A behavioural economics approach to personalised risk communication: nudging people with Type 2 diabetes towards better self-management



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Abstract

Type-2 diabetes mellitus (T2DM) is a major health burden, accounting for 12% of total healthcare expenditures worldwide. Recent forecasts estimate that 1 in 10 people will suffer from the condition by 2040. These figures are of great concern, particularly when T2DM has the potential to be well-controlled. By adhering to self-management behaviour, i.e. by making appropriate lifestyle changes, people with T2DM can delay and often prevent the onset of disease-related complications. However, in practice, many patients remain poorly controlled and the most effective method to motivate sustained adherence to self-management is unclear. Among recent motivation strategies, those informed by behavioural economics have raised growing interest in many different areas. The core principle of behavioural economics is to identify specific *errors* in the way people make decisions (i.e. specific deviations from standard economic rationality) and target these to steer people's choices towards a given (recommended) direction. Following this approach, I aim to develop an innovative behavioural economic-informed intervention for people with T2DM with the objective of motivating sustained adherence to self-management in this population.

The first part of the Thesis was concerned with investigating the factors known to have an influence on patients' decision to adhere to self-management. Firstly, I systematically reviewed all studies exploring the risk perceptions for diabetes-related complications in T2DM populations. I found that patients tend to largely underestimate their risks, especially those for cardiovascular complications, and express an optimistic bias (i.e. they tend to feel less at risk than their peers). Given the positive association between risk perceptions and adherence to self-management, these findings suggested the need for more effective risk communication addressed to this population. As a result, I further explored the decision-making processes of people with T2DM to identify relevant behavioural economic material. I measured the risk attitudes under prospect theory of 120 individuals, half of whom were diagnosed with T2DM. Focusing on a hypothetical trade-off between health outcomes and self-management, I found that patients express strong risk and loss aversion. This suggested that non-adherence to self-management may not be explained by a particular risk-seeking behaviour in this population. Consequently, this confirmed the need for better risk communication.

In the second part of the Thesis, I developed an innovative risk communication tool entitled PERCODIA[2]. Informed by the behavioural economic findings of the

[2]PERsonalised Risk COmmunication for people with Type 2 DIAbetes.

first part, PERCODIA is aimed to better deliver personalised risk information to people with T2DM. I first reviewed the risk communication literature to identify the best ways of *taking advantage* of these behavioural economic findings. Both the content and the format of the tool were selected for this purpose. I then designed and conducted a pilot randomised control trial (RCT) to assess the feasibility of using PERCODIA in a primary care setting and evaluate its impact on risk recall and self-management behaviour. The trial comprised 40 participants with poorly controlled T2DM randomly allocated to usual care supported by PERCODIA or usual care only. The results showed that using PERCODIA in routine primary care is not only feasible, but also well-received by both doctors and patients. The intervention significantly improved recall of risk information after 12 weeks and motivated intentions to improve dietary behaviour. These promising results support the development of a larger RCT to assess the impact of PERCODIA on long-term self-management behaviour in poorly controlled T2DM populations. Given the significant challenges and opportunities in this patient population, robust evidence of a positive effect, even moderate, would greatly benefit both patients and the healthcare system.

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List of Abbreviations

AADE . . . . . American Association of Diabetes Educators

AF . . . . . Atrial Fibrillation

AIC . . . . . Akaike Information Criterion

ARR . . . . . Absolute Risk Reduction

BIT . . . . . Behavioural Insights Team

BMI . . . . . Body Mass Index

CASP . . . . . Critical Appraisal Skills Programme

CE . . . . . Certainty Equivalent

CHD . . . . . Coronary Heart Disease

CI . . . . . Confidence Interval

COMRADE . Combined Outcome Measure for Risk communication And treatment Decision-making Effectiveness

CONSORT . . CONsolidated Standards Of Reporting Trials

CV . . . . . Cardiovascular

CVD . . . . . Cardiovascular Disease

DLBQ . . . . . Determinants of Lifestyle Behaviour Questionnaire

DSME . . . . . Diabetes Self-Management Education

DTU . . . . . Diabetes Trials Unit

eGFR . . . . . estimated Glomerular Filtration Rate

EU . . . . . Expected Utility

EUT . . . . . Expected Utility Theory

EV . . . . . Expected Value

GLM . . . . . Generalised Linear Model

GP . . . . . General Practitioner

HAPA . . . . . Health Action Process Approach

HbA1c . . . . Glycosylated Haemoglobin, type A1c

HBM . . . . . Health Belief Model

HDL . . . . . High-Density Lipoprotein

HERC . . . . . Health Economics Research Centre

HF . . . . . Heart Failure

HL . . . . . Hosmer-Lemeshow

IDF . . . . . International Diabetes Federation

IHD . . . . . Ischaemic Heart Disease

IQ . . . . . Interquartile

IT . . . . . Information Technology

ITT . . . . . Intention-To-Treat

LDL . . . . . Low-Density Lipoprotein

LMIC . . . . . Low- and Middle-Income Countries

MI . . . . . Myocardial Infarction

NCD . . . . . Non-Communicable Disease

NHS . . . . . National Health Service

NICE . . . . . National Institute for Health and Care Excellence

NNT . . . . . Number Needed to Treat

OLS . . . . . Ordinary Least Squares

PA . . . . . Physical Activity

PERCODIA . Personalised risk communication for people with diabetes

PMT . . . . . Protection Motivation Theory

PPI . . . . . Patient and Public Involvement

PRISMA . . . Preferred Reporting Items for Systematic Reviews and Meta-Analyses

PT . . . . . Prospect Theory

PVD . . . . . Peripheral Vascular Disease

QALY . . . . . Quality-Adjusted Life Year

QAT . . . . . Quality Assessment Tool for observational cohort and cross-sectional studies

QoL . . . . . Quality of Life

SEUT . . . . . Subjective Expected Utility Theory

RCT . . . . . Randomised Controlled Trial

RP . . . . . Reference Point

RPS-DM . . . Risk Perception Survey - Diabetes Mellitus

RRR . . . . . Relative Risk Reduction

SBP . . . . . Systolic Blood Pressure

SDSCA . . . . Summary of Diabetes Self-Care Activities

SF-STAI . . . Short Form Spielberger State-Trait Anxiety Inventory

SG . . . . . Standard Gamble

SMBG . . . . . Self-Monitoring of Blood Glucose

TPB . . . . . Theory of Planned Behaviour

T1DM . . . . . Type-1 Diabetes Mellitus

T2DM . . . . . Type-2 Diabetes Mellitus

TTM . . . . . TransTheoretical Model

TTO . . . . . Time Trade-Off

UK . . . . . United Kingdom

UKPDS . . . . United Kingdom Prospective Diabetes Study

UKPDS-OM . United Kingdom Prospective Diabetes Study - Outcomes Model

US . . . . . United States

VAS . . . . . Visual Analogue Scale

VPF . . . . . Value for the statistical Prevention of a Fatality

vNM . . . . . von Neumann-Morgenstern

WBC . . . . . White Blood Cell

WTA . . . . . Willingness To Accept

WTP . . . . . Willingness To Pay

WHO . . . . . World Health Organization

1
Introduction

1.1 Background and objective

Non-infectious diseases are now the primary threat to the health of populations. Until the end of the 20th century, most pandemics[1] were the result of exogenous agents, specifically human pathogens. For example, in the 14th century, Europe was devastated by the Black Death, losing between 30 and 60% of its population in a few years. [3] Almost six centuries later, the Spanish flu killed 3 to 5% of the world population (50 to 100 million of deaths). [4] Even nowadays, Malaria kills around 438,000 people every year, despite the existence of effective treatments. [5]

The 20th century was a watershed in the history of population health. Since the end of the Second World War, the lengthening of life expectancies, together with the drastic environmental and lifestyle changes widespread around the world, have favoured the emergence of a new kind of pandemic: a non-communicable disease (NCD) - or chronic disease - pandemic. NCDs, the four main types of which are cardiovascular diseases (CVD), cancers, respiratory diseases and diabetes, have become the major cause of morbidity and mortality globally. According to the World Health Organization (WHO), NCDs kill 38 million people each year; the four diseases mentioned above account for 82% of deaths. Low- and middle-income countries (LMIC) turn out to be strongly impacted by this phenomenon (three quarters of total NCD deaths). [6] Considering population ageing, Murray and Lopez (1996) estimated, about twenty years ago, that NCDs will be responsible for over three-quarters of all deaths by 2020. [7]

In the last WHO estimate, the number of people suffering from diabetes[2] is estimated at 422 million worldwide, which corresponds to around 3.7 million deaths annually (82% occurring in LMIC). [9] The disease is expected to become the 5th leading cause of death in the next 10 years. [10] The International Diabetes

[1] "An epidemic occurring worldwide, or over a very wide area, crossing international boundaries and usually affecting a large number of people". [1] The notion of contagiousness is not necessarily implied. For example, the WHO uses this term to describe the increasing incidence of obesity worldwide. [2]

[2] The WHO diagnostic criteria for diabetes is only based on plasma glucose levels: a) $\geq$7.0 mmol/L (126 mg/dL) following a fasting plasma glucose test; or b) $\geq$11.1 mmol/L (200 mg/dL) following a 2-h plasma glucose test (venous plasma glucose test 2-h after ingestion of 75g oral glucose load). [8]

Federation (IDF) has published even more alarming trends: 5 million individuals died from diabetes in 2015, and 1 in 10 people will have it by 2040. [11] The economic burden incurred by the disease is worrying: in 2015, the more conservative estimates suggest that health spending on diabetes accounted for 12% of total healthcare expenditure worldwide. [11]

Within this new epidemic, Type-2 diabetes mellitus[3] (T2DM, formerly called non-insulin-dependent or adult-onset) is by far the most common, accounting for around 90% of total cases in high income countries. [11] Because it is of slow progression and expresses symptoms less marked than those of Type-1 diabetes mellitus[4] (T1DM), the diagnosis may not be made until several years after onset, i.e. once complications have already arisen (cardiovascular, eye, kidney, and nerve damages, amongst others). It is estimated that almost half of people with T2DM do not know they have it. [11] This type of diabetes, traditionally diagnosed in middle-aged and older adults, is now also occurring in children.

Despite the substantial therapeutic arsenal that has been made available since metformin was launched in the 1950s [12], T2DM remains poorly controlled. The issue is not so much pathophysiological - the underlying mechanisms of the disease being quite well understood - but rather behavioural. As NICE[5] reports have emphasised, the first-line treatment for T2DM is first and foremost an appropriate lifestyle management, e.g. balanced diet, reduction in alcohol consumption, smoking cessation, and physical exercise. [13] However, because of the asymptomatic nature of the disease - at least in its first phase - multiple barriers hamper proper individual self-management behaviour.

Amongst those barriers, one which has been widely mentioned in the literature is the poor perception of the risks associated with the disease. People who underestimate their risks may be less likely to adopt recommended behaviours, while overestimating their risks may cause so much anxiety that it hampers people

[3]The hyperglycaemia results from an abnormal increased resistance to the action of insulin, and is reinforced by the incapacity of the body to produce enough insulin to overcome that resistance.

[4]In this form, the hyperglycaemia follows from the autoimmune beta-cell destruction in the pancreas and is characterised by a complete lack of insulin production.

[5]National Institute for Health and Care Excellence.

from taking precautionary measures. [14] There is strong evidence supporting the causality of the association between perceived risks and precautionary behaviours. [15, 16] In addition, perceived risks are signicant predictors of behavioural intentions. [17, 18] Although behavioural processes are complex, inaccurate risk perceptions are a major impediment to the adoption of self-care behaviours and, as a result, an additional risk for the occurrence of adverse outcomes.

This Thesis will investigate new ways of providing personalised risk information to people with T2DM so that it reduces risk misperceptions and encourages self-management behaviour. This introduction discusses the rationale behind the research question (section 1.2), explains the strategy adopted to address it (section 1.3), and finally provides an overview of the Thesis structure (section 1.4).

1.2 Justification for research

For the past twenty years, evidence has accumulated to show the impact of harmful effects caused by T2DM. Overall, people with diabetes have a 15% excess risk of death compared to people without diabetes. [19] Although this risk has significantly dropped in the last decade, it remains very high in certain patient groups, especially in those whose risk factor values are not kept within normal range. In particular, patients younger than 55 years old are twice as likely to die as their healthy peers. [19] Whereas individuals with T1DM tend to be more sensitive to microvascular complications[6], people with T2DM turn out to be more affected by macrovascular events.[7] When looking more closely at the nature of the macrovascular risk, it

[6]Due to damage to small blood vessels, which include eye diseases (retinopathy) – caused by damage to vessels supplying the retina -, leading to progressive loss of vision, and potentially blindness; kidney diseases (nephropathy), leading to kidney failure; nerve diseases (peripheral neuropathy) - through both direct damage caused by the hyperglycaemia and decreased blood flow to nerves -, leading to sensory loss, damage to limbs, and impotence in diabetic men. [20]

[7]Due to damage to larger blood vessels, which include CVD - through clogging of arteries (atherosclerosis process) -, leading to decreased blood flow to heart (coronary heart disease, or CHD, including MI) or to brain (stroke); peripheral vascular diseases - caused by the narrowing of blood vessels that carry blood to the extremities (arms, legs), stomach, and kidneys -, leading to pain and decreased healing of infections. Individuals with both peripheral neuropathy and peripheral vascular complications are at high risk for foot ulceration and subsequent lower-extremity amputation. [20]

appears that CVD are the most serious threat. Individuals with T2DM have a 2-4 times greater CVD risk - i.e. they are 2 to 4 times more likely to have heart disease or suffer a stroke - than people without diabetes, even after adjustment for recognised cardiovascular risk factors.[8] [21, 22] Moreover, women are at greater risk of developing CVD than men with diabetes and the general population without diabetes. For example, Lundberg *et al.* (1997) found the relative risk of myocardial infarction (MI) in diabetic women to be almost twice as high as the one in men (5.0 vs 2.9). [23]

In terms of mortality, after controlling for age and comorbidities, CVD death rates are about 2-5 times higher among adults with diabetes compared to individuals without diabetes. [24–26] CVD is the leading cause of mortality among people with diabetes, accounting for 50% of deaths. [27] Women turn out to be particularly affected, CVD mortality risk in diabetic women has been found to be similar to that in women with a history of cardiac event. [28–30]

Although they have lesser impact on overall mortality, other complications stemming from a lack of control of blood glucose are prevalent among individuals with T2DM. In terms of microvascular events, looking at recent statistics from Diabetes UK, 75% of people with T2DM have some degree of retinopathy within 20 years of diagnosis, and 20% of them develop overt kidney disease.[9] Peripheral neuropathy is estimated to affect between 30% and 50% of individuals with diabetes, regardless of their type. In this context, amputations of a foot or a leg due to an ulcer arise more often than one would expect: around one in twenty individuals with diabetes suffer from a foot ulcer in a given year, and more than 10% of the cases require an amputation. [31] Those figures are in line with those reported by Deshpande *et al.* (2008) for the prevalence of diabetes-related complications in the US. [20]

There is a consensus in the medical community that diabetes, whether Type

[8] In order to assess the independent effect of diabetes on the risk of CVD death, the authors controlled for age, ethnicity, income, cholesterol level, systolic blood pressure and smoking.

[9] Overall, nephropathies account for 11% of deaths in people with T2DM.

1 or Type 2, is not curable.[10] However, there is also a consensus that consistent *self-management* can delay the onset of complications. In the literature, definitions of self-management are wide-ranging and vary across fields. In healthcare, for Goodall and Halford (1991), self-management is "a set of skilled behaviours engaged in to manage one's own illness". [33] More recently, Barlow *et al.* (2002) refer to the concept as "the individual's ability to manage the symptoms, treatment, physical and psychosocial consequences and the lifestyle changes inherent in living with a chronic condition". [34] In the area of diabetes, seven self-care behaviours have been found to be correlated with good outcomes: healthy eating, physical activity (PA), medication adherence[11], self-monitoring of blood glucose (SMBG), problem solving (i.e. prevention, detection and treatment of acute complications), risk reduction activities (i.e. prevention, detection and treatment of chronic complications; e.g. quitting smoking, seeing the doctor regularly, following foot care guidelines), and healthy coping (e.g. seeking individually appropriate support from family/friends and/or medical team). [33, 37, 38] These behaviours are known as the AADE[12] 7 self-care behaviours, with the nomenclature reflecting patient-friendly and action-oriented terminology. [37] Therefore it seems reasonable to focus on these seven self-care behaviours when referring to diabetes self-management in the wider sense.

Having a clear definition of diabetes self-management allows evidence supporting its effect on clinical outcomes to be identified. In the literature, evidence supporting the positive impact of five essential self-care behaviours has been reported[13] (see Table 1.1).

Each of these self-care behaviours has been shown to improve glycaemic control (HbA1c levels). In the literature, there is strong evidence that strict glycaemic

[10] Although there is recent evidence that bariatric surgery can induce a significant and sustainable remission of T2DM in severely obese patients. [32]

[11] A glucose-lowering pharmacological treatment is initiated as soon as the aforementioned lifestyle measures become no longer sufficient to maintain an appropriate glycaemia. Therapeutic interventions might also include a lipid-lowering and/or a antihypertensive therapy. [35, 36]

[12] American Association of Diabetes Educators.

[13] Problem solving has more to do with the daily management of the disease to avoid acute complications, whereas measuring the impact of healthy coping is difficult.

control reduces the risk of developing micro and macrovascular complications.[14] [39–46]

Table 1.1: Impact of self-management behaviour on clinical outcomes.

Self-care behaviour	Study	Outcome impacted
Healthy eating	Povey *et al.* (2007) [47] (systematic review)	BMI, blood pressure, HbA1c, fasting blood glucose, HDL (mixed evidence)
Physical activity	Boulé *et al.* (2001) [48] (systematic review and meta-analysis)	HbA1c (significant), BMI (not significant)
	Boulé *et al.* (2003) [49] (systematic review and meta-analysis)	HbA1c (significant)
	Colberg *et al.* (2010) [50]	Many (acute and long-term effects of PA) (mixed evidence)
Medication adherence	Ho *et al.* (2006) [51]	HbA1c, blood pressure, LDL, increased risks for all-cause hospitalisation, all-cause mortality (all significant)
	Nichols *et al.* (2015) [52]	HbA1c (significant)
SMBG	Welschen *et al.* (2005) [53] (systematic review and meta-analysis)	HbA1c (significant)
Risk reduction activities	Facchini *et al.* (1992) [54]	LDL, HDL (significant)

Moreover, several studies have shown that diabetes self-management education (DSME) - i.e. interventions aiming at improving (multidimensional) self-management in patients – are associated with improved clinical outcomes (see Table 1.2).

Given its impact on biomarkers that predict the risk for adverse event, especially glycaemic control (and thus, indirectly, on the risk for adverse event itself), self-management has become the cornerstone for treating T2DM. [61] However, despite physician recommendations, implementing sustainable changes in the behaviour of newly diagnosed patients - who, for the most part, have suffered little or no tangible effect until then - is a major challenge. An extensive literature points out numerous barriers to self-management – such as psycho-cognitive, sociocultural,

[14] Interestingly, most improved clinical outcomes concern biomarkers that predict the risk for adverse event (e.g. MI, stroke, mortality), but do not concern the risk for adverse event *per se*. Apart from medication adherence, there is no evidence of a significant direct association between better self-care behaviour and lower risk for adverse event.

Table 1.2: DSME interventions associated with improved clinical outcomes.

Study	Outcome impacted
Ellis *et al.* (2004) [55] (systematic review and meta-analysis)	HbA1c (significant)
Gary *et al.* (2003) [56] (systematic review and meta-analysis)	HbA1c (significant), fasting blood glucose (significant), BMI (non-significant)
Norris *et al.* (2001) [57] (systematic review and meta-analysis)	HbA1c (significant), lipids (mixed), BMI (mixed), blood pressure (mixed)
Norris *et al.* (2002) [58] (systematic review and meta-analysis)	HbA1c (significant)
Tang *et al.* (2012) [59]	HbA1c (significant), serum cholesterol (significant), LDL (significant)
Yuan *et al.* (2014) [60]	HbA1c (significant), BMI (significant)

and environmental barriers (see e.g. Jerant *et al.*, 2005 [62]; Peyrot *et al.*, 2005 [63]; Bodenheimer *et al.*, 2002 [64]; Glasgow *et al.*, 1997 [65]). To date, no intervention strategies have been found to satisfactorily overcome them; the most effective DSME method is still unclear.[15] [57, 67] In particular, risk communication interventions targeting risk perceptions and self-care behaviours have shown mixed results, with many participants barely understanding the explanations of health professionals about risks and having poor recall of risk information. [68–72] To date, the effect of the most effective risk communication intervention lasts no longer than a few weeks. [69] An innovative, more effective way of communicating the risk information is needed.

Enhancing adherence to self-management is a key challenge to improve patients' long-term outcomes. The objective of this Thesis is precisely to investigate whether a new type of risk communication intervention can be developed to improve patients' self-management behaviour effectively.

[15] Several studies conducted both in the US and in India have reported low rates of compliance to self-management recommendations in T2DM populations. Compliance rates could vary between 37% and 52% for dietary prescriptions; between 26% and 52% for physical practice; and between 23% and 33% for glucose monitoring. Regarding treatment adherence, they could vary between 70% and 80% for US patients whose health insurance paid for prescribed drugs, and fall to 15% for US Medicaid-funded patients. [66]

1.3 Strategy approach

The lack of satisfactory DSME interventions has motivated the strategy adopted when designing this Thesis. I believe that DSME interventions could benefit from insights (both theoretical and practical) drawn from other fields, especially decision science and economics. Below, I explain the rationale behind this approach.

1.3.1 Rationale

For a long time, health promotion interventions were designed in accordance with psychosocial theories/models (e.g. the health belief model [73], or the protection motivation theory [74]; see Chapter 2). [57, 75] These models, despite involving and emphasising different constructs, agree on the key role played by risk perceptions in the behaviour change process.[16] Several studies have established the link between risk perceptions and self-care behaviours (see review by Waters *et al.*, 2013 [76]), and have thus contributed to support these models.

However, a major criticism is that psychosocial theories do not provide a satisfactory (realistically) rational account of how people form intentions and make decisions. Specifically, they are all based on normative models of decision-making that are systematically violated in empirical studies (see Chapter 3). [76, 77] In real life people do not always make rational decisions and express several systematic decision-making *errors*, or *biases* (deviations from standard economic rationality) that underlie, at least partially, their (suboptimal, as seen from the standard model) behaviour. For example, people tend to express *loss aversion*, meaning that they tend to dislike losses more strongly than they enjoy equivalent gains (i.e. that the pain of losing a given outcome is psychologically more powerful than the pleasure of gaining the same outcome). In standard economic theory, the utility associated with a given outcome should depend on its final value but not on the gains or losses involved in the process of obtaining that outcome.[17] People also tend to express a *present bias*, which is the tendency to value the immediate moment (such as having

[16] Whether they act as a direct influence or through other mediating constructs.
[17] *Asset integration* property (see Chapter 3).

a cigarette or an extra dessert now) more than future moments (such as avoiding cancer or heart disease), which are quickly discounted. This phenomenon can be reflected in standard economic analyses through (hyberbolic) discounting, using an exponential discounting with a fixed rate. However, in practice, present bias can lead to cases of dynamic inconsistency, where preferences change over time in such a way that they can become inconsistent at another point in time. This makes it difficult to predict what people will prefer in the future based on their stated preference now. For example, Read and Leeuwen (1998) showed that people will often choose healthy over tastier unhealthy snacks if they have to pre-commit to their choices for future consumption, but that many would switch to the unhealthy option, if given the opportunity, at the date of consumption. [78]

Increased awareness of people's decision-making biases, combined with the fact that traditional behaviour change interventions have failed to produce substantial beneficial effects [79], have encouraged the search for new tools to design more effective policies. Over the past decade, there has been increasing interest in using concepts and methods from behavioural economics to inform public policy.[18] Located at the interface of decision science, economics and psychology, behavioural economics can be thought of as "the set of observations that demonstrate that human decision making often conflicts systematically with the assumptions of standard economic theory". [80] The field is concerned with the development and application for policy-making purposes of alternative, more descriptively valid models of decision-making.

1.3.2 Behavioural economic-informed policies

Behavioural economic-informed policies aim to *manipulate* or *mitigate* the effects of decision-making biases in order to motivate behaviour change. Over the years, behavioural economists have developed different philosophical frameworks that underpin such a new approach in the design of public policy. For clarity purposes, I provide a brief overview of these frameworks and related behavioural

[18] This is particularly true in the UK, with the establishment of the Behavioural Insights Team (BIT) by the Cameron government in 2010.

economic-informed interventions, mostly based on the work of Adam Oliver (2015, 2018). [80, 81]

a Libertarian and asymmetric paternalism

The idea behind these concepts is that public policies can improve individual welfare without (or nearly without) restricting personal autonomy. *Libertarian paternalism*, which was developed by Thaler and Sunstein (2003) [82], is to try to influence people's choices in a way that will make them better off, as judged by themselves (paternalism), while ensuring that their freedom of choice is preserved (libertarianism). Similar to this framework, Camerer and colleagues' *asymmetric paternalism* aims to create "large benefits for the people who are boundedly rational [...] while imposing little or no harm on those who are fully rational". [83]

Policies underpinned by these frameworks are designed to help reduce *negative internalities*, i.e. longer-term harms that people impose on themselves "through their own ill-considered automatic decisions", resulting, at least partly, from deviations from standard economic rationality.[19] [80] In this regard, they focus upon influencing behaviour on the *demand-side* through the consumer, the patient, or the citizen. A significant example, in the health domain, is the use of commitment devices to encourage precautionary behaviours. Such instruments require people to voluntarily pre-commit small amounts of their own money to a given programme, which is returned to them if they adhere to that programme (e.g. smokers adhering to a smoking cessation plan). Commitment devices preserve people's freedom of choice but make them more likely to meet the programme's objectives because of loss aversion.

As noted by Oliver (2015), libertarian paternalism has been the most prominent framework in policy circles. This may be due to the "catchy buzzword" assigned to policies that derive from it: *nudge*. [81]

[19] Whether such policies truly aim to make people better off *as judged by themselves*, as is claimed, will be discussed in more detail in Chapter 6.

b Coercive paternalism

Coercive paternalists believe that libertarian paternalistic interventions, by preserving the autonomy of choice, may not be sufficiently effective.

A major argument in favour of freedom of choice is that all deviations from standard economic rationality may not be errors *per se*. For example, with respect to present bias, Sunstein (2013) states that enjoying the immediate moment might be "a large part of what makes life worth living". [84] For libertarian paternalists, imposing forward-looking behavioural policies on individuals who deliberately want to favour short-term over longer-term outcomes is not legitimate.

For coercive paternalists, such an approach is justified when "the broadly considered benefits of the regulation to the individuals themselves are perceived to outweigh the broadly perceived costs." Policies underpinned by this framework are referred to as *shoves*. For example, Conly (2013) considers that health and financial security are universal lifetime goals. Consequently, she advocates for a legal restriction of the activities that prevent people from reaching these goals (e.g. smoking). [85] As Oliver (2015) notes, such a position requires "a lot of confidence in deciding what people most value over their life course" and raises the issue of what constitutes objective well-being. [81] Putting aside the externality argument (that others might be negatively impacted by the behaviour in question), i.e. considering (negative) internalities only, the assumption that restricting myopic behaviours would make all individuals better off is a strong one.

c Behavioural regulation

Policies underpinned by paternalistic frameworks, whether libertarian or coercive, are concerned with targeting the harms imposed by the demand side.[20] However, it is also possible to use behavioural economic findings to inform supply side regulations, i.e. to address externalities stemming from the supply side. Such regulations are referred to as *budges*. [86] Budge policy intends to "counter the profit maximizing

[20] This is debatable regarding coercive paternalism, as some of Conly's arguments are motivated by negative supply-side externalities. For example, she advocates for a ban of trans-fats above maximum acceptable limit. [85]

behavioural economic-informed harmful manipulations of consumers by private organizations". An example is the enforcement of plain cigarette packaging to counter the salient effect of packaging designed by tobacco companies (effect on people's *availability heuristics*, see Chapter 3).

Although nudges, shoves, and budges belong to the same family of policies informed by behavioural economics, being aware of the different philosophical frameworks that underpin them is important. Nudges are, by nature, "softer" regulations, and implementing them will likely raise fewer ethical issues than shove or budge policies (please see Chapter 6 for more details).

1.3.3 Behavioural economic-informed health interventions

Behavioural-economic based interventions have shown promising results in helping people change their behaviour in the health domain (see e.g. Luoto and Carman, 2014 [79], or Arno and Thomas (2016) [87] for a review), including people with T2DM (see Kullgren *et al.*, 2017 [88] for a review). Because diabetes management is centred on people's daily behaviour, many decision-making biases identified in the economic literature also apply to T2DM populations (see e.g. Mørkbak *et al.*, 2017 [89]; Sultan *et al.*, 2003 [71]) and can be used to inform new interventions. So far behavioural economic-informed interventions to promote self-management behaviour have mostly been nudges aiming to mitigate present biasedness by means of incentives or rewards, with mixed results in the long term (see Chapter 4 for more detail).

In the risk communication field, however, using findings of behavioural economics to enhance the impact of interventions is not common practice. Although substantial work has been done to test the impact of *framing effects* (the tendency that people have to draw different conclusions from the same risk information, depending on whether it is expressed as gains or losses) on screening or precautionary behaviours (see Gallagher and Updegraff, 2012 [90], Edwards *et al.*, 2001 [91] for general reviews and Kullgren *et al.*, 2017 [88] for a review in T2DM populations),

most factors that influence health-related decisions have not yet been targeted. This is an issue since the effectiveness of risk communication may be enhanced by selecting adapted formats or metrics for this purpose. [92–94]

In this Thesis, the objective is to develop a new type of risk communication intervention, informed by behavioural economics, that will motivate sustained adherence to self-management in T2DM populations. The overarching objective is dual. On the one hand, helping patients whose deliberate preferences indicate that they would adopt a healthier lifestyle if they could, but who, for some reason inherent to human behaviour (e.g. decision-making errors mentioned above), fail to do so. In other words, addressing negative internalities of poorly controlled patients. On the other hand, contributing to alleviate the financial burden to the healthcare system caused by uncontrolled diabetes, i.e. addressing negative externalities.

Based on these objectives, I follow a libertarian paternalistic approach, i.e. I intend to develop a *nudge-based* intervention that does not penalise patients who deliberately prefer to live an unhealthy lifestyle if they wish to do so.

1.3.4 Nudge-based interventions

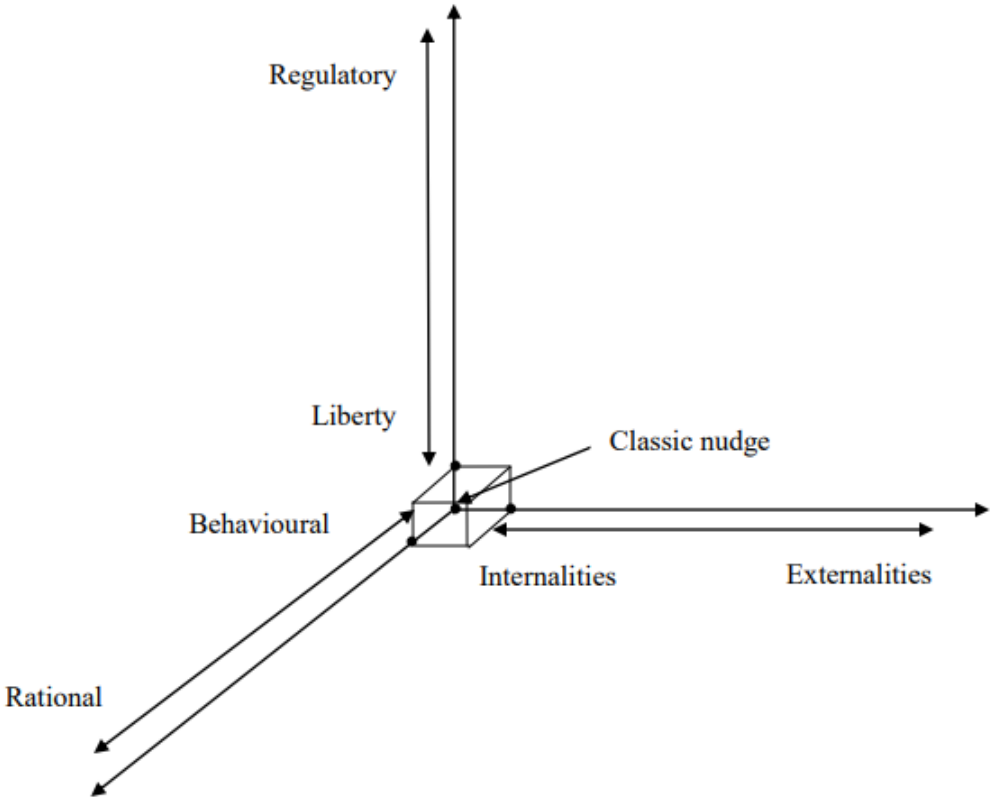
Oliver (2015) developed a handy diagram to quickly identify whether a behavioural economic-informed intervention can be defined as a nudge (see Fig. 1.1). [81] This diagram depicts the libertarian paternalism space according to three criteria represented by three axes:

- The behavioural-rational axis, i.e. whether the intervention is informed by behavioural or standard economics' insights.
- The liberty-regulatory axis, i.e. whether the intervention preserves the freedom of choice or not.
- The internalities-externalities axis, i.e. whether the intervention addresses internalities or externalities.

A classic nudge will be located at the centre of the diagram: it is a liberty-preserving behavioural economic-informed policy that intends to address negative internalities.

It is meant to improve the welfare of an individual who is considered in isolation from others. The ethical implications of such a policy will be discussed in more detail in Chapter 6.

Figure 1.1: Oliver's libertarian paternalism space (2015). [81]



1.3.5 Roadmap

Building behavioural economic-informed interventions require (a) exhaustive collection of all factors impacting the decision-making process, and (b) structuring them in a rational way, based on empirical evidence. Models such as the one developed by Sitkin and Pablo (1992) [95] have received extensive empirical support. [96–98] These models suggest that both risk perceptions and risk attitudes play a key role in the underlying processes that drive people's decisions.

Risk perception, on the one hand, is a complex construct whose role in driving health-related behaviour has been well-established. People who underestimate their

risks may be less likely to adopt recommended behaviours, while overestimating their risks may cause so much anxiety that it hampers people from taking precautionary measures. [14] There is strong evidence supporting the causality of the association between perceived risks and precautionary behaviours (see Chapter 2). Although behavioural processes are complex, wrong risk perceptions are a major impediment to the adoption of self-care behaviours and, as a result, an additional risk for the occurrence of adverse outcomes.

Risk attitudes (or risk preferences), on the other hand, can be regarded as the natural tendency of an individual to take or avoid risks. In the health domain, for example, people diagnosed with a lifestyle-related chronic disease such as T2DM must decide whether to adopt a healthier lifestyle or not, considering both the benefits (better health outcomes in the long run) and the costs (more commitments in their daily life, such as exercising regularly). As physicians observe low rates of adherence to healthy lifestyle in practice, one hypothesis may be that such patients are more inclined to "gamble with their health" (i.e. that they express a risk seeking behaviour, or a "taste for gambling") than an average individual. Because risky decisions made by people with T2DM may greatly impact their life expectancy and QoL, it is crucial to better understand the characteristics of and factors underlying their risk attitudes (see Chapter 3).

For example, Sitkin and Pablo's model relies on three main assumptions[21] (see Fig. 1.2):

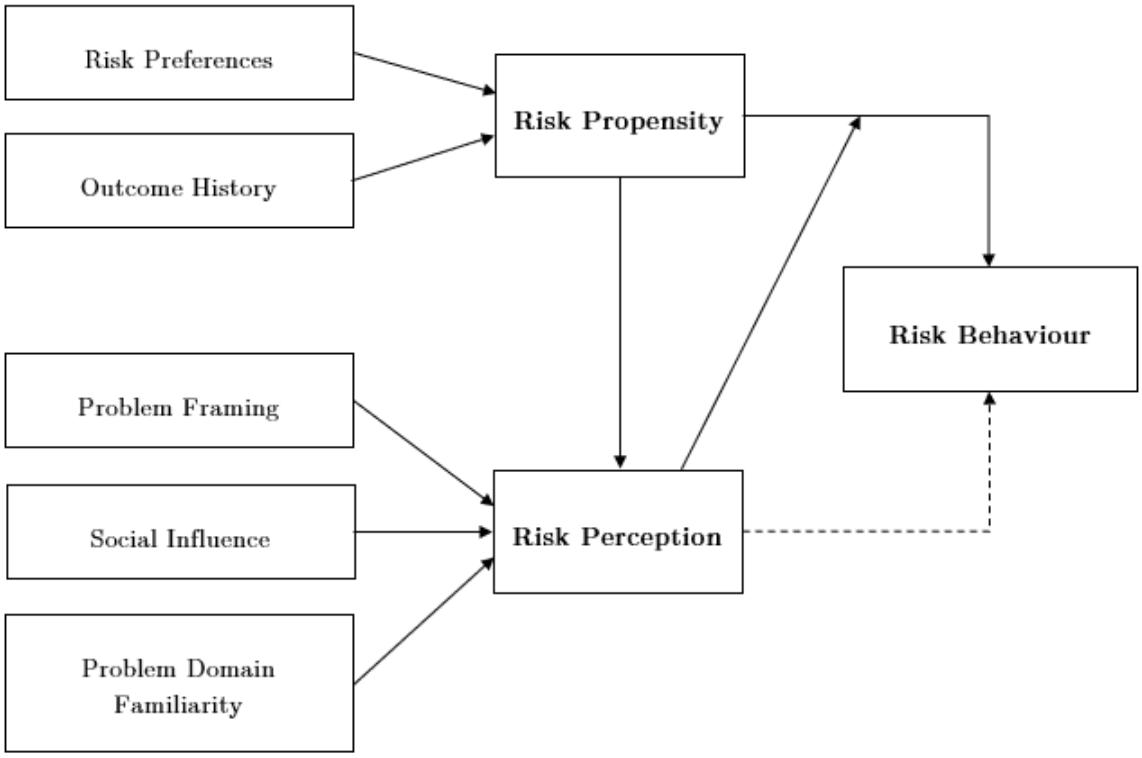
- Risky behaviour is mediated by two determinants: risk propensity (i.e. risk attitudes)[22] and risk perceptions.
- Risk propensity is determined by risk attitudes and prior outcome history.

[21] The detailed model relies on a total of 11 assumptions which govern the relationships between each factor and associated determinant of risky behaviour.

[22] In the literature, there are different interpretations of these two concepts that might cause confusion. In this Thesis, I will use the term risk attitudes to describe the natural tendency to take or avoid risk (i.e. the individual trait), whereas risk propensity will refer to a broader concept of attitude towards risk, which encompasses risk attitudes, among other factors.

- Risk perceptions are modulated by both decision-making biases (especially framing effects) and risk propensity.

Figure 1.2: Sitkin and Pablo's model (1992). [95]



Following this approach, which has also been encouraged by behavioural scientists who are focussing on preventive healthcare (see e.g. Fisher *et al.*, 2002 [99] for an application to diabetes prevention[23]), I suggest a similar structure adapted to T2DM self-management (see Fig. 1.3). The objective is not to propose a new model of health behaviour, but rather to summarise all the evidence that has accumulated, in various fields, on factors known to have an influence on the (risky) decision to adopt or not a precautionary behaviour. The idea is to ground future health promotion interventions on an enhanced methodological framework centred on people's decision-making process in the wider sense, and not only on classical

[23]Fisher and colleagues advocate for a better understanding of how people comprehend and make decisions regarding the complexities of diabetes prevention. In particular, they encourage examining risk perception and decision making in individuals at risk of diabetes. [99]

behaviour change models. In other words, to develop a behavioural economic-informed health intervention.

The nudge-based risk communication intervention that will be developed in this Thesis aims to target as many factors underlying the health-related decision-making processes of T2DM patients as possible (see details in the next section). I believe that such an approach will enhance the effectiveness of the risk communication and, as a result, optimise the chances to motivate adherence to self-management in this population.

1.4 Overview of the Thesis

The Thesis is divided into two main parts and includes six chapters (see Table 1.3), including this introductory chapter.

The first part of the Thesis (**Chapters 2** and **3**) is concerned with gathering the evidence that will support the design of a nudge-based risk communication intervention tailored for people with T2DM, in accordance with the strategy described in 1.3.

In **Chapter 2**, the *risk perceptions* determinant of health-related decisions is explored. The aim is to shed light on the causal association between risk perceptions and self-management (i.e. how risk perceptions influence the initiation and maintenance of appropriate self-care behaviours) and to better understand how risks of developing diabetes-related complications are perceived by people with T2DM. In this chapter, I conduct, to my knowledge, the first systematic review of risk perceptions for diabetes-related complications in T2DM populations. I perform a systematic search of nine electronic databases for peer-reviewed articles published on or before 1 March 2016. Data from 18 studies reporting lay perceptions of risks for complications in T2DM populations are reviewed.

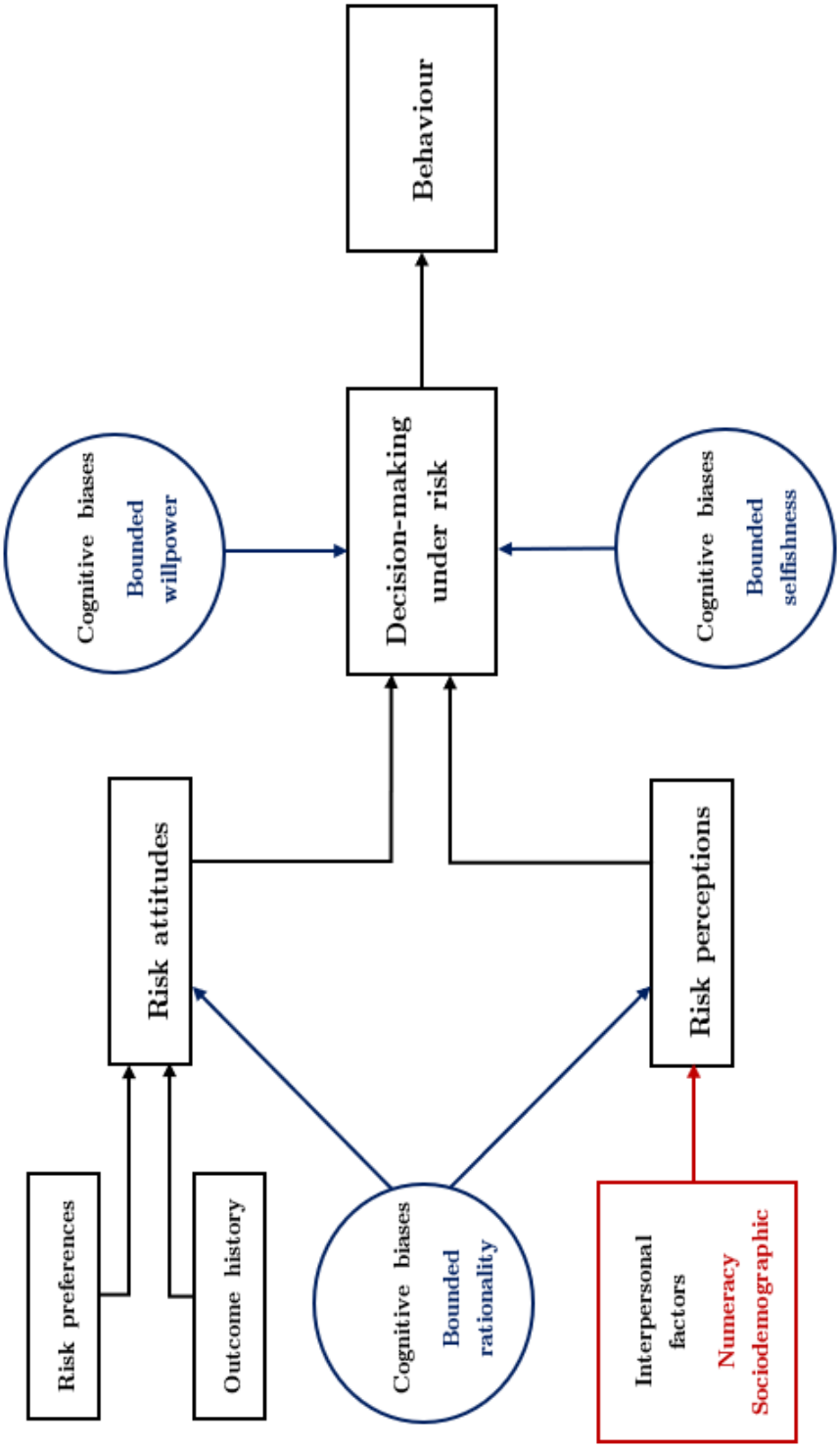


Figure 1.3: Determinants of self-management behaviour: a model of decision-making under risk.

Chapter 3 goes further in the exploration of patients' decision-making processes by investigating the *risk attitudes* determinant of health-related decisions. The objective is to better understand the rules that govern patients' suboptimal choices in the health domain and to identify specific behavioural economic material (decision-making biases) that could be targeted. I first explain how decision theory can help inform behaviour change interventions, and why prospect theory (PT) [100] is the descriptive theory of choice in this domain. Then, I design and conduct a study to measure the risk attitudes under PT of a T2DM population. The study includes 120 participants, half of whom were living with T2DM, and was approved by the competent NHS research ethics committees. For the first time, utility under PT is measured in the health domain using outcomes that include both life duration and quality of life. Focusing on a hypothetical trade-off between health outcomes and T2DM self-management, the study allows identification of behavioural economic materials specific to T2DM patients.

The findings of the first part of the Thesis shed light on a significant gap between a normative ideal of decision making (optimal decisions recommended by physicians) and the descriptive reality (actual decisions made by patients) regarding self-management of T2DM. Based on this observation, the second part of the Thesis (**Chapters 4** and **5**) is concerned with reducing this gap, so that adherence to self-management behaviour in T2DM populations can be improved. The objective of this second part is to design, develop and pilot a new type of risk communication intervention, informed by behavioural economics (*nudge-based*).

In **Chapter 4**, I first review the risk communication literature to identify the best ways of taking advantage of the behavioural economic elements uncovered in **Chapters 2** and **3**. After selecting the most appropriate content and format for this purpose, I design and develop an innovative tool, entitled PERCODIA, to deliver personalised risk information to people with T2DM. The objective of PERCODIA is to improve insight and recall of diabetes-related risks and, as a result, motivate sustained adherence to self-management. PERCODIA is developed

in collaboration with patients, clinicians, and digital health specialists for use in routine primary care consultation. Primary care is indeed the key point of contact with the healthcare system for this patient population.

