: 2.71**	**1.19**	**6.21**	-	
			Any other family structure				OR: 2.19	0.82	5.85	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. Variables are bolded where p<.05.

Table A55: **Women's association with deviant peers**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
29	Giordano (2016)	506	Frequency of friends attacking someone with the idea of seriously hurting them	Self-report	Unclear (Wave 1)	0	OR: 0.82	0.59	1.14	-	20
35	Vezina (2015)	443	Standardised mean of 9 items (e.g., best female friend ran away from home)	Self-report	Lifetime (Wave 2: Age 15)	0	Psych OR: 1.25 Phys/sex OR: 0.93	Psych OR: 0.85 Phys/ sex OR: 0.59	Psych OR: 1.84 Phys/ sex OR: 1.46	-	6

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A56: **Woman's parent relationship quality**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Brown (2008)	1 967	**Sum of 4 items regarding parental closeness**	**Self-report**	**None (Wave 1)**	**0**	**B: -0.07**	**0.02**	**-**	**<.01**	**16**
9	Magdol (1998)	317	**Negative family relations**	**Parent report, Observation**	**None (Waves 2, 4, 5, 8: Ages 3, 7-9, and 15)**	**1**	Phys β: 0.03 **Psych: β: 0.13**	**-**	**-**	Phys: NS **Psych: <.05**	**3**
29	Kaufman (2015)	507	Positive parent-child relationship quality	Self-report	None (Waves 1-5)	1	GEE (B): -0.01	0.02	-	NS	12

Note. Score is reliability score based on adapted Cambridge Quality Checklist. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A57: **Partner's parent relationship quality**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
5	Theobald (2012)	212 couples	**Partner had poor relationship with parents**	**Partner report**	**(Age 18)**	**0**	**B: 1.04**	**0.50**	**-**	**<.05**	**3**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A58: **Woman's experience of parental monitoring**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
35	Vezina (2015)	443	Lower parental monitoring (standardised mean of 5 items)	Self-report	None (Wave 2: Age 15)	0	Psych OR: 1.45 Phys/sex OR: 1.18	Psych OR: 0.79 Phys/sex OR: 0.60	Psych OR: 2.64 Phys/sex OR: 2.31	-	6

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A59: **Partner's experience of parental monitoring**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
5	Theobald (2012)	212 couples	**Poor parental supervision**	**Partner report**	**(Age 10)**	**0**	**B: 1.14**	**0.48**	**-**	**<.01**	**3**
20	Kerr (2011)	153 couples	**Experience of unskilled parenting**	**Partner and partner's parent report**	**None (Waves 1, 3, 5: Ages 10, 12, 14)**	**1**	**B: 0.14**	**0.05**	**-**	**<.01**	**4**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A60: Partner's employment

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Schumacher (2008)	634 couples	Employment	Partner report	None (Wave 1)	0	RRR: 1.20	0.89	1.61	-	12
17	Benson (2003)	3 006 couples	**# of intervals of unemployment**	**Partner report**	**Last 6 years (Waves 1-2)**	**0**	**B: 0.33**	**0.09**	**-**	**<.01**	**11**
27	Krishnan (2010)	653	Employment stability (ref: employment did not change)	Self-report	Last 6 months Waves 1-2 and Waves 2-3)	0					6
			Difficulty to no difficulty finding or keeping a job				**OR: 0.60**	**0.44**	**0.82**	-	
			No difficulty to difficulty finding or keeping a job				**OR: 1.82**	**1.19**	**2.79**	-	
32	Caetano (2005)	1 136 couples	Employment (ref: employed)	Partner report	Current (Wave 1)	1					16
			Unemployed				OR: 0.2	0.03	1.09	-	
			Retired/other				OR: 1.8	0.54	6.07	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A61: Partner's education

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
3	Smith (2014)	Not reported	**Education beyond high school**	**Partner report**	**Lifetime (Wave 1)**	**1**	**IRR: 1.15**	**1.04**	**1.28**	**-**	**10**
16	Bourey (2013)	4 749	Education (ref: none)	Self-report	Lifetime (Wave 1)	1					17
			Primary				RRR: 0.79	0.62	1.01	-	
			Secondary				**RRR: 0.80**	**0.65**	**0.99**	-	
17	Benson (2003)	3 006 couples	Education (no further details)	Partner report	Lifetime (Unclear)	0	B: 0.03	0.04	-	NS	11

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. IRR is incidence rate ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A62: Partner's IQ

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
5	Theobald (2012)	212 couples	**Low IQ (no further details)**	**Partner report**	**(Age 10)**	**0**	**B: 0.89**	**0.48**	**-**	**<.05**	**3**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A63: Parent education

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Lehrer (2006)	1 525	Education less than high school, (ref: otherwise)	Parent report	Lifetime (Wave 1)	1	OR: 1.70	0.88	3.28	-	6
7	McLeod (2014)	420	Mother did not have secondary or tertiary qualifications	Parent report	Lifetime (Birth)	1	OR: 2.28	0.85	6.09	-	13

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
			Father did not have secondary or tertiary qualifications	Parent report	Lifetime (Birth)	1	OR: 0.99	0.39	2.54	-	13
22	Jain (2010)	352	Less than college graduate (ref: College graduate)	Parent report	Lifetime (Wave 1)	1	B: -0.22	0.18	-	NS	5
29	Kaufman (2015)	507	Education (ref: High school)	Parent report	Lifetime (Wave 1)	1					12
			Less than high school diploma				GEE (B): 0.32	0.22	-	NS	
			College graduate				GEE (B): -0.22	0.20	-	NS	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. GEE is generalised estimating equation. Variables are bolded where p<.05.

Table A64: **Partner's SES**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
7	Fergusson (1986)	960	**Husband's SES (ref: Professional, managerial)**	**Self-report**	**None (Wave 1)**	**1**				**<.01**	**12**
			Clerical, technical, skilled				**Hazards: 1.66**	**-**	**-**		
			Semiskilled, unskilled				**Hazards: 2.75**	**-**	**-**		

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. Variables are bolded where p<.05.

Table A65: **Partner's occupation**

Cohort	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
23	Kouyoumdjian (2013)	6 916	Partner's occupation (ref: Agriculture)	Self-report	Last year (Waves 1-7, year prior to IPV)	0					13
			Shopkeeper/trading/vending				OR: 0.91	0.82	1.01	-	
			Housework				**OR: 0.82**	**0.71**	**0.95**	-	
			Professional				OR: 0.75	0.26	2.13	-	
			Student				OR: 1.13	0.80	1.60	-	
			Home brewing/bar worker/owner				OR: 0.82	0.66	1.03	-	
			Other				OR: 0.94	0.84	1.04	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. Variables are bolded where p<.05.

Table A66: **Women's family SES**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
6	Lansford (2007)	131	Family SES (Hollingshead Index)	Parent report	None (Wave 1)	1	F: 1.73			NS	7
7	McLeod (2014)	420	Low family SES (unskilled/semi-skilled occupational status) (ref: professional, managerial, clerical, technical, or skilled)	Parent report	None (Birth)	1	OR: 1.63	0.60	4.42	-	13
9	Magdol (1998)	317	Family SES (parents' occupations, family structure)	Parent report, official records	None (Waves 1, 4, 5, 8: Birth, Ages 7-9, and 15)	1	Physical β: 0.02 Psych: β: 0.03	-	-	Physical: NS Psych: NS	3
19	Menard (2014)	393	Family SES (Hollingshead Index, ref: working class)	Parent report	None (Wave 1)	1		-	-		6
			Upper/middle class				B: -0.14	-	-	NS	
			Lower class				B: 0.42	-	-	NS	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. Variables are bolded where p<.05.

Table A67: **Household poverty/economic stress**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
15	Hahn (2014)	20 089	**Female partner had received welfare, aid, and/or food stamps**	**Self-report**	**Last year (Wave 1)**	**0**	**OR: 1.77**	**1.39**	**2.25**	**-**	**6**
16	Bourey (2013)	4 749	Standard of living (ref: low)	Self-report	Current (Wave 1)	0					17
			Medium				**RRR: 0.81**	**0.67**	**0.98**	-	
			High				**RRR: 0.34**	**0.21**	**0.55**	-	
17	Benson (2003)	3 006 couples	Income to needs ratio: Total household income divided by the poverty line for households of that size	Self-report	Last year (Waves 1)	0	B: 0.03	0.03	-	NS	11
			Change in income to needs ratio (income increased)	Self-report	Last 6 years (Wave 1-2)	0	B: 0.02	0.02	-	NS	11
17	Fox (2002)	3 006 couples	Increase in number of debts between waves	Self-report	Last 6 years (Waves 1-2)	0	OR: 1.02	-	-	NS	5

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A68: **Family poverty**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
1	Fang (2007)	5 179	Annual household income ≤$20,000 in adolescence	Self-report	None (Wave 1)	0	Probit: 0.01	0.02	-	NS	9
7	McLeod (2014)	420	Childhood family living standards in lowest quartile	Parent report	None (Ages 0-10)	0	OR: 0.88	0.27	2.82	-	13

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A69: **Women's autonomy**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
16	Bourey (2013)	4 749	Respondent's economic contribution (ref: no change)	Self-report	Current (Waves 1-2)	0					17
			Decreased from T1 to T2				RRR: 1.27	0.93	1.73	-	
			Increased from T1 to T2				RRR: 1.11	0.78	1.56	-	
			Financial autonomy (Allowed to set aside money, ref: none)	Self-report	Current (Waves 1-2)	0					17
			Decreased from T1 to T2				RRR: 0.81	0.61	1.08	-	
			Increased from T1 to T2				RRR: 0.83	0.62	1.12	-	
			Continued from T1 to T2				**RRR: 0.72**	**0.55**	**0.93**	-	
			Freedom of movement (Need permission to go places, ref: none)	Self-report	Current (Waves 1-2)	0					17
			Decreased from T1 to T2				RRR: 0.72	0.48	1.08	-	
			Increased from T1 to T2				**RRR: 0.71**	**0.57**	**0.90**	-	
			Continued from T1 to T2				**RRR: 0.72**	**0.55**	**0.96**	-	
16	Sabarwal (2014)	4 904	Household decision making autonomy (ref: no autonomy at both time points)	Self-report	Unclear (Waves 1-2)	0					13
			High autonomy at both Waves				OR: 1.02	0.80	1.31	-	
			Gained autonomy from Wave 1 to 2				OR: 1.09	0.87	1.35	-	
			Lost autonomy from Wave 1 to 2				OR: 0.95	0.77	1.18	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. RRR is relative risk ratio. Variables are bolded where p<.05.

Table A70: **Partner's autonomy**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE	p-value (if no interval)	# Co-variates
17	Fox (2002)	3 006 couples	**Partner contributes a smaller share of the couple's earnings**	**Partner report**	**Last 6 years (Waves 1-2)**	**0**	**OR: 1.31**	**- -**	**<.05**	**5**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A71: **Household income**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
2	Gomez (2011)	1949	Parental income (ref: <US$16,000)	Parent report	Last year (Wave 1)	0					13
			US$16,000-US$29,999				OR: 1.01	0.66	1.52	-	
			US$30,000-US$49,999				OR: 0.76	0.45	1.28	-	
			US$50,000-US$79,999				OR: 0.55	0.30	1.02	-	
			More than US$80,000				OR: 0.65	0.35	1.22	-	
			No response				OR: 1.02	0.45	2.31	-	
3	Smith (2014)	Not reported	**Husband income**	**Partner report**	**Last year (Wave 1)**	**1**	**IRR: 0.89**	**0.86**	**0.93**	**-**	**10**
7	McLeod (2014)	420	In lowest quartile for averaged family income (ref: all quartiles)	Parent report	None (Ages 0-10)	0	OR: 0.57	0.15	2.12	-	13
15	Hahn (2014)	20 089	Annual household income (ref: ≥$100,000)	Self-report	Last year (Wave 1)	0					6
			<$10,000				OR: 1.35	0.81	2.26	-	
			$10,000-$29,999				OR: 1.55	0.98	2.45	-	
			$30,000-$49,999				OR: 1.34	0.84	2.13	-	
			$50,000-$64,999				OR: 1.46	0.91	2.34	-	
			$70,000-$99,999				OR: 0.74	0.43	1.26	-	
17	Jasinski (2001)	1 741 couples	Total couple income 'from all sources'	Partner and self-report	Last year (Wave 1)	1	OR: 1.00	0.00	-	NS	10
32	Caetano (2005)	1 136 couples	Couple income (ref: up to $10,000)	Self-report	Last year (Wave 1)	1					16
			$10,001–$20,000				OR: 3.50	0.77	16.03	-	
			$20,001–$30,000				OR: 0.70	0.19	2.91	-	
			$30,001–$40,000				OR: 1.00	0.24	4.60	-	
			$40,001 and more				OR: 3.90	0.88	17.62	-	

Note. Score is reliability score based on adapted Cambridge Quality Checklist. 'Ref' stands for referent category. OR is odds ratio. IRR is incidence rate ratio. Variables are bolded where p<.05.

Community factors

Table A72: **Neighbourhood disadvantage**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
2	Gomez (2011)	1 949	**Concentrated disadvantage index**	**Official records**	**Last year (Pre-study)**	**1**	**OR: 0.91**	**0.84**	**0.99**	**-**	**13**

17	Benson (2003)	3 006 couples	**Concentrated disadvantage index: Respondents who resided in advantaged neighborhoods in both waves=0. Those who moved out of a disadvantaged neighborhood in Wave 1 to an advantaged neighborhood in Wave 2=1. Those who resided in a disadvantaged neighborhood in Wave 2=2.**	**Official records, self-report**	**Current (Waves 1-2)**	**1**	**B: 0.31**	**0.13**	**-**	**<.05**	**11**
22	Jain (2010)	352	Concentrated poverty	Official records	Last year (1990 Census)	1	B: -0.02	0.33	-	NS	5
29	Giordano (2016)	506	Neighbourhood disadvantage: 10-item scale: 10 potential problems in neighbourhood (e.g., unemployment)	Parent report	Unclear (Wave 1)	1	OR: 0.98	0.86	1.10	-	20

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A73: Neighbourhood violence

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
19	Menard (2014)	393	Assaults and muggings were somewhat of a problem or a serious problem in the neighborhood	Parent and self-report	None (Waves 1 and 5-11)	1	B: -0.21	-	-	NS	6
22	Jain (2010)	352	Sum of 5 items assessing frequency of neighbourhood violence	Self-report	Last 6 months (Wave 1)	0	B: -0.20	0.33	-	NS	5

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A74: Residential stability

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
2	Gomez (2011)	1 949	Proportion of the population that has not moved since 1985 and houses that are owner occupied.	Official records	Last year (Pre-study)	1	OR: 0.94	0.85	1.05	-	13
14	Waltermaurer (2007)	19 610	**Lived in household for <6 months**	**Self-report**	**Last 6 months (Wave 1)**	**0**	**OR: 2.15**	**1.63**	**2.83**	**-**	**7**
17	Benson (2003)	3 006 couples	**Percent that had lived in current household for 5 years or less**	**Self-report**	**Last 5 years (Wave 2)**	**0**	**B: -2.02**	**0.84**	**-**	**<.05**	**11**

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A75: Economic disadvantage

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
17	Lanier (2009)	1 781 urban couples; 3 133 rural couples	County GINI index: Income inequality (0-100, where 100 indicates perfect inequality)	Official records	None (Between Waves 1-2: 1990 Census)	1	Rural poisson: -0.05 Urban poisson: 0.07	Rural poisson: 0.04 Urban poisson: 0.04	-	Rural poisson: NS Urban poisson: NS	14

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

Table A76: Immigrant concentration

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
2	Gomez (2011)	1 949	Proportion of population that are Latino and foreign born.	Official records	Last year (Pre-study)	1	OR: 0.91	0.77	1.08	-	13

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A77: **Social support**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
6	Lansford (2007)	131	Maternal social support during woman's childhood	Parent report	Last 5 years (Wave 1)	0	F: 2.24	-	-	NS	7
10	Huang (2010)	2 237	**Sum of positive responses to 3 items (e.g., whether the respondent could count on someone for money)**	**Self-report**	**Unclear (Wave 1)**	**0**	**OR: 0.82**	**0.07**	**-**	**<.05**	**9**
11	Staggs (2007)	825	Higher emotional and tangible social support	Self-report	Last year (Wave 2)	0	B: 0.01	0.02	NS	-	12
17	Benson (2003)	3 006 couples	Frequency that female indicated she had given help to or received help from anyone in any area	Self-report	Last month (Wave 1)	0	B: -0.04	0.04	-	NS	11

Note. Score is reliability score based on adapted Cambridge Quality Checklist. OR is odds ratio. Variables are bolded where p<.05.

Table A78: **Collective efficacy**

Cohort ID	First Author, Year	Sample size in analysis	Risk or protective factor	Informant	Time frame (Time point)	Score	Effect estimate	95% CI or SE		p-value (if no interval)	# Co-variates
22	Jain (2010)	352	Summary scale of social cohesion and informal social control	Self-report	None (Wave 1)	1	B: -0.34	0.28	-	NS	5

Note. Score is reliability score based on adapted Cambridge Quality Checklist. Variables are bolded where p<.05.

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Appendix B

Table B1: Polychoric correlations between ordinal IPV items

	Control	Humiliate	Push, slap	Punch, strangle	Coerced touch	Forced touch	Coerced sex	Forced sex
Control	1							
Humiliate	.764	1						
Push, slap	.728	.812	1					
Punch, strangle	.727	.786	.923	1				
Coerced touch	.592	.570	.630	.589	1			
Forced touch	.591	.569	.677	.649	.923	1		
Coerced sex	.627	.598	.640	.591	.882	.827	1	
Forced sex	.593	.602	.742	.720	.812	.890	.854	1

Note. N=3,158. Response categories for all variables were 0=never, 1=once, 2=a few times, and 3=often. Full item descriptions are shown in Table 1.

Table B2: Exploratory factor analysis (total sample)

	Single factor solution		Two-factor solution (r=64.97%)		
Item	**Factor 1**	**Uniqueness**	**Factor 1**	**Factor 2**	**Uniqueness**
Control	.771	.406	-	.705	.344
Humiliate	.792	.373	-	.842	.249
Push, slap	.881	.224	-	.877	.106
Punch, strangle	.855	.269	-	.905	.127
Coerced touch	.870	.243	.951	-	.098
Forced touch	.891	.207	.913	-	.092
Coerced sex	.866	.251	.862	-	.156
Forced sex	.898	.193	.770	-	.147
Factor eigenvalue:	**5.834**		**5.834**	**0.847**	
Proportion of variance explained	**.865**		**.865**	**.126**	

Note. N=3,158. Method is principal factors using a polychoric correlation matrix. Two-factor solution uses oblique rotation (promax). Factor loadings <.4 are suppressed. Full item descriptions are shown in Table 1.

Table B3: Exploratory factor analysis (women only)

	Single factor solution		Two-factor solution (r=63.86%)		
Item	**Factor 1**	**Uniqueness**	**Factor 1**	**Factor 2**	**Uniqueness**
Control	.786	.382	-	.714	.322
Humiliate	.805	.351	-	.863	.220
Push, slap	.886	.215	-	.887	.096
Punch, strangle	.860	.260	-	.928	.106
Coerced touch	.858	.264	.948	-	.107
Forced touch	.878	.230	.929	-	.093
Coerced sex	.860	.261	.853	-	.162
Forced sex	.892	.205	.755	-	.158
Factor eigenvalue:	**5.832**		**5.832**	**0.903**	
Proportion of variance explained	**.853**		**.853**	**.132**	

Note. N=2,050 women. Method is principal factors using a polychoric correlation matrix. Two-factor solution uses oblique rotation (promax). Factor loadings <.4 are suppressed. Full item descriptions are shown in Table 1.

Table B4: Exploratory factor analysis (men only)

Item	Single factor solution		Two-factor solution (r=59.60%)		
	Factor 1	Uniqueness	Factor 1	Factor 2	Uniqueness
Control	.790	.377	-	.751	.294
Humiliate	.749	.439	-	.896	.248
Push, slap	.869	.245	-	.739	.188
Punch, strangle	.776	.398	-	.934	.189
Coerced touch	.876	.233	.928	-	.086
Forced touch	.926	.142	.646	-	.132
Coerced sex	.822	.325	.876	-	.192
Forced sex	.822	.325	.992	-	.104
Factor eigenvalue:	**5.518**		**5.518**	**1.049**	
Proportion of variance explained	**.690**		**.690**	**.131**	

Note. N=1,108 men. Method is principal factors using a polychoric correlation matrix, forced to be positive definite. Two-factor solution uses oblique rotation (promax). Factor loadings <.4 are suppressed. Full item descriptions are shown in Table 1.

Table B5: Polychoric correlations between IPV impact items

	Frightened	Upset	Affect work	Sad	Anxious	Depressed	Substance use	Angry	No effect	Loved	Funny
Frightened	1										
Upset	.717	1									
Affect work	.621	.721	1								
Sad	.663	.893	.799	1							
Anxious	.701	.693	.724	.631	1						
Depressed	.646	.732	.776	.778	.747	1					
Substance use	.379	.400	.480	.406	.452	.531	1				
Angry	.343	.660	.318	.558	.401	.426	.297	1			
No effect	-.627	-.862	-.590	-.820	-.686	-.678	-.264	-.631	1		
Loved	-.178	-.468	-.162	-.376	-.148	-.164	-.018	-.301	.419	1	
Funny	-.478	-.522	-.318	-.422	-.472	-.454	-.093	-.392	.639	.432	1

Note. N=1,092. Participants only responded if they had experienced any IPV victimisation. Response categories for all variables were 0=no, 1=yes. Full item descriptions are shown in Table 2.