Chapter 5 is dedicated to piloting PERCODIA in a real-life setting. After a brief review of existing risk communication interventions for people with T2DM, I design and conduct a pilot randomised controlled trial (RCT) to assess the feasibility of using PERCODIA in routine primary care consultation and evaluate its impact on self-management in patients with poorly controlled T2DM. The overarching objective of this study is to inform the potential development of a larger RCT to be conducted in the future. Forty adults with poorly controlled T2DM (HbA1c>7.5%)are allocated to usual care supported by PERCODIA or usual care only. Recall of personalised risk estimates and self-management behaviour are evaluated before and 12 weeks after the intervention. Other measured outcomes include intentions to make lifestyle changes, emotional impact (anxiety and worry levels), and satisfaction with the intervention. The protocol was approved by the competent NHS research ethics committees.

Finally, **Chapter 6** summarises the Thesis findings and discusses their implications. A list of manuscripts that have been written based on the research presented in this Thesis is provided in Appendix A.

Table 1.3: Overview of the Thesis.

Chapter	Objectives
Chapter 1 Introduction	(a) Background and objective (b) Justification for research (c) Strategy approach
Part 1 - A new type of risk communication for people with T2DM: rationale and strategy	
Chapter 2 Risk perceptions and self-management of Type-2 diabetes	(a) Shed light on the link between health risk perceptions and self-care behaviour. (b) Establish an evidence-based synthesis of how people with T2DM perceive their risks for diabetes-related complications.
Chapter 3 Risk attitudes and self-management of Type-2 diabetes	(a) Explain how decision theory and behavioural economics can inform health behaviour interventions. (b) Measure risk attitudes and identify relevant behavioural economic material.
Part 2 - A new type of risk communication for people with T2DM: development and feasibility in primary care	
Chapter 4 Development of a nudge-based risk communication tool	(a) Review the literature on risk communication, in order to identify the best ways of taking advantage of the behavioural economic elements uncovered in **Chapters 2** and **3**. (b) Develop a nudge-based risk communication tool (PERCODIA) in collaboration with patients, clinicians and digital health specialists.
Chapter 5 Assessment of a nudge-based risk communication intervention in primary care: a pilot RCT	(a) Review the design of existing risk communication interventions. (b) Design and conduct a pilot RCT to assess the feasibility of using PERCODIA in primary care and evaluate its potential impact on risk perceptions and self-management.
Chapter 6 Conclusion	(a) Summary of findings and interpretation (b) Implications of findings

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2

Risk perceptions and self-management of Type-2 diabetes

As discussed in the introductory chapter, both risk perceptions and risk attitudes play a key role in the underlying processes that drive decisions under risk and, in turn, self-management behaviour. In this chapter, I am going to focus on the risk perception determinant of the decision-making process.

Risk perception is a complex construct that plays a central role in decision-making and behavioural processes. In this chapter, the aim is to shed light on the association between health risk perceptions and precautionary behaviour (i.e. how risk perceptions influence the initiation and maintenance of appropriate self-care behaviours); and to understand, in particular, how risks of developing diabetes-related complications are perceived by people with T2DM. Prior to giving a brief overview of the role of the construct in the main models of health behaviours, I first clarify the different notions of risk perceptions which are found in the literature. Then, I review the empirical evidence on the association between perceived health risks and precautionary behaviour. Finally, the last section of the chapter provides a systematic review of studies that investigate risk perceptions in T2DM populations.

2.1 Risk perceptions

2.1.1 Notions

Risks are an integral part of our environment. Each decision made by an individual – whether it implies her own interests or the interests of others – needs a preliminary risk assessment. In some cases this process is straightforward and does not require any reflection (e.g. checking for traffic before crossing the street), but in many others both the nature and the magnitude of the risk cannot be estimated with accuracy. Complication and mortality risks associated with a disease belong to this second category. In the medical literature, risk is often defined as "the product of the probability of an outcome (*perceived susceptibility*[1]) and the severity of that outcome (*perceived severity*)". [2, 3] However, most authors distinguish the two

[1] In the literature, the terms *perceived vulnerability* and *perceived likelihood* may also be used. [1]

constructs and tend to reduce the risk to its *perceived susceptibility* dimension only when investigating risk perception. Although epidemiologists – by benefiting from large datasets and powerful statistical tools – can provide physicians with reliable risk estimates at the population level; there are grey areas in the way individuals, and especially patients, process such information.

The construct of risk perception is not treated uniformly in the literature. Before going into further details, it seems appropriate to clarify the various terms encountered during the review process. First, perceived risk may refer to either absolute or relative (comparative[2]) risk. *Absolute risk* refers to the likelihood of experiencing an outcome over a specific period of time[3], whereas *comparative risk* refers to how likely a person is to experience a hazard compared to another person or group in a before/after comparison. [4] More precisely, there are two types of comparative risk measures. *Direct* comparative risk measures, which ask an individual to evaluate his own risk relative to an average person[4]; and *indirect* comparative risk measures, which require separate judgments of one's own absolute risk and others' absolute risk.[5] Indirect comparative risk is then computed as a difference score (own absolute risk - others' absolute risk). [5] Alwan *et al.* (2009) note that absolute risk perceptions, despite high-potential relevance for prevention, are more rarely assessed than comparative ones. [6] In some studies, risk perception may also be conditional on a particular event.[6] [7] One complexity is that those various notions of risk perception do not capture exactly the same information; this issue is discussed further in the next section.

Psychologists and behaviourists tend to agree on the two-dimensional nature

[2]Although the qualifiers *relative* and *comparative* are used interchangeably in the literature, some authors recommend not using the former to avoid any confusion with the epidemiological notion of relative risk, which is the extent to which a risk factor changes one's risk of a particular illness. [4]

[3]It may be expressed as numerical probability estimate (e.g. "Her risk of developing breast cancer within the next 10 years is 1%") or as verbal qualifier (e.g. "She is at moderate risk of developing breast cancer in the next 10 years").

[4]e.g. "I think that my risk of developing breast cancer is above average".

[5]e.g. "I think that my risk of developing breast cancer is 10% but that the average risk is only 5%".

[6]e.g. "Would your risk of developing heart disease increase, decrease, or stay the same if you were to exercise more?".

of risk perception. When facing a risk, individuals not only use cognitive skills to assess their likelihood of being affected (cognitive dimension), but also react to it emotionally (affective dimension). In other words, risk perception is a combination of how people *think* about the risk, and how they *feel* about it. [8, 9] There is strong evidence supporting the simultaneous existence of these two dimensions. [4, 7] However, whether one dimension prevails over the other in the assessment process is still open to discussion. Some authors think that cognitive risk perceptions are simply informed by affective states [9], whereas for others emotions are the main determinant of individual decisions. [10]

I decide to focus on the cognitive dimension of risk perception, considering that emotions, by nature, do not fundamentally differ from the other factors which influence the cognitive assessment process. From now on, the term *risk perception* will refer exclusively to cognitive risk perception; while any reference to its affective dimension will be made explicit. This approach is justified for two reasons. First, most of the literature on health risk perception follows the same logic by evaluating (cognitive) risk perception and feelings separately (see 2.1.2). Second, current theories of decision under risk – which will be used as an analysis tool in this Thesis – consider emotions as a classical cognitive bias, with no particular importance (as compared to other biases) in the decision-making process (see Chapter 3).

2.1.2 Health risk perceptions

Apart from affective states, multiple factors are likely to influence risk perception. [11, 12] Although the way of classifying those factors varies in the literature, two broad categories stand out. A first group of factors includes interpersonal characteristics[7] and social differences.

First, numeracy – defined as "the ability to process basic probability and numerical concepts" [13, 14], plays a major role in the comprehension of risk information. [15–17] As Peters *et al.* (2007) note, understanding numbers is essential for making informed health decisions. [18] An appropriate level of numeracy is

[7] Between-individual variability.

a prerequisite for the estimation of accurate disease-related risk estimates. The pernicious effect of low numeracy on assimilating health risk information has been reported in several studies. Schwartz *et al.* (1997), when examining the association between numeracy[8] and accuracy of perceived risks for breast cancer in a population of female veterans, found that high numeracy was strongly correlated to accurately assessing the benefit of mammography. [19] When investigating the influence of numeracy on the accuracy of adverse event risk estimation following the presentation of information about a medicine, Gardner *et al.* (2011) demonstrated that people with a lower numeracy level make larger errors in interpreting such information. [20]

Second, many studies have pointed out the impact of sociodemographic variables such as age, sex, race, and education, on risk perception. In a population of people with T2DM, Homko *et al.* (2010) found that women's perceived risks for cardiovascular diseases were significantly greater than men's. [21] Katapodi *et al.* (2004), when looking at the predictors of perceived risk for breast cancer, found evidence that younger age was associated with higher perceived risks. [22] Two studies also demonstrated that health risk perceptions vary among urban/rural populations and according to race/ethnicity. [23, 24] Moreover, several studies have underlined the positive correlation between family history of chronic disease and greater perception of the risks associated with that disease, especially in the area of cancer [22, 25] and T2DM. [26, 27]

A second category is formed of factors specific to each individual (intrapersonal level[9]). They are commonly referred to as (subjective) *beliefs*. When risks are disease-related, health risk perception results from matching personal risk factor information with so-called beliefs about those risk factors. [28–30] In this context, the literature has identified beliefs that matter more than others; including medical knowledge (if a risk factor does not fit within beliefs about disease causation, it is

[8] In this study, numeracy level was assessed through simple numerical tasks; one of them involved estimating the number of heads resulting from tossing a fair coin 1,000 times. One-third of the sample (n=500) believed that it would result in fewer than 300 heads.

[9] Within-individual (individual-specific) variability.

unlikely to increase perceived risk), illness perception (or perceived health status, a higher number of perceived negative consequences associated with the disease is likely to increase one's perception of risk for complications), and overconfidence (people do not see themselves as vulnerable, albeit they are aware of the risk). On a related note, economists have also reported evidence of the significant impact of *salience effects* on risk perceptions. In a study investigating preference-based values of safety programmes in different contexts (road safety, rail safety, domestic fires and public fires), before and after a tragic railway accident in London, Chilton *et al.* (2002) found a very significant upward shift in the priority given to the railway safety programme after the accident (measured in terms of *value for the prevention of a statistical fatality* or VPF). [31] This shift in preference was largely attributable to a higher perceived exposure to rail risks and to a decrease in the salience of baseline levels of risk. As value-based public preferences drive (in theory) the prioritisation of policies for public safety, such effects need to be understood and taken into account to adapt the VPF criterion.

As Waters *et al.* (2013) point out in their comprehensive review of the association between risk perceptions and health-related decisions, patients' perceived risks "often go awry". [4] Although this might stem from a lack of information at the root (i.e. from medical and nursing staff); a major cause is the impact of aforementioned beliefs on individuals' cognitive assessment process of risk estimates.

When asked to provide absolute numerical estimates, patients tend to overestimate chronic disease-related risks. Asimakopoulou *et al.* (2008) found evidence that people with T2DM were unrealistically pessimistic about their risk of developing coronary heart diseases and stroke. [32] Frijling *et al.* (2004), when looking at a diabetic population without distinguishing between Type-1 and Type-2 diabetes mellitus (T1DM and T2DM, respectively), showed that patients overestimated their actual 10-year risk for myocardial infarction and stroke by more than 20%. [27] This pessimistic bias has also particularly been highlighted with regards to breast cancer. Black *et al.* (1995) found that women aged 50 years and older who did not have a history of breast cancer, when asked to assess their risk of developing

the disease, provided a median estimate equivalent to 20 times higher than their actual risk. [33] More recently, a study reported that women at slightly increased risk of breast cancer (diagnosed with a precancerous condition) tended to greatly overestimate their lifetime risk of developing breast cancer. [34]

However, most studies investigating perceived risks have focused on comparative risks. In this context, quite paradoxically, it turns out that people generally underestimate their risks of developing health problems. Weinstein (1980) referred to this phenomenon as optimistic (or overconfidence) bias. [35] Evidence of optimistic bias has been widely supported in the literature, whether it regards risks associated with breast cancer [23], cardiovascular diseases [36, 37], or diabetes complications. [38–40]

The complexity of risk perception as a multidimensional construct has been largely discussed, in several fields. In the health domain, there is evidence that perceived risks – whether absolute or comparative – can be contaminated by a wide range of errors and biases. [11, 41] However, do biased risk perceptions matter? Previous research seems to suggest that they do, given the existence of a link between risk perceptions and people's propensity to engage in risky behaviour. [1, 42] Although the association is not yet fully understood and varies across areas of focus, accurate risk assessment plays a key role in the initiation of behaviours which aim at minimising this risk. [43] People who underestimate their health risk may be less likely to select less-risky options; while excessively high risk perceptions may cause so much anxiety that they hamper people from taking precautionary measures. [43, 44] The next section investigates the link between risk perception and risky behaviour, focussing on NCD-related protective behaviours.

2.2 Link between risk perceptions and precautionary behaviour

The aim of this section is to assess the causal association between health risk perceptions and precautionary behaviours, i.e. to estimate the extent to which

perceived risks may predict the initiation and maintenance of self-care behaviours. A brief review of the main models of health behaviour, along with associated empirical evidence of the impact of perceived risks, is provided.

2.2.1 Psychosocial theories

Since the 1950s, social psychologists and behaviourists interested in public health issues have developed theories/models[10] intended to predict, explain, and change health behaviours. As Sutton (2001) indicates in a comprehensive review, psychosocial theories can be divided into two main groups, respectively referred to as social cognition models and stage models. [45]

Social cognition models assemble similar theories according to which health behaviours are mostly determined by a small set of cognitive and affective factors. Four theories stand out in the literature; differences lie in the elements constitutive of this set of factors. The health belief model (HBM; Becker, 1974 [46]), which was a first attempt to explain why some people take preventive measures (such as vaccination or screening) while others do not, relies on four main constructs: perceived susceptibility and perceived severity of the disease; perceived benefits and perceived barriers of adopting behavioural recommendations (see Fig. 2.1). According to the HBM, higher perceived susceptibility is associated with a higher probability of adopting recommended behaviours.

In order to explain how people respond to health threat communications, Rogers (1983) set out the protection motivation theory (PMT) less than a decade later (see Fig. 2.2). [47] Unlike HBM, which focuses on determinants of health behaviour *per se*, PMT specifies predictors of people's *intention* to adopt a recommended action. Of those predictors, perceived vulnerability is one of the four that has received most empirical attention. The three others are perceived severity of the disease, response efficacy (the belief that the recommended action is effective in reducing the threat), and perceived self-efficacy (the confidence in one's own ability to achieve intended results). [45]

[10] In the literature, those two terms are used interchangeably.

Figure 2.1: Basic elements of the health belief model.

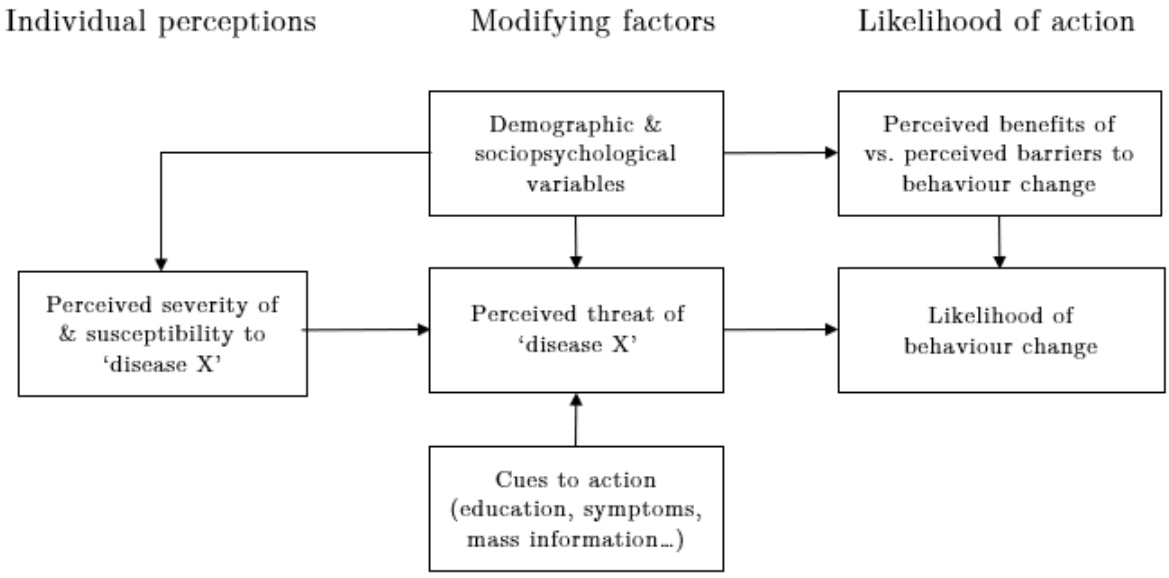
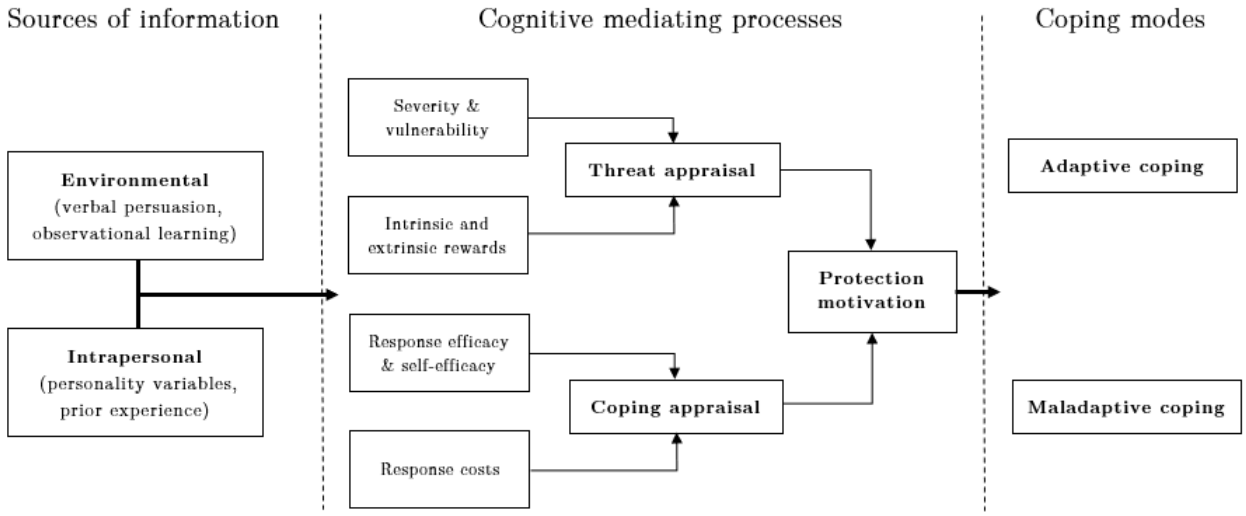


Figure 2.2: Overall model of protection motivation theory (derived from Floyd *et al.*, 2000 [48]



The theory of planned behaviour (TPB; Ajzen, 1991 [49]), on the other hand, focuses on how perceived behavioural control (how much control over a situation a person believes she has) explains one's intentions to change behaviour. However, TPB does not consider risk perception (see Fig. 2.3). Finally, Schwarzer and Fuchs' revised version of Bandura's Social Cognitive Theory [50] – which is called health action process approach (HAPA), asserts that self-efficacy (the extent to which one

believes in her own ability to complete tasks and reach goals, a notion similar to perceived behavioural control in TPB) and outcome expectancies are, along with risk perception, the two constructs at the root of health behaviour (see Fig. 2.4). [51]

Figure 2.3: Theory of planned behaviour.

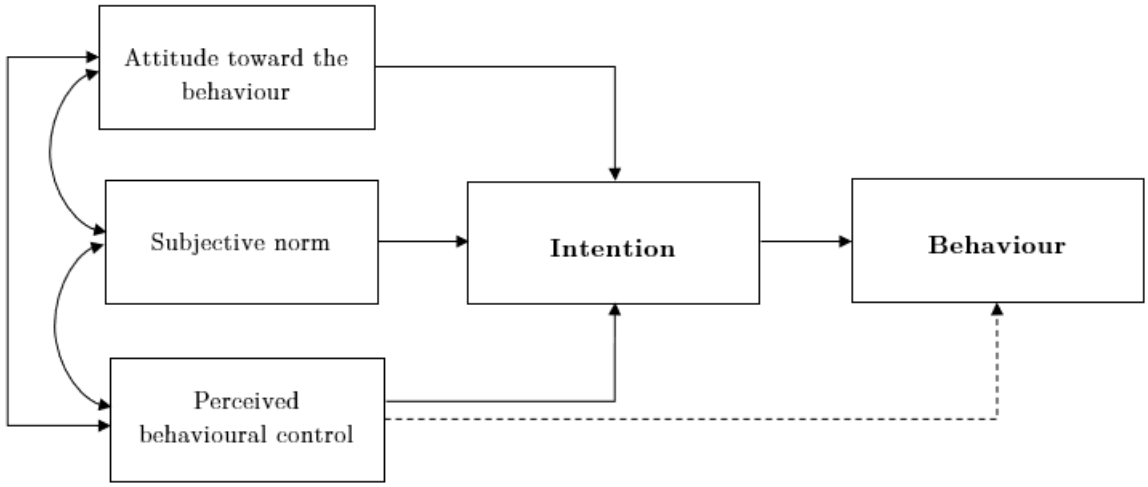
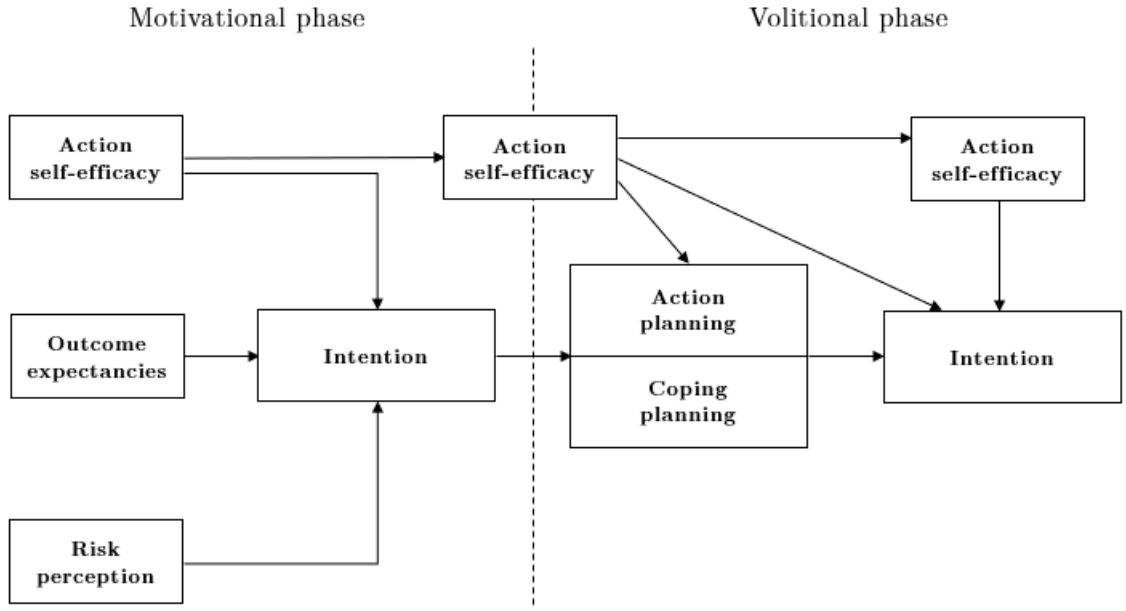


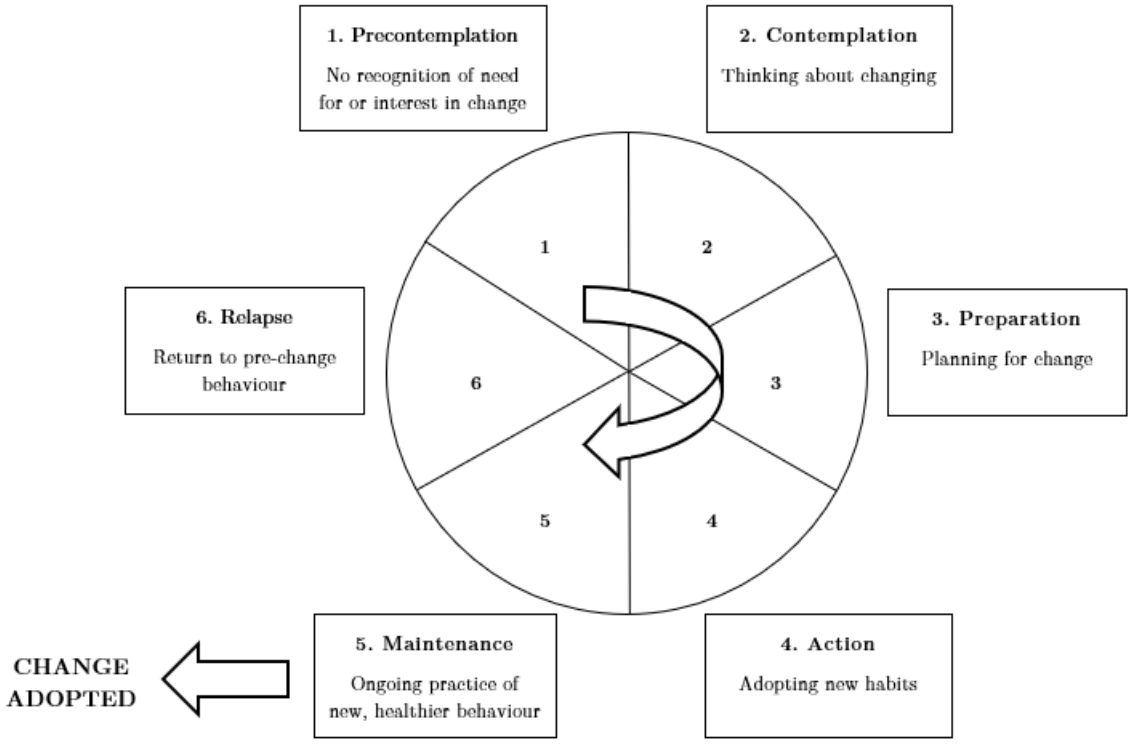
Figure 2.4: Health action process approach model (derived from Schwarzer, 2008 [52]).



Stage models use similar concepts to social cognition models but organise them in a different way. According to this approach, behaviour change involves movement through a sequence of discrete, qualitatively distinct, stages; and different

factors are assumed to be important at different stages. [45] This results from the willingness to gather relevant constructs drawn from various behaviour change theories together in a single model. The main stage model is the transtheoretical model (TTM; Prochaska *et al.*, 1992 [53]), which stems from studies investigating the processes of change in smoking cessation. Risk perception is involved in the two first stages of the model (respectively the *precontemplation* and the *contemplation* stages; see Fig. 2.5). Several variants of the TTM have been developed and applied to a wide range of other health behaviours, including physical exercise and healthy eating (e.g. see Prochaska and Velicer, 1997 [54]).

Figure 2.5: Transtheoretical model of change (derived from Prochaska *et al.*, 1992 [53]).



Despite lack of generalisability characterised by the existence of many disparate models (some intended to explain behaviour, others designed as framework for health interventions), psychosocial theories have become a reference methodological framework for health promotion programs. This is because each cognitive and affective factor specific to a given model is assumed to be amenable to change

(e.g. through the provision of relevant information), and thus susceptible to trigger behaviour change. [45] However, these social psychological theories are not universally accepted and other approaches to explain behaviour exist (e.g. ecological models of health behaviours; see Sallis *et al.*, 2015 [55]). For instance, a major criticism is that they do not provide a satisfactory (realistically) rational way of how people form intentions and make decisions. Specifically, they are all based – to a greater or lesser extent – on the expectancy-value principle[11], which itself derives from the classical expected utility model, a normative model of decision-making. [4, 45] Descriptive models of decision-making, which have been developed recently, may provide a useful complementary methodological framework for health interventions (see Chapter 3).

The role played by health risk perception in determining self-care behaviour is admitted by four of the five main psychosocial models of behaviour change. However, is this supported by empirical evidence? What is the nature of the correlation between risk perceptions and self-care behaviour (causality, direction, magnitude)? The objective of the next two subsections is to provide an overview of the accumulated evidence about this association.

2.2.2 Empirical evidence

Empirical evidence of the association between risk perception and precautionary behaviour is vast and disparate. In order to correctly interpret the data, it is first necessary to examine how this association has been conceptualised, over time, in the literature. Weinstein *et al.* (1998) suggested an analysis framework based on two assumptions. [56] The first assumption underlines the temporal order of the association and states that *initial* perceptions of risk influence subsequent protective action. Hence, higher perceived risk of harm (cause[12]) is thought to encourage people to take action to reduce their risk (effect[13]). This assumption,

[11] Also called *rational choice approach*, this principle states that people, when making a decision, combine both the subjective probability of each potential outcome with the subjective value they attribute to each outcome.

[12] Measured initially, at time 1.

[13] Measured later, at time 2.

sometimes called *motivational* [42] or *behavioural motivational* [57], is inherent to most psychosocial theories of health behaviour. The second assumption, also known as the *risk reappraisal hypothesis* [57], characterises the reciprocity of the association between risk perceptions and risky behaviour. It asserts that perceived risks are affected by current patterns of behaviour in a reciprocal way (e.g. after taking a precaution, people believe their risk is lower than it had been before). The risk reappraisal hypothesis also implies that risk perceptions adapt instantaneously to behaviour changes, so that, when measured, they incorporate the perceived effects of the behaviour that is measured simultaneously.

Because the motivational hypothesis is the one of interest for this Thesis, I focus on the evidence shedding light on this assumption. Most studies aiming at providing empirical support for the motivational hypothesis have obtained mixed results.[14] First, this has led some authors to call into question its validity. [59, 60] But according to the most recent literature, this is more likely due to the fact that only a few of them have used longitudinal design and related appropriate tests. [56, 57, 61] Indeed, due to its temporal dimension, the motivational hypothesis need to be tested in longitudinal design.[15] Panel data, by providing an additional (time-related) dimension, have the advantage of offering more guarantees against reverse causality issues.

Waters *et al.* (2013), aiming at testing the motivational hypothesis as effectively as possible, endeavoured to gather all the existing longitudinal evidence[16] published between 2006 and 2011 about the association between perceived risks and health-related behaviour. [4] I review their main findings and discuss evidence as well from relevant studies since 2011. It must be noted that risk perception measures in this literature take many forms: absolute risks, direct comparative risks, and indirect comparative risks are the main dimensions measured (see 2.1.1).

[14] I decide not to review the studies investigating the validity of the risk reappraisal hypothesis, as it is not directly relevant for the Thesis. However, it is worth mentioning that empirical support for this latter has been found in different contexts (see e.g. Glenn *et al.*, 2011 [58]; Brewer *et al.*, 2004 [57]).

[15] This is also true for the risk reappraisal hypothesis.

[16] To be included in the report, studies must have assessed perceived risks prior to the behaviour.

Questionnaires use either Likert scales (e.g. ranging from 0: "not likely at all" to 5: "very likely") or directly ask the respondent to estimate a percentage score.

In terms of risk-reducing behaviours (i.e. behaviours that reduce the likelihood of developing a disease/complication)[17], several studies have demonstrated that perceived risk can be a reliable predictor of behavioural intentions. Brewer *et al.* (2007) performed a meta-analysis of 34 studies examining the association between perceived risk and vaccination behaviour, and found that risk awareness significantly predicted vaccination. [1] This result was confirmed a few years later by a Japanese study, in which researchers aimed to determine potential predictors of the uptake of A (H1N1) influenza vaccine. [64] Perceived risks have also been associated with risk-reducing behaviours in the area of cancer. For instance, in a population of women at high risk of developing breast cancer, it has been shown that those with higher risk perceptions are more apt to undergo prophylactic mastectomy and/or oophorectomy than those with lower risk perceptions. [65, 66] In addition, Hay *et al.* (2007) found that higher perceived risk of developing a secondary cancer was associated with higher smoking abstinence rates among newly diagnosed cancer patients [67]; while Janssen *et al.* (2011) [68] and Hillhouse *et al.* (2008) [69] reported that people with higher perceived susceptibility of developing skin cancer are more likely to use sunscreen and less likely to use sunbeds, respectively. Focusing on a population of smokers receiving medical care, Borrelli *et al.* (2010) found that perceived vulnerability predicts smoking cessation whereas optimistic bias predicts a lower likelihood of cessation. [70] Last, from a large-scale study, Carman and Kooreman (2014) showed that perceived risk probabilities significantly affect the subsequent take-up rate of flu shots, mammograms, and aspirin; this even after controlling for individually predicted risk using epidemiological models. [71]

Focusing solely on dietary and physical exercise behaviours, the literature is

[17]Looking then at precautionary behaviours that are not directly risk-reducing (i.e. behaviours that do not impact the likelihood of developing a disease/complication, but rather increase the likelihood of curing it), the literature is sparse and results are quite mixed. For instance, McCaul *et al.* (1996) showed that women who feel more vulnerable to breast cancer are more likely to get screened [62]; whereas Stewart *et al.* (2009), despite having a racially and ethnically very diverse sample, found no effect of perceived risk on mammography behaviour. [63]

sparser and evidence is more mixed. Shreck *et al.* (2014), after implementing a one-year randomised control trial, reported that increased perceived risks and decreased optimistic bias in people with T2DM reduced significantly high-fat food consumption and improved exercise adherence. [72] Winkleby *et al.* (1994), almost two decades before, had also shown the positive impact of higher risk perceptions on long-term CVD risk factor scores. [73] However, some authors have reported null or negligible results. Schwarzer and colleagues, for example, found no evidence supporting the link between risk perception and physical exercise adherence, whether surveying members of the general public [74] or patients undergoing cardiac rehabilitation. [75] Two reasons have come up to explain those contradictory findings. First, it may be that "physical activity and diet are such complex repeated behaviours that situational factors and habit overwhelm any effect of perceived risk". [4] A second explanation could be that, regarding these very behaviours, perceived risk is more important for the formation of intentions than engagement in the behaviour. [74]

Nevertheless, identifying a reliable estimate of the causal effect[18] of risk perception on behaviour requires, in addition to the use of longitudinal data, to go one step further by also dealing with endogeneity[19] and, to a lesser extent, autocorrelation issues. In this regard, some authors assert that the only way of providing a definitive answer to the question "How do risk perceptions impact health-related behaviour?" is to conduct experimental studies. In fact, experiments allow testing of the causal impact of interest by, first, changing perceived risks and, second, examining whether there is a corresponding change in behaviour. [61]

Sheeran *et al.* (2014), for the sake of causality, endeavoured to focus exclusively on experimental studies. They meta-analysed 208 studies investigating the impact of any of the four components of the *risk appraisal* construct (risk perception, anticipatory emotion, anticipated emotion, perceived severity) on health-related

[18] i.e. an estimate which, according to a given requirement level, is both unbiased and consistent.

[19] The possibility that a third variable (e.g. conscientiousness, optimism) would cause both the risk perceptions and the behaviour. In this case, the association of interest would be spurious (i.e. not causal), because it would be the product of associations between risk perceptions, behaviour, and the true casual factor.

intention and/or behaviour.[20] Although of small-to-medium magnitude, the impact of heightening risk perceptions on health behaviour (56 studies involved) was highly significant.[21] [61] For example, amongst the studies included, there is a randomised control trial (RCT) of computer-based communications using imagery to alter representations of heart disease risk, which successfully increased diet efforts 2 weeks after intervention. [76]

In the literature, however (unfortunately), most studies investigating the link between risk perceptions and precautionary behaviour use correlational (cross-sectional) data. Both perceived risks and behavioural components are assessed at a single point in time, then correlations are examined.[22] In this context, Brewer *et al.* (2004) suggested a third assumption to evaluate the association between risk perceptions and risky behaviour: the *accuracy hypothesis*. [57] This states that, holding other risk factors constant, people who engage in risky behaviours will have higher perceived risks (since they have higher actual risks). This assumption does not imply any causal link – it rather simply describes the association between the two constructs *at a single point in time* – and, as a result, it can be tested using cross-sectional data.[23] Meta-analyses of correlational studies [1, 22, 42, 48, 77–80] have indicated that risk perceptions have significant, albeit small, associations with both intentions and behaviour.[24] [61]

Because of the reciprocal influence between risk perceptions and precautionary behaviour, particular caution is thus required when analysing their association.

[20]Three inclusion criteria: (a) Experimental study design, i.e. the treatment is randomly assigned among participants; (b) The treatment produces a statistically significant difference in risk appraisals among participants who receive the treatment compared to control participants; and (c) a statistical comparison of subsequent intention/behaviour between treatment and control participants is reported or otherwise obtainable. [61]

[21]Sample-weighted effect size of heightening risk perceptions on behaviour [CI] =.25 [0.18–0.33].

[22]Although this approach can provide useful insights in some cases, it also raises a number of issues which several authors have cautioned against. [41, 42, 45, 56, 57] In particular, coefficients are likely to be biased due to reverse causality issues.

[23]One should not confuse *testing the accuracy hypothesis* (*relative* accuracy), i.e. examining the sign and significance of the correlation between perceived risks and risky behaviour at a single point in time (a high degree of relative accuracy would be indicated by a strong negative correlation between perceived personal risk and the amount of precautionary behaviour a person is performing) with *testing the accuracy of risk perceptions* (*absolute* accuracy), which is comparing perceived risk estimates with actual ones. [56]

[24]Sample-weighted average correlations have ranged between .09 [22] and .26 [1]. [61]

Focusing on studies aiming at testing the motivational hypothesis, Weinstein *et al.* (1998), after reviewing 81 analyses of the risk perceptions-risky behaviour association, reported evidence of three main methodological and conceptual issues regularly found in the literature. First, a *causal inference problem* stems from the use of cross-sectional data, due to reverse causality (as mentioned above). Several researchers turn out to misinterpret correlations as a test of the motivational hypothesis when they actually provide the relative accuracy of risk perceptions. Then, authors who use prospective studies sometimes do not control for prior behaviours, which may bias the correlation coefficient (*prior behaviour problem*). Finally, some prospective studies have also been performed in situations of little behaviour change, which reduces de facto the chances of identifying any causal effect (*behavioural stability problem*). [56]

2.2.3 Risk perceptions and interrelated factors

As predicted by psychosocial theories, empirical data have confirmed that numerous constructs interfere with risk perceptions, making the link between perceived risks and risky behaviour more complex. Although cognitive risk perceptions are the most frequent form studied across the literature [1, 42], several authors have drawn attention to the importance of examining, in parallel, worry and related affective dimensions. Specifically, affect[25] may have a dual role. On one hand, it may act as a modulator of the effect of risk perceptions on behaviour.[26] Klein *et al.* (2009), for example, found that smokers with higher perceived risk of developing lung cancer had higher intentions to quit smoking only if they reported low levels of worry. [81] Alternatively, risk perceptions may be stronger predictors of behaviour when worry is high, given that affect may help to motivate individuals to adopt preventive measures. [8, 9, 61] On the other hand, and despite the fact that the two constructs are correlated[27], it has been shown that worry may also drive behaviour

[25] Assessed, in most studies, by measuring worry by means of self-reported questionnaires using a 5- or 7-point Likert scale, ranging from "not worried at all" to "extremely worried".

[26] Waters *et al.* (2013) refer to "a role in the conceptualisation of perceived risks". [4]

[27] Albeit only moderately (see e.g. Bjorvatn *et al.*, 2007 [82]; Zajac *et al.*, 2006 [83]; Lipkus *et al.*, 2005 [84]).

independently from perceived risks. [8, 85] Some studies have even found that it could be a better predictor of health-related behaviour than perceived risks. [86, 87] Another affective construct that has recently received greater attention is anticipated regret (the belief that one will feel regret at negative health outcomes in the future). [4, 88] It is usually measured using single or multi-item questionnaires that use the words *regret* or *wish*.[28] [88] In particular, Chapman and Coups (2006) reported that both anticipated regret of not being vaccinated and worry about influenza mediated the effect of perceived risk reduction on vaccination behaviour. [87]

As Ferrer *et al.* (2013) point out, investigating the way cognitive and affective dimensions of risk perceptions interact is important, because if such interactions influence behaviour, interventions and risk communications that are designed to increase either or both constructs may have unintended results. [85] Because affect may mitigate the rational association between risk perceptions and behaviour, or, conversely, cause synergistic effects, related affective constructs should be assessed and taken into consideration when designing interventions targeting behaviour change. [85] In fact, that reasoning can be extended to all factors (not only affective ones) likely to influence risk perceptions. Sheeran *et al.* (2014), in particular, note that heightening each element of risk appraisal separately has only small-to-medium effects on behaviour, whereas heightening several of them at the same time produces much larger effects. [61]

In sum, among the vast literature investigating the association between risk perceptions and precautionary behaviour, only a minority of studies have used a suitable design to test the motivational hypothesis (i.e. experimental studies, or, at least, longitudinal data). This hypothesis, being particularly relevant to describe that association, has been key in informing the literature on risk communication. [85] Studies that met the required criteria have provided strong evidence in favour of perceived risks as a predictor of engagement in protective health behaviours. However, despite high significance, the magnitude of the causal effect was modest.

[28] e.g. "If I did not vaccinate my child, I would regret it/wish I had".

The key point is that enhancing risk perceptions is necessary, but not sufficient, to promote behaviour change. Taking into account other factors that influence health-related decisions, i.e. using a broader assessment framework, is needed to design more effective behavioural interventions.

2.3 Risk perceptions of diabetes-related complications in T2DM populations

In T2DM, there is no clear understanding of how people perceive their risk of experiencing diabetes-related complications. Identifying particular trends and/or systematic biases characterising risk perceptions in this population would allow targeted and more effective risk communication interventions to be developed. To address this issue, I undertook an evidence-based synthesis of how people with T2DM perceive their risks for complications. [89]

2.3.1 Methods

a Search strategy and selection criteria

I conducted a systematic review to identify all studies that investigated lay risk perceptions for diabetes-related complications in people with T2DM. The review was performed in accordance with the PRISMA Statement methodology checklist. [90]

Nine electronic databases (MEDLINE, Embase, Global Health, PsycINFO, EconLit, Scopus, Web of Science, International Bibliography of the Social Sciences and Cochrane Library) were searched for peer-reviewed articles published on or before 1 March 2016. The search strategy consisted of different combinations of keywords related to (a) risk perceptions (e.g. awareness, susceptibility, likelihood); and (b) diabetes (e.g. Type 2 diabetes, t2dm, non-insulin dependent diabetes) (see Appendix B).

Two inclusion criteria were applied, the study had to: (a) include people diagnosed with T2DM and (b) examine lay risk perceptions for diabetes-related

complications (i.e. perceptions not influenced by a preliminary risk communication intervention). I excluded studies that: (a) were not peer-reviewed articles; (b) included people with T1DM or without diabetes; (c) reported perceptions measured after a risk communication intervention; and (d) reported perceptions of diabetes treatment benefits and risks, or general perceptions about T2DM. Screening by title, abstract and full-texts was also undertaken independently by a second reviewer. Results were compared and any disagreements regarding inclusion were resolved by consensus.

b Data extraction and quality assessment

For each study that met the inclusion criteria, information was extracted on sample size, population characteristics, types of perceived risks measured, types of actual risks used as comparators (when appropriate) and outcomes.

The quality of identified studies was also independently assessed by myself and a second reviewer, using two quality assessment tools. For qualitative studies without a quantitative component, the Critical Appraisal Skills Programme (CASP) Qualitative Checklist was used. [91] Each of the 10 items and the overall quality were rated as good, fair or poor. A study received a good global rating if it had no poor item ratings or at most one fair item rating, a fair global rating if it had one poor item rating or at most three fair item ratings, and a poor global rating if it had two or more poor item ratings or four or more fair item ratings. Other studies were evaluated using a adapted version of the Quality Assessment Tool (QAT) for Observational Cohort and Cross-Sectional Studies (those items not considered relevant to the design of included studies were removed; see Appendix C) [92], which is an instrument designed to assess risk of bias and methodological quality of cohort and cross-sectional studies. The score on each item of the tool allowed computation of an overall quality score in accordance with the QAT guidance. Similarly, the overall quality of a study was rated as good, fair, or poor, using the same rating system.

c Data synthesis and analysis

Given the expectation of significant heterogeneity in the methods and outcomes reported in the studies, I qualitatively synthesised the results for each study within summary tables. This allowed highlighting of similarities and differences among studies that addressed similar research questions. Finally, perceived risks may be absolute, comparative or conditional, depending on how they are measured. It has been shown that there is limited overlap across the three dimensions [93]; it is thus important to distinguish them and analyse each dimension separately.

d Study outcomes

I classified the selected studies according to their *outcomes*, i.e. to the specific dimension(s) of perceived risks they assessed. In total, 23 outcomes were identified and allocated into five categories. First, *absolute accuracy* refers to the difference between perceived and actual absolute risks. Second, studies measuring comparative risk were labelled as studies of *optimistic bias*. Third, some studies investigated multiple aspects of risk perceptions, either using questionnaires that measure several dimensions of perceived risks (and then generate an overall risk perception score) or through qualitative studies. Last, the *complementary evidence* category includes studies that focused on a very specific aspect of risk perceptions — for example, the measurement of perceived conditional risks — or studies providing valuable information that cannot be classified into one of the four previous categories.

2.3.2 Results

a Search results

A total of 5482 references were obtained from the search strategy. After eliminating duplicate records (n = 3373), 2109 references remained. These records were all screened and 2087 irrelevant studies were excluded. The remaining 22 full-text articles were assessed for eligibility. Of these, eight studies did not meet the inclusion criteria. In addition, the cited references of the 14 selected articles were searched for further studies of interest. Four new studies met the inclusion criteria,

bringing the total to 18 studies included in the review [7, 21, 26, 27, 32, 39, 40, 94–104], providing information for 23 different outcomes (see Fig. 2.6). Extracted data are summarised in Table 2.1.

b Study characteristics

Publication year ranged from 2002 to 2014, emphasising the recent nature of the research. Half of the studies were conducted in the US [7, 21, 26, 39, 94–98], followed by the UK [32, 40, 100, 101], The Netherlands [27, 102], Australia [103], Pakistan [99] and Spain. [104] Sample sizes varied widely, from 6 to 2008 individuals (median 143). Some studies reported in-depth semi-structured interviews and thus focused on a restricted number of persons, whereas others were large-scale studies with self-reported questionnaires.

c Study populations

The study populations consisted of people with T2DM, by decreasing order of frequency: (a) with no cardiovascular disease (CVD)/coronary heart disease (CHD) or no history of CVD/CHD; (b) recently diagnosed with T2DM; (c) socially deprived or from minority ethnic populations; and (d) with at least one additional cardiovascular risk factor, e.g. hypertension, hyperlipidaemia or self-reported smoking. Two studies did not specify whether people included in the study population had T1DM or T2DM (or if the study population was composed of both types). [27, 98] Despite a potential violation of the inclusion criteria, I decided not to exclude them because of the high probability that their study populations were composed of people with T2DM and their substantial sample sizes.

d Methods used to evaluate risk perceptions

Of 18 studies, four were *qualitative*, i.e. studies that used a purely qualitative research design (semi-structured or focus group interviews). [40, 97, 100, 101] I refer to the remaining 14 studies as *quantitative studies*. Of these, seven studies measured the absolute accuracy of risk perceptions, four studies investigated the

existence of optimistic bias, two studies assessed a risk perception score and four studies provided complementary evidence.

Figure 2.6: PRISMA statement flow diagram: summary of systematic search and review process.

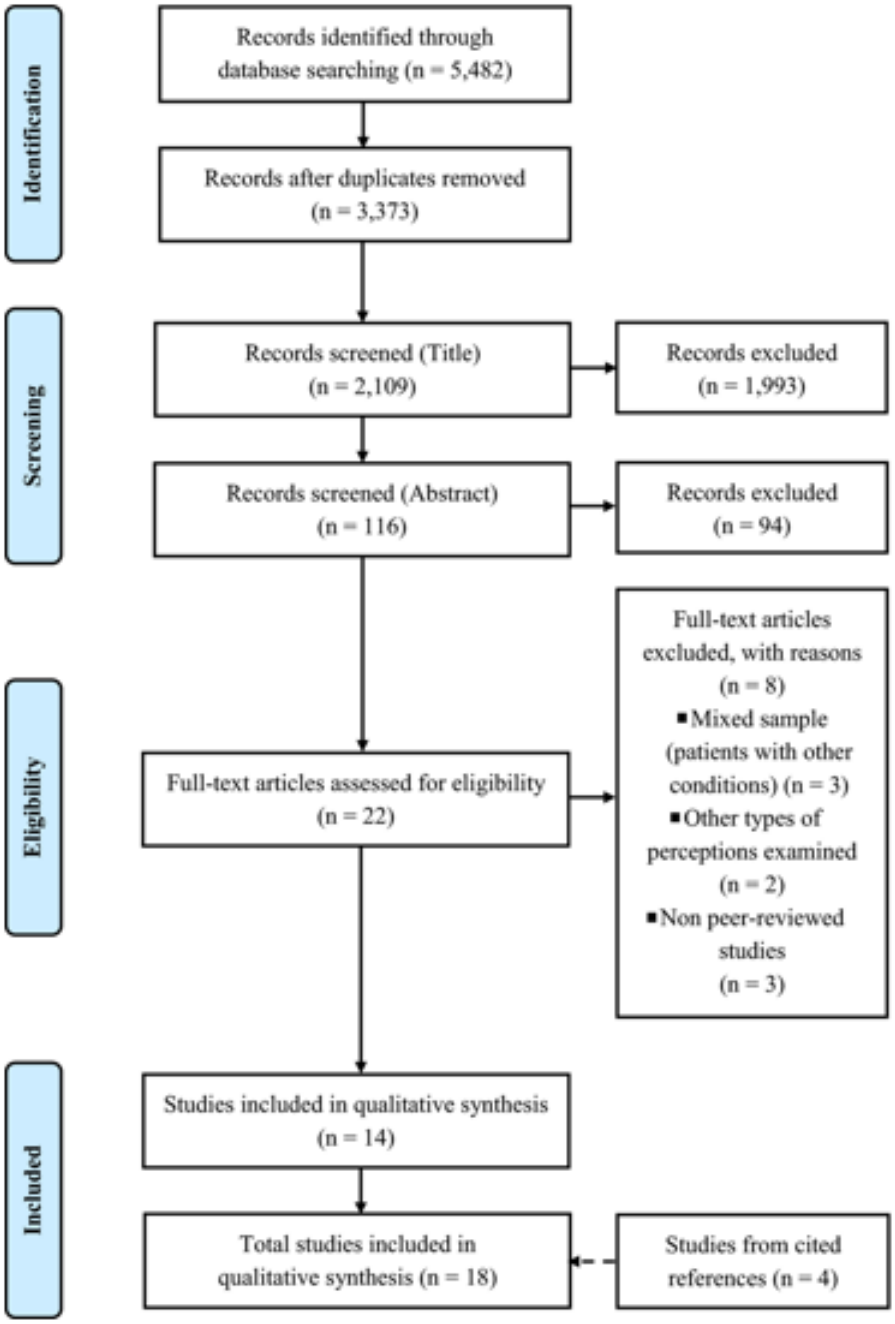


Table 2.1: Studies included in the review.

Authors	Population	Perceived risks measured	Comparator	Outcomes
A. Studies investigating absolute accuracy				
Allen *et al.* (2010) [94]	(a) US; (b) n=143; (c) Urban, low SES, middle-aged Black women; (d) No CVD; (e) At least 1 additional CV risk factor	Own absolute lifetime risk of CVD (percentage score)	Based on physiologic measures	Overestimation
Asimakopoulou *et al.* (2008) [32]	(a) UK; (b) n=95; (c) No CVD	Own absolute lifetime risk of CHD (percentage score) Own absolute lifetime risk of stroke (percentage score)	UKPDS risk estimate UKPDS risk estimate	Overestimation (factor 3.5) Overestimation (factor 5.5)
Frijling *et al.* (2004) [27]	(a) The Netherlands; (b) n=450; (c) No atherosclerotic disease	Own absolute 10-year risk of MI (percentage score) Own absolute 10-year risk of stroke (percentage score)	Framingham risk score Framingham risk score	Overestimation (factor 4) Overestimation (factor 4.7)
Hoffmann and Del Mar (2012) [103]	(a) Australia; (b) n=91	Average absolute 15-year risk of diabetes-related eye complication (percentage score)	Klein *et al.* (1984) [105]	Underestimation (factor 2.3)
Martell-Claros *et al.* (2011) [104]	(a) Spain; (b) n=511; (c) Diagnosed for less than 1 year; (d) No history of CV event or disease; (e) No insulin therapy	Own absolute lifetime risk of CVD (Likert scale)	Based on physiologic measures (ESH-ESC 2007 guidelines [106])	No agreement between perceived and calculated risks
Portnoy *et al.* (2014) [7] (a)	(a) US; (b) n=83; (c) Diagnosed for at least 1 year; (d) No history of CV event or CHD	Own absolute 10-year/lifetime risk of developing/dying from CHD (percentage score)	UKPDS risk estimate	No agreement between perceived and calculated risks

Table 2.1 Continued: Studies included in the review.

Welschen *et al.* (2012) [102]	(a) The Netherlands; (b) n=204; (c) No history of CVA or TIA	Own absolute 10-year risk of CVD, after being told estimates for average diabetic men and women (percentage score)	UKPDS risk estimate	No agreement between perceived and calculated risks
B. Studies investigating optimistic bias				
Choi *et al.* (2008) [96] (a)	(a) US; (b) n=143; (c) Diagnosed for at least 1 year; (d) Korean immigrants	Own risk perception score for CHD (optimistic bias section of the Becker & Levine index [107], Likert scale)	Perceived risk attributed to people of similar age and sex in the general population	Underestimation (optimistic bias)
Homko *et al.* (2010) [21]	(a) US; (b) n=211; (c) No history of CVD; (d) At least 10% 10-year risk of CVD (Framingham risk score); (e) No ESRD or dialysis; (f) No patients from nursing or boarding home	Own comparative 10-year risk of CHD, high BP and stroke (Schwartzer & Renner's risk score [108], Likert scale)	Perceived risk attributed to non-diabetic peers	Higher risk perception among women; No agreement between perceived and actual risks (Framingham risk score)
Portnoy *et al.* (2014) [7] (b)	(a) US; (b) n=83; (c) Diagnosed for at least 1 year; (d) No history of CV event or CHD	Own comparative 10-year/lifetime risk of developing/dying from CHD (percentage score)	Perceived risk attributed to the average person	Slight overestimation (pessimism)
Walker *et al.* (2007) [39]	(a) US; (b) n=250; (c) Socially deprived; (d) No dilated eye examination undergone over the past year	Own comparative risk perception (optimistic bias section of the RPS-DM [39], Likert scale)	Perceived risk attributed to diabetic peers	Higher optimistic bias in people born abroad and with lower education

Table 2.1 Continued: Studies included in the review.

C. Studies assessing a risk perception score				
Calvin *et al.* (2011) [95]	(a) US; (b) n=143; (c) Diagnosed for less than 5 years; (d) Urban African Americans; (e) No known complication	Own risk perception score for complications (RPS-DM score)	Based on physiologic measures	Low perception of risks
Choi *et al.* (2008) [96] (b)	(a) US; (b) n=143; (c) Diagnosed for at least 1 year; (d) Korean immigrants	Own risk perception score for CHD (Becker & Levine index)	Based on physiologic measures	Low perception of risks
D. Qualitative studies investigating risk perceptions				
Carroll *et al.* (2003) [40]	(a) UK; (b) n=20; (c) Half of patients with CVD	Own lifetime risk of CVD (semi-structured interview)	-	Low perception of the link between diabetes and CVD risk
Macaden & Clarke (2006) [100]	(a) UK; (b) n=20; (c) South Asian older people living in North East England	Perception of risks in relation to socio-cultural factors (focus group interview)	-	Bias induced by socio-cultural norms
McKenzie & Skelly (2010) [97]	(a) US; (b) n=6; (c) Diagnosed for at least 1 year; (d) Southern African American women; (e) No self-reported CHD	Lifetime risk of CVD (semi-structured interview)	-	Low perception of risks
Price *et al.* (2009) [101]	(a) UK; (b) n=16	Risks of CHD (focus group interview)	-	Low perception of the link between diabetes-related risk factors and CHD

Table 2.1 Continued: Studies included in the review.

E. Studies providing complementary evidence				
Kausar *et al.* (2013) [99]	(a) Pakistan; (b) n=100; (c) No history of psychiatric condition, cognitive impairment, any other chronic condition, terminal illness, speech or hearing problems, co-morbidities such as hypertension and obesity	Own risk perception for complications (RPS-DM score)	-	Higher risk perceptions were associated with higher emotional distress; men had better knowledge of diabetes complications
Merz *et al.* (2002) [98]	(a) US; (b) n=2,008	Own risks for CV complications (semi-structured interview)	-	Low perception of risks, especially in people aged $\geq$ 65 years old; higher risk perception for microvascular complications
Portnoy *et al.* (2014) [7] (c)	(a) US; (b) n=83; (c) Diagnosed for at least 1 year; (d) No history of CV event or CHD	Own conditional 10-year/lifetime risk of developing/dying from CHD (Likert scale, conditional on physical activity level)	UKPDS risk estimate	No agreement between perceived and calculated risks
Saver *et al.* (2014) [26]	(a) US; (b) n=202; (c) At least 1 additional CV risk factor	Ranking of 6 complications (including fatal events) by likelihood of occurrence	-	Underestimation of the likelihood of fatal events

Note: CHD = Coronary heart disease; CV = Cardiovascular; CVA = Cardiovascular accident; CVD = Cardiovascular disease; ESH-ESC = European Society of Hypertension - European Society of Cardiology [106]; ESRD = End stage renal disease; MI = Myocardial infarction; RPS-DM = Risk Perception Survey - Diabetes Mellitus [39]; SES = Socio-economic status; TIA = Transient ischemic accident; UKPDS = UK Prospective Diabetes Study.

Among the studies that measured absolute accuracy and/or optimistic bias, numerical probability estimates (e.g. "I think that my risk of developing a complication within the next 10 years is 1%") were used more frequently than verbal qualifiers (e.g. "I think that I am at moderate risk of developing a complication in the next 10 years") (nine vs. four outcomes, respectively).

e Types of actual risks used as comparator

Actual risks were used to measure absolute accuracy (nine outcomes), to interpret risk perception scores (two outcomes) and to compare with conditional risks (one outcome). Three types of actual risks were identified: personalised risk estimates calculated from simulation models (seven outcomes), risk estimates extracted from the literature (one outcome) and level of actual risk based on physiological measures (four outcomes). Of the four studies using calculated personalised risk estimates, two simulation models were identified: (a) the UK Prospective Diabetes Survey (UKPDS) risk engine [109, 110], which provides risks for diabetes-related complications based on a 20-year follow-up study (three studies [7, 32, 102]); and (b) the Framingham Risk Score [111], which provides 10-year risk estimates of developing cardiovascular diseases (one study [27]). Of the four studies that estimated actual risks based on physiological measures, only one indicated clearly how participants were classified into different risk categories. [104]

f Types of complications

Perceived risks for developing macrovascular complications were more frequently examined than perceived risks for experiencing microvascular events. Twelve of the 18 studies focused exclusively on cardiovascular risks (e.g. risks of stroke or myocardial infarctions), one focused exclusively on microvascular risks (risks of eye complications) [103], and five studies focused on both. [26, 39, 95, 99, 100]

g Study quality

Of the four qualitative studies, three were rated as good and one was rated as poor. Results were mixed regarding quantitative studies: 58% of these studies

were found to be of poor quality, 21% of fair quality and 21% of good quality. In particular, most studies were rated poorly with regard to presentation of quantitative data (40% poor, 33% fair, 27% good) and data analysis (31% poor, 44% fair, 25% good) (see Appendix C).

h Outcomes

Absolute accuracy

Seven studies measured the absolute accuracy of risk perceptions [7, 27, 32, 94, 102–104], reporting either people's own (eight outcomes) or average (one outcome) perceived absolute risks. One study clearly reported quantitative estimates of the difference between actual and perceived risks [32], two studies provided enough information (either numerically [27] or graphically [103]) to calculate it, and four studies did not report sufficient quantitative information for an estimate to be derived. [7, 94, 102, 104] Results were mixed, but tended towards overestimation: overestimation (three studies), underestimation (one study) and absence of a significant correlation between actual and perceived risks (meaning that no inference can be made towards under- or overestimation; three studies) were reported. For example, Frijling *et al.* (2004) [27] showed that people overestimated their 10-year risk of experiencing myocardial infarction (by a factor of 4) and stroke (by a factor of 4.7). Similar results were reported by Asimakopoulou *et al.* (2008) [32] and Allen *et al.* (2010) [94], in studies demonstrating that people were unrealistically pessimistic about their risks of developing both CHD and stroke (by factors of 3.5 and 5.5, respectively). Conversely, most people in Hoffmann and Del Mar's study underestimated their risk of eye complications. [103] In addition, three studies examining the association between perceived and actual risks found no evidence of correlation. [7, 102, 104] Finally, some studies reported that several people were unable to provide any (numerical) estimates of perceived risks. [27, 94]

Optimistic bias

Four studies assessed comparative perceived risks [7, 21, 39, 96], providing support

to the existence of optimistic bias. Choi *et al.* (2008) [96], focusing on Korean immigrants in the USA, observed that people underestimated their risks for CHD compared with those of the average person. Walker *et al.* (2007) [39] showed that the optimistic bias tended to be higher in minority ethnic populations and in people with a lower level of education. In addition, Homko *et al.* (2010) [21] provided further evidence on gender-based differences in risk factors and risk perceptions, finding that women perceived their risk for CVD to be significantly higher than did men. Finally, one study reported a slight overestimation of perceived comparative risks, when people were asked to compare themselves with the average person. [7]

Risk perception scores

The two studies that used composite scores (combination of different items to compute a risk perception score) as a proxy for perceived risks focused specifically on urban, socially deprived or minority ethnic populations. [95, 96] They reported low perception of risks for various diabetes-related complications.

Qualitative studies

Despite differences in study design and small sample sizes, findings from qualitative studies tended to be consistent, highlighting low risk perceptions. First, a focus group interview showed that people with T2DM often had a low perception of the link between diabetes-related risk factors and CHD. [101] Second, two semi-structured interviews exploring lifetime risk perceptions of CVD in two different populations (African American women [97] and Caucasian, UK residents [40]) pointed out that significant proportions of people did not even know that they were at risk. In addition, Macaden and Clarke (2006) [100], conducting a series of focus group and individual interviews, showed that sociocultural norms (e.g. beliefs, food and religion) had a significant influence on how certain populations underestimated risks.

Complementary evidence

Four studies provided complementary information on risk perceptions in people

with T2DM. In a large-scale study (n = 2008) where the dimensions of perceived risks measured were not specified, Merz *et al.* (2002) [98] found that more than two thirds of the persons did not consider CVD to be a serious complication of diabetes, and that very few respondents were able to name an approach to reduce their risk. Perceived risks were slightly higher for microvascular complications, but again were largely underestimated overall, especially in minority ethnic populations. In a study led in Pakistan, Kausar *et al.* (2013) [99] reported that men had better knowledge of diabetes complications, and that higher perceived risks were associated with higher levels of emotional distress. In addition, Portnoy *et al.* (2014) [7] did not obtain more accurate results when comparing perceived conditional risks for CHD. Lastly, a study by Saver and colleagues (2014) [26], in which people were asked to rank six complications—including death—by likelihood of occurrence, showed that the probability of experiencing fatal events was largely underestimated.

2.3.3 Discussion

This systematic review provides an overview of risk perceptions for diabetes-related complications in people with T2DM. I focused exclusively on lay risk perceptions to identify *genuine biases*, i.e. biases not contaminated by any type of communication intervention.[29] I found evidence that perceptions were generally biased, characterised by low risk awareness in most dimensions of perceived risks measured and contaminated by optimistic bias.

First, there was a clear lack of awareness concerning the risk of diabetes-related complications. People, particularly those from ethnic minorities [95–97], were often unaware of their risks of experiencing complications, and especially for macrovascular events. [40, 96–98, 101] In the largest study, nearly 70% of people with T2DM did not realise that they were at increased risk for CVD. [98] Two studies also showed that people had only a vague idea of (or ignored) the link between T2DM and cardiovascular risk factors. [27, 101] Such findings are consistent with research conducted in other populations [107, 112] and disease areas.

[29] Identifying such biases is crucial as a first step towards the development of new types of interventions specifically designed to address them.

[113] Second, studies examining comparative risk perceptions revealed the existence of an optimistic bias. [21, 39, 96] This is in line with evidence found for the general population. [114–117] However, some results also suggested that people tended to overestimate their risk of developing complications when the absolute dimension of perceived risks was measured. Three of seven studies measuring absolute perceived risks reported unrealistic pessimism. [27, 32, 94] Although apparently inconsistent with previous findings, this trend has also been documented in the recent literature [118, 119] and should be interpreted with caution, as absolute perceived risks are likely to be strongly affected by numeracy levels. Hence, people with T2DM had generally biased perceptions of their risks for complications, characterised by low risk awareness in most dimensions measured. The direction and magnitude of the biases were related to the dimension of perceived risks measured, the subpopulation concerned, and the type of complication considered.

A major finding was also the large heterogeneity in study designs and methods. Several dimensions of perceived risks (e.g. absolute, comparative, conditional) were measured using very different methods (e.g. self-reported numerical probability estimate, risk perception score, focus group interview), and various comparators were used to estimate actual risks (e.g. personalised estimates drawn from simulation models, estimates found in the literature). The heterogeneity in the aims of the identified studies partially explain the heterogeneity in study design. Although all studies assessed lay risk perceptions, their primary objectives varied widely. For example, Homko *et al.* (2010) [21] focused on gender differences in cardiovascular risk factors and risk perceptions; whereas Hoffman and Del Mar (2012) [103] examined how people with T2DM understand evidence-based health information. Furthermore, the heterogeneity in measurement methods was even larger. Despite a substantial, still growing, body of literature on risk perceptions, there has been no consensus about best practices for measurement and operationalisation so far. [93] One complexity is that different dimensions of risk perceptions measured do not exactly overlap. In fact, recent evidence suggests that these dimensions are only moderately related and not interchangeable. [5, 7, 83] Such heterogeneity made

data extraction and assessment of study quality challenging.

However, despite the heterogeneity in study designs and measurement methods, inclusion criteria were consistent across selected studies. More than half of the studies examined perceived risks for macrovascular complications, emphasising the importance of this issue for self-care behaviours. Several studies also focused on socially deprived or minority ethnic populations with T2DM. These populations are described as the most at risk of having uncontrolled diabetes. [120–122] Finally, almost half of the studies included people who were recently diagnosed. This population, by being more likely to experience the asymptomatic phase of the condition, may have lower perceptions of their risk of experiencing complications. The rationale for including recently diagnosed people may be driven by the expectation of higher gains from providing a risk communication intervention. Indeed, such an intervention could be expected to have a higher impact on those populations than on populations who self-manage their condition effectively, or on people who have already experienced a complication.

A major strength of this review is that it was conducted in accordance with PRISMA Statement guidelines. The selected studies provided a comprehensive overview of risk perceptions in T2DM populations; they were diverse in design, methods and populations (six countries were represented, including minority ethnic populations), but consistent in terms of inclusion criteria. In addition, study quality was evaluated by using two tailored assessment tools. Most studies presented a well-defined research question, provided a good description of their inclusion criteria and explained precisely how they measured lay risk perceptions in people with T2DM. Moreover, only two studies were funded [98] or involved researchers attached to pharmaceutical companies [104], which reduces the risk of publication bias. However, study limitations associated with other quality criteria should be noted. In studies that used actual risks as a comparator to perceived risks, I found that some did not describe how these actual risks were calculated or how they were attributed to participants. [94–96] For these studies, I reported how the authors interpreted their data (e.g. *overestimation*) without being able to quantify the

gap between actual and perceived risks. Also, I found that more than half of the studies did poorly in analysing and interpreting the data. For example, when analysing the absolute accuracy of risk perceptions, some studies only reported an *absence of association between perceived and actual risks*. This is mainly due to the heterogeneity in primary objectives across selected studies. Lay risk perceptions for complications were only a secondary outcome in some studies, and only partial analyses and interpretations were provided in that case. Another limitation of the review is that selected studies varied widely in terms of sample sizes (from 6 to 2008; median 143). Because of the heterogeneity in study designs and methods, I was unable to address quantitatively these variations in the analysis.

Further research on patient risk perceptions would benefit from the emergence of a standardised method to measure perceived risks. Encouraging a certain standardisation in the way researchers measure risk perceptions would provide a reliable way of comparing studies investigating the impact of risk communication interventions, and thus help identify the most effective ones. In practice, I would encourage the design of standardised questionnaires assessing multiple dimensions of risk perceptions but generating a single risk perception score. Comparing studies investigating the impact of risk communication interventions on risk perceptions through the modification of this risk perception score would add clarity and ease to the assessment of such interventions.

2.4 Conclusion

As discussed throughout this chapter, inaccurate perceptions of health risks may have significant consequences on self-management behaviour. Risk perception – a notion which combines perceived likelihood and perceived severity associated to a negative outcome – is a key component of most psychosocial theories for predicting and changing health behaviour, whether it acts as a direct influence on behaviour or through other mediating constructs. Studies which have rigorously investigated the link between risk perceptions and precautionary behaviour have shown a highly

significant, albeit small in magnitude, causal association.

It seems reasonable to assert, considering the evidence that has accumulated for more than two decades, that bringing patients' risk perceptions into line with their actual risks is a crucial prerequisite for effective management of NCDs. Nonetheless, because of the various beliefs and inter-individual differences interacting with risk perceptions which also play a role in decision-making processes, enhancing perceived risks is not sufficient to promote behaviour change. Identifying and targeting other decision-making biases specific to T2DM populations is also needed.

In this chapter, I conducted, to my knowledge, the first systematic review of risk perceptions for diabetes-related complications in T2DM populations. Despite a high degree of heterogeneity across selected studies, the results were in line with other research on the topic, and confirmed the need for more effective risk communication interventions. Worrying trends emerged from the data: unawareness of the risks associated with complications (especially macrovascular events), unawareness of diabetes-related risk factors, underestimation of comparative risks (optimistic bias), and overestimation of own absolute risks.

The next chapter will continue the exploration of the underlying mechanisms that govern patients' decision-making processes. It is assumed that identifying and using behavioural economic material specific to people with T2DM will enhance the effectiveness of risk communication, help reduce misperceptions, and, in turn, motivate sustained adherence to self-management.

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3

Risk attitudes and self-management of Type-2 diabetes

3.1 Introduction

As discussed in the previous chapters, both risk perceptions and risk attitudes play a key role in the underlying processes that drive decisions under risk. In the context of Type-2 diabetes mellitus (T2DM), patients are faced with the risk of experiencing diabetes-related complications, but also with the opportunity to reduce this risk by adopting an appropriate (but costly) self-management behaviour. In line with the strategy described in Chapter 1, I think that a better understanding how people with T2DM make these decisions will help in designing better targeted health behaviour interventions (the behavioural economic or *nudging* approach, see Chapter 1).

Chapter 2 was concerned with exploring the risk perception determinant of risky decision-making behaviour. The evidence accumulated over the past twenty years has shown that accurate risk perceptions are a crucial prerequisite for effective management of chronic health conditions. However, my systematic review of studies investigating how people with T2DM perceive their risks for diabetes-related complications highlighted substantial unawareness of these risks (especially risks of macrovascular events). In addition, I identified two systematic decision-making biases in T2DM populations: the optimistic bias and the tendency of people to overweight small probability events.

In addition to highlighting the need for better risk communication strategies, such information is of great value for the design of targeted interventions tailored to people with T2DM. Indeed, in the perspective of developing a fully tailored (*nudge-based*) intervention, we need to go further in understanding the rules which govern patients' suboptimal choices. Enhancing perceived risks is necessary but not sufficient to promote behaviour change (see Chapter 1).

In this chapter, I focus on the risk attitude determinant of risky decision-making behaviour. I perform a deeper analysis of patients' decision-making processes by exploring specific risk attitudes and rationality bounds. The idea is to have a comprehensive knowledge of the main channels through which patients' decision-making processes can be influenced, i.e. to identify as many levers for action as

possible for the design of a targeted risk communication intervention. I first explain in more detail how decision theory can inform health behaviour interventions (the behavioural economics approach). I then review the decision science literature to identify relevant behavioural economic material for the design of a targeted risk communication intervention. Finally, I conduct a study to measure the risk attitudes of people with T2DM under prospect theory, the theory that I consider the most appropriate to identify such material.

3.2 Theoretical considerations

3.2.1 Decision theory under risk: a brief historical review

It's a dangerous business, Frodo, going out your door. You step onto the road, and if you don't keep your feet, there is no knowing where you might be swept off to.

Bilbo Baggins (J.R.R. Tolkien, 1937) [1]

People are constantly faced with choices under conditions of risk or uncertainty, and the opportunity costs of those choices. Pursuing a PhD in health economics may lead to a prolific academic career, or to low recognition and unemployment. Similarly, diabetes patients who do not adopt precautionary behaviours may be freer in their daily life, but may subsequently suffer from complications that could have been avoided. Regardless of the nature of the choice, two axes of research have generated much interest over the last three centuries.[1] On one hand, the hunt for a *normative* theory of choice, i.e. a theory of how people *should* make decisions. On the other, the development of a credible *descriptive* (or *positive*) theory, i.e. a theory of how people *do* make decisions.[2] Modern decision theory

[1] Although roots of modern (formalised) decision theory are traced to the 18th century, there is no doubt that people have been interested in solving optimal choice problems for – at least – as long as betting exist.

[2] The term *prescriptive* may also be found in the literature. Prescriptive use of a decision theory refers to its interpretation as a practical aid to choice. [2] The focus is on helping people make better decisions (normative predictions), but with awareness of the limitations of human judgement (insights from descriptive models). By providing decision-makers with simplified andor

encompasses these two axes of research. Specifically, researchers are concerned with understanding, describing, and predicting how individuals make choices under conditions of risk or uncertainty.[3] Over the last 70 years, major milestones in the field have been achieved through the contributions of several disciplines, including economics, mathematics, and psychology. [5]

a The standard model: expected utility theory (EUT)

The issue of rationality has been at the heart of the development process of normative theories of choice. Although philosophers disagree on what is rational [6, 7], major theories define the *rational decision maker* through mathematical principles or axioms, which, if combined, imply rational behaviour. [3] In 1738, Swiss mathematician Daniel Bernoulli first stated the expected utility (EU) hypothesis, which has become the standard criterion for rational decision-making. This hypothesis asserts that, if certain axioms of rationality are satisfied, the subjective value (or *utility*[4]) associated with a gamble by an individual is a weighted average of that individual's valuations of all possible outcomes of that gamble. The utility of each outcome is weighted by the respective probability that it may occur.[5] [9]

During the first half of the 20th century, with the rise of modern consumer theory, interest in Bernoulli's EU hypothesis was revived. In particular, von Neumann and Morgenstern (1944), by providing necessary and sufficient conditions under which the hypothesis holds, gave birth to the eponymous benchmark model

debiased choice tasks, decisions closer to those predicted by normative models can be expected. [3]

[3]Under conditions of risk, agents have complete knowledge of the observed probability of each state of the world that can reveal after their choice. Under conditions of uncertainty, these probabilities become (at least partially) subjective. [4] In this Thesis, due to the use of a simulation model to obtain personalised risk estimates (see Chapter 4), we will focus our attention on choices under risk conditions.

[4]i.e. the extent to which an outcome is preferred, or preferable, to the alternatives.

[5]Until then, the procedure referred to as expected value, introduced at the end of the 17th century by French philosopher Pascal in his *Pensées* (1670), was considered as a normative reference. Derived from the principle of mathematical expectation, it consists in determining the (numerical) values and probabilities of occurrence of all outcomes that could arise from each possible action faced by an individual, and then multiplying the two to obtain an expected value. Then the action to be chosen should be the one that gives rise to the highest total expected value. [8] By taking into account the individual's subjective valuation, in addition to the sizes of the payoffs and the probabilities of occurrence, the expected utility hypothesis has proved useful to explain some popular choices that contradicted the expected value criterion.

of choice under risk. [10] von Neumann and Morgenstern consider preferences[6] of an individual agent faced with *lotteries*. Lotteries are gambles where each (mutually exclusive) outcome is expected to happen with a given probability, all probabilities summing to one. Their model relies on a set of appealing axioms on preferences to justify the EU hypothesis:

- **Axiom 1 (Completeness)**

  Any two lotteries L, M can be ranked according to preferences:

  $$L \prec M \ , \ M \prec L \ , \text{ or } \ L \sim M \tag{3.1}$$

  (either M is preferred, L is preferred, or the individual is indifferent).

- **Axiom 2 (Transitivity)**

  Preferences across any three lotteries L, M, N are consistent:

  $$\text{If } \ L \preceq M \text{ and } \ M \preceq N \ , \text{ then } \ L \preceq N \tag{3.2}$$

- **Axiom 3 (Continuity)**

  The upper and lower contour sets of a preference relation over lotteries are closed:

  $$\begin{gathered} \text{If } \ L \preceq M \preceq N \ , \text{ then there exists a probability } \ p \in [0,1] \\ \text{such that } \ pL \ + \ (1-p)N \sim M \end{gathered} \tag{3.3}$$

- **Axiom 4 (Independence)**

  Preference between two lotteries L, M holds independently of the possibility of another lottery N:

  $$\begin{gathered} \text{If } \ L \prec M \ , \text{ then for any } N \text{ and } \ p \in (0,1] \ , \\ pL \ + \ (1-p)N \prec pM \ + \ (1-p)N \end{gathered} \tag{3.4}$$

[6] In economics, the notion of preference refers to a subjective comparative evaluation of the form "agent A prefers X to Y".

To the extent that these axioms are perceived as sound principles of rational choice to which any reasonable person would subscribe, they provide grounds for interpreting EU theory both normatively and descriptively.[7] [2] The von Neumann-Morgenstern (vNM) theorem (1947) [11] operationalises EU theory as follows:

For any vNM-rational agent (i.e. satisfying axioms $(3.1) - (3.4)$**), there exists a function** u **assigning to each outcome** A **a real number** $u(A)$**, such that for any two lotteries** L, M**:**

$$L \prec M \textbf{ iff } \; E[u(L)] < E[u(M)] \tag{3.5}$$

where $E[u(L)]$ **denotes the expected value of** u **in** L**:**

$$E[u(p_1A_1 + ... + p_nA_n)] = p_1u(A_1) + ... + p_nu(A_n)$$

This function, which makes complete, transitive, continuous and independent preferences over risky outcomes representable by the expected utilities of those outcomes, is called vNM utility. Maximising vNM utility instead of the simple expected value associated with a lottery allows to capture additional dimensions underlying the decision-making process of the agent, such as risk aversion.[8]

Two important properties result from the vNM theorem:

- vNM utility function is *linear in probabilities* (a property directly associated with the independence axiom) [12], meaning that:

$$U(ap + (1 - a)q) = aU(p) + (1 - a)U(q) \tag{3.6}$$

- vNM utility depends on final outcomes rather than gains and losses occurring during the choice process (*asset integration*).

[7] Rationality, in this context, is often referred to as *von Neumann-Morgenstern rationality.*

[8] For example, let's consider an agent who possesses $\$1,000$ and consider trading it for a lottery $L = 0.2(\$10,000) + 0.8(\$0)$. According to the expected value criterion, the agent should accept the trade since $E(L) = \$2,000 > \$1,000$. However, in reality, the probability that the agent takes the risk of losing $\$1,000$ for a 20% chance of winning $\$10,000$ is very low. The vNM utility function, provided the agent is vNM rational, automatically accounts for these phenomena (here risk aversion). In this very example, we would then have $E[U(L)] = 0.2[U(\$10,000)] + 0.8[U(\$0)] < U(\$1,000)$.

In addition to the explicitly stated axioms, vNM-EU theory also requires additional structural assumptions.[9] [13, 14] In particular, two of these assumptions, which relate to the stability of preferences, are crucial[10]:

- **Assumption 1 (Description invariance)** Different representations of the same choice problem should yield the same preference. That is, preferences over lotteries should not depend on how the probability distributions of outcomes implied by those lotteries are described.
- **Assumption 2 (Procedure invariance)** The preference between lotteries should be invariant to the procedures used to elicit them.

Without stability across equivalent description and equivalent elicitation procedures, an individual's preferences cannot be represented by vNM utility.

b Violations of expected utility theory

vNM-EU theory became the standard theory of individual choice under risk, especially in economics. [2] Despite its normative nature[11], it has also emerged as a paradigm for modelling agents' behaviours in both micro- and macroeconomic models. [9] However, although the model does relatively well in predicting behaviours as compared to the expected value (EV) criterion, several studies aiming at testing its descriptive adequacy have identified systematic deviations from its predictions. From the early 1950s, a vast amount of empirical work has indeed revealed a variety of patterns in human choice that appear inconsistent with vNM-EU theory.[12] In different experiments, significant proportions of subjects have been found to violate at least one axiom or structural assumption. This subsection reviews the most

[9] These assumptions, also referred to as *principles*, are implicit but inherent to any normative theory of rational choice [2, 13]: *cancellation* ("any state of the world that results in the same outcome regardless of one's choice can be cancelled or ignored"), (*stochastic*) *dominance* ("within a given lottery, shifting positive probability mass from an outcome to a strictly higher outcome leads to a strictly higher evaluation of the transformed lottery"), *extensionality* ("people attribute the same value to objects that have the same external properties"), *description* and *procedure* invariance. [14, 15]

[10] Invariance condition is "absolutely necessary for any normative theory". [13]

[11] Some authors have argued against the normative interpretation of vNM-EU theory, see e.g. Anand (1995). [16]

[12] For a review, see Bryant (2014). [4]

significant ones.

Violations of the independence axiom

Violations of the independence axiom are probably the best-known deviations from vNM-EU theory. First evidenced by Allais (1953), they are commonly referred to as Allais common ratio and common consequence paradoxes.[13] [18] Hypothetical situations used by Allais to characterise them are summarised in Table 3.1.

Table 3.1: Allais paradoxes.

Common Ratio Effect	**Common Consequence Effect**
Situation A: Certainty of receiving 100 million.	*Situation C*: Certainty of receiving 100 million.
Situation B: 98% chance of 500 million; 2% chance of nothing.	*Situation D*: 10% chance of 500 million; 89% chance of 100 million; 1% chance of nothing.
Situation A': 1% chance of 100 million; 99% chance of nothing.	*Situation C'*: 11% chance of 100 million; 89% chance of nothing.
Situation B': 0.98% chance of 500 million; 99.02% chance of nothing.	*Situation D'*: 10% chance of 500 million; 90% chance of nothing.

When asked to choose between situations A (EV=100 million) and B (EV=490 million), and then between situations A' (EV=1 million) and B' (EV=4.9 million), a significant proportion of subjects choose A over B and B' over A'.[14] However, under vNM-EU theory, if A is preferred to B, then A' should be preferred to B'. Indeed, choice between situations A and B and choice between situations A' and B' share a common ratio of (winning) probabilities of 0.98 (between the two choices, the manipulation has only been to divide the probabilities by 100). Similarly, when asked to choose between situation C (EV=100 million) and D (EV=139 million), and then between situations C' (EV=11 million) and D' (EV=50 million), many

[13] "Their appeal is that even without experimentation they ring true, and with experimentation they are found to be robust." (Andreoni and Sprenger, 2010 [17]).

[14] For a review of the empirical evidence, see Camerer (1995) [19]

subjects choose C over D and D' over C'.[15] [16] However, under vNM-EU theory, if C is preferred to D, then D' should be preferred to C'. Situations C and D share a common consequence of winning 100 million with probability 0.89; situations C' and D' involve the exact same choice after this common consequence has been removed.

Different phenomena have been evoked to explain the observed violations of the independence axiom (see Oliver, 2003 [23] for a review), including:

- The *certainty effect*, i.e. the tendency to overweight outcomes that are certain as compared to those that are probable [21], an effect itself potentially grounded on phenomena such as *loss aversion* (the pain of losing a given outcome is psychologically more powerful than the pleasure of gaining the same outcome; see 3.2.2). [26] and *anticipated regret* (the tendency to incorporate the desire to eliminate or mitigate regret when making a decision). [27, 28]
- *Probability weighting*, which is the tendency to overweight small probabilities (less likely events) and underweight large probabilities (more likely events), meaning that the *linearity-in-probabilities* property is not respected (see 3.2.2 for more detail).

Violations of the structural assumptions

If there is no doubt that, in practice, people's choices do not obey pure vNM rationality, decision theorists have also evidenced the instability of their preferences. Violations of invariance have frequently been reported, whether procedural or descriptive. A significant cause of description invariance failure is the *framing effects* described by Tversky and Kahneman (1981). [29] In a famous experiment, they asked participants to choose between two programs, in case the following scenarios happen:

[15] Examples of empirical support include Camerer (1989) [20], Kahneman and Tversky (1979) [21], Slovic and Tversky (1974) [22]. The phenomenon has also been observed with health outcomes. [23]

[16] A funny anecdote was reported by Andreoni and Sprenger (2010): "Perhaps the most revealing subject was Leonard Savage who chose C over D and D' over C' and concluded that his preferences were *subtly* in error (Savage, 1954, p.103 [24]). This may have left Paul Samuelson in an uncomfortable situation as he had stated just before, *I sometimes feel that Savage and I are the only ones in the world who will give a consistent Bernoulli answer to questionnaires of the type that Professor Allais has been circulating* (Samuelson, 1952, p.678 [25])." [17]

Imagine that the U.S. is preparing for the outbreak of an unusual Asian disease, which is expected to kill 600 people. Two alternative programs to combat the disease have been proposed. Assume that the exact scientific estimate of the consequences of the programs are as in Table 3.2.

Table 3.2: Scenarios used by Tversky and Kahneman (1981) to illustrate framing effects. [29]

Situation A	**Situation B**
If Program A is adopted, 200 people will be saved.	*If Program C is adopted 400 people will die.*
If Program B is adopted, there is 1/3 probability that 600 people will be saved, and 2/3 probability that no people will be saved.	*If Program D is adopted there is 1/3 probability that nobody will die, and 2/3 probability that 600 people will die.*
Which of the two programs would you favor?	*Which of the two programs would you favor?*

These scenarios are equivalent, but programs are framed either in terms or gains (positive framing, scenario 1) or in terms of losses (negative framing, scenario 2). However, when presented with positive framing, program A was chosen by 72% of participants; whereas with negative framing, only 22% chose program C. These results also suggest violation of the *asset integration* property[17], since people's way to assess prospects vary whether they face gain or loss outcomes.

Regarding procedure invariance[18], several authors have reported systematic differences in utility functions elicited by different methods (see e.g. Hershey and Schoemaker, 1985 [30]). Most importantly, it has been shown that failure of procedure invariance leads to the well-documented *preference-reversal* phenomenon[19] (see e.g. Grether and Plott, 1979 [31]; Tversky *et al.*, 1990 [32]; Tversky and Thaler, 1990 [33]; Lichtenstein and Slovic, 1973 [34]; Lichtenstein and Slovic, 1971 [35]). A good example of this phenomenon is as follows [32]:

[17] vNM utility depends on final outcomes rather than gains and losses occurring during the choice process (see previous subsection).

[18] The preference between lotteries should be invariant to the procedures used to elicit them (see previous subsection).

[19] Different procedures for preference elicitation can lead to a switching of preference between two alternatives.

Let's consider a pair of gambles of comparable expected value:

- *The 'H bet', which offers a high probability of winning a modest sum of money: e.g. 28/36 chance to win $10*;
- *The 'L bet', offers a low probability of winning a relatively large amount of money: e.g. 3/36 chance to win $100.*

When offered a choice between the two gambles, most subjects choose the H bet (EV=$7.8) over the L bet (EV=$8.3). However, when asked to state their lowest selling price (i.e. the price at which they would sell each of these gambles assuming that they owned them), the majority state a higher price for the L bet than for the H bet, revealing a switch in preferences.

Violations of procedural invariance have also been observed in the health domain. A close replication to the original test was performed in by Oliver (2006), who assessed preferences over differential distributions of life expectancies across populations. [36] Respondents were asked to choose between two countries with different life expectancy distributions and then to give a certainty equivalent value in terms of life expectancy. The life expectancy distributions in the two countries were as follows:

- *The 'H bet': (64 years, 70%; 84 years, 30%)*
- *The 'L bet': (65 years, 3%; 70 years, 97%)*

Oliver observed that 36% of his respondents demonstrated a strict preference reversal, with all of those preferring the H-bet over the L-bet, but then valuing the L-bet higher. Oliver (2013) also found evidence of preference reversals over options defined as health care treatment options, although the patterns were non-systematic.[20] [37]

In addition to Oliver's studies, a substantial body of literature reports evidence of preference reversal across different elicitation methods, including:

[20]The author suggests that this was due to the complicated presentation of the options.

- Willingness to pay (WTP) for healthcare programmes vs. ordinal ranking of these programmes, with consistency rates among respondents ranging from 21% to 43%. [38–41]
- Choice task vs. matching task, with respondents first preferring option 1 (10 years of life with 5 days of migraine per week) over option 2 (20 years of life with 5 days of migraine per week) but then valuing option 2 more than option 1 in terms of their equivalent number of years in good health. [42]
- Visual analogue scale (VAS) task vs. choice task, with respondents first valuing poor health states as better than death via VAS but then preferring death over living for 10 years in those health states. [43]
- Ranking of health states vs. choice task, with significant preference reversal rates (between 9% and 43%) in which most respondents first preferred the (poor) health state over death in the ranking task but then preferred death over the (poor) health state in the choice task. [44]

The preference reversal phenomenon is challenging for health economists, who often use these methods interchangeably to elicit preferences.

Other types of violation

Interpreting vNM-EU theory as a descriptive theory requires that agents have preferences over all outcomes and knowledge of all probabilities associated with these outcomes. Although this concept makes sense theoretically, this requires, in practice, enormous memory and computational capacities. Consequently, many phenomena causing violations of vNM rationality merely stem from the fact that human cognitive abilities are limited.

Under the heading of *rationality bounds*, decision theorists and psychologists have included a large number of decision-making biases observed in practice. These include pure cognitive limitations, such as relying on heuristic or intuitions rather

than rational cognitive evaluation[21], making errors in deductive inferences, ignoring relevant information, or using irrelevant information; but also, other, non-cognitive limitations. [15] For instance, some authors have emphasised the crucial role played by emotions in the decision-making process (for a review, see Loewenstein and Lerner, 2003 [46]).

Subsection 3.2.2 provides further details regarding rational bounds that play a role in health-related decisions.

c Alternative models: non-expected utility theories

Although it would be unrealistic, according to Starmer (2000), to expect any decision theory to predict behaviours accurately 100% of the time, the systematic violations of vNM-EU theory – whether it concerns its axioms or structural assumptions, i.e. their *predictable* nature – has led to the development of alternative theories of choice intended to express a better descriptive power. [2]

A first category of models has attempted to extend vNM-EU theory, while staying within the realm of well-behaved theories of preference[22] (the *conventional strategy* [2]). It mostly involved the emergence of new models axiomatically based on weakening the independence axiom. For instance, given the evidence that individuals have subjective attitudes to probabilities which are distinct from attitudes to outcomes, a widely used method has been to insert probability transformation functions which convert objective probabilities into subjective decision weights. A well-known model of this type is Quiggin's rank-dependent expected (or anticipated) utility theory. [47] Other popular models aiming at generalising vNM-EU theory include Machina's generalised expected utility theory [48] and Yaari's dual choice model. [49]

A second approach has been to develop mathematical models of actual behaviours, grounded on empirical evidence, based on the argument that structural

[21]Kahneman (2003), who pioneered the identification of decision-making biases, notes that "central characteristic of agents is not that they reason poorly but that they often act intuitively. And the behaviour of these agents is not guided by what they are able to compute, but what they happen to see at a given moment." [45]

[22]Theories that satisfy the axioms and structural assumptions described in subsection 3.2.1. Preferences can be represented by a single well-behaved utility function.

failures of vNM-EU theory challenge the very idea that choices can, in general, be represented by a single well-behaved utility function (the *non-conventional* or *procedural strategy*). [2] These theories, inspired by the psychology literature, seek to model the mental processes that lead to choices.[23] Main examples are single [21, 50] and multiple reference point theories. [51, 52] In this context, one model has emerged and deserves special attention. Kahneman and Tversky's prospect theory [21, 50], the validity of which has been largely supported empirically, is a prime candidate to operationalise people's decision-making processes, including in the health domain. This will be discussed further in section 3.3.

3.2.2 How decision theory can inform health behaviour interventions

A large majority of studies assessing the validity of decision theories have used monetary outcomes, by virtue of their natural application to the economic and financial fields. However, the influence of vNM-EU theory has radiated out to many other areas involving human choices, including those implying non-monetary outcomes. Decision-making in the health domain, which involves both quantitative (e.g. survival time) and qualitative (e.g. well-being) non-monetary outcomes, fully belongs to this category.