Table B6: Age of occurrence among those who had experienced IPV

Item	Total N	N (%) Under 18	Over 18	Both
Told you who you could see and where you could go and/or regularly checked what you were doing and where you were (by phone or text)	953	201 (21.09)	569 (59.71)	183 (19.20)
Made fun of you, called you hurtful names, shouted at you	1,022	171 (16.73)	666 (65.17)	185 (18.10)
Used physical force such as pushing, slapping, hitting or holding you down	743	137 (18.44)	468 (62.99)	138 (18.57)
Used more severe physical force such as punching, strangling, beating you up, hitting you with an object	481	70 (14.55)	293 (60.91)	118 (24.53)
Pressured you into kissing/touching/something else	569	109 (19.16)	337 (59.23)	123 (21.62)
Physically forced you into kissing/touching/something else	437	74 (16.93)	255 (58.35)	108 (24.71)
Pressured you into having sexual intercourse	631	135 (21.39)	371 (58.80)	125 (19.91)
Physically forced you into having sexual intercourse	413	65 (15.74)	244 (59.08)	104 (25.18)

Appendix C

Measures

Table C1: 2010 English Indices of Deprivation[1]

Domain	Indicators	Weight in Index (2015)
Income deprivation	Adults and children in Income Support families; Adults and children in income-based Jobseeker's Allowance families; Adults and children in income-based Employment and Support Allowance families; Adults and children in Pension Credit (Guarantee) families; Adults and children in Child Tax Credit and Working Tax Credit families, below 60% median income not already counted; Asylum seekers in England in receipt of subsistence support, accommodation support, or both.	22.5%
Employment deprivation	Claimants of Jobseeker's Allowance, aged 18-59/64; Claimants of Employment and Support Allowance, aged 18-59/64; Claimants of Incapacity Benefit, aged 18-59/64; Claimants of Severe Disablement Allowance, aged 18-59/64; Claimants of Carer's Allowance, aged 18-59/64.	22.5%
Education, skills, and training deprivation	Children and young people: Key stage 2 attainment: average points score; Key stage 4 attainment: average points score; Secondary school absence; Staying on in education post 16; Entry to higher education. Adults skills: Adults with no or low qualifications, aged 25-59/64; English language proficiency, aged 25-59/64.	13.5%
Health deprivation and disability	Years of potential life lost; Comparative illness and disability ratio; Acute morbidity; Mood and anxiety disorders.	13.5%
Crime	Recorded crime rates for: Violence; Burglary; Theft; Criminal damage.	9.3%
Barriers to housing and services	Geographical barriers: Road distance to: post office, primary school, general store/supermarket, GP surgery. Wider barriers: Household overcrowding; Homelessness; Housing affordability.	9.3%
Living environment deprivation	Indoor living environment: Housing in poor condition; Houses without central heating. Outdoor living environment: Air quality; Road traffic accidents.	9.3%

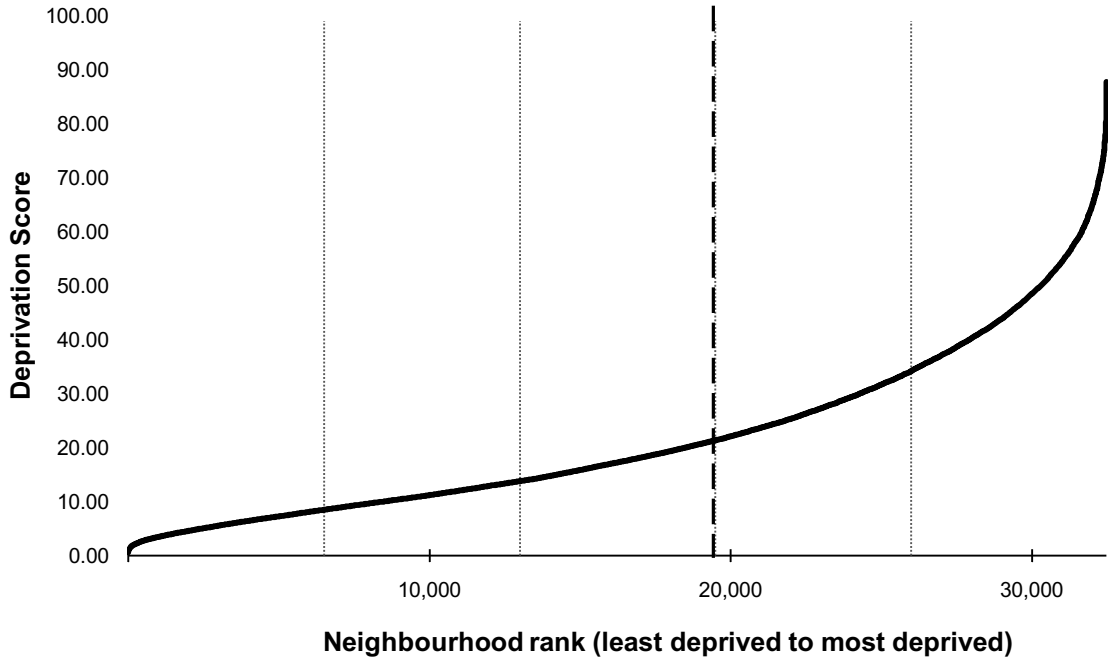

Figure C1: Exponential distribution of 2010 Indices of Multiple Deprivation scores by neighbourhood (lower-layer super output area) rank. The fine dashed lines indicate quintile markers. Neighbourhoods to the right of the thick dashed line are the 40% most deprived neighbourhoods in England. Data are from the official English Indices of Multiple Deprivation 2010 (available at: https://www.gov.uk/government/statistics/english-indices-of-deprivation-2010); further details on the measure and its construction are in the English Indices of Multiple Deprivation 2010 technical report.[1]

Table C2: Items used in composite perceived neighbourhood variables

	Poor social cohesion (0-4)
1	Neighbourhood people visiting home
2	Neighbourhood people argue with mum
3	Neighbourhood people look after mums CH
4	Neighbourhood people keep to themselves
5	Mum visits neighbours home
6	Mum argues with neighbours
7	Mum looks after neighbours CH
	Neighbourhood disorder (0-2): until age 10
1	Badly fitted doors and windows*
2	Poor ventilation*
3	Noise in rooms of home*
4	Noise from other homes
5	Noise from outside
6	Rubbish dumped in neighbourhood
7	Dog dirt on pavement is problem
8	Worry about vandalism
9	Worry about burglaries
10	Worry about attacks
11	Disturbance from youths
	Neighbourhood disorder (0-2): age 18
1	Noise from other homes
2	Noise from outside in the street
3	Rubbish or litter dumped around neighbourhood
4	Dog dirt on pavement/walkways
5	Worry about vandalism
6	Worry about burglaries
7	Worry about muggings or attacks
8	Disturbance from teenagers or youths
9	Traffic**
10	Parking**

*Item removed at age 18
**Item added at age 18

Sociodemographic and psychosocial factors

Variables were selected based on the literature and data availability in ALSPAC. I used the following sociodemographic and psychosocial variables reported by mothers at baseline. Parental education was coded as 1=at least the mother or her partner had higher than standard high school qualifications (i.e., higher than O-level – A-level or degree), 0=otherwise. Maternal marital status was coded as 1=married, 0=otherwise. Parental social class was coded using the standard occupational classification 2000, where 1=at least mother or partner were part of lower social class (partly or unskilled occupations), 0=otherwise (professional, managerial, or skilled occupations). Mothers' race/ethnicity was dichotomised as 1=non-White, 0=White due to the lower proportion of non-White race/ethnicities (e.g., Asian, black) in the sample. Maternal depressive symptoms were measured using the Edinburgh Post-Natal Depression Scale,[2] a 10-item scale which asks about the frequency of positive and negative behaviours/emotions in the last seven days (e.g., 'I have been anxious or worried for no good reason') on a 4-point scale from 0=never to 3=often. Items were all coded in the negative direction and summed so that higher scores indicate more depressive symptoms (α=.85). Residential mobility was measured based on mothers' reports of whether they had recently moved house before the baseline assessment (since becoming pregnant). Maternal social support was measured using a Social Network Index developed for ALSPAC, where mothers reported on 10 social situations (e.g., 'How many of your relatives or your partner's relatives do you see at least twice a year?'). Response categories were 0=none to 3=more than 4 and items were summed so that higher scores indicated a stronger social network (α=.79). Mothers reported on their difficulty in affording each of five items – food, clothing, heating, accommodation, or items for their child(ren) – on a 4-point scale from 0=not difficult to 3=very difficult. Responses were summed to create a composite for financial difficulties.

Dual trajectory analysis: Negative neighbourhood opinion (logit) and deprived neighbourhood exposure (binary IMD, logit model)

Table C3: Maximum likelihood parameter estimates for trajectory groups

Group	Parameter	Estimate (SE)	p-value
Negative neighbourhood opinion (logit)			
1 Stable positive opinion	Intercept	-1.37 (0.06)	<.001
	Linear	-0.30 (0.03)	<.001
	Quadratic	0.02 (0.00)	<.001
2 Moderate opinion	Intercept	-0.33 (0.11)	.003
	Linear	0.10 (0.03)	<.001
	Quadratic	-0.01 (0.00)	<.001
3 Improving opinion	Intercept	2.73 (0.24)	<.001
	Linear	-0.67 (0.07)	<.001
	Quadratic	0.02 (0.00)	<.001
4 Negative opinion with improvement	Intercept	2.18 (0.09)	<.001
	Linear	0.13 (0.03)	<.001
	Quadratic	-0.01 (0.00)	<.001
More deprived neighbourhood exposure (logit)			
1 Stable low deprivation	Intercept	-3.14 (0.07)	<.001
	Linear	-0.93 (0.06)	<.001
	Quadratic	0.05 (0.00)	<.001
2 Decreasing deprivation	Intercept	3.36 (0.14)	<.001
	Linear	-0.70 (0.04)	<.001
	Quadratic	0.01 (0.00)	<.001
3 Increasing deprivation	Intercept	-3.16 (0.19)	<.001
	Linear	0.61 (0.05)	<.001
	Quadratic	-0.02 (0.00)	<.001
4 Chronic high deprivation	Intercept	2.50 (0.10)	<.001
	Linear	0.59 (0.05)	<.001
	Quadratic	-0.03 (0.00)	<.001

Table C4: Diagnostics of models

Group (N)	Average posterior probability of group membership	Odds of correct classification	Estimated probability of group membership (observed probability)
Negative neighbourhood opinion (logit)			
1 Stable positive opinion (3666)	.84	16.71	.237 (.223)
2 Moderate opinion (2259)	.69	13.37	.146 (.172)
3 Improving opinion (1804)	.67	15.65	.117 (.123)
4 Negative opinion with improvement (3541)	.81	14.37	.229 (.212)
More deprived neighbourhood exposure (logit)			
1 Stable low deprivation (7386)	.97	33.56	.478 (.466)
2 Decreasing deprivation (1047)	.93	181.92	.068 (.082)
3 Increasing deprivation (459)	.92	370.65	.030 (.036)
4 Chronic high deprivation (2378)	.91	54.00	.154 (.145)

Dual trajectory analysis: Poor social cohesion (censored normal model) and deprived neighbourhood exposure (binary IMD, logit model)

Table C5: Maximum likelihood parameter estimates for trajectory groups

Group	Parameter	Estimate (SE)	p-value
Poor social cohesion score (censored normal)			
1 Strong to moderate cohesion	Intercept	1.53 (0.01)	<.001
	Linear	-0.06 (0.00)	<.001
	Quadratic	0.00 (0.00)	<.001
2 Moderate to weak cohesion	Intercept	1.78 (0.02)	<.001
	Linear	0.03 (0.00)	<.001
3 Weak cohesion with improvement	Intercept	2.34 (0.01)	<.001
	Linear	-0.14 (0.00)	<.001
	Quadratic	0.001 (0.00)	<.001
4 Weak cohesion	Intercept	2.59 (0.01)	<.001
	Linear	-0.034 (0.00)	<.001
	Quadratic	0.00 (0.00)	<.001
More deprived neighbourhood exposure (logit)			
1 Stable low deprivation	Intercept	-3.13 (0.07)	<.001
	Linear	-0.92 (0.06)	<.001
	Quadratic	0.04 (0.00)	<.001
2 Decreasing deprivation	Intercept	3.32 (0.14)	<.001
	Linear	-0.69 (0.04)	<.001
	Quadratic	0.01 (0.00)	<.001
3 Increasing deprivation	Intercept	-3.16 (0.19)	<.001
	Linear	0.61 (0.04)	<.001
	Quadratic	-0.02 (0.00)	<.001
4 Chronic high deprivation	Intercept	2.47 (0.10)	<.001
	Linear	0.62 (0.05)	<.001
	Quadratic	-0.04 (0.00)	<.001