Psychosocial models of behaviour reviewed in Chapter 2 (see 2.2.1) incorporate - to a greater or lesser extent - the expected value principle, by assuming that individuals are future oriented and weigh up the costs and benefits of possible future courses of action. [53] More specifically, most theories of behaviour (in general) and health behaviour (in particular) operationalise decision-making processes according to an extended version of vNM-EU theory: the subjective expected utility theory (SEUT).[24] [53–56] These include, amongst the most prominent examples, the theory

[23] In this regard, they build on observed empirical violations of vNM-EU theory such as bounded rationality.

[24] Savage's SEUT (1954) has been the standard theory of choice under uncertainty. [24] It has extended vNM-EUT by acknowledging that – in some situations, or under certain circumstances - it may not be reasonable to expect people to know exact probabilities associated with – at least - some events. In SEUT, therefore, probabilities are subjective and represent the extent to which a person believes the event is likely. These subjective probabilities are then multiplied by the

of reasoned action and the theory of planned behaviour. [53, 54]

However, even though a wide range of health promotion interventions rely on social cognition models, there is consensus that SEUT provides – at best - an incomplete framework through which these models should be operationalised (see e.g. Lejarraga *et al.*, 2016 [56]; Finucane and Holup, 2006 [55]; Frisch and Clemen, 1994 [57]; Lopes, 1981 [58]). Empirical evidence showing that SEUT does not accurately describe how people shape intentions and make decisions has accumulated. [55] The main issue - which comes back to the original question of descriptive validity - is that SEUT provides an unrealistically rational account of how people make decisions. It relies on a conscious, rational deliberation and evaluation of each outcome, but does not consider (or only partially) people's cognitive limitations, biases and emotions.[25] [53, 54] In practice, there are many situations where patients facing health choices may not be able to make a rational decision.[26] [54]

In a study of interest, Sultan *et al.* (2003) have identified, in clinical practice, recurrent elements explaining the lack of adoption of (secondary) preventive measures in people with T2DM. [60] They point out that:

a) Patients (like everyone) have limited cognitive skills, and experience difficulties in making decisions. This is especially the case when they face complex, uncertain situations. Although patients try to be rational, this rationality is subject to a series of cognitive biases which aim at simplifying the problems they face.[27]

b) Patients' actions are disproportionately ruled by short-term outcomes. They are rarely willing to accept present costs and constraints to protect themselves

perceived utility of the outcome, and each product is summed to create a single value. Then, for each option, the option with the highest subjective expected utility is expected to be chosen. [54, 55]

[25]This has led some researchers to revise the theories accordingly (see e.g. Fishbein, 2008 [59]).

[26]For instance, patients making treatment decisions about a life-threatening illness may "experience such stress that they are not able to conduct a complete evaluation of each benefit and cost". [54]

[27]See 6.5.2 for a discussion about the implications of standard economic rationality, in particular the issue of stable ("latent") preferences.

from a future hazard, especially if this hazard is uncertain.[28]

c) The roles of social pressures and self-esteem questioning should not be ignored with regards to preventive behaviours. It has been shown that most people only imitate other people who encounter the same situations, instead of collecting and analysing information about risks.

These findings are in line with what is usually described as classical causes of violations of vNM-EU theory in the economic literature[29], and hence contribute to legitimate non-conventional theories as a relevant framework for assessing risk attitudes associated with health behaviours.

Increased awareness of people's rationality bounds, combined with the fact that traditional health promotion interventions have failed to produce substantial beneficial effects [62], has encouraged the search for new tools, outside of traditional economics (vNM-EU paradigm), to design more effective interventions. Over the past ten years, a field has received particular attention for this purpose: behavioural economics. [62, 63] Located at the interface of decision theory, economics and psychology, behavioural economics is precisely concerned with both development and application of descriptive theories of choice for policy-making purposes. A major achievement of the field has been to identify a wide range of decision-making biases, i.e. to demonstrate that human decision-making is highly sensitive to context, including seemingly trivial details, and socio-environmental factors (see e.g. Kahneman, 2011 [64]; Marteau *et al.*, 2011 [65]; Zimmerman, 2009 [66]).

In a seminal paper of behavioural economics, Mullainathan and Thaler (2000) [61] have classified these decision-making biases (or bounds)[30] in three categories:

[28] This should be nuanced, as it depends on the nature of the protective behaviour. For example, high vaccination rates are sometimes observed in countries where there is no mandate. As compared to the costs associated with self-management (daily efforts), those associated with vaccination are very low (one-shot behaviour that does not require any particular effort or self-discipline). However, the benefits are still perceived as high, leading to a cost-benefit balance which is largely in favour of vaccination.

[29] In particular, it matches well with Mullainathan and Thaler's classification of human decision-making bounds (see further in this section). [61]

[30] i.e. deviations from vNM-rationality.

- **Bounded rationality**:

  The concept that people face limitations in how much information they can process and how much time they have to process it. This includes using decision heuristics and rules of thumb rather than computing the utility-maximising choice for each choice under risk they face. [62] Importantly, it has been shown that such errors can contribute to a variety of unhealthy behaviours. [63]

- **Bounded willpower**:

  The tendency that people have to favour the near term over the long term in their decisions, even though it is not in their best long-term interest.[31] In particular, O'Donoghue and Rabin (1999) have shown that people tend to pursue smaller immediate rewards instead of delayed but more highly valued rewards.[32] [69]

- **Bounded selfishness**:

  The fact that social norms and selflessness have a significant influence on how individuals make decisions.

Roberto and Kawachi (2015), in their book *Behavioural Economics and Public Health*, have listed the best-documented rationality bounds described by behavioural economists and psychologists in their research on health behaviour. [70] Their findings, along with the work of Luoto and Carman (2014) [62], have allowed me to identify seven biases that I believe are of great interest when trying to design better risk communication interventions. I provide a list of these biases and evidence of their influence on health behaviour in Table 3.3[33]

The other key research axis of behavioural economics, which is of primary

[31] Time inconsistent preferences, hyperbolic discounting, and present bias are usual terms found in the literature. Good examples of this phenomenon include cases where people are faced with the choice to exercise, to quit smoking.

[32] Recent evidence from the fields of neuroscience and neuroeconomics suggests that decisions involving immediate rewards activate different neural systems than decisions involving only future rewards (see e.g. DellaVigna, 2009 [67], McClure *et al.*, 2004 [68]).

[33] More details about each bias will also be provided in Chapter 4, when strategies to target them will be discussed.

interest for this Thesis, has been to find ways of taking advantage of these decision-making biases. Behavioural economists have designed interventions through which they aim to *manipulate*[34] or *mitigate* the effects of these biases.[35] Many of these interventions (nudge-based interventions or *nudges*) have shown promising results in the health domain (see Luoto and Carman, 2014 [62] for a review), including diabetes management (see Kullgren *et al.*, 2017 [74] for a review). This has raised interest and credibility for the use of inputs from descriptive theories for policy-making purposes.

Focusing on health policy in the diabetes area, several authors have recommended the use of decision theory to inform the understanding of patients' decisions and behaviours (see e.g. Downs *et al.*, 2011 [75]; Fisher *et al.*, 2002 [76]). Downs and colleagues (2011) claim that decision theory provides "basic tenets, general methods, and empirical results [...] that can inform our understanding of decisions relevant to managing diabetes". [75] They recommend developing new communication interventions that facilitate diabetes-related decisions by trying to close the gap between the normative ideal and the descriptive reality, arguing that current messages may not be providing relevant information in ways that motivate and facilitate action. [75]

In this Thesis, I aim to identify specific decision-making biases in the diabetic population in order to develop a nudge-based risk communication intervention informed by these biases (see Chapter 4 for more details about how each bias identified in Table 3.3 will be targeted).

[34]Such interventions derive from the concept of *libertarian paternalism* developed by Thaler and Sunstein (2003). [71] The idea of this approach is to try to influence people's choices in a way that will make them better off, as judged by themselves (paternalism), while ensuring that their freedom of choice is preserved (libertarianism). A significant example is the use of commitment contracts to encourage a variety of forward-looking behaviours. These contracts, by committing oneself today to a behaviour tomorrow (e.g. smoking cessation [72], weight loss [73]) can overcome present bias. [62]

[35]See Luoto and Carman (2014) for a comprehensive review of health interventions inspired by behavioural economics. [62]

Table 3.3: Decision-making biases presenting an interest as a lever for action to improve risk communication interventions.

Decision-making bias	Description	Empirical evidence	
		Health behaviour in general	**Specific to diabetes self-management behaviour**
Optimism	Overconfidence in own ability and intuition.	Waters *et al.* (2010) [77]; van der Weijden *et al.* (2007) [78]; Khwaja *et al.* (2007) [79]; Strecher *et al.* (1995) [80]; Weinstein (1980) [81]	See Chapter 2
Reference point dependence (sensitivity to framing effect)	Drawing different conclusions from the same information, depending on how that information is presented (e.g. gains or losses)	Gallagher and Updegraff (2012) [82]; Edwards *et al.* (2001) [83]; Rothman and Salovey (1997) [84]	See 3.3; Grady *et al.* (2011) [85]
Loss aversion	Tendency to dislike losses more strongly than enjoying an equivalent gain	Attema *et al.*, 2016 [86]; Attema *et al.*, 2013 [87]; Bleichrodt and Pinto (2002) [88]	See 3.3
Probability weighting	Tendency to overweight small probabilities (less likely events) and underweight large probabilities (more likely events)	Attema *et al.*, 2016 [86]; Attema *et al.*, 2013 [87]; Bleichrodt and Pinto (2000) [89]	See 3.3
Heuristics	Availability heuristic: Tendency to judge the likelihood of an event based on its salience (ease with which one can think of an example of it)	Pachur *et al.* (2012) [90]; An (2008) [91]; Triplet (1992) [92]	Williams *et al.* (2009) [93]; Sultan *et al.* (2003) [60]

Table 3.3 Continued: Decision-making biases presenting an interest as a lever for action to improve risk communication interventions.

	Affect heuristic (emotions): Emotional reactions guide decisions instead of cognitive analysis of risks	See Ferrer *et al.* (2015) for a review [94]; Suter *et al.* (2016) [95]; Pachur *et al.* (2014) [96]; Pachur *et al.* (2012) [90]; Beckjord *et al.* (2008) [97]	Portnoy *et al.* (2014) [98]
	Representativeness heuristic: Tendency to make intuitive judgements based on rules of similarity rather than objective measures of likelihood	Payne and Crowley (2008) [99]; Brannon and Carson, 2003 [100]; Triplet (1992) [92]	Sultan *et al.* (2003) [60]
Bounded willpower (present biasedness)	Tendency to have a stronger preference for more immediate payoffs relative to later payoffs	See Chabris *et al.* (2008) for a review [101]	Mørkbak *et al.*, 2017 [102]; Lebeau *et al.*, 2016 [103]; Reach *et al.* (2011) [104]; Sultan *et al.* (2003) [60]
Bounded selfishness (or bounded self-interest)	How others behave influence decisions	See McFerran (2015) for a review [105]	Schabert *et al.* (2013) [106]; Browne *et al.* (2013) [107]; Sultan *et al.* (2003) [60]

3.3 Risk attitudes towards self-management behaviour in T2DM populations

Complementing the work undertaken in Chapter 2 on risk perceptions, this section explores the risk attitudes of T2DM populations. I first explain why prospect theory is the optimal model to assess risk attitudes in the health domain. I then develop a tailored utility elicitation procedure, in line with the existing empirical literature, to measure both patients' risk attitudes and prospect theory biases in the health domain.

3.3.1 Prospect theory framework

Prospect theory (PT), developed in the 1970s by two Israeli psychologists, Daniel Kahneman and Amos Tversky, is the most widely used descriptive theory of decision-making under conditions of risk. [21, 50] It models people's preferences according to the following value function:

$$V = \sum_{i=1}^{n} \omega_i v(x_i) \tag{3.7}$$

Where $v(.)$ is the PT value function that assigns a (subjective) value to an outcome x_i.[36] The PT value function differs from a standard EU function by accounting for two decision-making biases:

- **Reference-point dependence**

  It assumes that people have reference-dependent preferences, meaning that they evaluate outcomes relative to a reference point (RP)[37] and then classify them as gains and losses (i.e. in terms of changes relative to this RP).[38]

[36] Kahneman and Tversky used the term *value* instead of the term *utility* to refer to this function. According to Starmer (2000), this is because they had in mind a conception of value independent of risk, and thus wanted to distance themselves from the notion of utility in EUT where utilities incorporate – at least partially – attitudes towards risk. [2]

[37] i.e. a reference outcome level (with monetary outcomes, a classical RP is 0).

[38] Under EUT, the RP does not matter, outcomes are evaluated independently.

- **Loss aversion**

  It assumes that people are more sensitive to losses than to gains as seen from this RP[39], i.e. that the pain of losing a given outcome is psychologically more powerful than the pleasure of gaining the same outcome.

These two biases, along with the assumption of *diminishing sensitivity*[40], give the PT value function a characteristic asymmetrical S shape (see Fig. 3.1). Passing through the RP, it is concave in the gain domain and convex in the loss domain (diminishing sensitivity), while being steeper for losses than for gains (loss aversion).

PT also accounts for the *probability weighting* bias. A probability weighting function $w(p_i)$ attributes a *decision weight* (or *transformed probability*) ω_i to an objective probability p_i. Under PT, people do not weight outcomes by their objective probabilities, but rather by these decision weights. The probability weighting function $w(p_i)$ typically has an inverted S shape (see Fig. 3.2) which reflects the tendency of people to overweight small probabilities (less likely events) and underweight large probabilities (more likely events).

Mostly applied in finance and insurance (where attitudes to risk play a central role)[41], the use of PT has radiated out to many other areas because of its descriptive accuracy. Numerous studies have provided and/or used empirical evidence of PT biases in fields as diverse as management [108], political sciences [109, 110], sport [111] and, importantly, healthcare. [84, 112–114] With monetary outcomes, much research has been done to measure utility and/or probability weighting under PT. [50, 115–118] Evidence is more scattered for loss aversion, as appropriate quantification methods have more recently been developed. However, despite some variations in the magnitude of the estimates, loss aversion for money has been found

[39] Losses loom larger than commensurate gains.

[40] Diminishing sensitivity holds that the psychological impact of a marginal change will decrease as we move further away from the RP. For example, relative to the status quo, the difference between a gain of \$10 and \$20 will seem larger than the difference between gains of \$1010 and \$1020. [62] More generally, applied to each outcome domain, this assumption entails diminishing marginal utility for gains (i.e., $u"(x) < 0$ for $x > 0$) and diminishing marginal disutility for losses (i.e., $u"(x) > 0$ for $x < 0$), which are properties not incompatible with EUT. [2]

[41] It was initially developed to explain gambling for money.

Figure 3.1: Prospect theory value function.

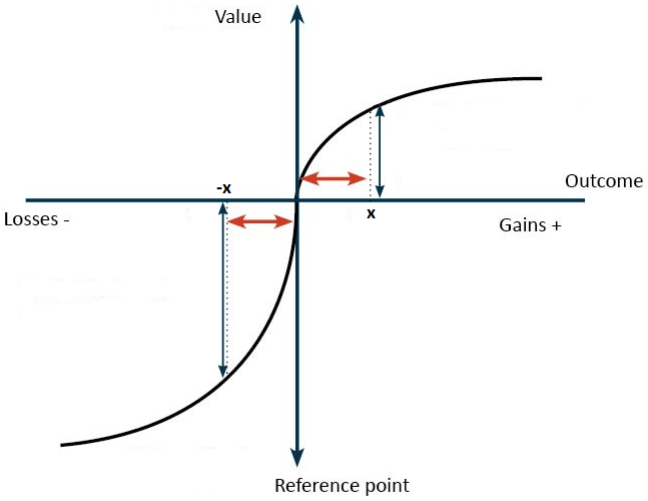
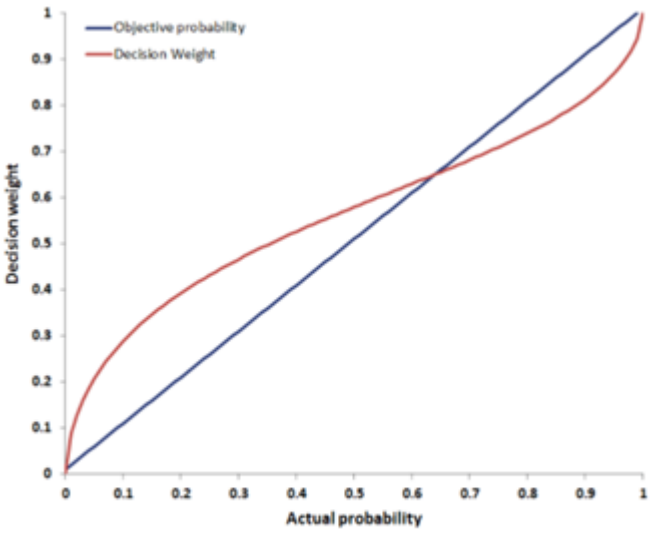


Figure 3.2: Probability weighting function.



to be quite robust in the lab. [119–121] More recent studies have also investigated the role played by loss aversion in decisions made outside of the lab. For example, Stephens and Tyran examined the role played by *nominal* loss aversion, i.e. the interaction of loss aversion and money illusion, in shaping evaluations of housing transactions. [122] Because people tend to think of transactions in terms of their nominal (monetary) values, real losses might loom larger when they are not "hidden"

by nominal gains.[42] They showed that evaluations of transactions involving real losses are systematically biased by nominal values, with nominal gains inducing more favourable evaluations ("nominal loss aversion"). On another note, a recent study by Andersson *et al.* (2014) investigated loss aversion in the context of decisions made for others. [123] Interestingly, the authors found that, on average, decision makers are less loss averse when their choices affect others.

In the health domain, however, the literature is much sparser. Although a few studies have reported some measures of utility [124, 125] and probability weighting [89], to my knowledge only two studies have quantified the whole PT function with health outcomes (i.e. including loss aversion). First, focusing on utility for life duration, Attema *et al.* (2013) [87] found evidence of loss aversion and risk aversion with respect to both gains (slightly concave utility) and losses (concave utility) in a study conducted on a student population. Evidence of probability weighting, more pronounced in the gain domain, was also reported. Second, a recent study conducted on a Dutch general population sample found similar trends with respect to the utility for quality of life (QoL). [86] Although evidence of PT biases has been reported, their findings challenge, in part, what is usually observed with monetary outcomes. In particular, they contradict the S-shaped utility function characteristic of PT, due to concave utility for losses.

These findings should also be nuanced, as several factors simultaneously impact the shape of the utility function, including risk and time preferences. For example, risk seeking behaviour has been found under both convex and concave utility for losses. [119] Importantly, it should be noted that these studies report measurements

[42] The authors give the following example: "Consider Anne, who buys a house for $200,000 in cash and sells it some years later for $170,000. She makes a nominal loss (of 15%) on this transaction. In an environment without inflation, the nominal loss corresponds to the real loss (15%). Compare this with Ben, who buys a house (somewhere else) for $200,000 in cash, and later sells it for $220,000. He makes a nominal gain (of 10%) on this transaction. However, if accumulated inflation in Ben's environment is substantial (30%, say) over the holding period, the transaction involves a substantial real loss (roughly 15%). Anne and Ben face equally disadvantageous situations in economic terms, with each facing the same real loss (15%). If they are equally prone to nominal loss aversion and at least partly evaluate the advantageousness of the transactions according to whether or not they lose money, Anne will be more reluctant to sell than Ben. The reason is that the real loss is more salient to Anne, who loses money, and less salient to Ben, who gains money." [122]

of PT parameters using mid-range probabilities (usually 0.5) in the design of the binary choice tasks used to elicit utility.[43] However, a major implication of PT is the prediction of a fourfold pattern of risk attitudes called the *reflection effect*. [50] The reflection effect is the consequence of the interaction between the PT value function (reference-point dependence) and the probability weighting function (underweighting of large probabilities and overweighting of small probabilities), and implies that:

- With large probabilities, people tend to express risk aversion over gains and risk seeking over losses. The reference-dependent phenomenon predicted by the PT value function is reinforced by the underweighting of large probabilities.
- With low probabilities, the converse happens, people tend to express risk seeking over gains and risk aversion over losses. The reference-dependent phenomenon predicted by the PT value function is reversed by the overweighting of small probabilities.

Table 3.4 illustrates the reflection effect by reporting the risk attitudes predicted by PT. [126] Although empirical support for the reflection effect is mixed (see Oliver, 2018 [126] for a review), some researchers have found evidence that supports this fourfold risk attitudes pattern, with respect to both money [127–129] and health outcomes. [126] In the health domain, Oliver (2018) has reported significant support for the reflection effect in an open valuation task[44] but his results were more mixed in a binary choice task. Hence, in view of these results, it is not excluded that risk aversion in both domains reported in Attema's studies can also hold under classic PT.[45]

[43] A binary choice task offers a choice between a risky outcome and a certain outcome, e.g. between a lottery and its expected value.

[44] An open valuation task directly asks the respondent to value a given lottery.

[45] However, because of loss aversion, it is unlikely to hold under EUT.

Table 3.4: Pattern of risk attitudes predicted by PT. [126]

	Gains	**Losses**
Large probability	**Risk aversion** (fear of missing gain)	**Risk seeking** (hope to avoid loss)
Small probability	**Risk seeking** (hope of gain)	**Risk aversion** (fear of loss)

This research is important to identify decision-making biases at the individual level that may be used to inform behavioural economic-informed policies, as in the case of this Thesis, but not only. Instruments used in health economics to elicit health-related preferences are based on the assumptions of standard rationality. Examples of common elicitation instruments include the certainty equivalence (CE) method, the standard gamble (SG), and the time tradeoff (TTO) method, which do not account for loss aversion and/or probability weighting. Consequently, empirical support for PT in the health domain may render such instruments invalid. Bleichrodt (2002), for instance, found that loss aversion exerts an upward bias on health state utilities elicited by SG and TTO, that utility curvature causes a downward bias on TTO utilities, and that underweighting of high probabilities often results in SG utilities that are too high (see also van Osch *et al.*, 2004 [130]). [131] Moreover, loss aversion is thought to be responsible for discrepancies observed between different versions of the TTO method [132, 133], and also between the WTP and the willingness to accept (WTA) elicitation methods. [134]

Using life duration as an outcome, Oliver (2003) reported that the internal consistency of the SG method could be improved with some adjustments for PT.[46] Among five alternatives to EUT considered (each one incorporating one or several PT components), he found that the one accounting for loss aversion improved the internal consistency of the SG. [23] More recently, Abellan-Perpiñan *et al.* (2009) found that evaluating health states under PT was significantly more consistent with ranking and choice tasks than under EUT. [135]

Hence, contributing to the literature on PT validity in the health domain

[46]The internal inconsistency of the SG could be due to a violation of the independence axiom. [23]

can also useful to reduce the biases inherent to health state valuation instruments currently used by health economists and, in turn, may improve policy-making decisions made based on these instruments.

The advantage of using PT to assess risk attitudes in my population of interest is thus threefold. First, as described above, previous studies have shown that PT is a suitable framework within which people's decision-making processes can be operationalised, including in the health domain. More than 30 years after Kahneman and Tversky's seminal paper, the model is still widely viewed as the best available description of how people evaluate risks.[47] [136] Second, despite such consensus, studies that have investigated PT preferences outside of the monetary domain are still rare. Analysing the risk attitudes of a patient population through the lens of PT will extend our knowledge of its validity in the health domain. Last, and most importantly, PT's key theoretical components incorporate three biases that have been identified as relevant to inform a behaviour intervention tailored to people with T2DM (see Table 3.3). Existing elicitation methods simply need to be adapted to fit the trade-off faced by T2DM populations regarding self-management. Identifying and characterising PT biases in this context will provide invaluable information for the design of a targeted behavioural intervention.

3.3.2 Objectives of the study

Focusing on a trade-off between health outcomes and behaviour change incurred by T2DM self-management, I measure the risk attitudes and quantify the full utility function under PT of a patient population. Using a recent method of utility elicitation under PT, I conduct a series of hypothetical elicitations over health outcomes associated with different self-management behaviours from a population of 120 individuals, half of whom live with T2DM. More specifically, I analyse risky decisions through the lens of three decision-making biases embedded into PT: RP dependence, probability weighting, and loss aversion.

[47] At least, in experimental settings.

3.3.3 Method

The method follows Attema and colleagues' semi-parametric method of utility elicitation in the health domain. [87] It uses *certainty equivalents*[48] (CEs) to measure the parameters of a specific utility function. CEs are elicited through a series of choices over binary prospects involving different life durations. However, I expand on this work by adding a QoL dimension to the outcomes, so that preferences based on more realistic scenarios for behaviour change can be captured. The use of quality-adjusted life years (QALY) outcomes allows quantifying similarly both the disutility of engaging in self-care behaviour and the disutility of suffering from diabetes-related complications.

a Model

Let $(p, x; y)$ denote a binary prospect that gives outcome x QALYs with probability p and outcome y QALYs with probability $1 - p$. Gain prospects [loss prospects, mixed prospects] imply that $x \geq y \geq 0$ [$x \leq y \leq 0, x > 0 > y$]. According to PT, evaluation of prospects can be represented by the following equations:

- $PT(p, x; y) = w^+(p) \times (u(x) - u(y)) + u(y)$ for gain prospects;
- $PT(p, x; y) = w^-(p) \times (u(x) - u(y)) + u(y)$ for loss prospects, and;
- $PT(p, x; y) = w^+(p) \times u(x) + w^-(1 - p) \times u(y)$ for mixed prospects.

The semi-parametric method entails three stages. First, eliciting CEs of a series of two-outcome gain prospects (different outcomes, but same probability for all prospects) allows me to identify simultaneously the utility function for gains and the decision weight $w^+(p)$ associated with the fixed probability. By assuming PT and a particular parametric shape of the utility function, it is indeed possible to estimate the parameters of the function and the decision weight of the probability that best fit the elicited data. Second, the same steps need to be performed in the loss domain. Finally, both utility functions can be connected through the elicitation

[48] The certainty equivalent is a guaranteed outcome (e.g. amount of money, years of life...) that one would accept rather than taking a chance on a better, but uncertain, outcome.

of the maximum loss amount that a subject is willing to accept in a mixed prospect including one of the gain outcomes used in the first stage. This enables estimation of the loss aversion coefficient λ, allowing comparison between gain utilities and loss utilities.[49][50] Details are provided in the following subsections. This semi-parametric method has several advantages compared to fully and non-parametric methods. On the one hand, measurements are not confounded by assumptions about the shape of the probability weighting function. On the other hand, it is less time-consuming because fewer questions are needed to elicit the CEs. [119]

b Elicitation of utility parameters and decision weights

Following Attema *et al.* (2013) [87], I consider the exponential family of utility functions for both utilities of gains and losses[51]:

$$\begin{cases} u(x) = \frac{1-\exp(-\gamma x)}{\gamma} & \text{for gains;} \\ u(x) = \frac{\exp(\delta x)-1}{\delta} & \text{for losses.} \end{cases}$$

with $\gamma, \delta \neq 0$. For $\gamma, \delta = 0, u(x) = x$.[52][53]

For each prospect i in the gain [loss] domain, I elicit a CE_i such that the respondent is indifferent between gaining [losing] CE_i QALYs for certain and the prospect that provides a 50% chance of gaining [losing] a higher amount x_i QALYs and 50% chance of gaining [losing] a lower amount y_i QALYs. Then, assuming PT and exponential utility functions, this indifference can be described by the following equations:

- **Gains**:

$$\frac{1-\exp(-\gamma CE_i)}{\gamma} = \omega^+ . \left(\frac{1-\exp(-\gamma x_i)}{\gamma} - \frac{1-\exp(-\gamma y_i)}{\gamma}\right) + \frac{1-\exp(-\gamma y_i)}{\gamma}; \tag{3.8}$$

[49] The method assumes that observable U is a composition of λ and a basic utility u, with: $\mathrm{U(x)}=\begin{cases} u(x) if x \geq 0 \\ \lambda u(x) if x < 0 \end{cases}$

[50] An individual is thus classified as loss averse when $\lambda > 1$.

[51] However, other parametric families could also be used (e.g. a power utility function).

[52] For gains, u(x) concave if $\gamma > 0$, convex if $\gamma < 0$, and linear if $\gamma = 0$.

[53] For losses, u(x) concave if $\delta < 0$, convex if $\delta > 0$, and linear if $\delta = 0$.

with $\omega^+ = w^+(1/2)$.

Solving this expression for CE_i gives the following regression equation:

$$CE_i = \frac{(ln[\gamma^+.(exp(-\gamma x_i) - exp(-\gamma y_i)) + exp(-\gamma y_i)])}{\gamma}. \tag{3.9}$$

From this equation, it is possible to estimate both the utility function parameter γ and the decision weight ω^+ simultaneously, through non-linear least squares.

- **Losses**:

$$\frac{\exp(\delta CE_i) - 1}{\delta} = \omega^-. \left(\frac{\exp(\delta x_i) - 1}{\delta} - \frac{\exp(\delta y_i) - 1}{\delta} \right) + \frac{\exp(\delta y_i) - 1}{\delta}; \tag{3.10}$$

with $\delta^- = w^-(1/2)$.

Solving this expression for CE_i gives the following regression equation:

$$CE_i = \frac{(ln[\omega^-.(exp(\delta x_i) - exp(\delta y_i)) + exp(\delta y_i)])}{\delta}. \tag{3.11}$$

Similarly, from this equation, it is possible to estimate both the utility function parameter δ and the decision weight ω^- simultaneously, through non-linear least squares.

c Elicitation of loss aversion parameter

I estimate the loss aversion coefficient λ by determining the loss outcome $L^*$for which the participant is indifferent between a prospect giving a 50% chance of gaining an outcome G^* and a 50% chance of losing the outcome L^*, and the status quo:

$$(1/2, G^*; L^*) \sim 0 \tag{3.12}$$

This mixed prospect gives:

$$\omega^+.U(G^*) + \lambda.\omega^-.U(L^*) = 0 \tag{3.13}$$

Which, in terms of the exponential utility function, translates into:

$$\omega^+.\frac{1 - \exp(-\gamma G^*)}{\gamma} + \lambda.\omega^-.\frac{\exp(\delta L^*) - 1}{\delta} = 0 \tag{3.14}$$

Solving this expression for L^* gives the following regression equation:

$$L^* = \frac{ln\left[1 - \left(\frac{\omega^+.\delta}{\omega^-.\gamma}\right)\frac{1-\exp(-\gamma G^*)}{\gamma}\right]}{\delta} \tag{3.15}$$

3.3.4 Experiment

a Stimuli

I aim to elicit preferences, both in the gain and loss domains, when the outcomes vary in terms of both life duration and QoL (see c). As a result, participants make choices over outcomes quantified in terms of QALYs.

The utility functions for both gains and losses were elicited by the use of 5 CE questions each and one outcome G^* was used to measure loss aversion (see Table 3.5). Importantly, I introduced an RP of living 20 more years. The length of the RP was the result of a trade-off between two elements: tractability and realism. On the one hand, a large RP was needed to allow variation in prospects over a sufficiently wide range of health outcomes. Setting up an RP of 20 years allowed me to design loss prospects involving the potential loss of up to 15 years of life. I decided not to design loss prospects involving the loss of up to 20 years of life (i.e. the totality of the RP), in order not to induce any psychological stress or negative emotional reaction. The prospect of dying *immediately*, that may have provoked anxiety, was thus ruled out as an option. On the other hand, because T2DM rarely occurs before the age of 50, the average life expectancy of the population did not provide me with much leeway to adopt a large RP. For the sake of realism, I decided to exclude individuals aged above 60 from the study (see b). In this way, setting up a RP of 20 years led to the best-case scenario of older participants reaching the maximum age of 95 years.[54] More details on the elicitation procedure are provided in c.

b Participants

The sample includes 120 individuals between 30 and 60 years of age. Sixty participants were living with T2DM, and 60 participants had no diagnosis of

[54]Patient of 60 years old + RP of 20 years of life + potential gain of 15 years of life.

Table 3.5: List of stimuli used in the experiment (years of life [QALYs]).

	j=1	**j=2**	**j=3**	**j=4**	**j=5**	G^*
$\|x\|$	6 [4.5]	10 [7.5]	12 [9]	15 [11.25]	15 [11.25]	10 [7.5]
$\|y\|$	0 [0]	0 [0]	0 [0]	5 [3.75]	9 [6.75]	

T2DM or any other chronic disease. T2DM is a good example of a manageable chronic disease: it often requires multiple drug therapies, frequent monitoring of risk factors and regular health care contacts, and good control is associated with better health outcomes (life expectancy and QoL). This study received ethics approvals from the competent NHS research ethics committees (reference 16/NE/0203) and from the University of Oxford's Central University Research Ethics Committee (reference R44523/RE001) (see Appendix D).

T2DM group

The inclusion criteria were: (1) aged between 30 and 60; (2) diagnosed with T2DM; (3) willing and able to give informed consent for participation in the study. The exclusion criterion was: non-English speaker. The rationale behind the lower age limit (30) is that T2DM is rarely diagnosed before that age, while the upper age limit (60) was set for experimental reasons, due to the RP of 20 years: the older the subject, the more unrealistic the hypothetical scenarios used to elicit risk preferences and the higher the risk of elicitation bias (see Appendix E). Subjects in this group were familiar with the daily management of T2DM (they had been informed and trained at diagnosis).

Control group

I formed a control group to examine risk preferences and PT parameters in a comparable population who is not concerned with the daily management of a chronic disease. The inclusion criteria were: (1) aged between 30 and 60; (2) not diagnosed with T2DM or any other chronic disease; (3) willing and able to give informed consent for participation in the study. The exclusion criterion was: non-English speaker.

c Procedure

Recruitment

Control participants were patients between 30 and 60 coming to the surgery practice for minor health issues and who kindly agreed to participate in the study. In order to balance the sample in terms of age and gender, I tried to recruit a similar proportion of subjects below and above 50 as well as a similar proportion of men and women in both groups. Because T2DM usually appears in the late 50 / early 60 years of age, a major challenge of the study was to find 'young' eligible people (i.e. aged below 50) diagnosed with the disease. Similarly, it was more difficult to recruit female participants than male participants in the T2DM group because of the higher prevalence of T2DM in the male population.

After receiving information about the study from their general practitioner (GP), consenting participants were presented with the online questionnaire during a one-to-one interview with an experimenter (myself). The study took place in a private area of the surgery practice. The experimenter carefully explained each scenario and made sure that the participant understood the instructions and the questions. It was made clear that there were no right or wrong answers. Each subject received a modest monetary incentive (£10) to participate in the study, as is commonly done in the field. The incentive was given at the end of the survey, independently of the answers of the participant.

Questionnaire

A questionnaire specifically designed to elicit participants' risk preferences was developed in collaboration with both health professionals and patients. I adapted existing questionnaires to fit the population and outcomes of interest. [87, 119] In particular, I took into account the suggestions of a small group of patients (4 volunteers) who were asked to provide feedback on user-friendliness (graphical features of the questionnaire) and comprehensibility (wording used in the instructions).

Indifferences (i.e. the CEs) were elicited through a series of binary choices. Each binary choice corresponded to an iteration in a bisection process (see example

in Table 3.6). In each choice participants were faced with two prospects, labelled *option A* and *option B*, where option A was always riskless. Based on volunteers' feedback, prospects were displayed as *smiley chains*, with each smiley symbolising a year of life, and accompanied by a short description. A green smiley represented a year lived with perfect QoL (100%), a yellow smiley a year lived with good QoL (75%), and a red smiley a year lived with moderate QoL (50%). Moreover, the wording used in the questionnaire was as simple as possible and two GPs provided feedback on the description of the self-management activities (see Appendix E, which provides three examples of the way the experimental questions were displayed).

The order of the 5 CE questions was randomised to avoid a potential order effect (for both gains and losses). Moreover, to control for response errors and check the reliability of collected data, I repeated the first iteration after the final iteration for 5 CE questions (j=3 and j=4 for both gains and losses, and the mixed prospect). [119]

Table 3.6: An illustration of the bisection method (gains; j=4).

Iteration	Offered choices in elicitation of G4	EV (QALY)
1	**10 years (75%)** vs. [½, 15 years (75%); 5 years (75%)]	**7.5**
2	5 years (75%) vs. **[½, 15 years (75%); 5 years (75%)]**	3.8
3	**8 years (75%)** vs. [½, 15 years (75%); 5 years (75%)]	**6.0**
4	6 years (75%) vs. **[½, 15 years (75%); 5 years (75%)]**	4.5
5	**7 years (75%)** vs. [½, 15 years (75%); 5 years (75%)]	**5.3**
Indifference value	**6.5 years (75%)**	**4.9**

In Table 3.6, the prospect that is chosen in each iteration is printed in bold. Starting values in the iterations were always selected so that prospects had equal expected value. Depending on the choice made, the certain outcome was increased or decreased (but was always an integer). The method resulted in an interval within which the indifference value should lie. The midpoint of this interval was taken as

the indifference value. In this first example, I started with prospects whose expected value was 7.5 QALYs[55] The indifference value elicited for the sure outcome was 4.9 QALYs[56], implying risk aversion.

Scenarios

The QoL dimension that I consider is a broad notion of health-related QoL, which describes the three hypothetical scenarios used to design the prospects. Each scenario is attributed with a score, expressed in percentage points, on a scale ranging from 0 (worst possible health-related QoL) to 100 (best possible health-related QoL), based on a description of the QoL associated with the scenario in question. The scenarios and their values were designed according to four criteria. First, the scenarios should depict significantly different QoL scores, in order to (a) benefit from sufficient leeway to design the prospects; and (b) create clear differences in the mind of participants. Second, the QoL scores should be as easy to understand as possible for the participants. Third, the QoL scores should also be as realistic as possible. Finally, the scenario descriptions should not be over-precise, in order to avoid rating biases. I wanted the participants to imagine living with the QoL score I set and to use a precise health state description to illustrate this score (e.g. "living with a QoL of 50% corresponds to living with a lower extremity amputation") would very likely create a bias, depending on how the person actually rates the QoL of living with an amputation. Such personal ratings could be much lower or higher than 50%, and studies such as Huang *et al.* (2007) [137] have shown that the scores patients with T2DM attribute to a given health state can be widely heterogeneous.

Scenarios and corresponding scores are displayed in Table 3.7. I used QoL scores drawn from a study which aimed to quantify patients' utilities (i.e. measuring preferences) for the full array of diabetes-related complications and treatments. [137] For scenario 1 (*behaviour change; no complication*), I used the utility attributed to

[55] $0.5 \times(15 \times 75\%) + 0.5 \times (15 \times 75\%) = 7.5$
[56] $1 \times(6.5 \times 75\%) = 4.9$

Table 3.7: Scenarios and associated QoL scores.

	Scenario 1 Behaviour change; no complication	**Scenario 2** No behaviour change; no complication	**Scenario 3** No behaviour change; complication
Score	QoL: 75% (good)	QoL: 100% (perfect)	QoL: 50% (moderate)
Source (mean [SD])	Utility attributed to conventional glucose control (0.76 [0.31]) [137]	-	Utility attributed to lower extremity amputation (0.55 [0.36]) [137]
Scenario description	Constraints in daily life linked to precautionary behaviour: "You will need to follow a diet, to exercise regularly, to see the doctor regularly, and to take daily medications. For example, you may have to eat less of some foods you like, or experience mild to moderate side effects associated with your medications."	No health-related constraints in everyday life	Constraints in daily life linked to a complication: "You might experience mild to moderate pain, tiredness, or difficulties in performing daily tasks such as driving or cleaning the house."

life under *conventional glucose control*[57] and rounded it down to 75%. For scenario 3 (*no behaviour change; complication*), I used the utility attributed to life with a lower extremity amputation and rounded it down to 50%. These scores (75%, 50%) reflect a compromise which satisfies each of the four criteria mentioned before, and quantify the (dis)utilities inherent in the three scenarios (and thus allow the elicitation of CEs) but are not intended to generate empirically grounded QALYs.

d Analysis

CEs and risk attitudes

A participant was classified into the risk averse [risk neutral, risk seeking] category for gains [losses] if he or she showed risk aversion [risk neutrality, risk seeking] for at least four out of five CEs. Otherwise, the person was classified into the mixed category for gains [losses]. This allowed me to take into account a small margin of error in the responses. Because the data were not normally distributed, I performed non-parametric statistical tests. Within-subjects comparisons were made using Wilcoxon signed-rank tests and between-subject comparisons were made using Mann-Whitney U tests. In both cases, two-tailed p-values are reported.

Utility and loss aversion parameters

The five types of individual parameters were estimated through non-linear least squares regressions. For each participant, utility parameters (risk aversion coefficients and decision weights) were calculated, in both domains, to best fit the elicited data (i.e., the CEs) to the non-linear functions described in equations (3.9) and (3.11). In the gain [loss] domain, I estimated the values of ω^+ and δ^+ [ω^- and δ^-] that minimised the sum of squared residuals, i.e. the difference between the elicited data and the fitted value provided by the model (ω^+ and ω^- were constrained between 0 and 1). After estimating the four utility parameters, I estimated the loss aversion coefficient based on equation (3.15). I also analysed

[57] i.e. following a recommended diet, exercising regularly, visiting the doctor 4 times/year, getting blood tests 2 times/year, adhering to glucose control therapy 1-2 pills, 1-2 times/day, possibly experiencing side effects.

the impact of observable characteristics by running ordinary least squares (OLS) regressions on each parameter estimate.

3.3.5 Results

a Reliability

In order to check for answer consistency throughout the experiment, I repeated the first iteration, after the final iteration, for five different CEs. Overall, 110 individuals provided consistent answers for at least four out of five CEs (92%), indicating good reliability. In the T2DM group [control group], 39 participants (65%) provided consistent answers for the five CEs in both domains [49 participants (82%)], 13 participants (22%) for four out of five CEs [9 participants (15%)] and only 8 participants (13%) for three out of five CEs [2 participants (3%)]. In both groups, no participants gave inconsistent answers for more than two out of five CEs. However, in order to minimise the bias due to unreliable answers, I decided to exclude participants who were not consistent for at least four out of five CEs. In the analysis, for subjects who answered inconsistently for one CE (e.g. they first expressed risk aversion for the CE, but then expressed risk seeking for the same CE when repeating the first iteration), I eliminated that CE and classified the subject according to their other answers. Therefore, the final sample sizes for analysis are 52 (T2DM group) and 58 (control group).

b Characteristics of the participants

Table 3.8 presents a summary of the participants' characteristics in the final sample (n=110). Chi-square tests show no significant between-group differences in terms of gender (p=0.14), employment status (p=0.23), education level (p=0.32) or reliability (p=0.14). According to Mann-Whitney U tests, age difference was significant (p=0.012) but time to complete the questionnaire was not (p=0.39).

Table 3.8: Characteristics of the participants.

	T2DM group	Control group	Total
Sample size	52	58	110
Sex, n(%)			
Male	32(61.5%)	28(48.3%)	60(54.5%)
Female	20(38.5%)	30(51.7%)	50(45.5%)
Age (years)			
median [IQ1-IQ3]	53.5 [46-57.75]	50 [36-56]	51.5 [40-57]
Education, n(%)			
High school or no degree	20(38.5%)	18(31%)	38(34.5%)
Bachelor or graduate degree	32(61.5%)	40(69%)	72(65.5%)
Employment, n(%)			
Employed	39(75%)	48(82.8%)	87(79.1%)
Unemployed or retired	13(25%)	10(17.2%)	23(20.9%)
Reliability, n(%)			
5/5	38(73.1%)	49(84.5%)	87(79.1%)
4/5	14(26.9%)	9(15.5%)	23(20.9%)
Time to complete the questionnaire			
min (mean[SD])	21.8 [5.9]	21.0 [7.5]	21.4 [6.8]

c Utility for gains and losses

CEs and risk attitudes

Table 3.9 classifies subjects according to their risk category in each domain. It shows that half of the subjects (49%; 30 in the T2DM group and 24 in the control group) were risk averse over the whole domain. The second largest group is made up of people who were risk averse in the gain domain and mixed in the loss domain (17% of the sample; 7 in the T2DM group and 12 in the control group). A large majority of subjects were classified into the risk aversion category for gains (70%), but only half of them for losses (55%). This difference in proportions is significant (p=0.02). Between-group comparisons do not show significant differences in terms of proportion of subjects classified into the risk averse and mixed categories with respect to gains (75% and 23% in the T2DM group vs 66% and 24% in the control group, p=0.14 and p=0.90, respectively); however, there is a trend towards significant difference for risk seeking subjects (2% vs 10%, p=0.07). In the loss domain, results

are more mixed but there are no significant differences in terms of proportions.

Table 3.9: Classification of subjects according to their risk category, for each domain.

	Losses				
	Risk averse	Risk neutral	Risk seeking	Mixed	Total
T2DM group					
Gains					
Risk averse	30	1	1	7	39
Risk neutral	0	0	0	0	0
Risk seeking	0	0	1	0	1
Mixed	3	0	4	5	12
Total	33	1	6	12	52
Control group					
Gains					
Risk averse	24	0	2	12	38
Risk neutral	0	0	0	0	0
Risk seeking	1	0	2	3	6
Mixed	3	1	1	9	14
Total	28	1	5	24	58

Table 3.10 reports the medians and interquartile ranges of individual CEs elicited in both groups. There is strong evidence for risk aversion, in both the gain and the loss domains. For four out of five CEs (j= 2, 3, 4, 5), risk aversion was higher in the gain domain. This difference is significant for j= 4, 5. Conversely, for j=1, risk aversion was significantly higher in the loss domain. Overall, 77% [7%, 16%] of the responses elicited CEs conveyed risk aversion [risk neutrality, risk seeking] for gains, whereas 65% [15%, 20%] conveyed risk aversion [risk neutrality, risk seeking] for losses. Median CEs elicited in the control group were often higher than and always at least as high as those elicited in the T2DM group, indicating higher risk aversion in the T2DM group. Differences are significant for one CE in the gain domain (j=5; p-value <0.01) and two CEs in the loss domain (j=2; p-value=0.034; j=5; p-value=0.093).

Table 3.10: Median CEs (absolute values) and Wilcoxon tests on the difference between gains and losses.

	T2DM group			**Control group**			**Total**		
	Gains	Losses	p-value	Gains	Losses	p-value	Gains	Losses	p-value
j=1 median (IQ1-IQ3)	2.50 (0.63-3.00)	0.75 (0.25-2.75)	<0.01 ***	2.50 (0.50-3.31)	2.00 (0.25-3.32)	0.06 *	2.50 (0.50-3.06)	1.25 (0.25-3.00)	<0.01 ***
j=2 median (IQ1-IQ3)	3.00 (1.25-4.50)	3.50 (0.63-5.00)	0.45	4.00 (2.00-5.00)	4.50 (2.44-5.50)	0.26	3.50 (2.00-4.50)	4.50 (2.00-5.50)	0.17
j=3 median (IQ1-IQ3)	3.00 (1.75-5.50)	3.00 (1.00-6.00)	0.85	4.00 (1.25-6.00)	5.25 (3.00-6.00)	0.86	4.00 (1.25-6.00)	4.50 (1.13-6.00)	0.96
j=4 median (IQ1-IQ3)	5.25 (5.00-8.25)	8.75 (5.00-10.00)	0.03 **	7.00 (5.00-9.00)	8.75 (6.31-10.00)	0.01 ***	6.50 (5.00-9.00)	8.75 (5.00-10.00)	<0.01 ***
j=5 median (IQ1-IQ3)	9.00 (9.00-10.00)	10.50 (9.00-12.38)	<0.01 ***	10.00 (9.00-12.00)	12.00 (9.00-12.13)	0.02 **	9.00 (9.00-12.00)	11.25 (9.00-12.13)	<0.01 ***

Note: *p<0.1; **p<0.05; ***p<0.01

Utility parameters

Table 3.11 reports the median utility parameters, based on the individual data. The median coefficients were significantly different from 0 for gains ($\lambda = 3.46$, p<0.01) and losses ($\delta = 1.91$; p<0.01), but were not significantly different from each other (p=0.84). The distributions of individual utility parameters are provided in Appendix F. Probability weighting was statistically significant in the gain domain ($\omega^+ = 0.49$; $p < 0.01$).

Overall, a large majority of subjects had concave utility functions in both domains (see Table 3.12). In the T2DM group, in particular, only 11 subjects (21%) had a convex utility in one of the two domains.

d Loss aversion

I observed a high degree of risk aversion in the mixed prospect, with 86% of the responses being risk averse. Only 11 [5] individuals (10% [5%]) were risk seeking [risk neutral]. The median coefficient for loss aversion is significantly different from 1 ($\lambda = 1.19$; p<0.01; see Table 3.11). Appendix F shows the distribution of individual coefficients for loss aversion.

Table 3.11: Estimation results for the parameters of interest.

		γ	ω^+	δ	ω^-	λ
T2DM group (n=52)	Median (IQ1-IQ3)	3.81 (1.45-9.94)	0.49 (0.48-0.50)	2.83 (0.77-8.14)	0.50 (0.49-0.50)	1.19 (0.91-2.43)
Control group (n=58)	Median (IQ1-IQ3)	2.86 (0.47-6.86)	0.50 (0.49-0.50)	1.29 (-0.07-3.69)	0.50 (0.49-0.51)	1.19 (0.56-1.92)
Total (n=110)	Median (IQ1-IQ3)	3.46 (1.03-8.91)	0.49 (0.48-0.50)	1.91 (0.36-5.23)	0.50 (0.49-0.50)	1.19 (0.62-2.00)

Note 1: In the gain domain, u(x) concave [linear, convex] if $\gamma > 0[\gamma = 0, \gamma < 0]$. In the loss domain, u(x) concave [linear, convex] if $\delta < 0[\delta = 0, \delta > 0]$.

Note 2: Loss aversion [neutral, seeking] when $\lambda > 1[\lambda = 1, \lambda < 1]$.

Note 3: All coefficients were significantly (p<0.01) different from 0 (γ and δ), 0.5 (ω^+ and ω^-), and 1 (λ).

Table 3.12: Classification of subjects according to their utility functions.

		Losses	
	Concave	Convex	Total
T2DM group			
Gains			
Concave	43	6	49
Convex	1	2	3
Total	44	8	52
Control group			
Gains			
Concave	38	8	46
Convex	5	7	12
Total	43	15	58

e Risk attitudes and observable characteristics

Table 3.13 shows the results of OLS regressions performed on each of the five parameters of interest. I studied the impact of five variables: (1) sex; (2) age; (3) T2DM status; (4) education; and (5) employment.

Firstly, I observed a clear age effect on risk aversion coefficients. The older the individual, the higher the risk aversion coefficient for both the gain ($p<0.01$ for both group categories) and the loss domain ($p=0.06$ for the senior group effect and $p=0.01$ for the veteran group effect). Secondly, people with T2DM were characterised by stronger probability underweighting in the gain domain, which suggests higher risk aversion than controls, on average, in this domain ($p=0.01$). Thirdly, significant interactions between education level and employment status suggest the presence of underlying subgroup effects. In order to further investigate the effects of age and T2DM status on risk aversion, I performed additional OLS regressions to check whether these results held when including only participants in the sample who provided fully consistent answers (i.e. excluding people who provided one inconsistent answer). Results displayed in Table 3.14 confirmed the existence of both effects.

Table 3.13: Ordinary least square regressions on the parameter estimates.

	Dependent variable				
	γ	ω^+	δ	ω^-	λ
Sex					
Male (ref.)	-	-	-	-	-
Female	2.683	-0.003	0.951	<0.001	0.948
	(3.029)	(0.010)	(3.126)	(0.004)	(0.916)
Age					
Junior (ref.)	-	-	-	-	-
Senior	9.330**	-0.012	7.303*	0.003	-0.022
	(3.368)	(0.010)	(3.822)	(0.005)	(0.534)
Veteran	11.327***	-0.022	12.258**	-0.003	1.507
	(3.719)	(0.016)	(4.673)	(0.005)	(1.379)
Group					
T2DM (ref.)	-	-	-	-	-
Control	-1.196	0.027**	-3.070	<0.001	1.103
	(2.935)	(0.010)	(3.240)	(0.004)	(1.577)
Employment					
Not employed (ref.)	-	-	-	-	-
Employed	2.699	-0.013	4.568	<0.001	-0.152
	(5.419)	(0.033)	(7.929)	(0.008)	(0.603)
Education					
Cat 1 (ref.)	-	-	-	-	-
Cat 2	-10.821*	0.034	-11.782	0.007	4.619
	(6.286)	(0.030)	(7.828)	(0.009)	(4.841)
Employment*Education					
Not employed/Cat 1 (ref.)	-	-	-	-	-
Employed/Cat 2	13.240*	-0.032	15.031	-0.005	-4.440
	(7.453)	(0.034)	(9.249)	(0.010)	(4.586)
Constant	-2.173	0.477***	2.165	0.495***	0.055
	(4.348)	(0.016)	(5.050)	(0.006)	(1.837)
Observations	110	110	110	110	110
R^2	0.107	0.113	0.097	0.028	0.084
Prob>F	0.031	0.321	0.090	0.892	0.909

Note 1: *p<0.1; **p<0.05; ***p<0.01

Note 2: Sex (male=0; female=1); age: junior (30 to 45 years old)=0; senior (46 to 55 years old)=1; veteran (56 to 60 years old)=2; group (T2DM=0; control= 1); education (cat 1: high school or no degree=0; cat 2: bachelor or graduate degree=1); employment (unemployed=0; employed=1).

Table 3.14: Ordinary least square regressions on the parameter estimates, when excluding individuals who provided one inconsistent answer.

	Dependent variable				
	γ	ω^+	δ	ω^-	λ
Sex					
Male (ref.)	-	-	-	-	-
Female	2.592	-0.006	-0.342	0.002	-0.031
	(3.342)	(0.013)	(3.591)	(0.004)	(0.263)
Age					
Junior (ref.)	-	-	-	-	-
Senior	13.050***	-0.014	9.431**	<0.001	0.310
	(3.957)	(0.012)	(4.582)	(0.006)	(0.306)
Veteran	13.792***	-0.024	14.955***	<0.001	0.246
	(4.129)	(0.017)	(5.283)	(0.006)	(0.347)
Group					
T2DM (ref.)	-	-	-	-	-
Control	-4.631	0.03**	-6.519	0.001	-0.200
	(3.238)	(0.013)	(3.801)	(0.004)	(0.306)
Employment					
Not employed (ref.)	-	-	-	-	-
Employed	3.698	-0.011	9.277	0.008**	-0.311
	(5.833)	(0.041)	(8.588)	(0.004)	(0.371)
Education					
Cat 1 (ref.)	-	-	-	-	-
Cat 2	-12.596*	0.034	-14.926	<0.001	0.333
	(7.489)	(0.039)	(9.189)	(0.006)	(0.528)
Employment*Education					
Not employed/Cat 1 (ref.)	-	-	-	-	-
Employed/Cat 2	15.741*	-0.027	22.085**	0.007	-0.432
	(8.584)	(0.044)	(10.702)	(0.008)	(0.610)
Constant	-1.725	0.471***	2.050	0.492***	1.466
	(4.296)	(0.018)	(6.055)	(0.006)	(0.394)
Observations	88	88	88	88	88
R^2	0.187	0.136	0.164	0.024	0.035
Prob>F	0.017	0.271	0.052	0.365	0.775

Note 1: *p<0.1; **p<0.05; ***p<0.01

Note 2: Sex (male=0; female=1); age: junior (30 to 45 years old)=0; senior (46 to 55 years old)=1; veteran (56 to 60 years old)=2; group (T2DM=0; control= 1); education (cat 1: high school or no degree=0; cat 2: bachelor or graduate degree=1); employment (unemployed=0; employed=1).

3.3.6 Discussion

a Novelty

This study extends previous research on decision-making under health risks in two ways. First, I expand on previous work investigating risk attitudes and PT utility in the health domain by building the prospects upon more realistic scenarios (decision on whether or not to adopt self-management behaviour faced by people living with a manageable chronic disease) and using health outcomes that include both dimensions of health utility: life duration and QoL. Second, while previous studies used a student or general population, my study population was selected to allow comparisons between people living with a manageable chronic disease (i.e. patients actually facing such risky choices) and people without chronic disease. These aspects are crucial in the perspective of bridging theoretical results and actual policy-making. Below I discuss the findings and their implications in more detail.

b Risk attitudes

My results show that participants expressed a high degree of risk aversion, both in the gain and in the loss domains, and significant loss aversion. Two phenomena seem to explain this trend. Firstly, participants had very concave utility functions, which translated into high utility parameters for both gains and losses. This is in line with previous studies conducted on student [87] and general populations [86] and suggests that the role of the RP may be less crucial for health than for money. Another possibility is that, despite making the RP as salient as possible in the instructions (use of visual aids), people's own idea of how long they may live was still stronger than the induced time horizon. More research that explicitly investigates this point is needed. Secondly, risk aversion was also reflected in probability underweighting of probability 0.5 (only significant in the gain domain). Similar effects were reported in the abovementioned studies. Regarding loss aversion, my coefficient is comparable with those reported in the literature for the health domain. [87]

However, a feature specifically related to the study of risk attitude for life years is that life years by definition involve a time component. Consequently, the

results will be affected by time preferences. In fact, the utility function for life duration can be regarded as reflecting both diminishing returns to lifetime and discounting of future life years, and it is hard to disentangle these effects. The incorporation of losses in this domain is particularly challenging, because only outcomes involving future (as opposed to past) lifetime are useful and, therefore, the RP has to be in the future in order to allow for losses. This creates two problems. First, because the changes in health occur at some point in the future, the observed choices may have been different if the changes had taken effect immediately. Second, the higher the loss, the closer one gets to the present, so that as the individual moves farther away from their RP, the influence of time preferences is reduced. As a result, the concavity resulting from positive time preference will neutralize any convexity inherent to losses, whereas there is no such influence of time preference for monetary outcomes. This may explain why I find concave utility for losses on balance.

I have also performed robustness analyses to test for any effect caused by the onset of the health changes being only in 15 years for gains. Table 1 in Appendix G shows the results, indicating no large differences when allowing for discounting these 15 years. As a result, one could argue that the S-shaped utility function typically found for money does not apply to health, where the distinction between gains and losses appears less influential. This does not detract from the robust evidence of loss aversion found for health outcomes. Hence, the framing of a question as a choice between the status quo and a mixed prospect clearly induces more risk aversion than framing questions as choices between two gain prospects or two loss prospects, even when the distinction between gains and losses appears less natural as in the case of life duration. Table 2 in Appendix G illustrates this in more detail, where the results of an additional analysis are presented that combines the CEs of both the gain and the loss prospects to elicit one single utility parameter and one single decision weight, while still using the results of the mixed prospect to estimate loss aversion.

Moreover, two interesting subgroup effects have been observed in this study.

Firstly, age was negatively related to risk attitudes. Splitting my sample into three groups of equivalent size, as a compromise between tractability (i.e. benefiting from sufficient variability in the data) and accuracy, significant effects were found on utility parameters for gains and losses. Such effects have already been reported in the health domain: Dohmen *et al.* (2011) showed that older individuals express, on average, higher risk aversion than younger individuals [138] and Rolison *et al.* (2013) found that health risk-taking reduces smoothly with age. [139] I examined a specific trade-off, in which choices were likely to be influenced by age. The median age of the sample, combined with the RP of living 20 more years, means that most subjects were asked to contemplate their life at (at least) age 70 and make decisions accordingly. From this perspective, it is likely that the QoL dimension had more weight than the life duration dimension in the decision-making process. For an older subject, the prospect of living the last years of his/her life with a constant, relatively good QoL (75%) was likely to be enough to offset the chance of living longer, i.e. the best outcome of the risky option. Conversely, following the same reasoning, the QoL dimension was less likely to prevail over the life duration dimension in decisions made by younger subjects. Hence, younger subjects seem to care relatively less about quality of life than older subjects, stressing the importance of implementing age-specific health promotion policies.

Secondly, I found that people with T2DM were, on average, underweighting probability 0.5 more strongly than controls, suggesting higher risk aversion in the T2DM group. However, caution is required about the interpretation of this effect, as it was no longer significant when including only fully consistent subjects. Rather than revealing different underlying risk preferences between patients and controls, a possible, alternative explanation is that the elicitation method could have enhanced the observed T2DM effect. Indeed, the higher degree of risk aversion found with T2DM patients could be interpreted as a difference in terms of perception of the task, and more specifically as an adjustment of their RP during the experiment. Since people with T2DM had already 'integrated' the cost of behaviour change (they had already had to change their behaviour), and although they were explicitly

told to forget about their condition and imagine living with perfect QoL at the beginning of tasks 1 and 2, they could have implicitly taken 75% of QoL, i.e. the QoL of the safe option, as their RP (as opposed to controls, who took 100% of QoL as their RP). It is assumed that each individual adjusts, to a certain extent, the RP according to her own situation, experience or perceptions; setting up a common RP allows to highlight these differences. In that case, gain and loss prospects were possibly interpreted as mixed prospects by some participants in the T2DM group. Option A was seen as the status quo, and option B was seen as a mixed prospect offering the chance to 'gain' or 'lose' 25% of QoL. As a consequence, because of loss aversion, this could have led to a higher degree of risk aversion in the T2DM group. Future research could try to avoid these problems; e.g. by eliciting respondents' reference points before starting the main experiment and subsequently using these individual-specific RP to elicit risk preferences.

c Strengths and limitations

The data in this study were collected following procedures intended to ensure high quality. Firstly, the questionnaire was developed in collaboration with both health professionals and patients in order to be as user-friendly as possible. I used visual aids that facilitated participants' understanding. Secondly, I was continually present as a support for the participant, throughout the questionnaire. I made sure that both the instructions and the questions were well understood. This was a key aspect of the procedure: no participant was asked to read the instructions or answer the questions on their own. The written instructions were accompanied by clear, additional verbal explanations. This translated into a large majority of participants providing consistent answers for at least four out of five CEs (92%). Despite a higher degree of complexity in the design of the prospects, the reliability of the procedure compares to those of earlier studies [87, 119, 140]

On the other hand, the sample size is small, which limits the data analysis. Because T2DM usually arises in the early 60s, a major difficulty of the study was to recruit patients with T2DM aged 60 or less, and even more challenging, aged 50 or

less. This constraint should be taken into account when looking at the final sample size of the T2DM group (n=52). I tried to ensure that controls were matched by age, while also trying to minimise the risk of selection bias by recruiting these participants randomly at the same surgery practice. Statistical tests showed no between-group differences in terms of gender, employment status, education level, or answer reliability. Only a slight difference in terms of age (the median age of the T2DM group was higher) was found after exclusion of individuals who provided more than one inconsistent answer. Individuals who were not fully consistent in their answers (n=30) turned out to be heterogeneous in terms of demographic and socio-economic characteristics, which minimizes the risk of selection bias and supports the elicitation procedure. However, significantly more individuals from the T2DM group had to be excluded from the analysis than from the control group (8 participants vs 2 participants). Although caution is required in the interpretation of this difference, it may be explained by underlying differences in terms of cognitive abilities between people with T2DM and controls. Recent studies have shown that diabetes impose a chronic negative effect on cognitive and decision-making skills. [141–143] This should be borne in mind when designing future studies involving cognitive tasks with T2DM populations.

Last, it is important to bear in mind other limitations inherent in the elicitation method. Firstly, I designed the prospects using a broad notion of health-related QoL, selecting three scores supposed to characterise three QoL states. These scores were the result of a trade-off between four criteria, including ease of understanding and realism. Despite my efforts to minimise rating biases, I could not completely rule out individual variations in the way these scores were perceived. For example, a QoL score of 100% is likely to be perceived differently according to whether a person is 30 or 60 years old. One way of dealing with this issue would be to elicit each individual's own QoL score before running the experiment, and then use these personalised scores in the prospects. Such an approach would, however, increase both the length and the complexity of the experiment.

Secondly, using only one single probability (p=0.5) in the prospects prevents

conclusions from being drawn about the decision weights attached to other probabilities. In particular, as mentioned earlier in this chapter, the tendency of people to overweight low probabilities and underweight large probabilities has been reported in the literature, including in the health domain (see 3.3.1). At first sight, my results undermine the validity of PT in this context because they challenge the traditional S-shaped curve of the PT value function (suggesting that the role of the RP may be less crucial for health than for money). However, these results must be put into perspective as it seems that PT does not mandatorily exclude (mild) risk aversion in the loss domain over mid-range probabilities. Over mid-range probabilities, it is not clear whether PT predicts people to be risk seeking for losses (as over large probabilities) or risk averse for losses (as over small probabilities). It is also possible, as suggested by Oliver (2018), that utilities elicited through binary choice tasks are more *reasonably consistent* with EUT than utilities elicited through pricing tasks. [126] However, because of robust loss aversion found in this study[58], I would not go so far as to say that EUT fits my results better than PT.

Thirdly, since separating the two attributes (quality of life and life duration) and assessing their separate utility functions would have caused a substantially higher response burden, I analysed utilities of QALYs. That is, I assumed that the risk attitude for quality of life was not systematically different from the risk attitude for life years. This is in contrast to the findings of van der Pol and Ruggeri (2008), who reported risk seeking for quality of life alongside risk aversion for life years. [145] However, the evidence with respect to risk attitudes regarding life years and quality of life is mixed, with other evidence suggesting risk aversion over quality of life. [86] Furthermore, I expressed quality of life in percentages whereas van der Pol and Ruggeri (2008) used EQ-5D classifications, and my gambles did not include immediate death as opposed to the lifetime gambles of van der Pol and Ruggeri (2008). It is therefore not clear that their results also apply to my setting.

[58] A recent study by Lipman *et al.* (2018) tends to show that loss aversion is stable across health states. [144]

d Implications of the results

In the health domain, risky decisions made by people living with a chronic disease may greatly impact their life expectancy and QoL. Evidence of risk aversion found in this study suggests that risk seeking behaviour may not explain lack of adherence to self-management observed in T2DM populations. Although there is no data allowing me to link the risk attitudes measured in this study with objective measures of patients' self-management, participants preferred choosing the safer option when facing scenarios that depict the risks inherent to each behavioural option. In other words, when they can clearly visualize (and thus be fully aware of) the risk associated with non-adherence, people seem averse to it and prefer adhering to self-management. These results suggest that in real life non-adherence to self-management may be explained, at least partly, by an underestimation of the risks associated with risky behaviour. In Chapter 2, it has been shown that T2DM patients largely underestimate the risks of diabetes-related complications. One may argue that, regardless of their level of risk perceptions, if these patients are risk seeking towards behavioural choices associated with the management of their condition, higher risk perceptions would not increase adherence to self-management. This study shows that this is not the case, i.e. that if patients had a higher awareness of the risks associated with non-adherence to self-management, they may change their behaviour. This does not necessarily contradict Simon-Tuval *et al.* (2016) who found an association between risk-seeking behaviour for money and non-adherence to T2DM self-management behaviour. [146] Putting aside the differences between utility for money and utility for health, it is possible that risk seeking patients (both towards money and health) are also among the ones who are the least adherent to self-management. However, this study shows that such a behaviour is uncommon when risky choices are specific to T2DM self-management and hence may not be relevant to explain the high rates of non-adherence observed in practice. Further research to link elicited risk preferences for health outcomes with objective measures of adherence to self-management behaviour would be interesting.

Finally, the age-related difference in risk attitudes found in this study suggests

that a different approach is needed for younger T2DM patients. Because of lower risk aversion, such interventions are likely to be less effective in this population. Efforts to develop alternative strategies should be of higher priority, as worrying differences between younger and older T2DM patients have been shown in terms of glycaemic control [147] and onset of complications. [148, 149] This is partly due to lower adherence to self-management [150] (that may be caused by lower risk aversion and present preferences) and partly due to younger-onset T2DM which is more aggressive by nature and thus more challenging to control. [148, 149]

3.4 Conclusion

In this chapter, I have gone further in the exploration of the underlying mechanisms that govern patients' decision-making processes. In line with the strategy defined in Chapter 1, I aimed to identify relevant behavioural economic material that will help better targeted interventions to be designed.

A review of the decision theory literature identified PT as the optimal theory to assess risk attitudes in the health domain. Besides, a range of factors that are known to play a role in how patients make decisions has been uncovered from the behavioural economic literature. As three of these factors are embedded into PT (reference-point dependence, probability weighting and loss aversion), measuring risk attitudes under PT allowed me to kill two birds with one stone, i.e. to also assess these factors in my population of interest.

Focusing on a hypothetical trade-off between health outcomes and self-management, I found that patients express strong risk and loss aversion. This suggests that non-adherence to self-management may not be explained by a particular risk-seeking behaviour in this population. I also found a significant age effect on risk attitudes, with young people being more risk seeking than older people on average.

These results are a crucial first step towards the development of a nudge-based intervention tailored to people with T2DM. Including the optimistic bias identified in Chapter 2, three factors underlying the decision-making processes of people with

T2DM have been evidenced in the first part of this Thesis.[59]

The second part of this Thesis will be dedicated to the development of a nudge-based risk communication intervention targeting the relevant factors identified in this chapter. A direct implication of the findings of this first part is that they support the design of interventions that raise awareness of diabetes-related health risks and take advantage of people's loss aversion. New risk communication strategies that make the risk look more concrete and proximal are a good example of how these two levers for action could be pulled.

Further research investigating risk attitudes of people living with different types of chronic disease should be encouraged, so that future health promotion interventions can be systematically tailored to specific patient populations.

[59]Out of seven behavioural economic phenomena identified as relevant to inform a tailored nudge-based intervention (see Table 3.3), and considering probability weighting as being negligible given he results of the study. More evidence had already been gathered about the three other factors in patient populations, including patients with chronic diseases (see Chapter 4).

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4

Development of a nudge-based risk communication tool

4.1 Introduction

The first part of this Thesis was concerned with exploring the factors that can influence Type-2 diabetes mellitus (T2DM) patients' decision to adhere to self-management behaviour. I found that people with T2DM have biased perceptions of their risks for diabetes-related complications, characterised by low risk awareness and contaminated by optimistic bias[1] (see Chapter 2). I also found that they express some systematic decision-making biases (deviations from standard economic rationality) when facing a risky trade-off between self-management and health outcomes: reference-point dependence[2] and loss aversion[3] (see Chapter 3).

These findings shed light on a significant gap between a normative ideal of decision-making (optimal decisions recommended by physicians) and the descriptive reality (actual decisions made by patients) regarding self-management of T2DM. Based on this observation, the objective of the second part of the Thesis is to contribute to the reduction of this gap, so that sustained adherence to self-management can be improved. In accordance with the strategy described in Chapter 1, I aim, in this chapter, to develop a nudge-based risk communication tool informed by the findings of Chapters 2 and 3. This has, to my knwoledge, never been done in the T2DM area.

I start the chapter with the presentation of this innovative risk communication tool, entitled PERCODIA[4] (section 4.2). Then, after a brief overview of the objectives and key concepts in health risk communication (section 4.3), I explain how the development of the tool results from the selection of appropriate elements of content (section 4.4) and format (section 4.5). These elements were chosen in order to best address the decision-making gap between normative ideal and descriptive reality observed in the first part of the Thesis.

[1] The tendency of patients to feel less at risk than their peers.

[2] People react to a particular choice in different ways depending on how it is presented.

[3] The pain of losing a given outcome is psychologically more powerful than the pleasure of gaining this outcome.

[4] PERsonlised Risk COmmunication for people with Type 2 DIAbetes.

4.2 A nudge-based risk communication tool named PERCODIA

PERCODIA was developed to communicate personalised risk information to people with T2DM, in line with the nudge strategy drawn out earlier in the Thesis (see Chapter 1). In this section, I introduce PERCODIA to the reader. I summarise the features that were selected to design it, i.e. its *content* and its *format*, and describe its development process. I also provide a user guide for PERCODIA, which includes illustrative screenshots and examples.

4.2.1 Content and format features

PERCODIA calculates and displays two types of risk estimates: the risk for cardiovascular (CV) event (largely underestimated in T2DM populations; see Chapter 2) and life expectancy. These estimates are calculated based on the equations of the UK Prospective Diabetes Study - Outcomes Model (UKPDS-OM), the validated prognosis model of reference in the T2DM area (see section 4.4). Moreover, PERCODIA was developed for use in routine primary care consultation, primary care being the key contact point with the healthcare system for people with T2DM.

Importantly, PERCODIA is a behavioural economic-based risk communication tool. In that, it differs from traditional risk communication interventions used to motivate sustained adherence to self-management in T2DM populations (see 4.3.2). When selecting its content and format features (see sections 4.4 and 4.5, respectively, for more detail), the objective was to target as many levers for action as possible to steer patients' choices towards adherence to self-management. Table 4.1 provides a summary of these features and link them to the lever they are aimed to target. In particular, PERCODIA is designed to take advantage of the relevant behavioural economic material identified earlier in the Thesis (see Table 3.3 in Chapter 3):

- In order to mitigate people's *optimistic bias* (the tendency that people have to underestimate their risks of developing health problems, compared with

their peers [1]), all risks are communicated in comparison to those of a similar, well-controlled individual (i.e. same age, sex, and medical history, but with all risk factor levels in normal ranges).

- In order to take advantage of people's *loss aversion* ("the pain of losing a given outcome is psychologically more powerful than the pleasure of gaining the same outcome", including in the health domain [2]) and mitigate their *present biasedness* [3], the impact of being poorly controlled on life expectancy is communicated with an innovative metric: the *hours of lifetime lost per day*, in comparison with a similar, well-controlled individual. [4]

- In an effort to increase the emotional impact and representativeness of the risk (i.e. target people's *affect heuristic* and *representativeness heuristic* [5], respectively), the 10-year CV risk was also expressed through a new metric: the effective heart age. [6, 7] A subject's effective heart age was calculated as the chronological age of a similar, well-controlled individual with the same predicted risk score. [8]

- In order to increase people's limited attention (i.e. target *availability heuristic* [5]), PERCODIA is interactive by showing how better adherence to self-management could lower the risks (see 4.2.3).

- Because people are more risk averse in the gain domain (*reference-point dependence*, or sensitivity to framing effects), variations in personalised risks are expressed using a (negatively) gain-framed message ("you would avoid losing X hours of lifetime per day if you manage to lower your HbA1c value to Y").

Table 4.1: PERCODIA: content and format features.

Objective	Content features	Format features	Lever for action targeted
Communicating personalised CV risk associated with self-management behaviour	Absolute 10-year risk of CV event (associated with current behaviour)	Coloured, simple bar graphs with numerical risk information (percentage)	Numeracy Affect heuristic
		Short summary statement with both numerical and verbal risk information	Numeracy
	Comparative 10-year risk of CV event (associated with recommended behaviour)[1]	Coloured, simple bar graphs with numerical risk information (percentage)	Numeracy Affect heuristic Optimism
		Short summary statement with both numerical and verbal risk information	Numeracy Optimism
	Heart age (associated with current behaviour)[2]	Coloured heart with numerical risk information and short summary statement (heart age)	Numeracy Heuristics (availability, affect, representativeness) Optimism
	Change in heart age (associated with variations of modifiable risk factors)[3]	Interactive statement inviting the patient to see how his/her heart age can change by varying four modifiable risk factors: smoking, HbA1c, SBP, LDL	Numeracy Heuristics (availability, affect, representativeness) Bounded willpower Loss aversion
Communicating personalised change in life expectancy associated with self-management behaviour	Loss of life expectancy as compared to a perfectly controlled individual (associated with current behaviour)	Clock with numerical risk information and short summary statement (daily hours of life lost per day)	Numeracy Heuristics (availability, affect, representativeness) Optimism

Table 4.1 Continued:PERCODIA: content and format features.

Gain or loss of life expectancy (associated with variations of modifiable risk factors)	Interactive statement inviting the patient to see how he/she can gain/lose daily hours of life by varying four modifiable risk factors: smoking, HbA1c, SBP, LDL	Numeracy Heuristics (availability, affect, representativeness) Bounded willpower Loss aversion Framing

[1]Risk of a similar individual who would be perfectly controlled, i.e. with the same characteristics and history of events, but with his/her modifiable risk factors within normal value ranges.

[2]A patient's heart age is calculated as the age of a well-controlled patient (i.e. with all risk factors in normal ranges) with the same predicted risk.

[3]Similar to the New Zealander *Your Heart Forecast* project [7], the tool offers the possibility to see how variations in modifiable risk factors impact an individual's heart age. By setting up personalised objectives that may differ from one patient to another (e.g. reducing HbA1c level from 8.0 to 7.0 given all other risk factors remain constant by the next visit), the user can see personalised variations in his/her heart age.

4.2.2 Development process

PERCODIA was developed in collaboration with a multidisciplinary team interested in promoting diabetes self-management: a general practitioner (GP) running a diabetic clinic in Oxford and conducting research activities in the area of T2DM and self-management, two patients with T2DM who volunteered to give feedback on the tool (recruited through the PPI[5] programme of the NIHR), and two programmers specialised in digital health at the Oxford Institute of Biomedical Engineering.

a Content features

I selected the content features of PERCODIA (CV risk and life expectancy) based on the review of the risk communication literature and the findings of the first part of the Thesis. These options were validated by the GP, based on his experience in following and communicating with T2DM patients in the long run.

b PERCODIA algorithm

Because the original UKPDS-OM lacks tractability and cannot be used in routine primary care consultation, I built simpler forecasting models (the PERCODIA algorithm) to allow rapid calculation of the risk information of interest (see 4.4 for more detail).

c Format features

I selected the format features of PERCODIA based on the review of the risk communication literature and the findings of the first part of the Thesis. Feedback on readability and user-friendliness was obtained from both the GP and the two volunteer patients.[6] Emphasis was given to the association between *change* in self-management behaviour[7] and *change* in health risks. Patients not only need to know how to interpret the information about a specific health risk, but must

[5]Patient and Public Involvement.

[6]No formal focus group was run during the development process. It was judged that Chapter 5's study would provide sufficient feedback in terms of user-friendliness and comprehensibility from both the patient and the doctor side.

[7]Proxied by change in modifiable risk factors.

also understand how their behaviour can affect this risk.[8] [9] To emphasise this link, PERCODIA was designed to show people with T2DM how variations in their modifiable risk factors can impact their risk.

d Programming

PERCODIA was coded by the two programmers specialised in digital health, based on the algorithm and format features previously selected. Considering the time constraints inherent to the use of PERCODIA in primary care consultation (5 minutes intervention) and the technical constraints associated with its programming, trade-offs had to be made in terms of formatting. For example, some graphical and interactive features were too time-consuming in terms of programming to be implemented at such an early development stage. Such features will be added to PERCODIA in the future, provided that its piloting in a real life setting shows positive results (see Chapter 5).

4.2.3 Use in practice

PERCODIA was designed for use in primary care consultation, as part of doctor-patient risk communication. Patients are not expected to go through the tool on their own. As recommended by several authors, risk calculators should be used in conjunction with health professionals who can guide the user through the calculator and help them use the resulting risk information as a source of motivation and encouragement. [10, 11] Although PERCODIA provides personalised risk estimates through formats intended to be easy to understand, the face-to-face interaction with a trusted source of information is necessary to impart meaning to the data. Hence, the use of PERCODIA must always be accompanied by personalised advice and strategies about how to improve self-management behaviour.

PERCODIA's user guide[9], including illustrative screenshots and examples, is

[8] The use of PERCODIA must lead people with T2DM to believe that they are at risk of developing complications, that the risk is serious, and that the risk can be reduced or prevented if they improve their self-management behaviour.

[9] A manual for user of the version used to conduct the pilot study in Chapter 5.

provided in Appendix H. No specific computer skills are required to use it.

4.3 Health risk communication

4.3.1 Objectives and key elements

If multiple definitions of risk communication can be found in the medical literature, the one by Edwards *et al.* (2002) seems to attain consensus. [12] The authors define risk communication as "the open two-way exchange of information and opinion about risk, leading to better understanding and better decisions about clinical management". One key aspect of this definition is that it moves away from the preconceived idea that information is communicated only from clinician to patient. [12] In the context of chronic disease management, with a focus on T2DM, Asimakopoulou (2008)[10] also provides a relevant definition: "risk communication is the social process of informing people about hazards, with the aim of enabling them to make decisions about risk issues, and influencing them towards behavioural change." [14]

Although these definitions may vary, several key aspects of health risk communication are common to all of them. One, in particular, is crucial in order to develop effective interventions: what is the objective of the risk communication.[11] [9, 15–17] In the literature, four objectives emerge for the communication of health risks: (a) Informing about treatment options; (b) Improving understanding about illness or treatment options; (c) Raising awareness about health risks; (d) Motivating behaviour change. These objectives are not mutually exclusive, and one might encompass another. For example, as discussed in Chapter 2, perceived risks are a significant predictor of intentions to change behaviour [18, 19] and behaviour change [20–22], meaning that improving the accuracy of risk perceptions may also favour behaviour change.

[10] Inspired by the work of Rohrmann (2000). [13]

[11] Some authors regret the lack of clear objectives in risk communication (see e.g. Waldron *et al.*, 2011 [15]).

Having a clear objective is essential to identify an appropriate assessment method. Despite a lack of guidelines on how to evaluate the efficacy of risk communication interventions [16], assessment criteria should depend on the defined objective. When the objective is to motivate behaviour change, a risk communication will be judged effective if it leads to an improvement in health-protective behaviour, regardless of any improvement in risk perception accuracy. Conversely, an intervention will be judged ineffective (or even detrimental) if it fails to trigger behaviour change (or causes the person to act inappropriately). [9]

In this chapter, I aim to develop a risk communication intervention that increases risk perceptions and motivates behaviour change. In order to achieve the defined objectives, two elements must be considered. First, the *content* of the risk message, i.e. the nature of the information to be communicated. A large body of evidence suggests that providing personalised risk information is more effective than generic information drawn from average population data to reach either of these objectives. A systematic review by Edwards *et al.* (2000) showed that the use of individualised risk estimates in risk communication was a key *effect modifier*, i.e. a characteristic of the most effective interventions (97 studies included). [23] Moreover, a Cochrane review of 22 randomised controlled trials (RCT) showed that personalised information, as compared to generic information (whether written, spoken, or visually presented), led to more accurate risk perceptions, improved knowledge, and increased uptake of screening tests. [24] Studies published after these reviews have confirmed this effect. [25–28] Some research suggests that personally relevant risk estimates increase people's attention and motivate more systematic information processing (see also section 4.4). [29]

However, if the use of individualised information is a prerequisite to help induce behaviour change, providing risk probabilities of health events alone is not enough to induce a sustainable, long-term effect. [9, 30, 31] Powers *et al.* (2011), who conducted a RCT to compare personalised risk communication based on Framingham stroke and coronary heart disease risk scores to standard risk factor education, reported no significant between-group differences in terms of medication

adherence, exercise, smoking cessation, or blood pressure. [30] In fact, the risk message should also include information on the causes of the risk, its consequences, and what can be done to reduce it. [9, 32–35] Providing a context within which the risk probability can be interpreted, for example explaining the link between a given health event and a person's behaviour or medical history, is thought to help the person relate to that risk and encourage behaviour change. [17, 35–37] Specifically, highlighting the consequences of the risk helps people recognise *what* could happen to them, whereas highlighting the causes of the risk helps them understand *how* it could happen. [9]

The second key element to consider is the *format* of the risk message, i.e. the visual features used to communicate the information. It has been shown that features motivating behaviour change may differ from those improving quantitative reasoning, while features liked by viewers may not support either of these goals. [38] Section 4.5 of this chapter explores the format features that may help increase risk perceptions and/or motivate behaviour change in people with T2DM by targeting the relevant behavioural economic findings identified in Chapters 2 and 3.

4.3.2 Effectiveness of risk communication interventions on risk perceptions and adherence to self-care behaviour: what is the evidence?

In this subsection, I provide an overview of the effectiveness of existing risk communication interventions on risk perceptions and adherence to self-care behaviour[12], both in T2DM (see Table 4.2) and broader patient populations, where relevant.[13] None of these interventions have been claimed to be informed by behavioural economics. Although it is true that most of them are not, it turns out that some of them, as we will see, do rely on behavioural economic insights, despite their authors not realising it (or not acknowledging it).

[12]Measured by change in specific self-care behaviour or change in specific risk factor.

[13]Although I did not perform a systematic review, I am confident that no major intervention conducted over the past 30 years in T2DM populations has been forgotten. Most studies have been identified from the recent reviews published by Harris *et al.* (2017) [11] and Usher-Smith *et al.* (2015) [39], as well as from the work of Welschen *et al.* (2012) [40] and Roach and Marrero (2005). [35]

a In T2DM populations

With regard to T2DM, there is surprisingly little evidence of the impact of risk communication given the importance of self-management in the treatment of the condition. Existing interventions have shown mixed results, with many participants barely understanding the explanations of health professionals about risks and having poor recall of risk information. [40–43] One high-quality RCT investigating the effects of CVD risk communication showed that the intervention (a thorough six-step procedure[14]) improved the accuracy of risk perceptions after two weeks, but its effect was no longer observable after 12 weeks. No effect was found on intentions to change behaviour. [40] Another study by Hoffmann and Del Mar (2012) reported that a one-day diabetes educational expo did not prevent people from underestimating their risk of eye complications. [45] And when Saver and colleagues asked people to rank complications by frequency of occurrence before and after they were provided with personalised risk estimates, they found that this intervention had no impact on risk perceptions. [41]

Conversely, two studies found a significant impact on risk perceptions post intervention. Using short time frames to communicate risks of CHD and stroke, Asimakopoulou *et al.* (2008) successfully improved the accuracy of perceived risks in their population after six weeks.[15] [46] Last, a recent RCT conducted in Egypt utilising an intervention similar to Welschen *et al.* (2012) [40] showed an improved accuracy of CVD risk perceptions after 12 weeks. [47] The authors also reported significant improvements in diabetes self-care activities and HbA1c levels, for both within- and between-group (control) comparisons.[16] However, the participants in this trial were provided with repeated communications (at baseline, after 4 weeks and after 8 weeks). In light of these findings, it appears that personalised risk

[14] The intervention consisted of the six following steps: 1) Introduction of the risk communication; 2) Communication of the absolute risk according to the UKPDS risk engine; 3) Visual communication by means of a risk card; 4) Positive message framing; 5) Communication with the patient for a reaction; 6) Thinking aloud. See Welschen *et al.* (2010) for more details. [44]

[15] Using shorter time frames to communicate the risk allows mitigating the effect of present biasedness.

[16] To my knowledge, this is the only study that investigated the effect of personalised risk communication on behaviour change in a T2DM population.

communication interventions for people with T2DM are not yet fully satisfactory. In particular, no effect lasting longer than six weeks has been shown.

b In broader populations

Studies including broader populations generally confirm the need for better risk communication interventions. On the one hand, several authors reported no or only marginal effects on risk perceptions or self-care behaviours. Price *et al.* (2011) assessed whether providing a personalised 10-year CVD risk estimate could increase physical activity in adults at high risk of CVD (19% of whom had diabetes) and found no evidence of behaviour change or positive impact on estimated CVD risk over a one-month period. There were no within- or between-group differences in intentions to increase physical activity either. [48] Powers *et al.* (2011), investigating the effects of both a standard and a personalised risk communication intervention for people with high risk of vascular events, found that the accuracy of CHD and stroke perceptions did not improve immediately or three months after the intervention. There was also no significant between-group difference for risk reduction behaviour or estimated vascular risk. [30] A recent study assessing the effect of a personalised intervention on the accuracy of perceptions for diabetes risk in the general population only showed a modest improvement after eight weeks.[17] [49] Similarly, van der Weijden *et al.* (2007) observed a mismatch between perceived and actual CVD risks after doctor-patient communication in primary care. Incorrect perceptions among people with diabetes were striking. [50] Moreover, a systematic review investigating the effect of providing CHD risk information for adults without a history of CHD only showed a modest improvement in the accuracy of risk perceptions. The authors mentioned a possible increase in the intention to initiate prevention strategies, but there was insufficient evidence to support this conclusion with certainty. [51] Last, a RCT by Hanlon *et al.* (1995) reported that giving feedback on personalised cardiac risk score (Dundee risk score) had no effect on self-care behaviours or risk factors. [52]

[17] The proportion of people having accurate risk perceptions went from 1.3% to 7.3%.

Table 4.2: Impact of risk communication interventions in T2DM populations.

Study	Design	Intervention	Control	Follow-up	Outcomes	Results
Asimakopoulou *et al.* (2008) [53]	Before and after study	N=95 patients with T2DM; Verbal and visual communication of CVD risks calculated by the UKPDS-OM, using different time frames.	N/A	6 weeks	Risk perceptions (absolute accuracy[1])	Significant improvement
Hoffmann and Del Mar (2012) [45]	Before and after study	N=91 patients with T2DM; Educational lectures including verbal communication of the average absolute 15-year risk of eye complication.	N/A	N/A	Risk perceptions (absolute accuracy[1])	Not significant
Saver *et al.* (2014) [41]	Before and after study	N=56 patients with T2DM; Verbal and visual communication of risks for 6 diabetes-related complication calculated by the UKPDS-OM.	N/A	N/A	Risk perceptions (ranking of 6 complications by likelihood of occurrence)	Not significant
Tawfik and Mohamed (2016) [47]	RCT	N=107 patients with T2DM; Thorough three-step procedure, involving verbal and visual communication of CV risk scores predicted by the WHO/ISH prediction chart. [54]	N=107 patients with T2DM; Usual care	12 weeks	Risk perceptions (absolute accuracy[1]) Behaviour change: (a) Self-care activities (b) HbA1c levels (c) CV risk score	Significant improvement (a) Significant improvement (b) Significant improvement (c) Not significant

Table 4.2 Continued: Impact of risk communication interventions in T2DM populations.

Welschen *et al.* (2012) [40]	RCT	N=131 patients with T2DM; Thorough six-step procedure, involving verbal and visual communication of 10-year CVD risks calculated by the UKPDS risk engine.	N=130 patients with T2DM; Usual care	12 weeks	Risk perceptions (absolute accuracy[1]) Intentions to change behaviour	Significant improvement at 2 weeks, but not at 12 weeks Significant

Note: CVD = cardiovascular disease; CV = cardiovascular; UKPDS = UK Prospective Diabetes Study; UKPDS-OM = UK Prospective Diabetes Study - Outcomes Model (UKPDS 68) [55]; WHO/ISH = World Health Organization/International Society of Hypertension

[1]Difference between perceived and actual absolute risks (see Chapter 2).

On the other hand, a few interventions have shown promising results. Fowler *et al.* (2017), assessing the impact of a breast cancer risk assessment tool in a population of women with no cancer history, reported beneficial effects in terms of accuracy of perceived risks and behavioural intentions (four-week follow-up). [25] In a large-scale trial, Grover *et al.* (2007, 2009) found that discussing coronary risk with patients was associated with a small but measurable improvement in the efficacy of lipid therapy [56] and with more appropriate blood pressure management. [57] Two other studies reported improvements in the serum lipid profile and/or the overall coronary risk score of high-risk patients after a personalised intervention involving written supports (one-page printout [58] or poster [59]). In patients with high risk of developing CVD, two RCTs reported contradictory results. Benner *et al.* (2008) found that providing CVD risk estimates led to behaviour change and risk reduction (after six months) [60], while Koelewijn-van Loon *et al.* (2010) did not observe any lifestyle change (after 12 weeks). [61] However, in these studies, risk communication was part of a larger intervention study, making it difficult to untangle the effects of risk communication only. The same conclusion can be drawn from a study by Engberg *et al.* (2002), which showed reductions in CHD risk, BMI, and cholesterol levels after five years in people who received CHD risk appraisal as part of a multiphase, broad-spectrum screening. [62] Two additional studies reported a beneficial effect on risk perceptions after using a risk assessment tool for cardiovascular (CV) risk, but in these studies perceived risks were only measured immediately after the intervention. [63, 64] Finally, two RCTs involving innovative tools to communicate personalised risks showed promising results. Lee et (2011) managed to increase intentions to change behaviour and healthy diet efforts after two weeks in a population of sedentary young adults by using personalised cardiac animations [65], while Shahab *et al.* (2007) reported significant improvements in risk perceptions, intentions to stop smoking and smoking cessation behaviours four weeks after presenting smokers with ultrasound images of their own damaged arteries.[18] [66]

[18]Such interventions, by being interactive and by making the risk look more concrete and impactful, rely on people's heuristics to motivate behaviour change.

In this context, it is reasonable to assert that risk communication interventions can be improved. To date, in the diabetes area, the most effective communication intervention lasts no longer than a few weeks. [40] An innovative, more effective, and tractable way of communicating risks to people with T2DM is needed. Efforts must be made to develop a short, easy-to-implement intervention for use in routine primary care consultation. In the next two sections, I discuss the development of a nudge-based risk communication tool for this purpose.

4.4 Tool design (1): content

Despite numerous calls in the literature, the communication of personalised risks for diabetes-related complications to patients with T2DM is not common practice. [23, 24, 46] In this chapter, the objective is to develop a tool allowing real-time risk communication in routine primary care consultation. This section focuses on the content of the tool, i.e. *what* is going to be communicated to people with T2DM. Based on the predictions of a risk calculator of reference, I select the risk estimates of interest and develop a method to calculate them, within a limited amount of time, during primary care visits.