Table C6: Diagnostics of models

Group (N)	Average posterior probability of group membership	Odds of correct classification	Estimated probability of group membership (observed probability)
Poor social cohesion score (censored normal)			
1 Strong to moderate cohesion (2584)	.85	28.63	.167 (.165)
2 Moderate to weak cohesion (1599)	.70	20.23	.104 (.125)
3 Weak cohesion with improvement (3937)	.74	8.17	.255 (.238)
4 Weak cohesion (3150)	.86	23.71	.204 (.201)
More deprived neighbourhood exposure (logit)			
1 Stable low deprivation (7405)	.97	32.55	.479 (.466)
2 Decreasing deprivation (1014)	.95	246.76	.066 (.083)
3 Increasing deprivation (460)	.91	350.22	.030 (.036)
4 Chronic high deprivation (2391)	.90	50.37	.155 (.145)

Dual trajectory analysis: Neighbourhood disorder (censored normal model) and deprived neighbourhood exposure (binary IMD, logit model)

Table C7: Maximum likelihood parameter estimates for trajectory groups

Group	Parameter	Estimate (SE)	p-value
Neighbourhood disorder (censored normal)			
1 Stable low disorder	Intercept	0.21 (0.01)	<.001
	Linear	-0.02 (0.00)	<.001
	Quadratic	0.00 (0.00)	<.001
2 Moderate disorder	Intercept	0.55 (0.01)	<.001
	Linear	-0.03 (0.00)	<.001
	Quadratic	0.00 (0.00)	<.001
3 High disorder with improvement	Intercept	1.08 (0.02)	<.001
	Linear	-0.04 (0.00)	<.001
	Quadratic	0.00 (0.00)	<.001
More deprived neighbourhood exposure (logit)			
1 Stable low deprivation	Intercept	-3.12 (0.07)	<.001
	Linear	-0.91 (0.06)	<.001
	Quadratic	0.04 (0.00)	<.001
2 Decreasing deprivation	Intercept	3.39 (0.14)	<.001
	Linear	-0.71 (0.04)	<.001
	Quadratic	0.01 (0.00)	<.001
3 Increasing deprivation	Intercept	-3.21 (0.19)	<.001
	Linear	0.62 (0.04)	<.001
	Quadratic	-0.02 (0.00)	<.001
4 Chronic high deprivation	Intercept	2.48 (0.10)	<.001
	Linear	0.59 (0.05)	<.001
	Quadratic	-0.03 (0.00)	<.001

Table C8: Diagnostics of models

Group (N)	Average posterior probability of group membership	Odds of correct classification	Estimated probability of group membership (observed probability)
Neighbourhood disorder (censored normal)			
1 Stable low disorder (6470)	.90	11.83	.419 (.407)
2 Moderate disorder (3974)	.85	15.91	.257 (.266)
3 High disorder with improvement (826)	.91	183.48	.053 (.057)
More deprived neighbourhood exposure (logit)			
1 Stable low deprivation (7315)	.97	34.12	.478 (.466)
2 Decreasing deprivation (1029)	.94	212.07	.066 (.082)
3 Increasing deprivation (438)	.92	361.41	.029 (.035)
4 Chronic high deprivation (2318)	.90	50.07	.156 (.146)

Additional data on the relationship between perceived and objective neighbourhood trajectory groups

Figure C2 and Table C9 summarise additional data from the dual trajectory analyses comparing objective neighbourhood deprivation and each perceived neighbourhood measure. In particular, Figure C2 shows the proportion of participants in each perceived measure trajectory group by their objective deprivation trajectory group. Table C9 shows the most common group pairings across the objective and perceived measures in the total sample.

Neighbourhood opinion and objective neighbourhood deprivation

Panel A of Figure C2 shows that, as expected, the majority of mothers (57%-92%) who held more positive opinions of their neighbourhoods over time (i.e., members of the moderate, improving, and stable positive opinion trajectory groups) experienced stable exposure to lower neighbourhood deprivation over time. Likewise, participants with the most negative opinion of their neighbourhood over the study period were more likely to experience chronic exposure to greater neighbourhood deprivation (45%). Additionally, among participants whose opinions of their neighbourhoods improved over time, 31% had congruently moved to less deprived neighbourhoods over the study period. Despite these areas of overlap, however, the dual trajectory analysis also highlighted important areas of divergence. For instance, 37% of participants in the negative opinion group were surprisingly in the stable low neighbourhood deprivation group.

As shown in Panel A of Table C9, participants were most commonly members of both the stable low objective deprivation group and stable positive opinion group (28%) – demonstrating convergence between the two measures. Likewise, 13% of all participants in the sample experienced chronic exposure to high neighbourhood deprivation and held more negative opinions of their neighbourhoods over the study period. However, the two measures diverged as well: for instance, 11% of participants were exposed to stable low deprivation but held more negative opinions of their neighbourhoods over time.

Perceived social cohesion and objective neighbourhood deprivation

As shown in Panel B in Figure C2, within each social cohesion trajectory group, there was a similar pattern in the distribution of membership across the objective deprivation trajectory groups, suggesting a weaker relationship between these two variables. Specifically, the highest proportion of participants in each social cohesion group belonged to the stable low neighbourhood deprivation group, followed by: chronic high deprivation, decreasing deprivation, and increasing deprivation. However, providing some evidence for a negative correlation between objective deprivation and perceived social coheison, participants in the most negative perceived social cohesion group (weak cohesion) were most often in the chronic high deprivation group (28%) and least often in the stable low deprivation group (49%). Likewise, participants in the most positive social cohesion group (strong to moderate) were most often in the stable low deprivation group (78%) and least often in the chronic high deprivation group (13%).

Panel B in Table C9 shows that the most common group pairings between social cohesion and neighbourhood deprivation all involved the stable low deprivation group (11%-22% of the sample), demonstrating no clear relation between the two variables. More in line with expectations of a negative correlation between objective deprivation and perceived social cohesion, the next most common group pairing among participants was between weak social cohesion and chronic high deprivation (8% of the sample).

Perceived neighbourhood disorder and objective neighbourhood deprivation

Panel C in Figure C2 shows that the majority of participants (78%) who followed a trajectory of low perceived neighbourhood disorder were exposed to stable low neighbourhood deprivation over

time. Similarly, most participants (62%) who perceived high neighbourhood disorder over time were in the chronic high deprivation group. However, unlike the objective neighbourhood measure trajectory groups, no group of participants experienced significant changes in their perceptions of neighbourhood disorder over time; participants who perceived moderate levels of disorder tended to be in either the stable low (53%) or chronic high deprivation groups (28%). Furthermore, one-fifth of participants who perceived higher neighbourhood disorder were conversely in the stable low deprivation group.

Panel C in Table C9 further demonstrates a positive relationship between objective neighbourhood deprivation and perceived neighbourhood disorder, with some areas of divergence. Demonstrating a strong relationship, the most common group pairing was by far stable low objective deprivation and stable low perceived disorder (43% of the sample). Demonstrating divergence, the next most common pairings were between perceived moderate disorder and each of low objective deprivation (19%) and high objective deprivation (10%).

Figure C2: Percentage of participants in each perceived neighbourhood measure trajectory group conditional on objective neighbourhood trajectory group membership

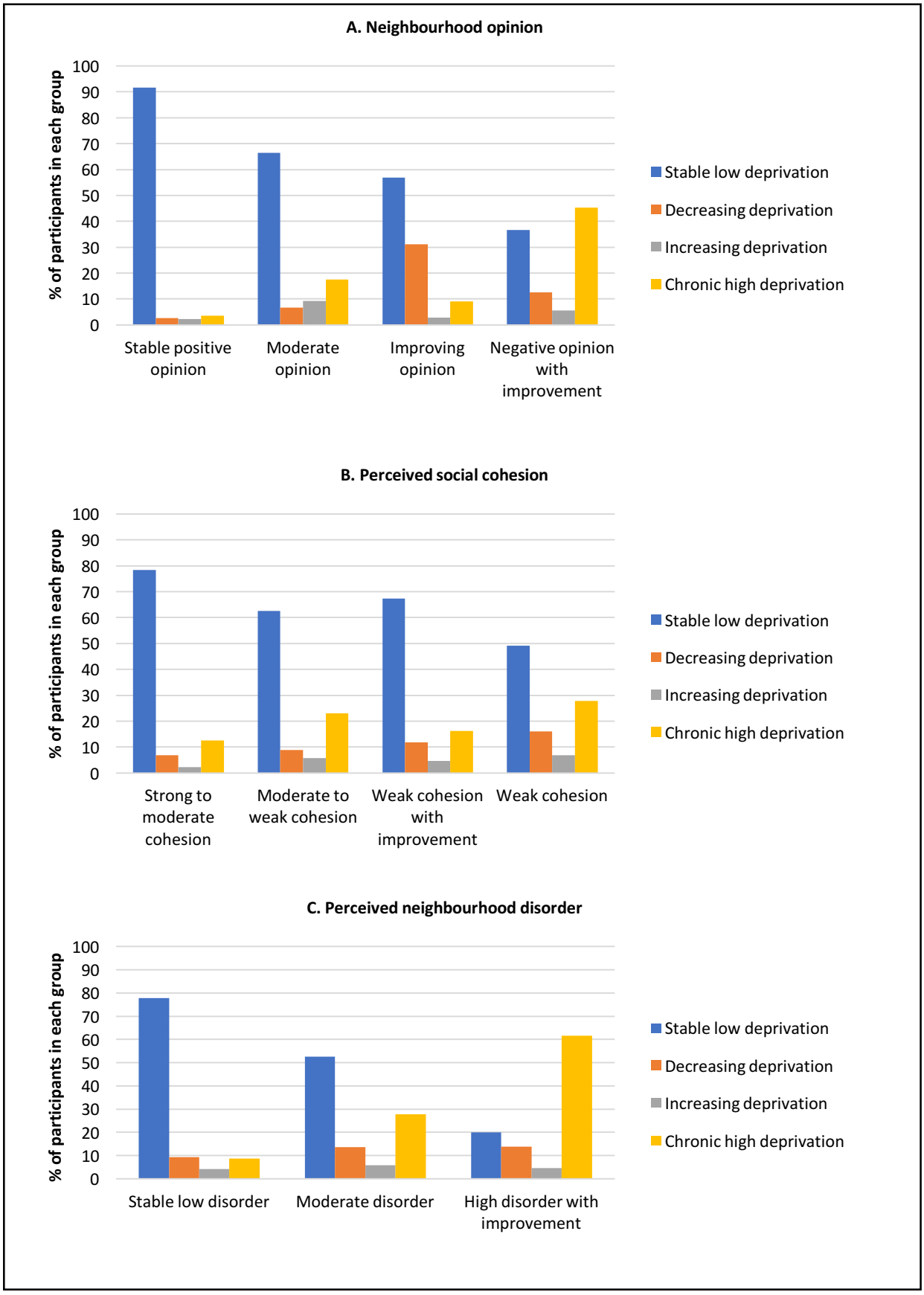

Table C9: Percentage of sample that belongs to each possible pairing of the objective neighbourhood trajectory groups and each of the perceived measure trajectory groups

		Objective deprived neighbourhood exposure			
		1 Stable low deprivation	2 Decreasing deprivation	3 Increasing deprivation	4 Chronic high deprivation
A. Negative neighbourhood opinion	**1 Stable positive opinion**	28.1	0.8	0.7	1.1
	2 Moderate opinion	15.6	1.6	2.2	4.1
	3 Improving opinion	9.6	5.3	0.5	1.5
	4 Negative opinion with improvement	10.6	3.6	1.6	13.1
B. Poor social cohesion score	**1 Strong to moderate cohesion**	17.8	1.5	0.5	2.8
	2 Moderate to weak cohesion	10.7	1.5	1.0	3.9
	3 Weak cohesion with improvement	22.0	3.9	1.5	5.3
	4 Weak cohesion	13.5	4.4	1.9	7.7
C. Neighbourhood disorder score	**1 Stable low disorder**	43.4	5.3	2.3	4.8
	2 Moderate disorder	19.2	5.0	2.1	10.1
	3 High disorder with improvement	1.5	1.1	0.3	4.7

Note. Each panel (A, B, and C) adds to 100%.