4.4.1 The UKPDS outcomes model

In health risk communication, personalised risk estimates are calculated via computer simulation models that provide a summary statistic of an individual's chances of experiencing a health event over a certain time frame (i.e. a single-event probability). As Lipkus (2007) reminds, a critical assumption of these models is that they rely on data measured on a specific population (collected from clinical trials or epidemiological studies). This means that the characteristics of an individual whose risk is estimated must be represented in this population.[19] [16] As a consequence,

[19]With rare exceptions (e.g. positive genetic test for the diagnosis of Huntington's disease, due to the nature of the condition), it is impossible to state an individual's risk with certainty. As a consequence, when talking about diabetes-related complications, the term *personalised risk estimate* might be incorrect if misinterpreted. [16]

risk calculators must be chosen according to how well the individuals from the original study reflect the targeted study population. [67] Moreover, as each calculator often has its own definition of the risk being considered, one must be careful when interpreting two different risk estimates calculated by two different calculators.[20] [68]

In the area of T2DM, recommended simulation models to forecast the risk of diabetes-related complications are generally based on data from the UK Prospective Diabetes Study (UKPDS).[21] [72] First, the UKPDS risk engine was designed for clinical use, to help physicians and patients decide on the most appropriate management of T2DM. [73, 74] It allows estimating the risk of a first coronary heart disease (CHD) event or stroke, over a defined period (usually 10 years), for a person recently diagnosed with T2DM without CHD or stroke history. Studies that have assessed the validity of the UKPDS risk engine found that it was more reliable [75, 76] or at least as reliable as other CV risk calculators. [77–80]

Subsequent to the development of the UKPDS risk engine, the Diabetes Trials Unit (DTU) and the Health Economics Research Centre (HERC) at the University of Oxford developed the UKPDS-OM (version 1 [55] and 2 [81]).[22] This model allows estimation of a wider variety of outputs, including life expectancy and quality adjusted life expectancy. It uses considerably more input data (including the history of previous events) and accounts for updated risk factor data from the post-trial monitoring phase of the UKPDS. Importantly, the UKPDS-OM can calculate personalised risk estimates for a wide range of T2DM-related complications. It has been shown to be internally valid and closely match the outcomes observed in the UKPDS trial for CV complications and all-cause mortality from diagnosis of diabetes to 12 years of follow-up. [55] External validity studies have also reported a

[20] For example, the broader the definition of a cardiovascular event, the more likely an individual will be to have one.

[21] The UKPDS involved 5,102 patients with newly diagnosed T2DM recruited between 1977 and 1991 and randomised to different blood glucose and blood pressure control regimens. [69, 70] Additional data were collected up to 10 years after the end of the initial study (post-trial monitoring period). [71]

[22] Version 2 is based on longer follow-up data. See also the UKPDS-OM (version 2) user manual. [82]

good predictive accuracy beyond the data used to estimate the model. [83–85]

So far, outcomes predicted by the UKPDS-OM have often been used to inform health economic evaluations or life expectancy tables [86], but not to directly inform patients through risk communication interventions. Despite offering less variety in its outcomes, the UKPDS risk engine was generally chosen to inform such interventions. In the next subsection, I focus on the UKPDS-OM to select the risk information that will inform the nudge-based communication tool.

4.4.2 Risk information to be communicated

The UKPDS-OM allows prediction of an individual's probability of first occurrence of eight complications: myocardial infarctions (MI), other ischemic heart disease (IHD), heart failure (HF), stroke, blindness, renal failure, amputation, and ulcer. Other outcomes forecasted by the model are the probability of death (all causes) and CVD-related death, life expectancies, quality adjusted life expectancies, and life-time health care costs. Inputs needed to calculate the outcomes are a combination of the individual's characteristics (ethnicity, sex, age, duration of diabetes, weight, height) and risk factors: smoking status, long-term blood glucose level (HbA1c), systolic blood pressure (SBP), low- and high-density lipoprotein cholesterol levels (LDL and HDL), atrial fibrillation (AF), peripheral vascular disease (PVD), albuminuria, estimated glomerular filtration rate (eGFR), haemoglobin, heart rate, white blood cell count (WBC), and history of the eight complications mentioned above.

Running the model requires setting the initial values for all these characteristics and risk factors, as well as defining the time period for event calculation and other calculation parameters (see UKPDS-OM user manual for more details [82]). This creates two challenges for its use in primary care consultation. First, the process is too time consuming to use in real-time with patients[23], because the current version of the model does not allow the user to restrict the number of input or output values. Second, not all the outcomes forecasted by the model are meant to be communicated to patients. Indeed, to attempt to do so would run counter to the

[23]Putting aside the fact that some of these patients' characteristics and/or risk factors might not be known by GPs at time of the consultation.

idea that risk messages must stay brief and simple. [87, 88] Offering a few, simple pieces of information is preferable to giving patients a mass of detailed, complex information (see 4.5 for more details). As a result, selecting the risk information of interest and developing a method to calculate this information within a limited amount of time is a prerequisite to the use of the tool.

a Risk for CV event

The systematic review conducted in Chapter 2 shed light on the underestimation of CV risks in T2DM populations. Although risks for microvascular complications (e.g. eye disease) are usually accurately estimated, patients tend to highly underestimate risks for CHD and stroke. [89] It is necessary to correct for these misperceptions, as CV events account for most complications experienced by people with T2DM (see Chapters 1 and 2). Following on previous studies investigating CV risk perceptions in T2DM populations [46, 90], and in line with the strategy of keeping the message as simple as possible, I decided to communicate the risks for MI and stroke. The time frame chosen to express the risk is 10 years (see 4.5).

b Life expectancy

A major asset of the UKPDS-OM is that it produces forecasts of life expectancy of individuals diagnosed with T2DM. Providing life time estimates in relation to different health behaviours is a promising way to communicate the consequences of risky behaviours (see refsection:4.5 for more details). [4, 91] Because this approach has never been used as an intervention with T2DM populations, I decided to include life expectancy as a second type of outcome in the content of the risk communication tool.

4.4.3 Development of two models to forecast the selected UKPDS-OM risk information in a limited amount of time

As discussed above, the UKPDS-OM lacks tractability for use in routine primary care consultation. This is mostly due to the inability to parameterise the model so

that only a restricted number of outcomes is calculated. To remedy this problem, I develop two simpler forecasting models that permit rapid calculation of the risk information of interest (10-year CV risk and life expectancy). This is the approach recommended by Royston *et al.* (2009).[24] [92]

Although there is no widely agreed approach to building a multivariable prognostic model from a set of candidate predictors[25], I followed the recommendations of Royston and colleagues by going through a five-step process: 1) Selecting clinically relevant candidate predictors for possible inclusion in the model; 2) Handling data related to pre-selected predictors[26]; 3) Selecting the predictors to be included in the final model by following a backward elimination approach[27]; 4) Modelling continuous predictors[28]; 5) Assessing the model's performance (predictive accuracy). See Royston *et al.* (2009) for further details. [92] The two forecasting models resulting from this process are displayed in Table 4.3 (Model 1: 10-year CV risk) and Table 4.4 (Model 2: life expectancy). Further details are also provided in Appendix I.

Data used to estimate these two models were obtained by running the UKPDS-OM on a dataset of (artificial) individuals whose continuous predictors had been selected to cover a wide range of values that can be found in T2DM populations (see Table 4.5). Variables required by the UKPDS-OM but not included in the models were held constant, within normal biological limits.[29] Due to the high number of individuals used to cover all value ranges (n=1,920,000), models for men and women had to be run separately with Stata software package. See Appendix J for further details.

As the Akaike information criterion (AIC) used to select Model 1 was not

[24] "[Predictive] models may be a complicated function of the predictors, as in weather forecasting, but in clinical applications considerations of practicality and face validity usually suggest a simple, interpretable model."

[25] i.e. characteristics and risk factors.

[26] This includes creating new variables (e.g. diastolic and systolic blood pressure may be combined to give mean arterial pressure) or new types of variable (e.g. turning a continuous variable into a categorical one).

[27] It requires starting with all the candidate variables (full model).

[28] e.g. transforming a simple predictor to detect and model a non-linear effect.

[29] Heart rate, hemoglobin, AF, PVD, albuminuria, WBC, history of ulcer, amputation, renal failure, blindness.

directly interpretable to measure the model's calibration, I further assessed its performance by using the Hosmer-Lemeshow (HL) test of goodness of fit. To do so, I created a random sample of individuals (n=200)[30] whose predictor values were randomly selected within the ranges described in Table 4.5. Then, I calculated each individual's CV risk through both the UKPDS-OM (observed value) and Model 1 (estimated value) for comparison. Model 1 clearly passed the HL test (two-tailed p-value=0.43), confirming high goodness of fit.[31] Model 2 also had a high goodness of fit ($R^2 = 0.9$). To double check it, I calculated the correlation between UKPDS-OM and Model 2 estimates using the same random sample of 200 individuals. The result was very satisfactory ($R^2 = 0.97$).

Having developed simpler, tractable models to rapidly calculate the risk information of interest offers much flexibility to design a tool for use in clinical practice. The next section focuses on how to best convey this information so that behaviour change is encouraged, in accordance with the nudge strategy.

[30]The Hosmer-Lemeshow test does not perform well when sample sizes are too large (n>25,000) or too small (n<100). [93, 94]

[31]The HL test is a goodness of fit test commonly applied to logistic regression results, especially used for risk prediction models. It addresses some limitations of the Pearson Chi-square test, which cannot be readily applied if there are only limited observations for each possible value (or combination of values) of an x variable(s), It can be seen as a Chi-square test for grouped data. Here I used the HL test to assess how well Model 1 does at predicting the data that have been used to estimate it (difference between the model estimates and the UKPDS-OM estimates). After estimating the 200 individual CV risk estimates, I split them into 10 equally-sized sections (quintiles) based on ascending order of probability and calculated the HL statistic based on these 10 groups. A large p-value indicates that the difference between the number of observed and expected values is insignificant and the model is therefore considered valid.

Table 4.3: Model 1: 10-year CV risk (GLM estimates)[1]

Predictor	Regression coefficient (male)	Regression coefficient (female)
Age	0.050	0.049
Age^2	<0.001	<0.001
Smoking status		
Non-smoker	-	-
Smoker	0.263	0.288
Duration of T2DM		
<5 years	-	-
5-10 years	-0.063	0.156
>10 years	-0.205	0.130
BMI	0.013	0.012
HbA1c	0.093	0.067
SBP	0.008	0.007
LDL	0.224	0.156
HDL	-0.330	-0.025
eGFR		
>60	-	-
≤ 60	0.119	0.599
Pre MI		
No	-	-
Yes	0.540	0.614
Pre stroke		
No	-	-
Yes	0.478	0.386
Pre IHD		
No	-	-
Yes	0.742	0.742
Pre HF		
No	-	-
Yes	0.357	0.425
Constant	-6.491	-6.701
Observations	960,000	960,000
AIC	31.912	31.373

Note: All coefficients are highly significant (p<0.01).
[1]Generalised linear model (see Appendix I).

Table 4.4: Model 2: life expectancy (OLS estimates)[1]

Predictor	Regression coefficient (male)	Regression coefficient (female)
Age	-1.020	-1.115
Age^2	0.005	0.005
Smoking status		
Non-smoker	-	-
Smoker	-2.252	-2.541
Duration of T2DM		
<5 years	-	-
5-10 years	-0.009	-0.201
>10 years	0.095	-0.154
HbA1c	-0.215	-0.179
SBP	-0.026	-0.023
LDL	-0.838	-0.637
HDL	0.091	-0.501
eGFR		
>60	-	-
≤ 60	-3.077	-3.040
Pre MI		
No	-	-
Yes	-4.326	-5.123
Pre stroke		
No	-	-
Yes	-6.368	-7.365
Pre IHD		
No	-	-
Yes	-2.544	-4.208
Pre HF		
No	-	-
Yes	-7.107	-8.525
Constant	71.171	76.617
Observations	960,000	960,000
Prob>F	<0.001	<0.001
R^2	0.883	0.900

Note: All coefficients are highly significant (p<0.01).
[1]Ordinary least squares (ordinary linear model, see Appendix I).

Table 4.5: Value ranges used for selected continuous predictors.

Continuous predictor	Value range (step)
Age (years)	35-75 (10)
BMI	18-38 (7)
HbA1c (%)	5-12 (1)
SBP (mm Hg)	110-170 (15)
LDL (mmol/L)	1-5 (1)
HDL (mmol/L)	0.4-2.2 (0.6)

4.5 Tool design (2): format

In this section, I explore the literature on risk communication to find the best ways of targeting the interpersonal factors and decision-making biases identified in the first part of the Thesis. The idea is to select the formats – i.e. the visual features - that will best help reduce the gap between the normative ideal and the descriptive reality, and hence encourage the adherence to self-management behaviour. As discussed in 4.2, having a clear objective is essential to design an effective risk communication.

4.5.1 Targeting strategy (1): numeracy

Numeracy is defined as "the ability to process basic probability and numerical concepts". [95, 96] It has a significant impact on risk perceptions and influences the understanding of risk messages. [96, 97] Several authors have shown that people often do not understand numerical probabilities, and thus fail to use them properly to make decisions. [95, 97] This includes basic numeracy. For example, Lipkus and colleagues (2001) found that only 21% of a sample of highly educated American adults could correctly identify one in 1000 as being equivalent to 0.1% [98], and Yamagishi (1997) showed that death rates of 1286 out of 10 000 were rated as more risky than rates of 24.14 out of 100. [99]

This lack of numeracy (or *innumeracy*) is especially worrying in the context of health risk communication. [16, 98, 100, 101] If numbers are to be used, it is crucial to select formats that facilitate their understanding by the target audience (including

people *at the extremes of the numeracy continuum*). [16, 102, 103] However, although choosing the most appropriate format is key in risk communication, there is little – and sometimes contradictory – evidence about which formats are most effective and/or preferred by patients. Importantly, the appropriateness of a given format depends on the nature of the risk information to be communicated. In the medical literature, we can distinguish four main types of risk message: risk for a single event[32], change in risk[33] or comparison between two risks[34], and evolution of a risk over time[35]. In what follows, I take these considerations into account to review the three main formats used in practice: numerical, visual, and verbal.

a Numerical formats

Numerical probabilities are not easily understood, and thus are difficult to communicate.[36] However, they should not be neglected by the risk communicator, as they tend to lead to more accurate risk perceptions than simpler, verbal formats. [15] Numerical formats are especially needed to present two types of risk information: a risk for a single event and a change in risk (or a comparison between two risks).

Risk for a single event

Although *percentage*[37] is the most common way of communicating health risks and benefits [108], many studies have shown that using *natural frequency*[38] (or *ratio*) improves understanding among patients [103, 109–114], or is at least equivalent to the use of percentage. [115] Ratios also have a higher impact on people's judgments [102, 116], risk perceptions and intentions to make lifestyle changes.[39] [119]

[32] e.g. "Your risk of getting a heart attack in the next ten years is 10%".

[33] e.g. "Your risk of getting a heart attack in the next ten years has decreased by 2% since you started your treatment".

[34] e.g. "Your risk of developing a heart attack in the next ten years if you take (don't take) your treatment is 5% (10%)".

[35] e.g. "Your risk of getting a heart attack over the next ten years is 1% per year in the first five years, and then 2% per year in the last five years".

[36] Although a few studies have found a preference for numerical formats in different populations (students [104, 105], patients [106, 107]).

[37] e.g. "You have a 30% chance of getting a stroke".

[38] e.g. "3 out of every 10 patients get a stroke".

[39] This should be nuanced, as some studies have also reported mixed experimental findings. [117, 118]

Another advantage of natural frequencies is that both patients and health professionals find them easier to understand than percentages or probabilities. [16, 103, 120] One explanation is that they make the relation between the numerator and the denominator clearer. [108, 121] When the denominator is a power of ten (such as *1 in 10* or *1 in 100*), natural frequencies are particularly recommended to communicate risks for single events. [16]

Change in risk or comparison between two risks

The three main ways of numerically presenting a change in risk or a difference between two risk magnitudes are (a) the *absolute risk reduction* (ARR)[40], (b) the *relative risk reduction* (RRR)[41], and (c) the *number needed to treat* (NNT)[42]. In 2011, a Cochrane review compared these three formats and reported that RRR and ARR were better understood by both patients and clinicians than NNT (35 trials included). [122] Importantly, RRR was perceived to be larger and more persuasive[43] than ARR and NNT.[44] Besides, it has been shown that patients generally prefer to receive risk information expressed as RRR. [123, 124] This is in line with Gianinazzi and colleagues' recommendations, who encourage using this format to communicate risk reduction to people with diabetes. [125]

b Visual formats

The use of graphics and other visual displays has been strongly encouraged as complement to numerical formats [16, 32, 38, 100, 126–130], both in younger and older populations. [131, 132] Although more studies are needed to clearly understand how visual displays affect risk perceptions and behaviour, the rise of IT in recent

[40] i.e. the difference between two absolute risks (e.g. pre and post intervention).

[41] i.e. ARR divided by the absolute risk of reference (e.g. pre intervention).

[42] i.e. the number of people who need to be treated for 1 person to benefit, or 1/ARR (e.g. 10 people need to take the treatment for 1 person to experience a benefit).

[43] Persuasiveness in this review refers to how likely participants are to make a hypothetical decision in favour of an intervention (e.g. hypothetical decision to treat cholesterol). [122]

[44] For example, considering a decrease in risk from 8% to 6%, risk reduction was perceived as much larger in terms of relative risk (33% less risk) than in terms of absolute risk (2% less risk).

years has supported the emergence of user-friendly, interactive, and infographic[45] formats. This is a big opportunity to improve the delivery of health risk information.

- **Graphs**

Graphical displays have the advantage of summarising a large amount data and revealing hidden patterns in these data that might not have been detected otherwise. [16, 130, 134] They allow attention to be gained and held by enlivening the information. [24, 133] Waldron *et al.* (2011), in a systematic review of the strategies used to communicate CV risks, found that graphs improved risk perceptions and favoured the reduction of risk factors. [15] However, in populations with poor numeracy, graphs may also create confusion. [133] As a result, they must be tailored to their target audience.[46] As discussed by Ancker *et al.* (2006) in a dedicated review[47], a key point is that different graphical features have different effects on viewers. For instance, graphs that improve the accuracy of quantitative reasoning may differ from those that induce behaviour change. [38]

Change in risk or comparison between two risks

Simple bar graphs are a prime candidate to convey a change in risk or a relative difference between two risk magnitudes. [16, 38, 97, 130, 135] According to Edwards *et al.* (2006), they are "probably understood more easily than any other graphical presentation method". [24] Another useful format is pie charts. [100, 133] By using areas to represent probabilities, pie charts are usually familiar and acceptable to a general audience. However, they may create confusion when multiple risks are to be compared. [133]

Evolution of a risk over time

Line graphs are largely recommended for this purpose. [100, 130, 133] Generally

[45] i.e. graphical representations of data intended for a nontechnical audience. [133]

[46] The effectiveness of graphs depends on the relative numeracy of an audience. [133] Also, graphical literacy has been shown to be strongly affected by expertise and familiarity with specific graphical formats (interactions with education level, literacy, numeracy, and culture). [38]

[47] To date, there have been two comprehensive reviews on the use of graphs to communicate risks. [38, 130]

well understood, it is worth noting that people tend to focus on the direction of the line (i.e. whether it goes up or down) rather than on the numbers. [136]

- **Other visual aids**

Numerous visual aids that cannot be classified as graphs have been created to help communicate risks, especially in the health domain. For example, grids, diagrams, icon arrays (pictographs), and other decision aids have been found to help reduce reasoning and judgment errors. [137, 138] These formats have been shown to be effective and user-friendly in numerous studies [38, 130, 139–142], although, like graphs, they must be tailored to the target audience. [143] Displays which emphasise the numerator of a risk ratio (number of people harmed, or *foreground*) over its denominator (total number that can be harmed out of a population, or *background*) are more likely to increase risk perceptions [16] and, as a result, to promote behaviour change. [16, 142, 144, 145]

Risk for a single event

When communicating individual statistics, numerous studies suggest that pictographs are more quickly and better comprehended than other graphical formats. [87, 102, 113, 139, 146–151] Pictographs express the risk as a natural frequency and highlight the numerator. Experts tend to favour a horizontal, ordered presentation of the icons (over a scrambled presentation) which helps convey a meaningful representation of the risk magnitude. [16, 100, 139, 152, 153] Nevertheless, some studies found that pictographs were not noticeably easier to understand [108, 154], or any better at increasing risk perceptions than other visual formats. [155] As people with low numeracy do not rely on numbers when looking at pictographs, it is important to complement their use with simple numerical information. [147]

Risk ladders are a possible alternative to pictographs. In the spirit of bar graphs, they take advantage of individuals' sensitivity to height to express the risk magnitude. [16, 130, 156]

Change in risk or comparison between two risks

Pictographs [100, 150, 152] and risk ladders [16, 130, 156] have been described as the most effective way of comparing two risk magnitudes. Other, more complex formats have also been developed, such as Venn and Euler diagrams [16, 100], risk characterisation theatres [125], or Paling palettes [100], however they may create confusion in low numeracy populations. [9, 24, 35, 44, 118]

c Verbal formats

Expressing risks verbally (e.g. using risk categories, such as *high*, *moderate*, *low*) generally leads to less accurate risk perceptions than numerical or graphical information. [15] This is partly explained by the high degree of variability in interpreting probability phrases [16]; terms such as *probable*, *unlikely*, or *rare* have been shown to convey "elastic concepts". [157] Indeed, one person may understand the term *likely* as having a 1 in 10 chance, where another person interprets it as equivalent to a 1 in 2 chance. [12] Nevertheless, the systematic communication of simple statements that summarise the gist of information to be remembered from the presentation of numbers or visual formats is recommended. [16, 135, 158, 159]

4.5.2 Targeting strategy (2): bounded rationality

People's decision-making processes, which include the interpretation of risk information, is subject to a broad range of systematic biases (see Chapter 3). Biases belonging to the bounded rationality category (according to Mullainathan and Thaler's classification [160]), are mostly due to people facing limitations in how much information they can process and how much time they have to process it. In what follows, I provide, for each bias identified in Chapters 2 and 3, an overview of the formatting strategies that manipulate or mitigate their effect, in ways that raise risk perceptions and/or encourage behaviour change (nudging).

a Optimism

In the health domain, the phenomenon of overconfidence in one's own ability and intuition (optimistic bias) translates into the tendency of people to feel less at risk than their peers. As there is evidence of optimistic bias in T2DM populations (see Chapter 2), there is a need to increase the personal relevance of the risk, by helping people to realise that complications *could happen (to them)*.

The first option is to communicate personally tailored messages using comparative risk information. Several studies have shown that comparative risks were not just informative, but also persuasive. [87, 119, 155, 161, 162] Making reference to a group of similar individuals – e.g. people of the same age/sex [9, 15, 163], same ethnicity/culture [16, 164], in a similar family situation [15, 16], or geographically close [165] – enhances risk perceptions. The closer a person identifies him/herself to the comparable population, the higher his/her perceived risks.[48] [166] Besides, the display of comparative risk information may be complemented by the provision of case histories and/or testimonials of people seen as similar. These formats have been shown to increase the salience of outcomes associated with a given health condition [9, 35], and to be more persuasive than similar information presented as numbers or didactic facts. [9, 167] Last, it is also recommended to give information about the causes and consequences of the risk associated with a given complication. This is thought to enhance the feeling that such complication could happen (to them). [9, 168]

b Reference-point dependence (sensitivity to framing effects)

Framing refers to the cognitive bias in which people react to a particular choice in different ways depending on how it is presented.[49] As first introduced by Tversky and Kahneman (1981) [169], people may be sensitive to whether a behavioural alternative is framed in terms of its associated costs (loss frame) or benefits (gain frame), even

[48] Raghubir and Menon (2008) showed that people perceived their best friend (most similar) to be less at risk that an average person belonging to the same social category (e.g. undergraduate student, less similar) who, in turn, was perceived to be less at risk than the average person (least similar). [166]

[49] Less numerate participants show larger framing differences. [118]

when the two frames describe objectively equivalent situations. Studies have shown that people tend to avoid [seek] risk when a gain-framed [loss-framed] alternative is presented. [169] However, in the health domain, results of framing experiments have been more mixed. Improvements in adherence to self-care behaviours and/or accuracy of risk perceptions have been observed following exposure to both gain-framed (e.g. Rothman *et al.*, 1993 [170]) and loss-framed information (e.g. Banks *et al.*, 1995 [171]; Meyerowitz and Chaiken, 1987 [172]). Moreover, some studies have failed to find any association with either frame. [173]

According to Rothman and Salovey (1997), the relative effectiveness of a gain-framed or loss-framed message depends on the function of the behaviour under consideration. [174] If the behaviour is perceived as risky (such as detection or screening behaviours), loss-framed messages should be more persuasive, as people favour risky prospects over sure prospects in the loss domain. In contrast, if the behaviour is perceived as safe (such as preventive behaviours), gain-framed messages should be more persuasive, people preferring sure prospects over risky prospects in the gain domain. Several empirical studies have supported these predictions (see e.g. Apanovitch *et al.*, 2003 [175]; Moxey *et al.*, 2003 [176]; Rivers *et al.*, 2005 [177]; but also O'Keefe and Jensen, 2007 [178] or Finney and Iannotti, 2002 [179] for contradictory evidence). With regard to T2DM, patients have expressed risk aversion in both the gain and the loss domains (see Chapter 3), meaning that both types of framing would theoretically be equivalent. However, because risk aversion coefficients were higher in the gain domain, and because self-management is largely perceived as a safe behaviour, a gain-framed message will be chosen.

c Loss aversion

Loss aversion is one of the decision-making biases embedded into prospect theory (see Chapter 3). The idea behind this concept is that *losses loom larger than gains*, i.e. that the pain of losing a given outcome is psychologically more powerful than the pleasure of gaining the same outcome. [180] Phenomena such as the endowment

effect[50] and the status quo bias[51] can be explained, at least partly, by loss aversion. Empirical evidence of loss aversion has been found both with monetary [183–185] and health outcomes. [2, 186] In line with these studies, we found that people with T2DM expressed significant loss aversion when facing a trade-off between health outcomes and behaviour change incurred by T2DM self-management (see Chapter 3).

Strategies that take advantage of people's loss aversion to motivate behaviour change have been increasingly used. For example, commitment contracts, whereby people deposit money that they receive back only if they fulfil their commitment (e.g. smoking cessation [187], weight loss [188]), have shown encouraging results.[52] Regarding risk communication for people with T2DM, two options are promising. First, the evidence of loss aversion found in this population suggests using negatively gain-framed messages to deliver the risk message. Gain-framed messages (see b) may indeed take two forms, according to whether gains or losses are to be highlighted: 1) positively gain-framed ("if you adopt recommended behaviour X, desirable outcome Y will be obtained") or 2) negatively gain-framed ("if you adopt recommended behaviour X, undesirable outcome Z will be avoided"). Because negatively gain-framed messages highlight the avoided losses (complications) of adopting a given behaviour, they may be more effective to encourage adherence to self-management. Second, providing loss of life expectancy estimates (see 4.3) as part of the risk communication intervention is expected to help nudge patients towards better self-management behaviour.

d Probability weighting

The tendency of individuals to overweight small probabilities (less likely events) and underweight large probabilities (more likely events) has been well-described (see Chapter 3). In the health domain, although evidence is sparser, underweighting

[50] Overvaluing a good that is owned, regardless of its objective market value. [181]

[51] The fact that people prefer things to stay the same by doing nothing or by sticking with a decision made previously. [182]

[52] See also the website Stickk (https://www.stickk.com/), which offers personalised commitment contracts.

of probability 0.5 has been reported with both general and patient populations. These biases are in line with people's general difficulties in interpreting numerical expressions of risk. Formats that help deliver the numerical risk information, such as those described in the numeracy section, are recommended. Interactive graphics, for instance, by being adjustable to people's abilities and preferences, offer valuable feedback and assessment options. [167] Zooming facilities, or font size proportional to probability, are examples of recent innovations in infographics that could help improve risk communication interventions. [133]

e Heuristics

The term *heuristic* refers to people's propensity to use mental shortcuts (*rules of thumb*) when making decisions or judgments. Because time and information are limited, heuristics allow people to make rapid decisions if needed. Although these mental shortcuts may sometimes be helpful, they can also lead to judgment errors. Three main types of heuristic have been identified in the literature; I first introduce each of them and then discuss communication strategies accordingly.

- **Availability heuristic**

The concept of availability heuristic (or limited attention) is the tendency to judge the likelihood of an event based on its salience, i.e. based on how easily an example or case related to this event comes to mind. As a result, such events are thought to be more common than they may be in reality.[53] Tversky and Kahneman (1974) suggested that availability heuristic occurs unconsciously and operates under the principle that "if you can think of it, it must be important". [5] In the health domain, studies have shown that people were more likely to base their judgments on concrete, case-based information than on abstract, statistical information. [9] This may partly explain why personalised risk information, by increasing attention, has been shown to be more effective than generic risk information in inducing behaviour change (see 4.2).

[53]For example, after seeing several TV programs on shark attacks, one may think that such attacks are common and, in turn, decide to no longer swim in the ocean.

- **Affect heuristic**

Finally, people also rely on their emotions (good or bad feelings experienced in relation to a stimulus) when making judgments and decisions. Affect plays an important role in how people perceive risks (see e.g. Xie *et al.*, 2011 [189]; Klein and Stefanek, 2007 [163]; Slovic *et al.*, 2004 [190]; Loewenstein *et al.*, 2001 [191]; Finucane *et al.*, 2000 [192]), with different emotions leading to different behavioural intentions. [193] Individuals experiencing positive affect tend to make more optimistic likelihood estimates or judgments than those experiencing negative affect. [194, 195] For instance, movies can sometimes make unhealthy activities look both positive and appealing (e.g. smoking), which may, in turn, favour risky behaviours. Conversely, messages intending to provoke negative affect have been used to heighten perceived risks on a wide range of public health issues. They usually evoke an unpleasant state linked to the negative consequences of a given risky behaviour, so that the opposite, precautionary behaviour is encouraged. However, evidence has shown that fear-arousing messages do not always lead to expected outcomes, at least in targeted populations (those most at risk are most likely to reject the message). Examples include driving [196] or smoking behaviour. [197]

- **Representativeness heuristic**

Representativeness heuristic refers to people's tendency to make judgements based on rules of similarity rather than objective measures of likelihood. In Kardes and colleagues' example, a consumer may infer a relatively high product quality from a store (generic) brand if its packaging is designed to look like a national brand. [198]

Heuristics are important elements to be considered when designing risk communication interventions. Enhancing or mitigating their effect may help facilitate behaviour change and, although not explicitly expressed, several authors have used this lever for action in their intervention. Three tools can help pull this lever; they are detailed below.

Imagery

Due to the close link between imagery and affective processes, images and videos can trigger strong emotional responses.[54] [189, 200, 201] They can make the risk (or the negative consequences of the risk) look more concrete to the receiver and, consequently, easier to understand.[55] [9, 193, 202] By automatically catching attention, images and videos stimulate the receiver's *working memory*, making them more memorable over time. [203] By enhancing both cognitive and affective aspects of risk representation, they also have the potential to motivate behaviour change. [65, 66, 200] For example, promising results have been obtained when presenting smokers with ultrasound images of their own damaged arteries [66] or sedentary young adults with personalised cardiac animations. [65]

Colours play an important role in transmitting emotions and enhancing representativeness. For example, most people automatically link red with danger. [193] In a study exploring preferred formats for risk communication, Hill *et al.* (2010) found that thermometers showing comparative risks with a traffic light system of colours were easily understood and rated as the top choice by both GPs and patients. [135] Other authors have confirmed the importance of such features. [35, 119, 204]

Interactivity

Understanding and memorising of information can be improved with the use of interactive contents (e.g. animation, sound, buttons, sliders). [133] As opposed to static visualisation, interactive features actively engage the receiver in the learning process. Such features offer a wide range of options to help correct for risk misperceptions and motivate behaviour change. [167] It also presents the advantage of counteracting, at least partially, differences in numeracy between individuals. [205]

[54] It is worth noting that more classical visual formats such as graphs and icon arrays [108, 116], as well as simple verbal probability terms (e.g. *low*, *high*, *very high*) [199] can also provoke emotional responses.

[55] See e.g. advertisement campaigns against the risk of smoking or the risk of not wearing a seat belt.

Low amount of information and plain language

For communication purposes, a limited amount of simple information is preferable to detailed, complex information. [87, 88] Restricting clinical or statistical jargon and using plain language make the informational content more understandable. [206]

Finally, it is also worth considering innovative metrics to further target people's heuristics. In recent years, promising metrics have been developed to deliver the risk information. For example, health risks can be converted into *organ ages*. An individual with increased CV [respiratory] risk would have an *effective* heart [lung] age higher than his/her chronological age. The idea behind this concept is to map the increased risk onto a characteristic more easily grasped by patients. [6] Both heart age [56, 207, 208] and lung age [209] are thought to have the potential to motivate behaviour change.

Another innovative way of delivering the risk message is the provision of life time estimates. Galesic and Garcia-Retamero (2011), for example, recommend using changes in life expectancy rather than changes in absolute risk to communicate the consequences of risky health behaviours. [91] This was also suggested by Spiegelhalter (2012) in a paper introducing the concept of *microlife* (see 4.4.3). [4] Communicating losses or gains of life time instead of hazard ratios or increased risks of complication may help motivate behaviour change, as these concepts are easier to imagine and recall. [4, 15]

4.5.3 Targeting strategy (3): other bounds

a Bounded willpower

People make choices that are not always in their long-term interest. [160] There is a tendency to favour short-term outcomes over long-term outcomes, including in the health domain. People with T2DM, because of the nature of the disease[56], are especially subject to bounded willpower[57] when making health-related decisions. [3, 42]

[56] The first phase is asymptomatic, i.e. patients do not feel at risk.

[57] *Present biasedness*, or *time inconsistency* are also found in the literature.

Over the past ten years, strategies have been developed to mitigate the overvaluation of short-term outcomes and motivate behaviour change. In particular, the use of monetary-based reinforcers (rewards) has shown promising results in encouraging weight loss (Volpp *et al.*, 2008 [188]), enhancing exercise (Petry *et al.*, 2013 [210]), or improving medication adherence (Petry *et al.*, 2015 [211]), at least in the short term.[58] Regarding diabetes management, Petry *et al.* (2015) found that reinforcing adolescents with T1DM in their self-monitoring of blood glucose may increase testing frequency and decrease HbA1c levels. [213] In their review, Kullgren *et al.* (2017) reported that four of the six studies that tested financial incentives on T2DM populations showed increased adherence to self-care behaviours or improved biometric measures. [214]

However, there is a question whether these money rewards should be considered as behavioural economic- or rather as standard economic-informed interventions (see Chapter 1). According to the simple principle of price mechanism, standard economic rationality does predict that individuals will be more or less likely to adopt a certain behaviour according to whether they are rewarded or penalized for that behaviour (e.g. tax exemption or surcharge).

Oliver (2017) notes that the *amount* of the financial incentive is determinant to decide on the nature of an intervention. [215] Rather than offering a genuine material benefit, a money-reward nudge is aimed to offset present bias by reminding the person who receives it that their immediate action, although enjoyable, is potentially harmful. Despite offering a small immediate compensation for engaging [not engaging] in beneficial [detrimental] behaviour, it is not meant to significantly change the economic incentives of those who receive them.[59] For example, Volpp *et al.* (2008)'s study mentioned above involved high incentives (each participant could earn $750), meaning that the effectiveness of the intervention is to be attributed more to the classic price mechanism of standard economic theory than to behavioural

[58] For example, in the case of Volpp's study, participants were found to regain the weight upon withdrawal of the incentives. [212]

[59] "[...] any aspect of the choice architecture that alters people's behavior in a predictable way without forbidding any options or *significantly changing their economic incentives.* To count as a nudge, the intervention must be easy and cheap to avoid. Nudges are not mandates." [216]

economics.

Moreover, empirical support for self-determination theory (Ryan and Deci, 2000 [217]) has shown that financial incentives may be viewed as a form of control when introduced to drive a behaviour that relies on intrinsic motivation. Some studies have found that monetary incentives can crowd out intrinsic motivation and consequently lead to results opposite to those anticipated by standard economic rationality (e.g. Gneezy and Rustichini, 2000 [218]; Ariely *et al.*, 2009 [219]). For this reason, economic incentives are allowed insofar as they do not drive material concerns, but rather serve as "almost unconscious supportive – not controlling – reminders that the incentivised action is beneficial to the individual." [60] [215]

Importantly, the effectiveness of small financial incentives in motivating health-related behaviours seems to depend on the nature of the behaviours in question. When individuals are required to act *at a single point in time* (e.g. getting vaccinated [220] or screened for a disease [221]), or at least a limited number of times (e.g. adhering to short-term treatment course), such incentives tend to work well. However, when money rewards are aimed to motivate a sustained behavioural change, evidence suggests that they are less effective. For example, only one out of 17 studies that tested the impact of financial incentives on smoking has shown higher cessation rates after six months. [222] Regarding dietary behaviour, a meta-analysis of randomised trials on financial incentives aimed at encouraging loss weight showed that, compared to no incentive, an incentive of less [more] than 1.2% of personal disposable income was associated with no [negligible] weight loss after 18 months. [223]

Regarding risk communication options, several authors have recommended presenting risks over a short timeline to mitigate bounded willpower. [15, 46, 125, 135] Hill *et al.* (2010) suggest that longer time frames are seen as being too far away, and may not encourage people to make lifestyle changes. [135] Although most risk communication interventions use either lifetime or 10-year time horizons, several authors suggest using shorter time frames. [96, 224, 225] Asimakopoulou *et*

[60] There is understandably a blurred line concerning the threshold at which people change their behaviour due to pure financial reasons.

al. (2008), in particular, found that 5-year time frames had a better impact on risk perceptions in a T2DM population. [46] Chandran and Menon (2004) go further and claim that day frames, by making the threat more concrete and proximal, should be favoured over year frames. [226] Interestingly, they observed a reversal effect which is reminiscent of Tversky and Kahneman's framing effect [169]: with negatively-framed outcomes (e.g. developing a heart disease), the day frame is more persuasive than the year frame; whereas the converse is true with positively-framed outcomes (e.g. avoiding a heart disease). In terms of patients' preferences, it is not clear whether short (5-year or less) or long (10-year or lifetime) time frames are preferred. Age seems to have an influence, with younger people preferring shorter time frames and older people preferring longer ones. [113]

Another strategy to make the risk look more concrete and proximal has recently been introduced by David Spiegelhalter. [4] The concept of *microlife*, which represents half an hour of adult life expectancy[61], is an innovative way to communicate lifestyle-related risks. By expressing the daily effect of health behaviours as changes in microlives, the idea is to use the concept of *ageing faster or slower than an average person* to describe the level of risk. As explained by Spiegelhalter, the issue with current presentations of change in life expectancy (e.g. "a lifetime habit of an extra portion of red meat per day is associated with a reduction in male life expectancy of around one year, from age 80 to 79") is that "they do not look very impressive, as people tend to dismiss effects that are perceived to lie in the distant future."[62] Converting changes in life expectancy[63] into daily hours (or minutes) of life lost or gained is a promising way to mitigate the effect of bounded willpower and motivate behaviour change in T2DM populations.

[61] Calculated from hazard ratios if a lifelong effect from a specified age is assume, see Spiegelhalter (2012) for more details. [4]

[62] A quote from Kingsley Amis was reported by Spiegelhalter (2012) to illustrate this phenomenon: "No pleasure is worth giving up for the sake of two more years in a geriatric at Weston-super-Mare." [4]

[63] For example, associated to different levels of self-management behaviour.

b Bounded selfishness

Bounded selfishness (or bounded self-interest) refers to the fact that people care about the well-being of others. While neoclassical theory stresses self-interest as the primary motivator of decisions, behavioural economists have shown that individuals may act out of concern for others or based on conceptions such as fairness, revenge, or on other altruistic motives. [227]

Bounded selfishness and, to another extent, social norms, play an important role in the process of behaviour change. As noted by Rothman and Kiviniemi (1999), people's behaviour is largely affected by whether "a response format leads them to believe that the frequency of their behaviour is above or below the perceived norm." [9] This is to be taken into consideration when encouraging people with T2DM to adopt self-management behaviour. The farther away the behaviour in question from their perceived norm, the lower the chances of it being adopted.

Moreover, although the general public does not perceive diabetes to be a stigmatised condition, people with diabetes tend to feel negatively judged and constantly monitored due to their condition. [228] Such perceived stigma may have negative consequences on self-care behaviour and clinical outcomes. [228] Importantly, social context influences the ways individuals respond to risk information. The relevance and the trustworthiness of information sources are key elements that people assess in making everyday decisions. [229] In the medical literature, studies have shown that physicians were the most trusted by people when it comes to risk communication. [230, 231] In particular, the use of risk calculators must always be used in conjunction with health professionals who can guide the user through the calculator and help them interpret the resulting risk information (see Chapter 5). [10, 11]

Table 4.6 summarises the levers for action and targeting communication strategies (formatting) discussed throughout this section.

Table 4.6: Levers for action and targeting communication strategies (formatting).

Lever for action	Targeting strategy (formatting)
Numeracy	
Risk for single event	(a) Natural frequencies; (b) Pictographs, risk ladders; (c) Short summary statement
Change of risk, comparison of risk	(a) RRR; (b) Simple bar graphs; (c) Short summary statement
Evolution of risk over time	(a) Line graphs; (b) Short summary statement
Optimism	Comparative risks (personalised vs similar individuals) Testimonials
Framing	Gain-framed message
Probability weighting	See Numeracy
Loss aversion	Innovative metrics: loss of life expectancy, microlife Negatively gain-framed message ("if you adopt recommended behaviour X, undesirable outcome Z will be avoided")
Heuristics	Imagery, interactivity, colours, plain language Innovative metrics: loss of life expectancy Innovative format: organ ages
Bounded willpower	Short time frame Innovative outcome: loss of life expectancy
Bounded selfishness	Comparison to similar groups of individuals in terms of behaviour Trusted source of information

4.6 Conclusion

Risk communication interventions aiming to motivate sustained adherence to T2DM self-management have only shown modest results so far. The effect of the most effective intervention lasts no longer than a few weeks after people receive the risk message. In this context, given the importance of risk perceptions in the initiation and maintenance of self-care behaviours (see Chapter 2), there is a need to improve these interventions.

Over the past decade, increased awareness of people's decision-making biases (deviations from standard economic rationality) has motivated the use of findings from behavioural economics to inform new types of health promotion interventions. Specifically targeting people's decision-making biases has shown promising results in enhancing the impact of such interventions, including in the diabetes area (see 4.5). Following this approach, I developed in this chapter a nudge-based risk communication tool, tailored to people with T2DM, informed by the behavioural economic evidence gathered in the first part of the Thesis.

The development of the tool, named PERCODIA, results from the selection of appropriate elements of content (section 4.4) and format (section 4.5) to make the best use of relevant behavioural economic materials (see summary in Table 4.1). Two types of risk estimates are calculated and displayed by PERCODIA: the risk for CV event (largely underestimated in T2DM populations; see Chapter 2) and life expectancy, both correlated to patient self-management level. These estimates are calculated based on the equations of the UKPDS-OM, the validated prognosis model of reference in the T2DM area. Moreover, PERCODIA was developed for use in routine primary care consultation, primary care being the key contact point with the healthcare system for people with T2DM.

The next chapter will be dedicated to piloting PERCODIA in a primary care setting. I will conduct a pilot randomised controlled trial (RCT) to assess the feasibility of using the tool in routine primary care consultation and evaluate its impact on risk recall and self-management in patients with poorly controlled

T2DM. The ultimate objective is to inform the development of a larger RCT to be conducted in the future.

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5

Assessment of a nudge-based risk communication intervention in primary care: a pilot randomised controlled trial

People can foresee the future only when it coincides with their own wishes, and the most grossly obvious facts can be ignored when they are unwelcome.

Orwell (London Letter, 1945) [1]

5.1 Introduction

The fact that many patients with Type-2 diabetes mellitus (T2DM) do not engage in self-care behaviours suggests that current risk messages may not provide relevant information in ways that motivate such behaviours (see Chapter 1). In the previous chapter, I developed a risk communication tool informed by behavioural economics (*nudge-based*) in order to motivate sustained adherence to self-management in this population. This tool, entitled PERCODIA, is informed by behavioural economic material relevant for T2DM patients identified earlier in the Thesis (see Chapters 2 and 3). Such an approach is expected to improve the effectiveness of current risk communication interventions (see Chapter 4).

To my knowledge, PERCODIA is the first nudge-based risk communication tool built to motivate adherence to self-care behaviours. So far, behavioural economic-informed interventions aimed to promote self-care behaviours have mostly relied on mitigating present biasedness by means of incentives or rewards, with mixed results in the long run (see Chapter 4). In the risk communication field, however, using findings of behavioural economics to enhance the impact of interventions is not common practice. Although substantial work has been done to test the impact of *framing effects* (the tendency that people have to draw different conclusions from the same risk information, depending on whether it is expressed in terms of gains or losses) on screening or precautionary behaviours (see Gallagher and Updegraff, 2012 [2], Edwards et al, 2001 [3] for general reviews and Kullgren *et al.*, 2017 [4] for a review in T2DM populations), most factors that influence health-related decisions have not yet been targeted. This is an issue since the effectiveness of risk communication may be enhanced by selecting adapted formats or metrics for this purpose. [5]

Existing risk communication interventions for people with T2DM have shown

mixed results, with many participants barely understanding the explanations of health professionals about risks and having poor recall of risk information (see Chapter 2). There is an urgent need to improve risk communication in this area, making people with T2DM prime candidates to receive an intervention informed by behavioural economics.

This chapter reports a pilot study assessing the feasibility of using PERCODIA in a primary care setting. PERCODIA was designed for use in primary care consultation, as part of doctor-patient risk communication. Current health promotion interventions tailored for people with T2DM are often complex and multidimensional (where risk communication is only one part of the intervention), making them hardly compatible with a large-scale use in primary care (see Chapter 4).

5.2 Objectives

The objectives of this pilot study are to assess the feasibility of using PERCODIA in a primary care setting and investigate its effect on risk insight and recall and, in turn, on self-management behaviour. The results of the pilot study aim to inform the development of an adequately powered randomised controlled trial (RCT) to be conducted in the future.

5.3 Research design and methods

Study design, methods and results are reported in accordance with the CONSORT statement for pilot and feasibility trials. [6]

5.3.1 Recruitment

Forty adults with poorly controlled T2DM (HbA1c>7.5% measured at least once over the past year) between 30 and 75 years of age were recruited from a single-site surgery

practice in Oxford, UK. Subjects were excluded if (1) they were non-English speakers; (2) unable to give informed consent; or (3) diagnosed with T2DM for less than a year. The study was approved by the competent UK NHS research ethics committee (ref. 17/NW/0267; see Appendix K), and each participant gave written consent.

5.3.2 Design

a Randomisation

Participants were randomly assigned to either the intervention group, receiving the nudge-based risk communication intervention in addition to the standard care provided in routine primary care consultation, or to the control group, receiving standard care only (see Fig. 5.1).