References

1 Lad M. The English Indices of Multiple Deprivation 2010. London: Department for Communities and Local Government; 2011.
2 Cox JL, Holden JM, Sagovsky R. Detection of postnatal depression: Development of the 10-item Edinburgh Postnatal Depression Scale. British Journal of Psychiatry. 1987;150:782-6.

Appendix D

Measures

Indices of Multiple Deprivation

As described in Chapter 6, the Indices of Multiple Deprivation are an official measure of area-level deprivation in England, which considers deprivation beyond economic poverty alone, using indicators across seven domains: income, employment, education, health, crime, housing, and living environment.[1-3] Table D1 describes the indicators used to construct each domain of deprivation and the domain's weight in the final index. Based on these indicators, each lower-layer super output area (~1500 residents or 650 households designed to approximate residential neighbourhoods) is assigned a domain-specific and total deprivation rank score *relative* to all other neighbourhoods. A neighbourhood is thus defined as more or less deprived based on the extent of inequality in its living conditions compared to other neighbourhoods in England (i.e., *relative* deprivation) as opposed to whether it falls above or below an objective standard of conditions (i.e., *absolute* deprivation). The Indices of Multiple Deprivation is constructed using an exponential transformation of neighbourhoods' rank scores; this standardises the scores for different indicators, allowing them to be combined, and makes the most deprived neighbourhoods easier to identify: the 10% most deprived neighbourhoods make up 50% of the score distribution (i.e., have a transformed-rank score between 50-100). See Figure 6.1 in Chapter 6 for a summary of this distribution and the technical reports of the Indices of Multiple Deprivation for further details on the measure's construction and distribution.[1,3,4]

Table D1: **2010 English Indices of Deprivation[1]**

Domain	Indicators	Weight in Index (2015)
Income deprivation	Adults and children in Income Support families; Adults and children in income-based Jobseeker's Allowance families; Adults and children in income-based Employment and Support Allowance families; Adults and children in Pension Credit (Guarantee) families; Adults and children in Child Tax Credit and Working Tax Credit families, below 60% median income not already counted; Asylum seekers in England in receipt of subsistence support, accommodation support, or both.	22.5%
Employment deprivation	Claimants of Jobseeker's Allowance, aged 18-59/64; Claimants of Employment and Support Allowance, aged 18-59/64; Claimants of Incapacity Benefit, aged 18-59/64; Claimants of Severe Disablement Allowance, aged 18-59/64; Claimants of Carer's Allowance, aged 18-59/64.	22.5%
Education, skills, and training deprivation	Children and young people: Key stage 2 attainment: average points score; Key stage 4 attainment: average points score; Secondary school absence; Staying on in education post 16; Entry to higher education. Adults skills: Adults with no or low qualifications, aged 25-59/64; English language proficiency, aged 25-59/64.	13.5%
Health deprivation and disability	Years of potential life lost; Comparative illness and disability ratio; Acute morbidity; Mood and anxiety disorders.	13.5%
Crime	Recorded crime rates for: Violence; Burglary; Theft; Criminal damage.	9.3%
Barriers to housing and services	Geographical barriers: Road distance to: post office, primary school, general store/supermarket, GP surgery. Wider barriers: Household overcrowding; Homelessness; Housing affordability.	9.3%
Living environment deprivation	Indoor living environment: Housing in poor condition; Houses without central heating. Outdoor living environment: Air quality; Road traffic accidents.	9.3%

Time-invariant covariates

All covariates were measured by mother-report, with time-invariant covariates measured at baseline, unless otherwise noted. Parental education was coded as 1=at least the mother or her partner had higher than O-level (A-level or degree), 0=otherwise. Maternal marital status was coded as 1=married, 0=otherwise (including widowed, divorced, separated, or never married). Parental social class was coded using the standard occupational classification 2000, where 1=at least mother or partner were part of lower social class (partly or unskilled occupations), 0=otherwise (professional, managerial, or skilled occupations). Mothers reported on their own, their partners', and their parents' race/ethnicities at baseline and on the young person's race/ethnicity when the child was age 11.5. To use all available data, participants coded as 'non-white' at age 11.5 were coded as 'non-white' on the final ethnicity variable and, in addition, those who were missing at the age 11.5 assessment but whose mothers, fathers, grandmothers, or grandfathers were reported as 'non-white' were also coded as such. Race/ethnicity was dichotomised as 1=non-white, 0=white because of the lower proportion of non-white race/ethnicities (e.g., Asian, Black) in the sample. I also used the mother's number of children at baseline as a time-invariant covariate.

Time-varying covariates

Maternal depressive symptoms were measured using the Edinburgh Post-Natal Depression Scale, a 10-item scale which asks about positive and negative behaviours/emotions in the last seven days (e.g., 'I have been anxious or worried for no good reason').[5] Response categories were 0 'Never' to 3 'Often'. Items were all coded in the negative direction and summed so that higher scores indicate more depressive symptoms (α=.85). Residential mobility was measured based on mothers' reports of whether they had moved house between questionnaire assessments. Maternal social support was measured using a Social Network Index developed for ALSPAC, where mothers reported on 10 social situations (e.g., 'How many of your relatives or your partner's relatives do you see at least twice a year?'). Response categories were 0 'None' to 3 'More than 4' and items were summed so that higher scores indicated a stronger social network (α=.79). Parental employment was typically indicated by mother-report of whether her partner was currently employed, except at baseline, when mothers reported on whether they themselves were currently employed. Family structure was coded as 1=both biological parents live with child, 0=only one or neither biological parent lives with child. Mothers reported on their difficulty in affording each of five items – food, clothing, heating, accommodation, or items for their child(ren) – on a 4-point scale from 0=not difficult to 3=very difficult. Responses were summed to create a composite for financial difficulties. Mothers reported on the take-home family income as a weekly average, apart from when children were age 18 when the monthly average was reported. Response categories varied over time, as expected with inflation, from baseline (weekly average, 1=<£100 to 5=>£400) to age 18 (monthly average, 1=<£899 to 10=>£4000).

Computing the stabilised inverse probability weights and estimating marginal structural models

Constructing inverse probability weights involves estimating a denominator that is the product of the probabilities that each participant received the exposure she actually did at time t conditional on prior exposure up until time t-1, time-varying covariates up until time t or time t-1 (depending on the study), and baseline covariates (including time-invariant covariates).[6-9] In practice, inverse probability weights tend to be very variable; thus stabilised weights are typically used for more efficient effect estimates.[7,9] Stabilised weights involve estimating a numerator as well, which is typically a function of prior exposure and baseline covariates (i.e., excluding time-varying covariate history beyond baseline). The analysis in the weighted sample (i.e., marginal structural model) is then conducted conditioning on the baseline covariates.

In Chapter 6, to compute the denominator of the stabilised weights, I regressed the level of neighbourhood deprivation at time k (A_k) onto the level of previous exposure (A_{k-1}) and time-varying covariates (L_{k-1}) at time k-1 and baseline covariates (X and L_0) using a pooled binary logistic regression in a long dataset (i.e., with participant-observations as the unit of analysis).[8,9] The only

exception was for the weights constructed for T_9, which used time-varying covariates measured at T_9 because none were measured at T_8. I then estimated the predicted probabilities of exposure from the corresponding regression and derived participants' probabilities of their *observed* exposure status. The denominator for the final exposure weight at each time t is then the product of probabilities up until time t (i.e., the estimated probability of participants' observed exposure histories up until time t conditional on prior exposure and covariate history up until time t-1). In a wide dataset, where the unit of analysis is participants, this final weight would equate to the product of probabilities over all time points. I computed the numerator in the same way as the denominator but excluding the time-varying covariates beyond baseline (L_{k-1}) from the regression. The final form for the stabilised weights for the *i*th participant was thus:

$$SW_i = \prod_{k=1}^{K} \frac{p(A_{ki} = \alpha_{ki} | A_{(k-1)i} = \bar{\alpha}_{(k-1)i}, L_{0i} = l_{0i}, X_i = x_i)}{p(A_{ki} = \alpha_{ki} | \bar{A}_{(k-1)i} = \bar{\alpha}_{(k-1)i}, \bar{L}_{(k-1)i} = \bar{l}_{(k-1)i}, X_i = x_i)}$$

I estimated censoring (i.e., permanent attrition) weights (and where relevant weights for intermittent missingness) in the same way as the exposure weights, but instead predicting the probability of being censored (or missing). The final weights were the product of the stabilised exposure and censoring weights (and, where relevant, intermittent missingness weights).

As in any longitudinal analysis of an exposure-outcome association, estimating marginal structural models further requires defining the exposure trajectories (and potential outcomes) of interest. In simple settings (e.g., two time points of binary exposure), marginal structural models can be estimated non-parametrically, where the expected outcomes are compared for each possible exposure trajectory (e.g., $E(Y_{0,0})$, $E(Y_{0,1})$, $E(Y_{1,0})$, and $E(Y_{1,1})$).[7,9] However, in more complex settings, for instance due to many more time points, parametric models are required. In Chapter 6, with 10 time points of binary exposure data, there were 2^{10} or 1,024 potential exposure trajectories. I thus estimated parametric marginal structural models. Based on prior marginal structural model studies of neighbourhood disadvantage, I used a cumulative or duration-weighted exposure, defined as the average exposure over the study period ($\sum_{k=0}^{9} a_k /10$).[10,11] As noted by these studies and the broader neighbourhood effects literature, this specification is of theoretical interest because it allows for estimation of the effects of sustained exposure to neighbourhood deprivation: for instance, in Chapter 6, differences in IPV risk between women who spent more of their childhood in the most deprived neighbourhoods in England versus the least.

I estimated two primary marginal structural models in my weighted sample. One was a negative binomial regression, where the discrete IPV frequency score (Y) was modelled as a function of duration-weighted exposure to more severe neighbourhood deprivation ($\bar{\alpha}$) and (time-invariant and time-varying) covariates measured at baseline (X and L_0):

$$log[P(Y_{\bar{\alpha}i} = y_{\bar{\alpha}i} | X_i = x_i, L_{0i} = l_{0i})] = \theta_0 + \theta_1 \left(\frac{\sum_{k=0}^{9} a_{ki}}{10} \right)$$

The second was a log-binomial model, where the risk of experiencing any IPV (Y) was modelled as a function of duration-weighted exposure to more severe neighbourhood deprivation ($\bar{\alpha}$) and time-invariant/baseline factors (X and L_0):

$$log[P(Y_{\bar{\alpha}i} = 1) | X_i = x_i, L_{0i} = l_{0i}] = \theta_0 + \theta_1 \left(\frac{\sum_{k=0}^{9} a_{ki}}{10} \right)$$

Typically, when the outcome is an end-of-study outcome as opposed to repeated measures, researchers will conduct their marginal structural models in a wide dataset, where each participant is weighted by the product of the time-specific weights.[7,11-14] This is analogous to the practice used in repeated-measures marginal structural models, where researchers weight each participant-observation

at time k by the product of the time-specific weights up until time k.[8,10,13,15] However, in a wide dataset, this means that participants without complete data at all time points are listwise deleted. In the current study, this would have resulted in an analytic sample of n<200 participants. Therefore, to make the most of the available data, I conducted my analyses at the time-point level (i.e., in a long dataset), where each participant-observation was weighted by the appropriate time-varying weight (i.e., the cumulative probability of observed exposure and censoring history until time t) with cluster-robust (conservative) standard errors. This allowed me to include participants who did not have complete exposure and covariate data at all time points, but did have at least 50% exposure data, IPV data, and time points with complete covariate data (see Figure D1 for further details). Participant observations are resultantly included in the analysis up until the time point with missing data (i.e., when the time-specific weight is missing). I explored alternative strategies in my sensitivity analyses.

STATA syntax for basic marginal structural model analysis

```
/*Denominator: computing pooled logistic regression to predict total IMD using only variables measured at all time points */
set more off
logit imd_b i.imd_b1 i.moved1 i.ptnremploy1 epds1 epds0 i.moved0 i.mumemploy0 i.eduparents0 i.mummarried0 nochild0 i.ethnicity0 i.parentsc0

*Predicting probabilities from logit for the estimation subsample
predict pimd if e(sample)
replace pimd=pimd*imd_b+(1-pimd)*(1-imd_b)

/*Creating cumulative probabilities (multiplying each probability by the one before it, except for the first probability), clustered by participant id */
sort id time
by id: replace pimd=pimd*pimd[_n-1] if _n!=1

/*Numerator: computing pooled logistic regression to predict total IMD using only variables measured at all time points */
set more off
logit imd_b i.imd_b1 epds0 i.moved0 i.mumemploy0 i.eduparents0 i.mummarried0 nochild0 i.ethnicity0 i.parentsc0

predict pimd0 if e(sample)
replace pimd0=pimd0*imd_b+(1-pimd0)*(1-imd_b)

sort id time
by id: replace pimd0=pimd0*pimd0[_n-1] if _n!=1

/* Calculating weights (stabilised and unstabilised)

*Non-stabilised
gen ipt_w=1/pimd
summarize ipt_w, detail

*Stabilised
gen ipt_sw=pimd0/pimd
summarize ipt_sw, detail

*Checking distribution of weights
summarize ipt_w, detail
summarize ipt_sw, detail
```

```
/*Now creating censoring weights*/

/*Denominator: computing pooled binary logistic regression to predict censoring using only variables measured at all time points*/
set more off
logit censor i.imd_b1 1 i.moved1 i.ptnremploy1 epds1 epds0 i.moved0 i.mumemploy0 i.eduparents0 i.mummarried0 nochild0 i.ethnicity0 i.parentsc0

*Predicting probabilities for the estimation subsample
predict pcens if e(sample)

*Calculating probability of being uncensored
replace pcens=1-pcens

/*Calculating cumulative probabilities (multiplying each probability by the one before it, except for the first probability), clustered by participant id*/
sort id time
by id: replace pcens=pcens*pcens[_n-1] if _n!=1

/*Numerator: computing pooled binary logistic regression to predict censoring using only variables measured at all time points */
set more off
logit censor i.imd_b1 epds0 i.moved0 i.mumemploy0 ///
i.eduparents0 i.mummarried0 nochild0 i.ethnicity0 i.parentsc0

*Predicting probabilities for the estimation subsample
predict pcens0 if e(sample)

*Calculating probability of being uncensored
replace pcens0=1-pcens0

*Calculating cumulative probabilities
sort id time
by id: replace pcens0=pcens0*pcens0[_n-1] if _n!=1

*Calculating weights (stabilised and unstabilised)
gen cens_w=1/pcens
gen cens_sw=pcens0/pcens

*checking distribution of weights
summarize cens_w, detail
summarize cens_sw, detail

*Creating total weight (product of exposure and censoring weight)
gen w=ipt_w*cens_w
gen sw=ipt_sw*cens_sw

*Checking distribution of weights
summarize w, detail
summarize sw, detail

/*Estimating parameters from pooled negative binomial regression or log-binomial generalised linear model (weighted by sw)*/
glm v_ freqsum18 imd_cum epds0 i.moved0 i.mumemploy0 i.eduparents0 ///
```

```
i.mummarried0 nochild0 i.ethnicity0 i.parentsc0 [pw=sw], cluster(id) family(nbinomial ml) link(log)
eform

glm v_18freqallb imd_cum epds0 i.moved0 i.mumemploy0 i.eduparents0 ///
i.mummarried0 nochild0 i.ethnicity0 i.parentsc0 [pw=sw], cluster(id) family(binomial) link(log)
eform
```

Longitudinal variation in exposure to neighbourhood-level deprivation over time

Figure D1 shows the proportion of the sample living in the most deprived (IMD quintiles 4 and 5) versus the least deprived (IMD quintiles 1-3) neighbourhoods over the exposure period. Over time, the proportion of participants in the most deprived neighbourhoods decreased, likely influenced, at least in part, by non-random attrition.

Figures D2-D4 demonstrate the longitudinal variation in exposure to neighbourhood-level deprivation within participants. Figure D2 compares participants' IMD quintiles at gestation (baseline) and age 18. It shows that within each IMD quintile at baseline, 31-51% of participants were in a different IMD quintile by age 18. Figure D3 shows the absolute sum of changes in the level of exposure to neighbourhood-level deprivation (IMD quintile) experienced by participants over the study period: 46% of participants experienced any change in their IMD quintile over the study period. Finally, Figure D4 summarises the net or directional change in participants IMD quintile over the study period: 26% of participants experienced a net decrease and 16% a net increase in the severity of their neighbourhood deprivation exposure.

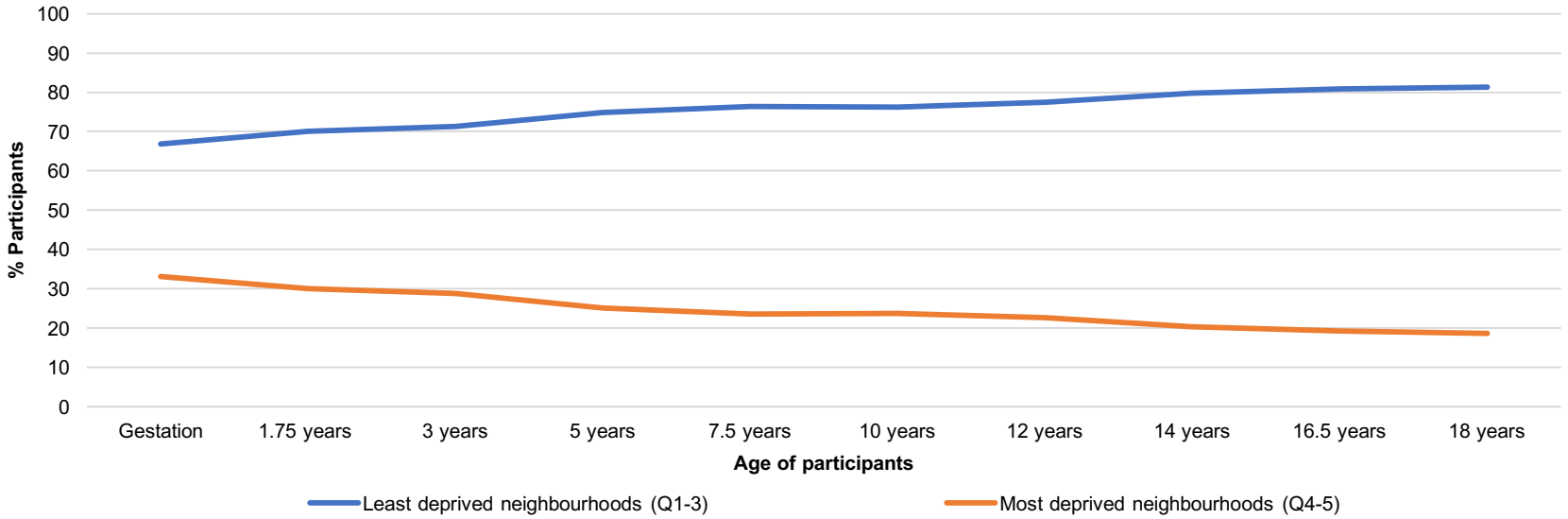

Figure D1: **Percent of participants by exposure to most versus least deprived residential neighbourhoods during the period of exposure under study**

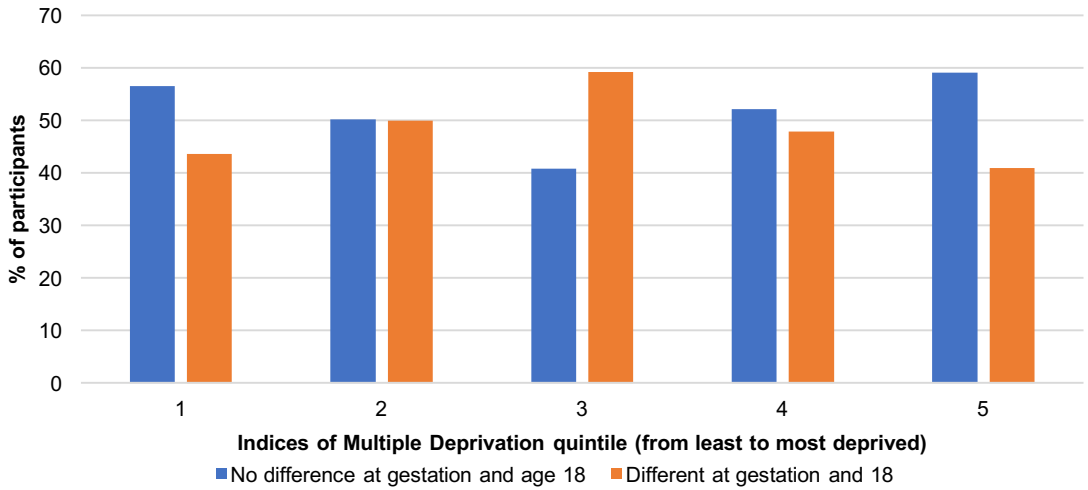

Figure D2: **Percent of participants whose level of exposure to neighbourhood deprivation at gestation was different at age 18**. N=1,756 women with complete IMD data at gestation (baseline) and age 18.