In order to balance the two groups in terms of sex and age, I used a stratified randomisation method so that each group had the same number of males and females aged above and below 57 years old. This age threshold was chosen in accordance with the distribution of age at diagnosis of diabetes.[1] [7] Group allocation sequence was determined using a computerised generated random number table (www.randomizer.org). Given the nature of the study, participants were blinded to group assignment, but the research team was not.

b Intervention group

The PERCODIA intervention consisted of communicating the impact of being poorly controlled on two types of outcome: (1) absolute 10-year risk of experiencing a cardiovascular event, i.e. heart attack or stroke (10-year CV risk); (2) life expectancy. PERCODIA estimates are calculated based on the UKPDS outcomes model (version 2). [8]

c Control group

Participants in the control group were given an estimate of their absolute 10-year CV risk based on the UKPDS outcomes model (version 2), but were not given information on life expectancy or access to the PERCODIA intervention.

[1] According to US data, as there is, to my knowlege, no available data for the UK.

d Protocol

Primary care setting

PERCODIA was developed to allow real-time, individualised risk communication in routine primary care consultation. All consultations were conducted by the same general practitioner (GP). In the intervention group, PERCODIA was running by the GP on his surgery screen. In both groups participants were also given personalised lifestyle advice by the GP. They were orally reminded of the importance of adhering to self-care behaviours in order to reduce their risks. Emphasis was placed, where appropriate, on increasing consumption of fruits and vegetables, decreasing consumption of high-fat foods, increasing physical activity, maintaining adherence to medications, and decreasing alcohol consumption and smoking, depending on each participant's familial or financial constraints. The GP also provided information about the causes and consequences of the risk.

Timeline of events

- **Visit 1**

  Patients who gave consent to participate to the study were given three questionnaires (demographics, intentions to adhere to self-management, and self-management) by an experimenter (myself). After filling out the questionnaires, they attended their routine clinical consultation with the GP. During the consultation, participants allocated to the intervention group received usual care and the PERCODIA intervention, while participants allocated to the control group received usual care only. Right after the consultation, two questionnaires (risk recall and intentions to adhere to self-management) were given to the participants by the experimenter.

- **Visit 2**

  At participants' next consultation visit (12 weeks after Visit 1), participants

were given two questionnaires (risk recall and self-management) by the same experimenter (myself) before their consultation.

5.3.3 Primary outcomes

Primary outcomes were selected to demonstrate that PERCODIA can be used in a real-world setting. The objectives were to assess the feasibility of implementing the intervention in routine primary care consultation and to obtain patient outcome data (satisfaction, worry and anxiety) for the development of a larger RCT.

Study feasibility was assessed by examining consent rate, retention rate, and rates of missing data. In addition, I measured participants' satisfaction with the intervention using adapted questions from the COMRADE[2] scale, which was validated for use in the UK. [9] This scale consists of 10 questions on a 5-point scale ranging from *strongly disagree* to *strongly agree* and was used immediately after the intervention. Special attention was devoted to the question asking participants to evaluate the ease of understanding of the information they were communicated. Acceptability of the intervention was further assessed by measuring participants' worry and anxiety levels immediately after the intervention. Following Welschen *et al.* (2012) [10], I used questions adapted from Claassen *et al.* (2010) [11] to assess how worried or anxious they were about their personalised risk information (7-point Likert scale ranging from *not worried/anxious at all* to *very much worried/anxious*). General anxiety was assessed by the validated Short Form Spielberger State-Trait Anxiety Inventory (SF-STAI) [12], where participants had to evaluate how *calm*, *tense*, *upset*, *relaxed*, *content*, and *worried* they felt on a 4-point Likert scale. Higher scores indicate a higher level of anxiety.

5.3.4 Secondary outcomes

Secondary outcomes were selected to estimate the effect of the intervention on risk recall and self-management behaviour. First, recall of personalised risk information was measured immediately and 12 weeks after the intervention using questionnaires

[2]Combined Outcome Measure for Risk communication And treatment Decision-making Effectiveness.

specifically developed for this purpose (binary outcome: recalled or not recalled). Second, intentions to make lifestyle changes were measured immediately before and after the intervention using the validated Determinants of Lifestyle Behaviour Questionnaire (DLBQ). [13] The DLBQ consists of three parts, investigating respectively dietary intake, physical activity and smoking habits. Intentions are assessed by a 5-point Likert scale ranging from *strongly agree* to *strongly disagree*. Third, change in self-management behaviour from baseline to 12 weeks was measured using the validated Summary of Diabetes Self-Care Activities (SDSCA) questionnaire. [14] This instrument explores six dimensions of self-management (healthy eating, physical activity, medication adherence, self-monitoring of blood glucose, foot checks and smoking behaviour), using the self-reported frequency of completing recommended activities during the past seven days as an outcome (SDSCA score ranging from 0 to 7 for each dimension). All questionnaires were delivered to the participants by me. Copies of these questionnaires are provided in Appendix L. Finally, HbA1c levels (%) were measured before and 12 weeks after the intervention.

5.3.5 Data analysis

a Demographics and baseline characteristics

Between-group baseline differences were tested using Student t-test (means) and Chi-square test (proportions). For each test, two-tailed p-values were reported.

b Recall of personalised risk information

Between-group differences were assessed using Chi-square test. In the intervention group, differences in recall of the different types of personalised risk information were also tested using Chi-square test, taking 10-year CV risk score as the reference category. Within-group differences in risk recall after 12 weeks were tested using McNemar's test.

c Behavioural outcomes

Within-group pre-post intervention differences for intentions to make lifestyle changes and self-management behaviour (SDSCA scores and HbA1c levels) were tested using paired t-test. Between-group pre-post intervention differences for intentions to make lifestyle changes and self-management behaviour (SDSCA scores and HbA1c levels) were investigated using regression analysis to model secondary outcomes as a function of intervention status while adjusting for potential confounders (age, sex, ethnicity, education, duration of diabetes) and the corresponding baseline measure. General linear models were used for continuous outcome variables (all self-care behaviours except medication adherence) and a logistic model was used for the only dichotomous variable (medication adherence).

Intention-to-treat (ITT) analyses were conducted, both with complete cases only (excluding participants with missing follow-up data) and with all cases (including participants with missing follow-up data). In the analysis including all cases, missing data for follow-up measures were replaced by baseline data. [15]

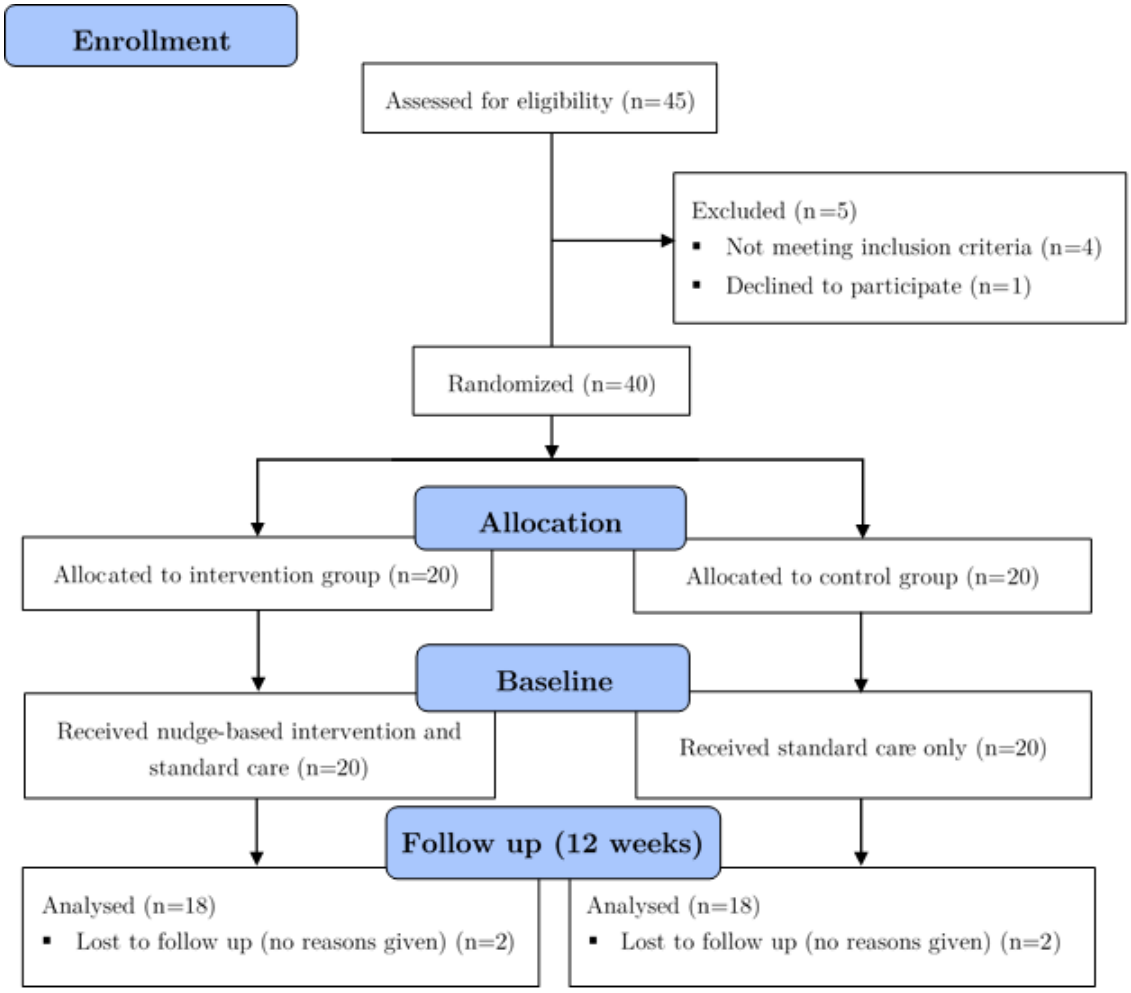
5.4 Results

5.4.1 Demographics and baseline characteristics

Forty-five individuals with poorly controlled T2DM were invited to take part in the study; 44 consented and 40 were randomised to the intervention (n=20) or control group (n=20) after further assessment of eligibility. Four patients were lost to follow-up (see Fig. 5.1).

Statistical tests showed no between-group differences in terms of age, ethnicity, employment status, education level, duration of diabetes, smoking status and baseline HbA1c level, indicating an effective randomisation process (see Table 5.1).

Figure 5.1: CONSORT flow chart of participants.



5.4.2 Primary outcomes

One eligible individual did not give consent to be enrolled in the study, leading to a final consent rate of 98%. Of 40 participants, four individuals did not attend the follow-up visit (without giving any reason). Therefore, the study retention rate was 90%. Missing data were confined to the drop-outs, and to participants who could not recall personalised risk information and so were logically not able to indicate how anxious or worried they felt about it.

Table 5.2 summarises the results for the outcomes used to evaluate the acceptability of the intervention. Participants rated both their satisfaction with the intervention (4.4 and 4.6 out of 5 in the intervention and control group, respectively)

Table 5.1: Demographics and baseline characteristics.

	Intervention group (n=20)	**Control group** (n=20)	**p-value**
Age (years), mean [SD]	57.4 [12.5]	61.1 [11.2]	p=0.32
Sex, n(%)			
Male	12(60%)	12(60%)	p=1.00
Female	8(40%)	8(40%)	
Ethnicity, n(%)			
White	16(80%)	16(80%)	p=1.00
Asian British	4(20%)	4(20%)	
Education, n(%)			
High school or no degree	11(55%)	8(40%)	p=0.35
Bachelor or graduate degree	9(45%)	12(60%)	
Employment, n(%)			
Employed	10(50%)	11(55%)	p=0.75
Unemployed or retired	10(50%)	9(45%)	
Duration of T2DM (years), mean [SD]	9.7 [7.1]	12.1 [9.8]	p=0.38
Smoking status, n(%)			
Smoker	2(10%)	2(10%)	p=1.00
Non-smoker	18(90%)	18(90%)	
HbA1c (%), mean [SD]	8.6 [1.6]	8.4 [1.3]	p=0.73

and the ease of understanding of the information (4.7 out of 5 in both groups). There was no significant between-group difference regarding these two outcomes (p=0.79 and p=0.49, respectively). In the intervention group, communicating heart age was associated with higher levels of anxiety and worry than 10-year CV risk (p=0.01 and p=0.02, respectively). However, the lowest levels of anxiety and worry were associated with the communication of daily hours of lifetime lost (p=0.02 and p=0.01, respectively).

5.4.3 Secondary outcomes

a Recall of personalised risk information

A summary of the results for risk recall is provided in Table 5.3 (ITT analysis with complete cases only) and Table 5.4 (ITT analysis with all cases). No significant difference was found between the two groups regarding recall of 10-year CV risk score. In the intervention group, recall for effective heart age was significantly better than recall for any of the other formats, both immediately (as compared to 10-year CV risk score) and 12 weeks after the intervention. Effective heart age was

also the only format that was satisfactorily recalled after 12 weeks (no significant difference in terms of proportion of individuals remembering the information between baseline and follow-up).

b Behavioural outcomes

A summary of the results for intentions to make lifestyle changes and self-management behaviour (SDSCA scores and HbA1c levels) is provided in Table 5.5 (ITT analysis with complete cases only) and Table 5.6 (ITT analysis with all cases). In addition, results of the regression analyses are presented in Tables 5.7 (complete cases only) and 5.8 (all cases). Controlling for potential confounders (age, sex, ethnicity, education, duration of diabetes) and the corresponding baseline measure, the intervention had a positive effect on participants' intentions to change their diet (complete cases only, p=0.07). I also observed an improvement in self-monitoring of blood glucose, under ITT analysis with both complete cases (p=0.07) and all cases (p=0.08). No other between-group significant difference was detected.

Table 5.2: Summary of results (primary outcomes and between-group comparisons).

	Intervention group (n=20)	**Control group** (n=20)	**p-value**
Satisfaction[1], mean [SD]	4.4 [0.7]	4.6 [1.1]	p=0.79
Ease of understanding[2], mean [SD]	4.7 [0.7]	4.7 [0.5]	p=0.49
Anxiety, mean [SD]			
General[3]	11.4 [3.8]	9.9 [4.8]	p=0.29
10-year CV risk[4]	3.6 [2.5] (n=14)	2.9 [2.1] (n=17)	p=0.34
Effective heart age[4]	4.2 [2.2]	-	-
Number of hours lost[4]	2.6 [1.6] (n=16)	-	-
Worry, mean [SD]			
10-year CV risk[4]	3.8 [2.5] (n=14)	2.8 [2.0] (n=17)	p=0.21
Effective heart age[4]	4.3 [2.2]	-	-
Number of hours lost[4]	2.7 [1.7] (n=16)	-	-

Note: Variations in sample size for anxiety and worry outcomes are explained by variations in recall of personalised risk information.
[1]Assessed by an adapted version of the COMRADE scale [9], which consists of 10 questions on a 5-point scale ranging from *strongly disagree* to *strongly agree*. [2]Assessed by one of the COMRADE questions. [3]Measured by the SF-STAI, a 6-item questionnaire to assess people's emotional state on a 4-point scale. [12] Higher scores indicate a higher level of anxiety. [4]Measured by a 7-point Likert scale, ranging from *not anxious/worried at all* to *very worried/anxious*. [11]

Table 5.3: Summary of results (risk recall, complete cases only).

	Intervention group (n=18)					Control group (n=18)		
	After intervention		After 12 weeks			After intervention	After 12 weeks	
	Score	p-value	Score	p-value	p-value (within)	Score	Score	p-value (within)
Recall, n(%)[1]								
10-year CV risk	14(78%)	-	6(33%)	-	p=0.01**	16(89%)	2(11%)	p<0.01***
10-year CV risk well-controlled	14(78%)	p=1.00	2(11%)	p=0.11	p<0.01***	-	-	-
Effective heart age	18(100%)	p=0.04**	16(89%)	p<0.01***	p=0.48	-	-	-
Number of hours lost	16(89%)	p=0.38	4(22%)	p=0.47	p<0.01***	-	-	-

Note: *p<0.1; **p<0.05; ***p<0.01

[1]Number of individuals who accurately recalled their personalised risk information.

Table 5.4: Summary of results (risk recall, all cases).

	Intervention group (n=20)					Control group (n=20)		
	After intervention		After 12 weeks			After intervention	After 12 weeks	
	Score	p-value	Score	p-value	p-value (within)	Score	Score	p-value (within)
Recall, n(%)[1]								
10-year CV risk	14(70%)	-	6(30%)	-	p=0.01**	17(85%)	4(20%)	p<0.01***
10-year CV risk	14(70%)	p=1.00	3(15%)	p=0.25	p<0.01***	-	-	-
well-controlled								
Effective heart age	20(100%)	p<0.01***	18(90%)	p<0.01***	p=0.48	-	-	-
Number of hours lost	16(80%)	p=0.47	5(25%)	p=0.73	p<0.01***	-	-	-

Note: *p<0.1; **p<0.05; ***p<0.01
[1]Number of individuals who accurately recalled their personalised risk information.

Table 5.5: Summary of results (intentions and self-management behaviour, complete cases only).

	Intervention group (n=18)				Control group (n=18)			
	Baseline	**After intervention**	**After 12 weeks**	**p-value**	**Baseline**	**After intervention**	**After 12 weeks**	**p-value**
Intentions[1], mean[SD]								
Diet	3.1 [1.6]	4.3 [1.1]	-	p<0.01***	4.5 [0.8]	4.5 [0.8]	-	p=0.80
Exercise	3.7 [1.5]	4.3 [1.0]	-	p=0.05*	3.9 [1.3]	4.3 [1.0]	-	p=0.07*
Self-management score[2]								
Diet (fruits and veg.), mean[SD]	4.2 [1.8]	-	4.7 [2.2]	p=0.48	5.2 [1.8]	-	4.6 [2.2]	p=0.02**
Diet (high-fat foods), mean[SD]	2.8 [2.2]	-	2.9 [1.6]	p=0.75	4.2 [2.3]	-	3.6 [2.4]	p=0.19
Exercise[3], mean[SD]	6.3 [3.9]	-	5.2 [4.1]	p=0.16	5.7 [3.5]	-	5.5 [3.8]	p=0.86
SMBG, mean[SD]	3.5 [3.3]	-	4.2 [3.1]	p=0.27	3.4 [3.1]	-	3.2 [2.8]	p=0.35
Foot, mean[SD]	1.9 [2.5]	-	2.3 [2.7]	p=0.71	3.1 [2.9]	-	4.1 [2.7]	p=0.14
Med. adherence								
No missed, n(%)	15(83%)	-	14(70%)	-	16(89%)	-	17(94%)	-
Missed, n(%)	3(17%)	-	4(30%)	-	2(11%)	-	1(6%)	-
HbA1c, mean[SD]	8.8 [1.6]	-	8.5 [1.9]	p=0.37	8.5 [1.3]	-	8.4 [1.7]	p=0.65

Note 1: *p<0.1; **p<0.05; ***p<0.01

Note 2: Given the small proportion of smokers in my sample, I did not model smoking status as an outcome variable.

[1]The DLBQ consists of three parts, investigating respectively dietary intake, physical activity and smoking habits. Intentions are assessed by a 5-point Likert scale ranging from *strongly agree* to *strongly disagree* [13]; [2]The SDSCA questionnaire explores six dimensions of self-management with 11 items, using the self-reported frequency of completing recommended activities during the past seven days [14]; [3]Combination of two items: walk and specific exercise sessions.

Table 5.6: Summary of results (intentions and self-management behaviour, all cases).

	Intervention group (n=20)				Control group (n=20)			
	Baseline	**After intervention**	**After 12 weeks**	**p-value**	**Baseline**	**After intervention**	**After 12 weeks**	**p-value**
Intentions[1], mean[SD]								
Diet	3.0 [1.6]	4.0 [1.4]	-	p<0.01***	4.4 [0.8]	4.5 [0.8]	-	p=0.38
Exercise	3.5 [1.6]	4.1 [1.1]	-	p=0.02**	3.9 [1.2]	4.2 [1.0]	-	p=0.07*
Self-management score[2]								
Diet (fruits and veg.), mean[SD]	4.3 [1.9]	-	4.7 [2.2]	p=0.48	4.8 [2.2]	-	4.2 [2.4]	p=0.02**
Diet (high-fat foods), mean[SD]	2.7 [2.2]	-	2.8 [1.6]	p=0.75	4.1 [2.3]	-	3.6 [2.4]	p=0.19
Exercise[3], mean[SD]	6.1 [4.0]	-	5.0 [4.1]	p=0.16	5.5 [3.6]	-	5.4 [3.8]	p=0.86
SMBG, mean[SD]	3.6 [3.3]	-	4.2 [3.1]	p=0.27	3.8 [3.1]	-	3.5 [2.8]	p=0.35
Foot, mean[SD]	2.3 [2.7]	-	2.7 [2.8]	p=0.48	3.4 [2.9]	-	4.4 [2.7]	p=0.14
Med. adherence								
No missed, n(%)	17(85%)	-	16(80%)	-	17(85%)	-	18(90%)	-
Missed, n(%)	3(15%)	-	4(20%)	-	3(15%)	-	2(10%)	-
HbA1c, mean[SD]	8.6 [1.6]	-	8.4 [1.9]	p=0.45	8.4 [1.3]	-	8.3 [1.6]	p=0.69

Note 1: *p<0.1; **p<0.05; ***p<0.01
Note 2: Given the small proportion of smokers in my sample, I did not model smoking status as an outcome variable.
[1]The DLBQ consists of three parts, investigating respectively dietary intake, physical activity and smoking habits. Intentions are assessed by a 5-point Likert scale ranging from *strongly agree* to *strongly disagree* [13]; [2]The SDSCA questionnaire explores six dimensions of self-management with 11 items, using the self-reported frequency of completing recommended activities during the past seven days [14]; [3]Combination of two items: walk and specific exercise sessions.

Table 5.7: Regression coefficients (intentions and self-management behaviour, complete cases only).

	Dependent outcome variable (n=36)								
	Intentions[1] (Diet)	**Intentions[1] (Exercise)**	**Diet 1[2]**	**Diet 2[3]**	**Exercise[4]**	**SMBG**	**Foot check**	**Medication adherence[5]**	**HbA1c**
Age (years)	<0.01	<0.01	0.05**	<0.01	0.07	<0.01	0.04	0.94	<0.01
	(0.01)	(0.01)	(0.02)	(0.03)	(0.05)	(0.03)	(0.03)	(0.06)	(0.02)
Sex Male (ref.)	-	-	-	-	-	-	-	-	-
Female	-0.09	-0.28	0.88	-0.21	-1.13	0.67	1.11	5.49	-0.14
	(0.27)	(0.24)	(0.75)	(0.57)	(1.17)	(0.55)	(0.73)	(6.40)	(0.30)
Duration T2DM	0.01	<0.01	-0.03	-0.02	-0.04	<0.01	0.11**	0.99	0.01
	(0.01)	(0.01)	(0.05)	(0.03)	(0.07)	(0.02)	(0.05)	(0.06)	(0.02)
Group status									
Control (ref.)	-	-	-	-	-	-	-	-	-
Intervention	0.40*	0.10	0.80	-0.07	-0.61	1.01*	-0.86	3.92	-0.11
	(0.22)	(0.23)	(0.69)	(0.51)	(1.07)	(0.55)	(0.66)	(4.87)	(0.38)
Education									
High school or no degree (ref.)	-	-	-	-	-	-	-	-	-
Bachelor or grad degree	-0.21	-0.58**	1.00	0.33	-0.50	-1.26**	-0.96	1.16	0.03
	(0.27)	(0.22)	(0.69)	(0.55)	(1.12)	(0.54)	(0.63)	(1.44)	(0.42)
Baseline score	0.49***	0.47***	0.53***	0.48***	0.63***	0.75***	0.25*	6.39	0.96***
	(0.16)	(0.14)	(0.17)	(0.10)	(0.16)	(0.10)	(0.14)	(8.57)	(0.10)
Constant	2.87***	2.88***	-2.00	1.93	-0.85	1.19	-0.71	0.60	0.01
	(0.93)	(0.91)	(1.58)	(1.84)	(3.41)	(1.81)	(2.02)	(2.00)	(1.43)

Note 1: *p<0.1; **p<0.05; ***p<0.01. *Note 2*: Given the small proportion of smokers in my sample, I did not model smoking status as an outcome variable.
[1]Measured immediately after the intervention. [2]Consumption of fruits and vegetables. [3]Consumption of high-fat foods. [4]Combination of two items (walk and specific exercise sessions). [5]Interpreted as a dichotomous variable (no missed dose = 0; missed dose = 1).

Table 5.8: Regression coefficients (intentions and self-management behaviour, all cases).

	Dependent outcome variable (n=40)								
	Intentions[1] (Diet)	**Intentions[1] (Exercise)**	**Diet 1[2]**	**Diet 2[3]**	**Exercise[4]**	**SMBG**	**Foot check**	**Medication adherence[5]**	**HbA1c**
Age (years)	-0.01 (0.01)	<0.01 (0.01)	0.05** (0.02)	<0.01 (0.03)	0.07 (0.05)	-0.01 (0.03)	0.03 (0.03)	0.95 (0.05)	<0.01 (0.02)
Sex Male (ref.)	-	-	-	-	-	-	-	-	-
Female	0.01 (0.28)	-0.20 (0.26)	0.78 (0.67)	-0.06 (0.53)	-0.97 (1.02)	0.85 (0.50)	0.77 (0.65)	2.05 (2.39)	0.05 (0.29)
Duration T2DM	<0.01 (0.02)	<0.01 (0.02)	-0.03 (0.05)	-0.02 (0.03)	-0.05 (0.06)	<0.01 (0.02)	0.11** (0.04)	0.95 (0.06)	0.01 (0.02)
Group status									
Control (ref.)	-	-	-	-	-	-	-	-	-
Intervention	0.22 (0.22)	0.13 (0.24)	0.85 (0.57)	-0.05 (0.45)	-0.55 (0.93)	0.87* (0.49)	-0.88 (0.61)	2.54 (2.83)	-0.08 (0.33)
Education									
High school or no degree (ref.)	-	-	-	-	-	-	-	-	-
Bachelor or grad degree	-0.24 (0.29)	-0.33 (0.25)	0.98 (0.64)	0.40 (0.50)	-0.23 (0.98)	-1.18** (0.50)	-1.31** (0.57)	0.38 (0.52)	0.05 (0.38)
Baseline score	0.54*** (0.14)	0.51*** (0.13)	0.60*** (0.13)	0.53*** (0.10)	0.67*** (0.13)	0.79*** (0.08)	0.33*** (0.13)	21.53** (26.76)	0.94*** (0.09)
Constant	2.79*** (0.90)	2.24*** (0.88)	-1.95 (1.45)	1.39 (1.69)	-1.45 (3.05)	1.36 (1.69)	0.20 (1.87)	1.64 (5.54)	0.18 (1.34)

Note 1: *p<0.1; **p<0.05; ***p<0.01. *Note 2*: Given the small proportion of smokers in my sample, I did not model smoking status as an outcome variable.
[1]Measured immediately after the intervention. [2]Consumption of fruits and vegetables. [3]Consumption of high-fat foods. [4]Combination of two items (walk and specific exercise sessions). [5]Interpreted as a dichotomous variable (no missed dose = 0; missed dose = 1).

5.5 Discussion

The objective of this study was to pilot PERCODIA in a primary care setting. PERCODIA is a nudge-based risk communication tool that aims to improve insight and recall of diabetes-related risks and, in turn, motivate adherence to self-management in poorly controlled T2DM populations. The overarching objective was to inform the development of an adequately powered RCT.

5.5.1 Feasibility and acceptability of the intervention

Primary care is the key point of contact with the healthcare system for individuals with T2DM. [16] Recent studies have shown that GPs play a crucial role in the long-term management of T2DM [17], making routine primary care consultations a logical focus for promoting lifestyle change in this population. The risk communication intervention presented in this study, relying on real-time calculation of personalised risk estimates, was developed for this purpose. As also recently discussed in the literature, the use of health risk calculators should always be accompanied by personalised advice and strategies about how to reduce the risk, regardless of whether the material is personalised or presented in a vivid way. [18] In this regard, the role of the health professional who delivers the risk information is crucial to help individuals impart meaning to the data and use it as a source of motivation and encouragement. [18, 19] Due to a privileged relationship with T2DM patients, I believe that GPs are prime candidates to conduct such interventions.

This study showed a very good acceptability of the intervention. Only one eligible individual did not give consent to be enrolled in the study (98% consent rate). Retention after 12 weeks of follow-up was very high (90%), especially for a study population involving poorly controlled individuals with T2DM. Previous studies have reported that no-show rates to scheduled medical appointments in diabetic populations range from 4% to 40%. [20] Importantly, higher no-show rates are associated with poorer glycaemic control and suboptimal diabetes self-management behaviour. [21, 22] Comparable studies testing risk communication or lifestyle interventions for people with T2DM have reported study retention rates ranging

from 77% to 88% after 12 weeks. [10, 23, 24] In light of these findings, the observed retention rates are promising when designing a future RCT.

PERCODIA was received very positively by participants (average COMRADE score of 4.4 out of 5). No significant difference was found in terms of satisfaction with the consultation between participants receiving the intervention and controls. In particular, the ease of understanding of the personalised risk information received was rated equally high in both groups (4.7 out of 5). These satisfactory results were obtained despite a rise in worry and anxiety levels, due to the communication of heart age, in the intervention group. Indeed, significant differences emerged according to the type of risk information communicated, with effective heart age triggering more worry and anxiety than information on 10-year CV risk score ($p=0.01$) or hours of lifetime lost per day ($p<0.01$). This is in line with what has been reported by Soureti *et al.* (2010) in a study investigating the impact of heart age estimates on risk perceptions and intentions to make lifestyle changes among UK adults with increased cardiovascular disease (CVD) risk. [25] The impact of using heart ages needs to be taken into account when communicating personalised risks and it should be used with caution. In particular, I recommend using alternative formats such as icon arrays to communicate CVD risks to people with anxiety disorders. However, it should also be noted that no significant between-group difference was found in terms of general anxiety after the consultation.

Importantly, PERCODIA was also very well received by the GP who conducted the intervention. Although not formally assessed with the use of a validated questionnaire, his feedback was a main source of information to identify the improvements needed to ensure PERCODIA will be adopted and used on a regular basis by GPs. The key parameter that will determine its regular use in primary care is the time required to run it in routine consultation. In section 5.5.4, I discuss some minor adaptations to PERCODIA that should be carried forward to reduce this time to a minimum and, as a result, favour its adoption and regular use by GPs.

Overall, my results showed that, both in terms of feasibility and acceptability, the PERCODIA intervention is very satisfactory.

5.5.2 Effects of the intervention on risk recall and behavioural outcomes

Although not powered to identify statistically significant effects, this pilot study showed promising results on the impact of PERCODIA on risk recall and on intentions to change behaviour.

First, I found that one of the formats used in the intervention had a very significant effect on risk recall: a very high proportion of participants in the intervention group accurately remembered their effective heart age 12 weeks after the intervention. The idea behind the concept of effective organ age is to map the increased risk onto a characteristic that will be more easily grasped by patients (i.e. increase the emotional impact and representativeness of the risk). [26] To date, the effects on recall of the most effective (non-repeated) risk communication interventions in T2DM populations have lasted between two weeks [10] and six weeks [27] after people received it. Hence, by using heart age estimates, the diabetes risk communicator can expect to double this effect. My results confirmed those observed by Lopez-Gonzalez *et al.* (2015), who showed that using effective heart age increased recalled CVD risk in a general population, as compared to absolute risk score. [28] On the contrary, no significant improvement in risk recall could be attributed to the provision of comparative risk estimates or daily hours of lifetime lost. These results differ from those reported by Galesic and Garcia-Retamero (2011), who found that the consequences of health-related behaviors were recalled better when expressed as changes in life expectancy rather than as changes in risk of diseases. [29] The absence of effect in my study may be due to the use of a daily time frame to express the variations in life expectancy. Despite making the risk more proximal (in an effort to mitigate the effect of present biasedness), this may also have softened the magnitude of the risk as compared to a monthly or yearly period (as used in Galesic and Garcia-Retamero's study. [29]

Second, the intervention had a statistically significant effect on participants' intentions to improve dietary behaviour (ITT analysis with complete cases only). In terms of risk-reducing behaviours, risk perception is a reliable predictor of behavioural intentions. [30] It is likely that the impact of PERCODIA on risk perceptions also translated into a higher motivation to eat healthier. Nevertheless, no significant effect was found on intentions to increase physical activity.

Importantly, despite higher risk perceptions and intentions to eat healthier, I did not observe statistically significant effects on behavioural outcomes as measured by SDSCA score and HbA1c value change. This is likely to be explained by the small sample size of the study, which was not powered to investigate between-group differences in terms of behavioural outcomes. Although evidence is still scarce, previous studies using organ age estimates to motivate behaviour change have reported promising results, both in terms of intentions to make lifestyle changes [25] and behavioural outcomes. [28, 31] Only one study found no superiority of heart age over absolute CVD risk scores. [32] Communication of changes in life expectancy, despite improving risk recall [29], has to my knowledge not yet been tested on risk-reducing behaviour. This is an issue since concepts such as *ageing faster or slower* or *gaining/losing daily hours of lifetime* have potential to improve people's understanding of the consequences of their risky behaviours. [33]

5.5.3 Limitations

This study was conducted at a single-site surgery practice in Oxford, which limits the degree to which the findings can be generalised to the whole T2DM population. However, the local population in this area of Oxford is diverse, both socially and ethnically, and this was reflected in my sample. Although the intervention could be beneficial to all patients with T2DM, the rationale behind the exclusion of people diagnosed with the condition for less than a year was to make sure that each participant was familiar with T2DM self-management. Due to the nature of the study, the underpowered sample size is also a factor limiting the generalisability of the results (I followed Hertzog (2008) [34], who found sample sizes between 20 and

40 sufficient for pilot and feasibility studies). Moreover, as mentioned above, having such a small sample may explain the absence of significant effects on behavioural outcomes.

On the one hand, the follow-up period (12 weeks), selected to allow comparisons with similar studies [10, 23, 27], was short to evaluate changes in lifestyle behaviour and improvements in health outcomes as a response to these changes. On the other, it could be argued that the motivating impact of the intervention may decrease after a few weeks, meaning that reinforcement activities may be necessary to maintain the initial impact and trigger a sustained effect. [28] So far, only repeated risk communication interventions have shown effects longer than six weeks. [23, 35] PERCODIA could be more effective as a long-term intervention (repeated use) in primary care, as the provision of updated risk scores at regular intervals may enhance its impact on behavioural outcomes. It would be interesting to assess if regularly receiving an improved heart age or a gain in life expectancy after the intervention would result in further reinforcement and motivation to adhere to self-management behaviour. Moreover, in order to maximise people's engagement and memorisation, such repeated intervention could be supplemented with the provision, at each visit, of a written support (e.g. one-page printout) summarising the updated risk information. Finally, integrating the intervention into the clinic workflow may also help address clinical inertia ("the tendency to maintain current treatment strategies despite results demanding escalation") in the management of T2DM. [36]

In terms of outcome measurements, two limitations should be mentioned. First, regarding the recall of personalised risk information, individuals in the intervention group had more information content to remember than individuals in the control group (four numbers vs one number, respectively). This could have biased the recall results (memory bias), leading to a lower recall rate in the intervention group. However, the absence of significant between-group difference in terms of recall of 10-year CV risk score, both immediately and 12 weeks after the intervention, suggests that this memory effect was negligible.

Second, regarding the measurement of self-management behaviour, there is uncertainty concerning the best method to use (see Chapter 1). Seven diabetes self-care behaviours have been found to be correlated with improved biometric measures or better health outcomes: healthy eating, physical activity, medication adherence, self-monitoring of blood glucose, problem solving (i.e. prevention, detection and treatment of acute complications), risk reduction activities (i.e. prevention, detection and treatment of chronic complications; e.g. quitting smoking, seeing the doctor regularly, following foot care guidelines), and healthy coping (e.g. seeking individually appropriate support from family/friends and/or medical team). [37] Most instruments that have been developed to assess diabetes self-management are multidimensional, based on these self-care behaviours (see Lu *et al.*, 2016 [38] for a review). Although many different instruments exist (in their systematic review, Lu *et al.* (2016) [38] identified no less than 18), the SDSCA questionnaire has gained the status of a gold standard. [14] Although not free from weaknesses (e.g. there is evidence of unsatisfactory inter-item correlations within the specific diet subscale [14]), it was the only instrument judged positively by three reviews assessing the psychometric properties of diabetes self-management measurement methods. [38–40] Quick, easy to use and efficient, the SDSCA questionnaire is the most commonly used tool to evaluate diabetes self-management today [41] and the one I chose to use in this study. However, in practice, I observed that several participants found it difficult to quantify their daily intake or fruits and vegetables or high-fat food. Consequently, I suggest complementing the use of the SDSCA questionnaire, when possible, with more objective measures of behaviour. Such measures include, for example, plasma vitamin C as a robust measure of fruit and vegetable intake [42], or serum cotinine as a reliable indicator of the quantity of tobacco smoked. [43]

5.5.4 Conclusion

This pilot study provides the first evidence of the feasibility of using PERCODIA, a nudge-based risk communication tool for people with T2DM, in a primary care setting. Based on its results, an adequately powered RCT to determine

the effectiveness of PERCODIA on longer term behavioural and health outcomes of UK patients with poorly controlled T2DM is judged feasible. Importantly, the study allowed to identify some minor adaptations to the PERCODIA intervention and study methods that should be carried forward to conduct a longer term RCT. I recommend the following adjustments:

- Modifying the inclusion criteria: excluding individuals diagnosed with anxiety disorders from the trial.
- Communicating variations in life expectancy associated with self-management in terms of months (over a period of five years, for example) rather than in terms of daily hours.
- Limiting the content of PERCODIA to the communication of effective heart age and loss of life expectancy (i.e. removing the communication of absolute CV risk from the intervention).
- Complementing the self-reported measure of self-management with more objective measures of behaviour.
- Implementing a repeated intervention (with update of the risk scores at each visit) in place of a single intervention and increasing the follow-up period to maximise the chances of triggering a sustained effect on behavioural outcomes.
- Providing participants with a printout of their personalised risk information to facilitate memorisation.

It is worth highlighting that the use of PERCODIA is simple, economical, and not time consuming (between 5 and 10 min per patient). This makes it an ideal candidate to be used in routine primary care consultation with people with T2DM. The personalised risk information is easy and quick to calculate, and expressed in a way that makes it readily understandable by everyone. Provided that its impact on longer term behavioural and health outcomes is positive, its simplicity and ease of understanding also makes the approach a prime candidate to be adapted to other clinical areas.

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6
Conclusion

6.1 Summary of findings and interpretation

Good self-management behaviour is the first-line treatment of Type-2 diabetes mellitus (T2DM). [1] Increased adherence to self-care behaviours, such as balanced diet, regular physical exercise, or medication adherence, has been associated with improved biometric measures (especially glycaemic control) and, as a result, improved clinical outcomes in people with T2DM (see **Chapter 1**). [2] Despite this consensus, a high proportion of people with T2DM remain poorly controlled for they fail to change their behaviour [3–5], and the most effective method to trigger sustained adherence to self-care behaviours in this population is still unclear.

Among the multiple barriers that hamper the adoption of self-care behaviours, inaccurate perceptions of the risks of disease-related complications have been the subject of considerable research. Risk perception is a complex construct that plays a central role in decision-making and behavioural processes. People who underestimate their risks may be less likely to adopt recommended behaviours, while overestimating their risks may cause so much anxiety that it hampers people from taking precautionary measures. [6] Studies which have rigorously investigated the link between risk perceptions and precautionary behaviour have shown a highly significant, positive association (see **Chapter 2**). Bringing patients' risk perceptions into line with their actual risks seems to be a crucial prerequisite for the management of non-communicable diseases (NCD). Given this evidence, I first focused on investigating how people with T2DM perceived their health risks.

In **Chapter 2**, I conducted, to my knowledge, the first systematic review of risk perceptions for diabetes-related complications in T2DM populations. I performed a systematic search of nine electronic databases for peer-reviewed articles published on or before 1 March 2016. Eighteen studies reporting lay perceptions of risks for complications in T2DM populations were identified, and data from these studies were included in my analysis. I found that risk perceptions of people with T2DM are largely biased, characterised by low risk awareness according to most measurement methods. In particular, unawareness of the risks associated with macrovascular events, unawareness of diabetes-related risk factors,

and underestimation of comparative risks (also called *optimistic bias*, i.e. the tendency of people to feel less at risk than their peers) were observed. Despite a high degree of heterogeneity in study designs and measurement methods across selected studies, these results were in line with research in other disease areas and indicated a need for more effective risk communication interventions.

Based on these findings, I focused on the development of an enhanced risk communication intervention for people with T2DM, with the objective of reducing risk misperceptions and motivating sustained adherence to self-management in this population. I use the term *enhanced* because, although improving risk perceptions is necessary to promote behaviour change, the literature has also shown that this was not a sufficient condition (see **Chapter 2**). A wide range of psychological factors, both cognitive and affective, have been found to interact with risk perceptions and influence the way people make decisions. In order to maximise the chances of having an effect on behaviour, these factors have to be considered when designing behaviour change interventions.

The field of behavioural economics, which is concerned with understanding and influencing how people make decisions, offers a promising way to improve behavioural interventions (see **Chapter 3**). Behavioural economics integrates insights from economics, psychology and behavioural science to identify systematic *biases* (i.e. deviations from standard economic rationality) in people's decision-making processes. Targeting these biases to develop more effective policies has raised growing interest, especially in the health domain (see e.g. Luoto and Carman, 2014 [7] for a review). Some of these behavioural economic-informed policies have shown promising results in helping people change their behaviour, including people with T2DM (see Kullgren *et al.*, 2017 [8] for a review).

In recent years, several systematic biases underlying health behaviour have been documented. For example, people tend to discount delayed rewards, such as avoiding cancer or heart disease, relative to more immediate, smaller rewards, such as having a cigarette or an extra dessert now (*present biasedness*). Because diabetes management is centred on people's daily behaviour, many of these biases also apply

to T2DM populations (see e.g. Mørkbak *et al.*, 2017 [9]; Rouyard *et al.*, 2017 [10]; Sultan *et al.*, 2003 [11]) and can be used to inform new interventions.

So far, behavioural economic-informed interventions to promote self-care behaviour have mostly relied on mitigating present biasedness by means of incentives or rewards, with mixed results in the long run (see Chapter 4). However, in the risk communication field, using behavioural economics to inform interventions is not common practice. Although substantial work has been done to test the impact of *framing effects* (the tendency that people have to draw different conclusions from the same risk information, depending on whether it is expressed as gains or losses) on screening or precautionary behaviours (see Gallagher and Updegraff, 2012 [12], Edwards *et al.*, 2001 [13] for general reviews and Kullgren *et al.*, 2017 [8] for a review in T2DM populations), most relevant behavioural economic materials have not yet been targeted. This is an issue since the effectiveness of risk communication may be increased by taking advantage of such materials. [14–16] Existing risk communication interventions for people with T2DM have shown mixed results, with many participants barely understanding the explanations of health professionals about risks and having poor recall of risk information (see **Chapter 2** and Rouyard *et al.*, 2017 [10] for a review). There is an urgent need to improve risk communication in this area, making people with T2DM prime candidates to receive an intervention informed by behavioural economics.

With the objective of developing a *nudge-based* risk communication intervention (see **Chapter 1**), **Chapter 3** focused on exploring the risk attitudes of people with T2DM to identify additional systematic decision-making biases to the optimistic bias identified in **Chapter 2**. Risk attitude, which is the natural tendency of an individual to take or avoid risks, is known to play a key role in the decision-making process. [17–19] In the health domain, risky decisions made by individuals living with a chronic disease may greatly impact their health outcomes. For example, a recent study showed that risk-seeking people with T2DM reported lower general adherence to self-care behaviours. [20] In this context, it was crucial to better understand the characteristics of and factors underlying the risk attitudes

of people with T2DM.

In a brief review of the history of decision theory, I identified prospect theory (PT) as the optimal theory to measure these risk attitudes (see **Chapter 3**). PT also presents the advantage of being built around three biases that are good targets to improve the effectiveness of risk communication: reference-point (RP) dependence[1], probability weighting[2] and loss aversion[3]. Hence, in **Chapter 3**, I designed and conducted a study to measure the risk attitudes under PT of a T2DM population. The study included 120 participants, half of whom were living with T2DM, and was approved by the competent NHS research ethics committees. For the first time, utility under PT was measured using real-life scenario prospects and outcomes that include both life duration and quality of life.

My results showed that people with T2DM express a high degree of risk aversion when facing a trade-off between health outcomes and self-management. I also found evidence of significant loss aversion. This suggested that non-adherence to self-management may not be explained by a particular risk-seeking behaviour in this population. Consequently, this confirmed the need for better risk communication. Including the optimistic bias identified in **Chapter 2**, the work developed in the first part of this Thesis allowed me to identify six relevant decision-making biases to inform a nudge-based risk communication intervention for people with T2DM: optimism, RP dependence, loss aversion, heuristics, bounded willpower and bounded selfishness.

In **Chapter 4**, I developed an innovative, nudge-based risk communication tool entitled PERCODIA to better communicate risks to people with T2DM. PERCODIA calculates personalised risk estimates based on the equations of the UKPDS outcomes model (UKPDS-OM), the validated prognosis model of reference in the T2DM area. [21, 22] So far, the UKPDS-OM has mostly been used to

[1]The tendency of people to evaluate outcomes relative to a RP and then classify them as gains and losses (i.e. in terms of changes relative to this RP).

[2]People tend to overweight small probabilities (less likely events) and underweight large probabilities (more likely events).

[3]People tend to be more sensitive to losses than to gains as seen from the RP, i.e. that the pain of losing a given outcome is psychologically more powerful than the pleasure of gaining the same outcome.

inform health economic evaluations or life expectancy tables [23], but more rarely to directly inform patients through risk communication interventions. PERCODIA was informed by both a thorough review of the literature on risk communication and the behavioural economic evidence gathered in **Chapters 2** and **3**. It is aimed to improve insight and recall of diabetes-related risks and, in turn, motivate sustained adherence to self-management. PERCODIA was developed in collaboration with patients, health professionals, and digital health specialists for use in routine primary care consultation. Primary care being the main contact point with the healthcare system for people with T2DM, these consultations are a logical focus for promoting lifestyle change in this population.