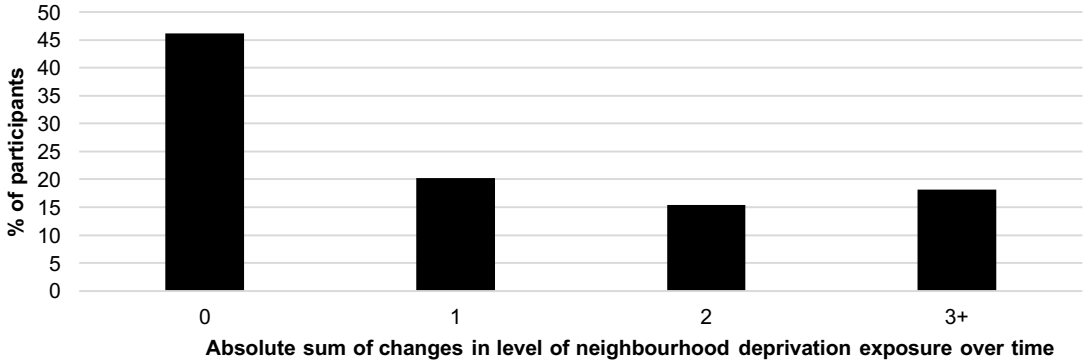

Figure D3: **Percent of participants by absolute sum of changes in neighbourhood deprivation exposure experienced between ages 0-18**. N=1,692 women who were not missing data on neighbourhood deprivation exposure (IMD quintile) for more than 50% of time points between ages 0-18. For this figure, any missing data on neighbourhood deprivation exposure for these remaining participants were carried forward from the last available time point.

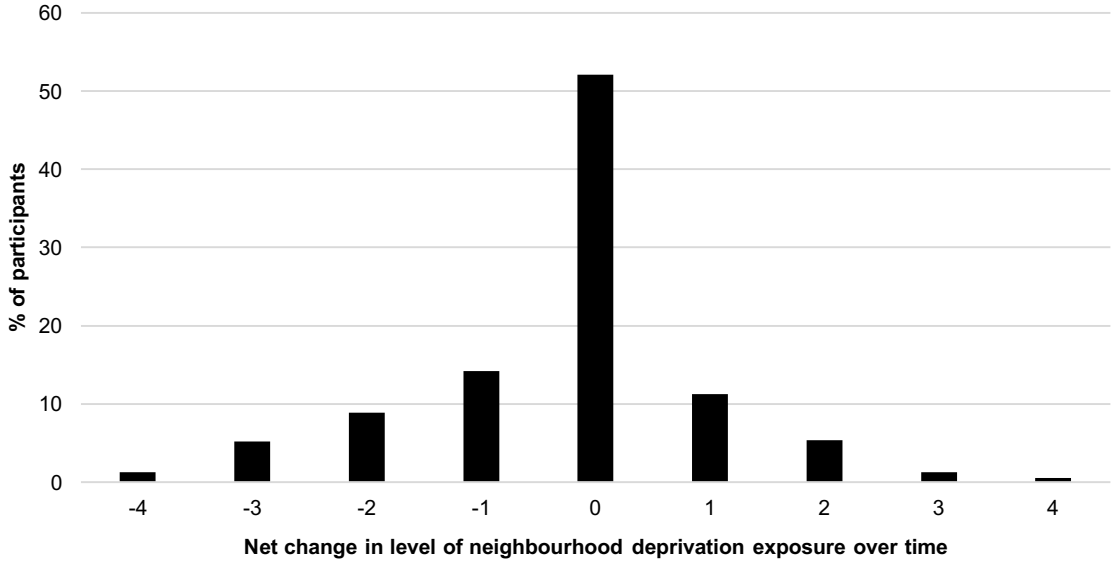

Figure D4: **Percent of participants by net change in level of neighbourhood deprivation exposure between ages 0-18**. N=1,692 women who were not missing data on neighbourhood deprivation exposure (IMD quintile) for more than 50% of time points between ages 0-18. For this figure, any missing data on neighbourhood deprivation exposure for these remaining participants were carried forward from the last available time point.

Table D2: **Prevalence of IPV victimisation and impact items and overall types between ages 18-21**

Victimisation items	**Experienced at least once (n=2,014): N (%)**
Told you who you could see, where you could go, or regularly checked what you were doing and where you were	346 (17)
Made fun of you, called you hurtful names, shouted at you	443 (22)
Used physical force such as pushing, slapping, hitting or holding you down	245 (12)
Used more severe physical force such as punching, strangling, beating you up, hitting you with an object	96 (5)
Pressured you into kissing/touching/something else	144 (7)
Physically forced you into kissing/touching/something else	72 (4)
Pressured you into having sexual intercourse	192 (10)
Physically forced you into having sexual intercourse	64 (3)
Impact items	**Experienced after IPV (n=588 who experienced any IPV): N (%)**
Scared	288 (49)
Upset/unhappy	465 (79)
Angry/annoyed	441 (75)
Made me feel sad	425 (72)
Affected my work/studies	187 (32)
Anxious	272 (46)
Depressed	231 (39)
No effect/not bothered	74 (74)
Made me drink more alcohol/take more drugs	92 (16)
Thought it was funny	48 (8)
Felt loved/protected/wanted	80 (14)
IPV typologies	**Any IPV (n= 2128): N (%)**
Any physical, psychological, or sexual IPV	683 (32)
Any physical, psychological, or sexual IPV with at least one of eight self-reported negative impacts	608 (29)
Any physical, psychological, or sexual IPV, excluding made fun of you, call you names, shouted	561 (26)
Any physical, sexual, or moderate-intensity (at least one item occurring at least a few times) psychological IPV	625 (29)
Any physical, sexual, or severe-intensity (at least one item occurring often) psychological IPV	443 (21)
Any physical or psychological IPV	617 (29)
Any sexual IPV	252 (12)

Table D3: **Distribution of IMD quintiles in categorical confounders at baseline**

	IMD quintiles: N (%)				
	1 (Least disadvantaged)	**2**	**3**	**4**	**5 (Most disadvantaged)**
Lives with biological parents, N (%)					
Both parents	1 173 (96)	974 (92)	673 (91)	653 (88)	445 (79)
Either biological mother or father or neither	52 (4)	85 (8)	65 (9)	92 (12)	121 (21)
Mother recently moved house, N (%)					
Yes	148 (11)	124 (11)	86 (10)	106 (12)	93 (14)
No	1 187 (89)	1,041 (89)	763 (90)	765 (88)	574 (86)
Parental employment[2], N (%)					
Yes	841 (65)	765 (66)	531 (60)	544 (61)	329 (46)
No	454 (35)	387 (34)	349 (40)	342 (39)	384 (54)
Ethnicity					
Non-White	22 (2)	17 (2)	11 (2)	24 (5)	30 (9)
White	929 (98)	760 (98)	527 (98)	475 (95)	312 (91)
At least one parent has higher than O-level education					
Yes	959 (70)	764 (62)	479 (52)	418 (45)	254 (35)
No	420 (30)	462 (38)	444 (48)	503 (55)	482 (65)
At least one parent is part of lower social class					
Yes	172 (14)	201 (19)	163 (21)	225 (30)	250 (45)
No	1 022 (86)	851 (81)	602 (79)	523 (70)	303 (55)
Mother married					
Yes	1 224 (81)	993 (81)	734 (80)	667 (71)	414 (54)
No	164 (12)	239 (19)	189 (20)	273 (29)	355 (46)

Table D4: **Distribution of IMD quintiles in categorical confounders at Age 18**

	IMD quintiles: N (%)				
	1 (Least disadvantaged)	**2**	**3**	**4**	**5 (Most disadvantaged)**
Lives with biological parents, N (%)					
Both parents	401 (78)	245 (75)	160 (70)	83 (60)	37 (61)
Either biological mother or father or neither	116 (22)	83 (25)	69 (30)	56 (40)	24 (39)
Mother recently moved house, N (%)					
Yes	19 (4)	11 (3)	11 (5)	3 (2)	4 (6)
No	500 (96)	319 (97)	217 (95)	138 (98)	60 (94)
Parental employment[2], N (%)					
Yes	365 (70)	229 (69)	154 (67)	77 (55)	34 (52)
No	156 (30)	104 (31)	76 (33)	64 (45)	31 (48)

Sensitivity and secondary analyses for the effect of neighbourhood deprivation on IPV among women

In addition to the secondary analyses to explore alternative hypotheses described in Chapter 6, I ran three types of sensitivity analyses (n=38 analyses) to test the robustness of my findings from my main analyses:

(1) ***Outcome operationalisation:*** I tested the robustness of my findings to several stricter definitions of IPV. These included: (a) 1=any IPV with at least one of the eight self-reported negative impacts versus 0=otherwise; (b) both (i) the average frequency and (ii) any experience of IPV, excluding emotional abuse (i.e., made fun of, insulted, shouted); and (c) based on a recent measurement study,[16] any physical, sexual, or (i) moderate-intensity (i.e., at least one item occurring at least a few times since age 18) or (ii) severe-intensity (i.e., at least one item occurring many times) psychological IPV.
(2) ***Additional missing data strategies***, including: (a) creating stabilised weights for intermittent missingness (so that final weights were the product of cumulative probabilities of exposure, censoring, and intermittent missingness histories);[13] (b) based on prior marginal structural model studies,[8] imputing missing covariate data with the last available observation (if t-1 missing, data set to missing), and (c) setting missing weights to 1, so that all complete participant-observation cases would be included (even when t-1 was missing), in both (i) long and (ii) wide data format to demonstrate consistency.
(3) ***Alternative model specifications***. This included (a) re-running analyses using time-varying, point-in-time exposure, similar to a prior marginal structural model study.[10] This estimated the average effect of living in more versus less deprived neighbourhoods at each time point up until age 18 on IPV risk between ages 18-21. I also (b) used covariate and exposure data from the last available time period (T_6) to estimate the T_9 weights, to test the influence of using cross-sectional data in the main analyses, and (c) re-ran analyses using the available-covariate weights but excluding T_8, to test the influence of this time period (which was not included in analyses using consistent-covariate weights). Additionally, (d) I conducted conventional generalised linear models in the unweighted sample (i.e., not accounting for time-varying confounding) to compare methods. In a crude model, I regressed each primary outcome onto exposure and all time-invariant and time-varying covariates measured at baseline and in an adjusted model I added the average value of time-varying covariates over the remaining time periods (T_1-T_9).

Validity checks

The weight distributions for each relevant sensitivity and secondary analysis are shown in Table D5.

Table D5: **Mean, standard deviation, and range of stabilised weights by sensitivity analysis**

	M (SD)	1st percentile, 99th percentile
Stabilised weights including weights for intermittent missingness		
Consistent-covariate exposure weights	1.0 (0.1)	0.8, 1.2
Consistent-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Consistent-covariate intermittent missingness weights	0.9 (0.1)	0.6, 1.0
Consistent-covariate exposure*censoring*missing weights	0.8 (0.1)	0.4, 1.0
Available-covariate exposure weights	1.0 (0.1)	0.7, 1.2
Available-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Available-covariate intermittent missingness weights	0.8 (0.1)	0.4, 1.0
Available-covariate exposure*censoring*missing weights	0.7 (0.2)	0.3, 1.0
Missing data on all available covariates imputed with last observation carried forward		
Available-covariate exposure weights	1.0 (0.1)	0.8, 1.3
Available-covariate censoring weights	0.9 (0.1)	0.7, 1.1
Available-covariate exposure*censoring weights	0.9 (0.1)	0.6, 1.2
Missing weights set to 1		
Consistent-covariate exposure weights	1.0 (0.1)	0.5, 1.2
Consistent-covariate censoring weights	0.9 (0.1)	0.6, 1.0
Consistent-covariate exposure*censoring weights	0.9 (0.1)	0.4, 1.1
Available-covariate exposure weights	1.0 (0.2)	0.1, 1.2
Available-covariate censoring weights	0.8 (0.1)	0.6, 1.0
Available-covariate exposure*censoring weights	0.8 (0.2)	0.04, 1.1
Main analysis but with T9 weights using covariate data from T6 (instead of T9)		

	M (SD)	1st percentile, 99th percentile
Consistent-covariate exposure weights	1.0 (0.1)	0.8, 1.3
Consistent-covariate censoring weights	0.9 (0.04)	0.8, 1.0
Consistent-covariate exposure*censoring weights	0.9 (0.1)	0.7, 1.2
Available-covariate exposure weights	1.0 (0.1)	0.7, 1.3
Available-covariate censoring weights	0.9 (0.1)	0.7, 1.2
Available-covariate exposure*censoring weights	0.9 (0.1)	0.6, 1.2
Main analysis with available-covariate weights but excluding T8		
Available-covariate exposure weights	1.0 (0.1)	0.8, 1.2
Available-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Available-covariate exposure*censoring weights	0.9 (0.1)	0.6, 1.1
Crime deprivation (Quintile 4 and 5 vs. Quintiles 1, 2, and 3)		
Consistent-covariate exposure weights	1.0 (0.1)	0.8, 1.2
Consistent-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Consistent-covariate exposure*censoring weights	0.9 (0.1)	0.7, 1.1
Available-covariate exposure weights	1.0 (0.1)	0.8, 1.2
Available-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Available-covariate exposure*censoring weights	0.9 (0.1)	0.6, 1.1
Income deprivation (Quintile 4 and 5 vs. Quintiles 1, 2, and 3)		
Consistent-covariate exposure weights	1.0 (0.1)	0.8, 1.3
Consistent-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Consistent-covariate exposure*censoring weights	0.9 (0.1)	0.6, 1.2
Available-covariate exposure weights	1.0 (0.1)	0.8, 1.2
Available-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Available-covariate exposure*censoring weights	0.9 (0.1)	0.6, 1.1
Ordinal neighbourhood deprivation (quintile variable)		
Consistent-covariate exposure weights	1.0 (0.1)	0.8, 1.1
Consistent-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Consistent-covariate exposure*censoring weights	0.9 (0.1)	0.7, 1.1
Available-covariate exposure weights	1.0 (0.1)	0.6, 1.2
Available-covariate censoring weights	0.9 (0.1)	0.7, 1.0
Available-covariate exposure*censoring weights	0.9 (0.1)	0.5, 1.0

M is mean. SD is standard deviation.

Effect estimates

Table D6 shows the effect estimates from all sensitivity analyses.

Table D6: **Sensitivity analyses for effect estimate of neighbourhood deprivation on IPV among women testing the robustness of the main findings**

	N_{women} ($N_{observations}$) in analysis	Relative risk	95% CI
Outcome: Any IPV with a negative impact (binary)			
Consistent-covariate weights	1,226 (6,279)	1.27	0.96, 1.69
Available-covariate weights	1,040 (5,067)	1.39	1.00, 1.93
Outcome: IPV, excluding emotional abuse			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,228 (6,294)	1.74	1.05, 2.86
Available-covariate weights	1,041 (5,088)	1.68	1.04, 2.71
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,228 (6,294)	1.42	1.05, 1.92
Available-covariate weights	1,041 (5,088)	1.37	0.97, 1.94
Outcome: Any of physical, sexual, or moderate psychological IPV			
Consistent-covariate weights	1,228 (6,294)	1.35	1.02, 1.78
Available-covariate weights	1,041 (5,088)	1.35	0.98, 1.86
Outcome: Any of physical, sexual, or severe psychological IPV			
Consistent-covariate weights	1,228 (6,294)	1.34	0.93, 1.93
Available-covariate weights	1,041 (5,088)	1.34	0.89, 2.03
Stabilised weights including weights for intermittent missingness			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,228 (6,294)	1.61	1.11, 2.33
Available-covariate weights	1,041 (5,088)	1.72	1.10, 2.69
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,228 (6,294)	1.38	1.07, 1.79
Available-covariate weights	1,041 (5,088)	1.39	1.04, 1.86
Missing data on all available covariates imputed with last observation			
Outcome: IPV frequency score (count)	1,041 (6,454)	1.69	1.09, 2.65
Outcome: Any IPV (binary)	1,041 (6,454)	1.37	1.01, 1.86
Missing weights set to 1			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,228 (9,824)	1.61	1.14, 2.27
Available-covariate weights	1,041 (9,369)	1.68	1.13, 2.51

	N_{women} ($N_{observations}$) in analysis	Relative risk	95% CI
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,228 (9,824)	1.35	1.07, 1.70
Available-covariate weights	1,041 (9,369)	1.42	1.08, 1.87
Missing weights set to 1 in wide dataset (unit of analysis is participant)			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,228 (N/A)	1.59	1.11, 2.26
Available-covariate weights	1,041 (N/A)	1.78	1.20, 2.65
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,228 (N/A)	1.31	1.03, 1.66
Available-covariate weights	1,041 (N/A)	1.46	1.11, 1.92
Exposure: Point-in-time exposure (binary, time-varying)			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,291 (6,400)	1.44	1.11, 1.87
Available-covariate weights	1,112 (5,237)	1.40	1.01, 1.94
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,291 (6,400)	1.29	1.06, 1.57
Available-covariate weights	1,112 (5,237)	1.23	0.99, 1.55
T9 weights using covariate data from T6 (instead of T9)			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,228 (6,340)	1.64	1.12, 2.41
Available-covariate weights	1,041 (5,180)	1.65	1.03, 2.63
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,228 (6,340)	1.40	1.08, 1.82
Available-covariate weights	1,041 (5,180)	1.37	1.01, 1.86
Main analysis with available-covariate weights but excluding T8			
Outcome: IPV frequency score (count)	1,066 (4,862)	1.62	1.04, 2.52
Outcome: Any IPV (binary)	1,066 (4,862)	1.36	1.01, 1.83
Un-weighted (conventional) analyses of cumulative exposure			
Outcome: IPV frequency score (count)			
Crude analysis (adjusting for only baseline covariates)[a]	1,123 (N/A)	1.63	1.12, 2.35
Adjusted analysis (for baseline and time-varying covariates)[b]	1,021 (N/A)	1.56	1.04, 2.34
Outcome: Any IPV (binary)			
Crude analysis (adjusting for only baseline covariates)[a]	1,123 (N/A)	1.44	1.11, 1.86
Adjusted analysis (for baseline and time-varying covariates)[b]	1,021 (N/A)	1.44	1.09, 1.91

N is number. CI is confidence interval. MSM is marginal structural model. N/A is not applicable.
Unless otherwise noted, analysis is negative binomial regression (for count outcome) or log-binomial generalised linear model (for binary outcome), weighted as indicated, conducted in a long-form data set (with participant-time as the unit of analysis) with clustering accounted for with robust (conservative) standard errors and adjusting for baseline time-invariant and time-varying covariates. Relative risk in the log negative binomial regression is the incidence rate ratio and in the log-binomial model is the risk ratio.
[a]Conventional generalised linear model adjusting for all available covariates at baseline in unweighted sample in wide format.
[b]Conventional generalised linear model adjusting for all available covariates at baseline and the average value of all available time-varying covariates in unweighted sample in wide format.

Table D7 shows the effect estimates from all secondary analyses exploring alternative hypotheses. As I expected given the exponential distribution of the deprivation scores, I did not find a meaningful association between the ordinal exposure variable and risk of IPV. To confirm the robustness of these findings, I also re-ran my analyses using exposure weights constructed by multinomial rather than ordinal logistic regression (in case of violations of the proportional odds assumption). However, results did not differ and are thus not shown.

Table D7: **Secondary analyses for effect estimate of neighbourhood deprivation on IPV among women exploring alternative hypotheses**

	N_{women} ($N_{observations}$) in analysis	Relative risk	95% CI
Crime deprivation			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,185 (6,196)	1.18	0.87, 1.62
Available-covariate weights	1,041 (5,088)	1.25	0.89, 1.76
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,185 (6,196)	1.12	0.89, 1.41
Available-covariate weights	1,041 (5,088)	1.16	0.90, 1.50
Income deprivation			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,185 (6,196)	1.20	0.88, 1.64
Available-covariate weights	1,041 (5,088)	0.99	0.63, 1.57
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,185 (6,196)	1.13	0.85, 1.51
Available-covariate weights	1,041 (5,088)	1.00	0.71, 1.37
Ordinal neighbourhood deprivation			

	N_{women} ($N_{observations}$) in analysis	Relative risk	95% CI
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,185 (6,196)	1.04	0.92, 1.18
Available-covariate weights	1,041 (5,088)	1.01	0.87, 1.16
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,185 (6,196)	1.07	0.97, 1.18
Available-covariate weights	1,041 (5,088)	1.06	0.94, 1.18
Physical or psychological IPV			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,228 (6,294)	1.54	1.06, 2.24
Available-covariate weights	1,041 (5,088)	1.49	0.95, 2.33
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,228 (6,294)	1.51	1.15, 1.99
Available-covariate weights	1,041 (5,088)	1.41	1.02, 1.95
Sexual IPV			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,228 (6,294)	1.86	0.99, 3.49
Available-covariate weights	1,041 (5,088)	2.28	1.13, 4.60
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,228 (6,294)	1.33	0.81, 2.21
Available-covariate weights	1,041 (5,088)	1.59	0.88, 2.87
No exposure versus past versus most recent (ordinal)[a]			
Outcome: IPV frequency score (count)			
Consistent-covariate weights	1,101 (5,786)		
Past exposure to deprived neighbourhood		1.70	1.22, 2.35
Living in deprived neighbourhood at age 18		1.25	0.82, 1.91
Available-covariate weights	951 (4,714)		
Past exposure to deprived neighbourhood		1.39	0.95, 2.01
Living in deprived neighbourhood at age 18		1.18	0.72, 1.92
Outcome: Any IPV (binary)			
Consistent-covariate weights	1,101 (5,786)		
Past exposure to deprived neighbourhood		1.64	1.25, 2.14
Living in deprived neighbourhood at age 18		1.21	0.88, 1.66
Available-covariate weights	951 (4,714)		
Past exposure to deprived neighbourhood		1.55	1.14, 2.10
Living in deprived neighbourhood at age 18		1.10	0.76, 1.60

N is number. CI is confidence interval. MSM is marginal structural model. Unless otherwise noted, analysis is negative binomial regression (for count outcome) or log-binomial generalised linear model (for binary outcome), weighted as indicated, conducted in a long-form data set (with participant-time as the unit of analysis) with clustering accounted for with robust (conservative) standard errors and adjusting for baseline time-invariant and time-varying covariates. Relative risk in the negative binomial regression is the incidence rate ratio and in the log-binomial model is the risk ratio.

[a]Referent is never lived in most deprived neighbourhoods. Only n=2 participants were living in a deprived neighbourhood at age 18 without ever previously living in a deprived neighbourhood. As such, these participants were analysed in the same category as all other participants living in a deprived neighbourhood at age 18.

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