Chapter 5 was dedicated to piloting PERCODIA in a real-life setting. I designed and conducted a pilot randomised controlled trial (RCT) to assess the feasibility of using PERCODIA in routine primary care consultation and evaluate its impact on self-management behaviour in patients with poorly controlled T2DM. Current behaviour change interventions tailored for people with T2DM are often complex and multidimensional (where risk communication is only one part of the intervention), making them hardly compatible with a large-scale use in the primary care setting (see **Chapter 4**). The aim of this study was to inform the potential development of a larger trial to be conducted in the future. The trial comprised 40 participants with poorly controlled T2DM (HbA1c>7.5%) randomly allocated to usual care supported by PERCODIA or usual care only. Recall of personalised risk estimates and self-management behaviour were evaluated before and 12 weeks after the intervention. Other measured outcomes included intentions to make lifestyle changes, emotional impact (anxiety and worry levels), and satisfaction with the intervention. The protocol was approved by the competent NHS research ethics committees. The consent and retention rates of the study were high (98% and 90%, respectively) and participants rated positively both their satisfaction with the intervention (4.4 out of 5 on the COMRADE scale) and the ease of understanding of the information (4.7 out of 5), reflecting a very good acceptability of the intervention. Moreover, although not powered to identify significant effects, this study showed

promising results on the impact of the intervention on risk recall and behavioural outcomes. Recall of personalised risk information after 12 weeks and intentions to change dietary behaviour were significantly improved as compared to controls. Based on these results, an adequately powered trial to determine the effectiveness of the intervention on UK individuals with poorly controlled T2DM was judged feasible. The study also allowed identification of a number of adaptations to the intervention and methods that should be carried forward to conduct this longer term trial.

6.2 Implications of findings

6.2.1 Contribution to literature

This Thesis makes a contribution to research in several areas. A tabulated summary is provided in Table 6.1.

First, the systematic review in **Chapter 2** provides the first evidence-based synthesis of the trends and biases that characterise lay risk perceptions for diabetes-related complications in T2DM populations. The review alerts clinicians and researchers to the lack of risk awareness and highlights the need for better risk communication interventions tailored to this population. I suggested that further research on health risk perceptions would benefit from the emergence of a standardised method to measure perceived risks. Encouraging a certain standardisation in the way researchers measure risk perceptions would provide a reliable way of comparing studies investigating the impact of risk communication interventions. In practice, I would encourage the design of standardised questionnaires assessing multiple dimensions of risk perceptions while generating a single risk perception score. Comparing risk communication interventions through the modification of this risk perception score would add clarity and ease to the assessment of such interventions. In line with the evidence discussed in **Chapter 4**, I also particularly recommend the use of personalised risk estimates informed by validated prognostic models rather than population risk estimates as proxies for actual risks in future

studies.

Second, **Chapter 3** contributes to the decision theory literature by expanding on recent work done in the field. Most studies investigating people's risky choices under PT have measured utility for monetary outcomes. For health outcomes, evidence on people's risk attitudes and decision-making biases is still sparse. This is an issue since significant differences exist between health utility and utility for money (see Attema *et al.*, 2013 [24]). This lack of evidence is worrying since current applications of decision theory in the health domain rely on research done with monetary outcomes (see 6.2.2 for more details). For example, this may partly explain why interventions aiming to nudge people towards healthier behaviour have shown mixed results so far (see Luoto and Carman, 2014 [7] for a review). Investigating risk attitudes and utility for health outcomes is crucial to link theoretical results and actual policy-making.

Third, the Thesis adds evidence to the application of behavioural economics to the healthcare field. In **Chapter 4**, the design of PERCODIA is informed by six decision-making biases specifically identified for this purpose. In health risk communication, targeting decision-making biases to enhance the impact of interventions is not common practice and most biases influencing behaviour change are yet to be exploited (see 6.1). To my knowledge, PERCODIA is the first tool built to do so. This also contributes to the risk communication literature in general, as such approach may be used in areas other than healthcare (e.g. communication of environmental risks).

Fourth, **Chapter 5** provides the first evidence on the feasibility of using PERCODIA in primary care, as part of routine primary care consultations. In addition to being well-received by patients, the use of PERCODIA showed potential improvements on risk recall and intentions to make lifestyle changes.

Finally, and most importantly, this Thesis contributes to a better long-term management of people with T2DM. PERCODIA can provide a valuable support to primary care physicians and diabetologists by facilitating the doctor-patient communication. Using PERCODIA on a regular basis, and setting personalised

goals from one consultation to the other, may enhance the motivation of patients to adhere to self-care behaviours. It will be worth investigating whether the positive effects on long-term self-management behaviour can be confirmed in a larger RCT.

6.2.2 Policy implications

In the health domain, risky decisions made by people living with a chronic disease may greatly impact their life expectancy and QoL. Evidence of risk aversion found in **Chapter 3**'s study suggests that risk seeking behaviour may not explain lack of adherence to self-management observed in T2DM populations. The results rather suggest that in real life non-adherence may be explained, at least partly, by an underestimation of the risks associated with risky behaviour. In **Chapter 2**, it has been shown that people with T2DM largely underestimate their risks of developing complications and that better risk communication interventions are needed. One may have argued that such interventions are doomed to fail because these patients are "naturally inclined to gamble with their health". This study shows that this is not the case, i.e. that if patients had a higher awareness of the risks associated with non-adherence, they may change their behaviour.

However, the age-related difference in risk attitudes found in the study suggests that a different approach is needed for younger T2DM patients. Because of lower risk aversion, such interventions are likely to be less effective in this population. Efforts to develop alternative strategies should be of higher priority, as worrying differences between younger and older T2DM patients have been shown in terms of glycaemic control [25] and onset of complications. [26, 27] This is partly due to lower adherence to self-management [28] (that may be caused by lower risk aversion and present preferences) and partly due to younger-onset T2DM which is more aggressive by nature and thus more challenging to control. [26, 27]

Chapter 3's study also sheds light on behavioural economic information relevant to T2DM populations. In particular, significant loss aversion has been identified. Such information is crucial as a first step towards the development of behavioural economic-informed policies tailored to this population. On a more

theoretical side, this study is the first to provide estimates of PT utility parameters for health outcomes that include both life duration and quality of life. Only Attema and colleagues had previously reported PT estimates for health utility, with one-dimension outcomes. [24, 29] As the authors explain, the lack of research is worrying since the assumptions of PT render various common utility elicitation methods invalid (see 3.3.1 for more detail). In particular, the certainty equivalence (CE), the standard gamble (SG), or the time trade-off (TTO) methods, which are health state valuation techniques widely used for health economic evaluations, do not account for loss aversion or probability weighting. [30] Bleichrodt (2002), for instance, found that loss aversion exerts an upward bias on health state utilities elicited by SG and TTO, that utility curvature causes a downward bias on TTO utilities, and that underweighting of high probabilities often results in SG utilities that are too high. [30] For this reason, eliciting the PT function for health outcomes is important to obtain unbiased estimates of health utility and apply them to reduce the biases inherent to these methods. [24, 31, 32]

Chapter 5 provides preliminary results on the use of PERCODIA in primary care. Although it is too soon to draw any conclusion about its effects on behavioural outcomes in T2DM populations, the results of the pilot RCT are promising and support the implementation of a larger trial to further investigate these effects. I believe that these results will be of interest for health professionals interested in increasing adherence to self-management behaviour, especially general practitioners (GP) and diabetologists, and health policymakers interested in enhancing patient education in primary care. Provided that positive results are found in the larger trial, expansion to other clinical areas where patients would benefit from better adherence to self-care behaviours may be considered.

Table 6.1: Thesis contributions and findings.

Chapter	Originality of contribution	Findings
Chapter 2	The systematic review is the first to provide an evidence-based synthesis of risk perceptions for diabetes-related complications in T2DM populations.	- Risk perceptions of people with T2DM are largely biased, characterised by low risk awareness with most measurement methods. - Risk perceptions of people with T2DM are contaminated by optimistic bias. - Study designs and methods used to assess health risk perceptions are very heterogeneous.
Chapter 3	The study is the first to measure the risk attitudes of people facing a trade-off between self-management behaviour and health outcomes. It is also the first to measure health utility under prospect theory using outcomes that include both life duration and quality of life.	- People show risk aversion when facing a trade-off between self-management behaviour and health outcomes. - The older the individual, the higher the risk aversion coefficient. - People also express significant loss aversion when facing such a trade-off. - Risk aversion observed in the loss domain suggests that the impact of the RP may be less crucial for health outcomes than for monetary outcomes. - Results suggest that non-adherence to self-management may not be explained by risk seeking behaviour.

Table 6.1 Continued: Thesis contributions and findings.

Chapter 4	PERCODIA is the first risk communication tool informed by behavioural economics principles (nudge-based) ever developed.	It is possible to target systematic decision-making biases while communicating personalised health risk estimates, using adapted communication formats and innovative metrics.
Chapter 5	PERCODIA, an innovative risk communication tool tailored to people with T2DM, is assessed for usability in primary care via a pilot RCT.	- PERCODIA is suitable for use in primary care and well-accepted by people with T2DM. - Although the pilot sample is underpowered, the study shows that: (a) Communicating effective heart age in place of absolute risk for CV event significantly improves risk recall after 12 weeks; (b) PERCODIA significantly motivates intentions to improve dietary behaviour. - A larger, adequately powered RCT is judged feasible and is recommended, providing some adjustments are made to the study design and to PERCODIA.

6.3 Limitations and generalisability

6.3.1 Personalised risk information calculated with PERCODIA

Limitations inherent in the UKPDS-OM should not be overlooked when using PERCODIA. Although a large number of people with T2DM were recruited in the UKPDS, patients who participated in the study may not be representative of all people with T2DM living in the UK. For example, participants in the UKPDS were newly diagnosed with T2DM in the 1970s, 1980s and early 1990s, at a time when the number of available therapeutic strategies was more limited than today. As a consequence, despite the satisfactory results reported by studies investigating the internal and external validity of the UKPDS-OM (see **Chapter 4**), the personalised risk estimates calculated by PERCODIA should be interpreted with caution.

6.3.2 Representativeness of study participants

a Elicitation study (Chapter 3)

In the study conducted in **Chapter 3**, putting aside the drawbacks inherent in the elicitation method (see 3.3.6), two limitations should be borne in mind. First, data analysis was limited by the small sample size (n=120, half of whom were living with T2DM). In order to obtain realistic estimates of PT parameters, I could not include older individuals (aged above 60) in the study (see 3.3.4). However, as T2DM usually arises in the late 50s/early 60s, the recruitment of patients aged 60 or less (or even more challenging, 50 or less) was very difficult. This constraint should be taken into account when looking at the final sample size of the T2DM group (n=52). Moreover, a direct consequence is that most subjects in my sample may have been diagnosed with T2DM for less than 10 years[4] (however, those diagnosed for less than a year were excluded to make sure that each participant was familiar with T2DM self-management). Although this may limit the generalisability of the findings, these patients are those for whom behavioural decisions can have the highest impact on

[4]Unfortunately, it was difficult to collect data on duration of diabetes, as some of the patients were not registered at the surgery practice at time of diagnosis.

health outcomes (they can still prevent the onset of complications). Hence, in terms of policy-making, this is potentially the most interesting subgroup. Despite small sample size, efforts were made to minimise the risk of selection bias by randomly recruiting age-matched controls. No significant differences were found in terms of age, sex, education level, employment status, or answer reliability between patients and controls.

Second, the recruitment took place at a single-site surgery practice in Oxford, UK. Although this certainly limits the external validity of the results, the diabetic clinic of this surgery practice treats patients from many different neighbourhoods of the local urban area. As a result, its regular T2DM population is very diverse, both ethnically and socio-economically. This was reflected in the demographic and socio-economic characteristics of the study participants (see 3.3.5). Diversity was also observed in the group of individuals excluded from the analysis because of answer unreliability, which minimises the risk of selection bias based on higher cognitive ability and provides support for the elicitation method used in this study.

b Pilot RCT (Chapter 5)

The pilot RCT conducted in **Chapter 5** shares common limitations with the elicitation study in **Chapter 3**. It was conducted at the same primary care practice, hence the drawbacks associated with a single-site recruitment have to be considered. It is also worth mentioning that patients diagnosed with T2DM for less than a year had to be excluded from the study to guarantee that all participants were familiar with T2DM self-management. This exclusion criterion was necessary so that self-management outcomes could be reliably collected, but does not mean that newly diagnosed patients could not also benefit from the use of PERCODIA. Indeed, I would recommend using the tool early in the management process to facilitate the set-up of quantifiable self-management objectives.

6.3.3 Generalisability of the results

a Elicitation study (Chapter 3)

In light of the findings, and despite the sample limitations discussed above, I believe that the risk attitudes observed and the biases measured in the elicitation study are generalisable to T2DM populations and, to a greater extent, populations diagnosed with any *manageable* chronic disease (i.e. a chronic disease that requires adherence to self-management behaviour). In the study, the hypothetical risky trade-off between self-management and health outcomes used to measure people's risk attitudes was designed to fit the management of any condition requiring self-management behaviour, not specifically T2DM (see 3.3.4). Lack of adherence to self-management has been observed with other chronic disease populations (e.g. obesity [33], arthritis [33], chronic obstructive pulmonary disease [34]). Because the barriers to self-management are similar, it is likely that the risk attitudes and biases underlying the decision-making processes of these populations are, at least in part, similar to the ones identified in this study. In older populations, however, more caution must be taken about generalising these results. As mentioned above, the elicitation method did not allow me to include people above 60 in the study. Moreover, I observed a very significant age effect on risk attitudes (controlling for group status and other covariates), with younger individuals being more risk-seeking on average. Other studies have also reported significant differences in risk-taking across the adult life span. [35–37]

b Pilot RCT (Chapter 5)

Due to the nature of the study, the underpowered sample size (n=40) limits the generalisability of its results. However, the main objective of the study was to assess the feasibility of using PERCODIA in primary care, rather than providing generalisable findings of the effect of PERCODIA on behavioural outcomes. The pilot RCT allowed identifying a certain number of limitations inherent in the study design and methods (see 5.5). Adjustments to the intervention and study methods that should be carried forward to conduct the definitive RCT have been

recommended accordingly.

6.4 Further research

6.4.1 Uncertainty in risk perceptions

In **Chapter 2**, the systematic review of studies investing the risk perceptions for diabetes-related complications in T2DM populations highlighted the fact that none of these studies reported uncertainty ranges along with the elicited perceived risks. Eliciting uncertainty ranges around patient risk perceptions would add valuable information to assess the gap between perceived and actual risks. For the risk communicator, this may also provide additional levers for action to design more effective interventions.

6.4.2 Reference point in health domain

In **Chapter 3**, one finding of the elicitation study is that people express risk aversion in both the gain and the loss domain for health outcomes. This is in contrast with the results of studies that measured utility under PT for monetary outcomes, in which people tend to be risk averse for gains and risk seeking for losses.[5] [41–43] These results would suggest that the role of the RP is less crucial for health than for money. However, another possibility is that, despite making the reference point as salient as possible in the instructions for participants (use of visual aids), people's own idea of how long they may live (*personal* RP) was still stronger than the induced time horizon (*artificial* RP) (see **Chapter 3** for more details). More research that explicitly investigates the role of the RP in the health domain is needed.

Moreover, because of the study limitations, these results must be put into perspective. In particular, I used only one single probability (p=0.5) in the prospects, which prevents conclusions from being drawn about the decision weights attached

[5] Although the evidence is sometimes a little mixed, especially because of the interaction between the PT value function and the probability weighting function [38, 39], see Oliver (2018) for a review of the empirical support for the reflection effect. [40]

to other probabilities (see discussion about the reflection effect in 3.3.1). At first sight, my results undermine the validity of PT because they challenge the traditional S-shaped curve of the PT value function. However, it seems that PT does not mandatorily exclude (mild) risk aversion in the loss domain over mid-range probabilities. Over mid-range probabilities, it is not clear whether PT predicts people to be risk seeking for losses (as over large probabilities) or risk averse for losses (as over small probabilities). It is also possible, as suggested by Oliver (2018), that utilities elicited through binary choice tasks are more *reasonably consistent* with EUT than utilities elicited through pricing tasks. [40] However, because of robust loss aversion found in this study, I would not go so far as to say that EUT fits my results better than PT. More research is needed to investigate the reflection effect in the context of risky choices incurred by the management of chronic disease.

6.4.3 Risk attitudes and decision-making biases

Importantly, further research investigating the risk attitudes and decision-making biases of patients is needed. Risk attitudes of older people, for example, may differ from those identified in this study. If so, the most appropriate risk communication strategy to be adopted with this population would need to be fine-tuned. Further research also needs to be conducted with people living with other types of chronic disease, so that future health promotion interventions can be systematically tailored to specific patient populations. In particular, the exploration of decision-making biases not yet identified in the health domain - such as the projection bias[6] or the Peltzman effect[7] - would make an important contribution to both the theoretical and applied behavioural sciences literature. Finally, investigating the decision-making biases that influence the behaviour of health professionals may also be considered. For example, the *curse of knowledge* phenomenon (i.e. the bias that occurs when an individual unknowingly assumes that other individuals with whom they are communicating have the background to understand their explanations), described

[6] People's assumption that their tastes or preferences will remain the same over time. [44]

[7] The tendency of people to adjust their behavior in response to the perceived level of risk, becoming more careful where they sense greater risk and less careful if they feel more protected. [45]

by Camerer *et al.* (1989) [46] in economic settings, may be targeted in doctor populations to improve the doctor-patient communication.

6.4.4 PERCODIA: further development

PERCODIA can be improved, both in terms of content and in terms of format. In terms of content, it would be ideal to benefit from the original UKPDS-OM equations to calculate patients' personalised risk estimates. As discussed in **Chapter 4**, running the current version of the UKPDS-OM is too time consuming to be used in clinical practice. As a result, PERCODIA's risk estimates are based on two simpler forecasting models that allow real-time calculation in primary care by approximating the UKPDS-OM risk estimates. The development of a simpler, more tractable version of the UKPDS-OM would be very useful for this purpose. Moreover, updated data collected on current T2DM populations in the UK are needed to calculate more accurate personalised risk estimates. In addition to the representativeness issues mentioned in 6.3.1., the PERCODIA risk estimates result in a positive correlation between BMI and life expectancy (see Appendix H), although BMI is positively associated with risk of CV events in the original UKPDS-OM equations. As a result, it is delicate to include BMI as a modifying risk factor for the communication of changes of life expectancy associated with self-management behaviour.[8] Given the importance of weight loss in the management of T2DM, such data would allow showing patients how changes in BMI impact their personalised risks and hence significantly improve the tool.

In terms of format, PERCODIA may benefit from the work of a webdesigner specialised in digital health to make all features perfectly patient-friendly and interactive. For example, sliders could replace the drop-down menus on the output pages, and fonts and colours may be adjusted. Importantly, a key improvement would be to directly link PERCODIA to the GP database, so that the input page can be automatically filled out with patients' personal information before starting

[8] As the objective of the tool is to encourage self-management behaviour in T2DM populations, which often includes weight loss, it is hardly conceivable to communicate improved outcomes associated with weight gain.

the intervention. This would save considerable time in practice and improve the reliability of the calculated risk information.

6.4.5 Larger RCT to confirm findings from pilot study

The results of the pilot study in **Chapter 5** are favourable to the development of a larger, adequately powered RCT. They show that using PERCODIA in routine primary care with poorly controlled people with T2DM is not only feasible, but also favourably experienced by these patients. Promising results on risk recall and intentions to make lifestyle changes have been observed. Provided that a few adjustments to the study design and methods are made (see **Chapter 5** for more details), it is worth investigating the effect of PERCODIA on long-term behavioural outcomes in this population through the implementation of a larger RCT. In the event that significant, positive effects on long-term self-management behaviour are identified, expansion to other patient populations would also be worth considering.

6.4.6 Populations with anxiety disorders

Further research is needed on how best to communicate personalised risk estimates to people with anxiety disorders. The pilot RCT showed that the effective heart age format may increase anxiety and worry levels, which is why alternative formats should be considered for this patient population.

6.5 Ethical considerations

Over the past 10 years, public health interventions informed by behavioural economics have been increasingly used to promote healthier lifestyles and to reduce the health care costs of obesity and NCDs. In particular, nudge-based interventions, which are grounded on an appealing philosophical framework (libertarian paternalism, see Chapter 1) and have a catchy name, have attracted a lot of attention. Although there has been growing evidence of their effectiveness in contributing to reach these objectives (see extended overview by Sunstein and Reisch, 2017 [47]),

the use of nudges has also raised ethical concerns in the literature.

Nudges have been defined by Sunstein and Thaler as *choice-preserving* interventions that steer people's behaviour in specific directions while not impeding them from going their own way (libertarian paternalism, see Chapter 1). [48] They are supposed to affect choices using neither rational persuasion nor restrictive means (like standard economic regulations such as like mandates or fines) and cannot include lies, deceit, subliminal advertising, or other powerful manipulation. [49] According to Sunstein and Thaler, nudges are aimed to make the *better option, as judged by the decision-maker*, more convenient or salient to select - i.e. to make the decision-maker *better off, as judged by himself* - without jeopardising his freedom of choice.

Below I discuss some of the criticisms that have been voiced about this claim of welfare, mostly based on the work of Robert Sugden. What is the better option, as judged by the decision-maker? How to identify it? Do nudges really make people better off as jugded by themselves? Sugden contests this statement and discusses the validity of this "as judged by themselves" (AJBT) criterion. He challenges both the legitimacy of the paternalistic framework [50] and the AJBT criterion in the context of health nudges. [51, 52]

6.5.1 Critique of the paternalistic framework

Paternalistic frameworks go against what became the cornerstone of normative economic analysis in the 20th century: welfare economics, i.e. a welfarist approach to normative analysis. Welfare economics is concerned with the assessment of social welfare, i.e. the evaluation of (economic) policies in terms of their effects on the well-being of the society. From its standpoint, social well-being is taken as the sum of all individual well-being in the society. Importantly, welfare economists traditionally rely on standard economic theory (axioms of van Neumann-Morgenstern (vNM) rationality, see Chapter 3) to analyse decisions at the individual level. In this context, individuals' own judgements are considered to be decisive in assessing the relative well-being that those individuals derive from alternative options. [50]

However, as discussed in Chapter 3, the findings of behavioural economics from the second half of the 20th century have shed light on systematic deviations from standard economic theory revealed by individuals when making decisions. While standard economic theory assumes that people have *stable* preferences (as part of the standard vNM rationality), behavioural economists have shown that in real life people's preferences can be context-dependent or inconsistent (e.g. sensitive to framing effect). In other words, people's preferences can sometimes be unstable, or *incoherent*, from a standard economic perspective.

As Sugden (2008) notes, this poses a problem for the welfarist approach to policy-making. How to interpret a "standard of well-being" for individuals whose judgements reveal incoherent preferences? How to design policies that maximize social welfare in this context? [50]

Behavioural economists have developed paternalistic frameworks as a response to this policy-making challenge. Under such frameworks, despite some variations in the degree of freedom left to the decision-maker (from Sunstein and Thaler's libertarian paternalism to Conly's coercive paternalism), the policy-maker end up playing a key role in interpreting this "standard of well-being" (see next subsection for more detail on this point).[9] The rationale is as follows: if people do not come to a decision problem with pre-existing preferences, i.e. if their preferences are incoherent, paternalism cannot be avoided because it is not possible to respect those preferences. Based on this observation, the policy-maker is "forced" to steer the individual's choice towards a given direction. This direction is the one that *he* (the policy-maker) favours, the one that *he* thinks is the best decision for the individual.

Interestingly, Sugden contests this approach and argues that it is possible to develop viable, alternative frameworks to paternalism that incorporate findings of behavioural economics. Without going into details, he brings into question the rationale behind the "social planner approach" traditionally used in welfare

[9]Whether this subjective interpretation is acknowledged or not by the advocates of soft paternalism. For example, Sunstein and Thaler claim that, if an individual's preferences are incoherent, paternalism is "inevitable". [53]

economics. Such an approach puts the policy-maker in the shoes of an omniscient social planner (or "benevolent despot") concerned with optimizing the allocation of resources to maximize social welfare given each individual's preferences. Instead, he advocates for a more "Hayekian" viewpoint[10] of normative economics which would require everyone to be responsible for promoting his/her own good. Resources would then be allocated between individuals based on voluntary exchange and mutual advantage. Structuring the economy in this way would, according to Sugden, allow findings from behavioural economics to be incorporated in a normative theory of decision-making, such as incoherent preferences would not be considered as errors *per se*. [50]

The fact that seemingly incoherent preferences are currently treated, by most behavioural economists, as *errors* of the decision-making process, poses some fundamental problems. These problems are discussed in the next subsection.

6.5.2 Critique of the behavioural welfarist approach to normative economics (the "better off as judged by themselves" criterion)

According to Thaler and Sunstein's definition of the nudge, the AJBT criterion implies a welfarist-like approach to normative economics. The policy-maker (the social planner) is supposed to be able to identify what's best for each individual (what choices maximise their well-being), even though people's actual choices do not always reveal it.

The idea is that, in real life, people sometimes make "bad decisions" due to a series of decision-making errors (e.g. present bias, see Chapter 3), and that such errors affect their well-being. Had these errors been corrected, i.e. had people's underlying *true* preferences not been contaminated by these errors, it is assumed that better decisions would have been made and, as a result, that people's well-being would have been increased. Sunstein and Thaler refer to these bad decisions as "inferior decisions in terms of their [i.e. the individuals'] own welfare". [53]

Therefore, from this viewpoint, the policy-maker's task is to reconstruct

[10] From Friedrich Hayek, laureate of the Nobel Prize in Economics in 1974.

individuals' true ("latent") preferences so as to identify (and favour) the choices that would maximise people's welfare. This approach is referred to as *preference purification*. [54] The criterion of individual well-being is not the preferences revealed by the individual in his choices, but the ones that would have been revealed had his choices not been contaminated by decision-making errors.

a The latent preference issue

For preference purification to provide an acceptable welfare criterion, latent preferences need to be *themselves stable*. Because the satisfaction of latent preferences is taken as the normative criterion by defenders of nudges, they need to be consistent and context-independent, i.e. to satisfy the axioms of standard economic rationality. Although this assumption seems to be accepted by many behavioural economists, the question of its validity remains.

Sugden and colleagues' argument is as follows. If latent preferences are a subjective concept, i.e. if they are "judgements that are formed within the minds of human beings", then they "do not correspond directly with objective properties of the external world". This supposes that people are capable of producing a mode of *latent reasoning* that generates subjective preferences that comply with standard economic theory principles. However, because there is no evidence of how such a mode of reasoning works, the validity of the preference purification approach endorsed by defenders of nudges is *de facto* limited.

Indeed, how to be sure that the satisfaction of purified preferences would maximize people's welfare AJBT?

On this point, Hausman (2011)'s contribution is interesting. [55] He argues that theories of well-being that rely on the satisfaction of preferences (even stable) are not tenable. Preference satisfaction only "usually contributes to well-being [...] because preferences provide reliable information about well-being". Hausman asserts that, for some choices, even "sound" decision-making processes (in the standard economic theory sense of the term) is unable to determine a preference ranking

(violation of the completeness axiom). A direct implication of this assumption is that what seems to be suboptimal choices from a standard economic standpoint might not be errors *per se* that can be corrected by preference purification.[11]

b Assuming that latent preferences exist, how to elicit them?

The preference purification approach underpins Sunstein and Thaler's model of the *inner rational agent* [54] that is used to justify nudge policies. In this model, rational decision-making is affected by a wide range of errors (decision-making biases) resulting from human psychology. "The rational inner agent is trapped inside and constrained by an outer psychological shell."[12] Normative analysis in this context is thought of as an attempt to reconstruct and respect the preferences of this inner rational agent.

In some cases, such an approach could be regarded as fairly uncontroversial. Infante *et al.* (2016) gives the example of a unique product priced differently by competing suppliers (according to different tariffs). It has been shown that consumers often fail to choose the cheapest option when tariffs are complex. [57] However, "it seems unexceptionable to assume that, for any given quantity bought, consumers have an underlying preference for paying less rather than more." [54] Hence, representing such choices as mistakes, defined relative to latent preferences for low prices, may be legitimate.

In some other cases, however, this model is problematic because no best solution (no "most rational solution") can be derived from the choice problem. These are cases, for instance, where the completeness of preferences axiom cannot be satisfied. Sugden and colleagues discuss Sunstein and Thaler's famous cafeteria example [48], in which people's choices between food items (e.g. apples or cakes)

[11] As pointed out by Kahneman (1996) himself, *rational choice is not self-explanatory*: cases in which behaviour is consistent with the standard economic theory are just as much in need of psychological explanation as are deviations from that theory.

[12] This model echoes Kahneman's idea that the workings of the mind can be separated into two 'systems' – the fast and automatic System 1 (the psychological shell) and the slow and reflective System 2 (the inner rational agent). [56]

may vary according to the display chosen by the cafeteria director.[13]

Assuming that (coherent) latent preferences do exist, the decision-making process of the inner rational agent implies that those preferences are complete. However, how to elicit these preferences? How to know whether the inner rational agent prefers eating an apple or a cake at the cafeteria? In fact, choices can reasonably be interpreted as errors only when these errors "*equate an individual's subjective ranking of options* (e.g. different tariffs) *with an objective ranking* (e.g. in inverse order of their prices) *that is independent of the consumer's perceptions or judgements*." There is no obvious analogue to this objective ranking in cases such as the choice between eating an apple and eating a cake. For these cases, "it is not at all obvious which (if any) of these choices is correct – or even how 'correctness' should be defined."

Sugden's conclusion is that latent preference is not a useful concept for normative economics. The fact that many policy-makers have been using it may result from the misconception that standard economic theory accounts for the reasoning behind people's choices, when it merely provides a first approximation to these choices.

Hence, rather than presenting nudges as an instrument to "correct for people's errors" or "make people better off AJBT", Sugden recommends policy-makers to acknowledge that they aim to "regularize revealed preferences that are inconsistent with standard economic theory." In other words, to acknowledge that they aim to make individuals better off as judged by the 'choice architect' who implements the nudge, i.e. the policy-maker.

6.5.3 Transparency and democratic acceptance

Discussion on the legitimacy of nudges has also focused on issues of transparency and democratic accountability. [59, 60] As opposed to legal or fiscal instruments, there is a risk that nudges may be manipulative, or at least insufficiently transparent.

[13] People tend to choose items that are put in easier-to-reach places. A nudge that aim to make people eat healthier would then consist in placing healthy foods at eye level while putting less-healthy junk food in harder-to-reach places (however, there is mixed evidence about the effectiveness of such interventions, see e.g. review by Wilson *et al.* (2016). [58]

Defenders of nudges have argued that transparency about their mechanisms of action neither affects their acceptance, nor their effectiveness [61–63] and therefore may not be incompatible with their use. [64, 65]

Putting aside policies that are mistakenly referred to as nudges (i.e. policies that are labelled as nudges but do not satisfy the criteria defining by the libertarian paternalism space; see Chapter 1 and Oliver, 2015 [66] for examples), a question remains whether classic nudges are really accepted by the people they target.

In his response to Sugden's critique of nudging (see 6.5.2), Sunstein (2018) asserts that the response to the question "Do people really want to be nudged toward healthy lifestyles?" is "Yes", given that in numerous countries - including the USA, the UK, Germany, France, Italy and Australia - strong majorities endorse nudges towards healthy lifestyles, including when people are specifically asked about whether they *themselves* would like to be nudged (not only whether they approve of nudges in general). [67]

More specifically, citizens (1) generally approve of nudges, unless they perceive them to be inconsistent with the interests or values of most choosers [68]; (2) do not approve of nudges that are perceived as having an illicit goal (e.g. religious or political favoritism) [69]; (3) object to manipulation, but only in very specific cases (e.g. subliminal advertising). [68] Importantly, when the objective is to promote health-related behaviours, the concept of nudging tends to be largely supported. [70–72]

Although I fully agree with Sugden's concerns regarding the validity of the AJBT criterion, including his critique of Sunstein's counterarguments on the matter [52], I tend to follow Sunstein (2016) when he notes that in a democratic society[14], the final word should anyway lie with public officials who represent the citizens' interest. [69] If strong objections against nudges emerge in the population, measures should be taken to further reassess their acceptability.

[14]There would be much to say about whether representative democracies can truly be considered as democratic, but this is another debate.

6.5.4 Is PERCODIA a nudge *per se*?

As discussed in Chapter 1, it can sometimes be challenging to tell whether an intervention is a nudge or not. Oliver (2015)'s diagram allows policy-makers to assess the nature of an intervention according to three criteria (represented by three axes) that define the libertarian paternalism space [66]:

- The behavioural-rational axis, i.e. whether the intervention is informed by behavioural or standard economics' insights.
- The liberty-regulatory axis, i.e. whether the intervention preserves the freedom of choice or not.
- The internalities-externalities axis, i.e. whether the intervention addresses internalities or externalities.

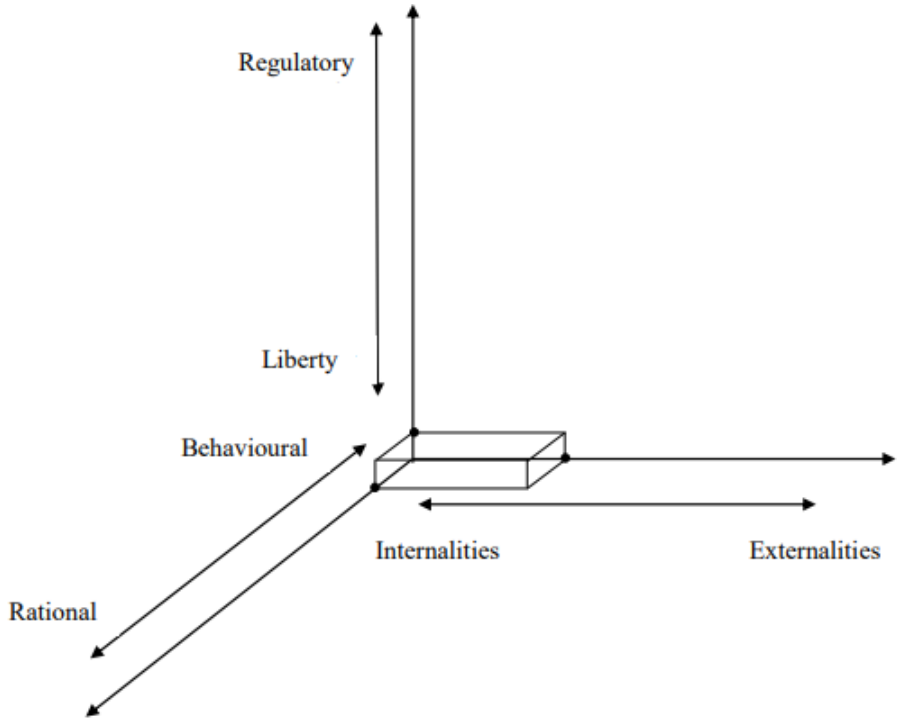
A classic nudge will be located at the centre of the diagram: it is a liberty-preserving behavioural economic-informed policy that intends to address negative internalities. A legitimate question is to ask whether PERCODIA satisfies these criteria.

In designing PERCODIA, my objective was dual. On the one hand, I aimed to address the negative internalities expressed by people with poorly controlled T2DM. My intention was to help patients whose deliberate preferences indicate that they would adhere to a healthy lifestyle if they could, but who, for some reason, fail to do so. On the other hand, a major motivation was to contribute to alleviating the financial burden to the healthcare system caused by uncontrolled diabetes, i.e. addressing associated negative externalities.

In that respect, and in the vein of health nudges in general, PERCODIA may not be a *pure*, classical nudge as defined by Oliver's criteria. It would rather lie further away from the origin on the internality-externality axis, as shown in Fig. 6.1 (arbitrarily judged by me).

PERCODIA relies on loss aversion, heuristics, and overconfidence to steer patients' choices towards improved adherence to self-management. It is therefore a behavioural economic-informed intervention that does not impose any constraints (in the standard economic sense of the term) on those who receive it. Assuming that

Figure 6.1: PERCODIA in Oliver's libertarian paternalism space (2015). [66]



patients with poorly controlled T2DM do wish to change their behaviour (it is likely that some of them do not wish to do so, see 6.5.2 for more detail about latent preferences), it is also meant to address negative internalities. However, the motivation stemming from the externality argument - saving money for the National Health Services (NHS) - cannot be denied. Hence, in terms of satisfaction of the three criteria, PERCODIA conforms reasonably closely to the characteristics of a nudge.

A few words on the externality argument are worthy of consideration. In a society like the UK, where public universal coverage implies that health care costs are borne by all members of the society, the social contract justifies in my opinion the use of policies to address the negative externalities imposed on others by people with poorly controlled T2DM. Behavioural economic-informed interventions such as PERCODIA seem to be justified according to the interests of those who bear the costs of the healthcare system. Such a policy is more problematic in societies where private insurance companies or managed care organizations already account for people's risk factors to set up bespoke premiums and deliver care accordingly. In that

case, inducing behaviour change in situations in which the targeted individual does not seem to be making mistakes in satisfying his own preferences (or in maximizing his own welfare) is harder to justified.

Finally, I note that PERCODIA could potentially be subject to ethical concerns associated with health nudges that use salience and affect effects. There is a risk that such interventions be perceived by patients as scare tactics and, as a result, alienate them from the physician. [73] This will then have a negative impact on the mutual trust and therapeutic alliance between the doctor and the patient. In order to minimise that risk, it is important that the information being represented saliently is as true and accurate as possible, and not exaggerated or misrepresented. This is the case of PERCODIA, which relies on a validated prognosis model of reference to deliver the risk information.

In light of these elements, I believe that health nudges such as PERCODIA should usually be encouraged as a complement to standard economic policies. They constitute a cheap, easy-to-implement, and generally approved weapon to fight lifestyle-related chronic diseases that undermine the healthcare system in societies where health coverage is public and universal.

6.6 Concluding remarks

Diabetes will become the 5th leading cause of death worldwide by 2025. [74] If the trend is not reversed, 1 in 10 people will live with the disease in 2040. [75] T2DM, which accounts for around 90% of total diabetes cases in high income countries [75], has the potential to be well-controlled - and in most cases even prevented – by adhering to self-management behaviour. Despite substantial efforts made to improve self-management in this population, a high proportion of people with T2DM fails to change their lifestyle and remains poorly controlled. The work developed in this Thesis aimed to add new weapons to the arsenal of measures available to fight this trend. Health nudges present a formidable opportunity to improve the health of populations living with manageable chronic diseases. PERCODIA is a cheap,

easy-to-implement tool that can help primary care physicians to nudge patients towards sustained adherence to self-management. Hopefully a larger RCT will be developed to assess its long-term impact. Given the challenge in improving self-management behaviour, any positive effect, even moderate, would greatly benefit both patients and the healthcare system.

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Appendices

A

Dissemination of research

Published manuscripts

Rouyard, T., Attema, A., Baskerville, R., Leal, J. & Gray, A. Risk attitudes of patients with 'manageable' chronic disease: an analysis under prospect theory. *Social Science & Medicine* 214, 144-153 (2018).

Rouyard, T., Leal, J., Baskerville, R., Velardo, C., Salvi, D. & Gray, A. Nudging people with Type 2 diabetes towards better self-management through personalised risk communication: a pilot randomized controlled trial in primary care. *Endocrinology, Diabetes & Metabolism*, e00022 (2018).

Rouyard, T., Kent, S., Baskerville, R., Leal, J. & Gray, A. Perceptions of risks for diabetes-related complications in Type 2 diabetes populations: a systematic review. *Diabetic Medicine* 34, 467–477 (2017).

Oral presentations

Rouyard, T. *et al.* Nudging people with Type-2 diabetes towards better self-management through personalised risk communication: a pilot randomised trial in primary care. *NIHR CLAHRC Self-Management Theme, Nuffield Department of Primary Care Health Sciences, University of Oxford, Oxford, October 2017.*

Rouyard, T. *et al.* Risk attitudes of patients with 'manageable' chronic disease: an analysis under prospect theory. *12th World Congress of the International Health Economics Association, Boston University, Boston, July 2017.*

Rouyard, T. *et al.* Risk attitudes of patients with 'manageable' chronic disease: an analysis under prospect theory. *Health Economics Seminars (Invited Speaker), Erasmus University, Rotterdam, February 2017.*

Rouyard, T. *et al.* Type-2 diabetes and risks for complications: risk perceptions, risk attitudes. *DPhil Seminar Series, Green Templeton College, University of Oxford, Oxford, May 2016.*

Rouyard, T. *et al.* Type-2 diabetes and risks for complications: risk perceptions, risk attitudes. *Health Economic Research Centre Seminar Series, University of Oxford, Oxford, October 2015.*

Rouyard, T. *et al.* Perceptions of risks for diabetes-related complications in Type 2 diabetes populations. *NIHR CLAHRC Self-Management Theme, Nuffield Department of Primary Care Health Sciences, University of Oxford, Oxford, February 2015.*

B

Search strategies

Table B.1: MEDLINE (via OvidSP) 1946 to February week 4, 2016.

#	Search terms
1	(*risk*$ OR *susceptib*$ OR *vulnerab*$ OR *likel*$) ADJ3 (*perce*$ OR *aware*$ OR *estimat*$)
2	"*type 2 diabet*$" OR "*type-2 diabet*$" OR "*type-2-diabet*$" OR "*t2dm*" OR "*tiidm*" OR "*niddm*" OR "*non insulin dependent diabet*$" OR "*noninsulin dependent diabet*$" OR "*non-insulin dependent diabet*$"
3	1 AND 2

Table B.2: Embase (via OvidSP) 1974 to February week 4, 2016.

#	Search terms
1	(*risk*$ OR *susceptib*$ OR *vulnerab*$ OR *likel*$) ADJ3 (*perce*$ OR *aware*$ OR *estimat*$)
2	"*type 2 diabet*$" OR "*type-2 diabet*$" OR "*type-2-diabet*$" OR "*t2dm*" OR "*tiidm*" OR "*niddm*" OR "*non insulin dependent diabet*$" OR "*noninsulin dependent diabet*$" OR "*non-insulin dependent diabet*$"
3	1 AND 2

Table B.3: Global Health (via OvidSP) 1973 to February week 4, 2016.

#	Search terms
1	(*risk*$ OR *susceptib*$ OR *vulnerab*$ OR *likel*$) ADJ3 (*perce*$ OR *aware*$ OR *estimat*$)
2	"*type 2 diabet*$" OR "*type-2 diabet*$" OR "*type-2-diabet*$" OR "*t2dm*" OR "*tiidm*" OR "*niddm*" OR "*non insulin dependent diabet*$" OR "*noninsulin dependent diabet*$" OR "*non-insulin dependent diabet*$"
3	1 AND 2

Table B.4: PsycINFO (via OvidSP) 1967 to February week 4, 2016.

#	Search terms
1	(*risk*$ OR *susceptib*$ OR *vulnerab*$ OR *likel*$) ADJ3 (*perce*$ OR *aware*$ OR *estimat*$)
2	"*type 2 diabet*$" OR "*type-2 diabet*$" OR "*type-2-diabet*$" OR "*t2dm*" OR "*tiidm*" OR "*niddm*" OR "*non insulin dependent diabet*$" OR "*noninsulin dependent diabet*$" OR "*non-insulin dependent diabet*$"
3	1 AND 2

Table B.5: SCOPUS (all years to 2016).

#	Search terms
1	(*risk** OR *susceptib** OR *vulnerab** OR *likel**) W/3 (*perce** OR *aware** OR *estimat**)
2	"*type 2 diabet**" OR "*type-2 diabet**" OR "*type-2-diabet**" OR "*t2dm*" OR "*tiidm*" OR "*niddm*" OR "*non insulin dependent diabet**" OR "*noninsulin dependent diabet**" OR "*non-insulin dependent diabet**"
3	1 AND 2

Table B.6: EconLit (all years to 2016).

#	Search terms
1	(*risk** OR *susceptib** OR *vulnerab** OR *likel**) NEAR/3 (*perce** OR *aware** OR *estimat**)
2	"*type 2 diabet**" OR "*type-2 diabet**" OR "*type-2-diabet**" OR "*t2dm*" OR "*tiidm*" OR "*niddm*" OR "*non insulin dependent diabet**" OR "*noninsulin dependent diabet**" OR "*non-insulin dependent diabet**"
3	1 AND 2

Table B.7: IBSS (all years to 2016).

#	Search terms
1	(*risk** OR *susceptib** OR *vulnerab** OR *likel**) NEAR/3 (*perce** OR *aware** OR *estimat**)
2	"*type 2 diabet**" OR "*type-2 diabet**" OR "*type-2-diabet**" OR "*t2dm*" OR "*tiidm*" OR "*niddm*" OR "*non insulin dependent diabet**" OR "*noninsulin dependent diabet**" OR "*non-insulin dependent diabet**"
3	1 AND 2

Table B.8: Web of Science (all years to 2016).

#	Search terms
1	(*risk** OR *susceptib** OR *vulnerab** OR *likel**) NEAR/3 (*perce** OR *aware** OR *estimat**)
2	"*type 2 diabet**" OR "*type-2 diabet**" OR "*type-2-diabet**" OR "*t2dm*" OR "*tiidm*" OR "*niddm*" OR "*non insulin dependent diabet**" OR "*noninsulin dependent diabet**" OR "*non-insulin dependent diabet**"
3	1 AND 2

C
Study quality

Table C.1: Study quality: studies assessed using the CASP.

Authors	Aim	Methodology	Design	Recruitment	Data collection	Relationship	Ethics	Analysis	Findings	Value	Global
Carroll et al (2003)	+	+	+	+	+	0	+	+	+	+	+
Macaden & Clarke (2006)	+	+	+	0	+	-	+	+	+	+	-
McKenzie & Skelly (2010)	+	+	+	+	+	0	+	+	+	+	+
Price et al (2009)	+	+	+	+	+	+	+	+	+	+	+
Sum good (%)	4 (100%)	4 (100%)	4 (100%)	3 (75%)	4 (100%)	1 (25%)	4 (100%)	4 (100%)	4 (100%)	4 (100%)	3 (75%)
Sum fair (%)	0 (0%)	0 (0%)	0 (0%)	1 (25%)	0 (0%)	2 (50%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Sum poor (%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (25%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (25%)

Note (a): '+': Good; '0': Fair; '-': Poor.

Note (b): Aim: 'Was there a clear statement of the aims of the research?'; Methodology: 'Is a qualitative methodology appropriate?'; Design: 'Was the research design appropriate to address the aims of the research?'; Recruitment: 'Was the recruitment strategy appropriate to the aims of the research?'; Data collection: 'Was the data collected in a way that addressed the research issue?'; Relationship: 'Has the relationship between researcher and participants been adequately considered?'; Ethics: 'Have ethical issues been taken into consideration?'; Analysis: 'Was the data analysis sufficiently rigorous?'; Findings: 'Is there a clear statement of findings?'; Value: 'How valuable is the research?'

Table C.2: Study quality: studies assessed using an adapted version of the QAT*.

Authors	Research question	Study population	Inclusion criteria	Data collection: perceived risks	Data collection: actual risks	Quantitative data	Analysis	Global
Allen et al (2010)	+	+	+	+	-	-	0	-
Asimakopoulou et al (2008)	+	0	+	+	+	+	+	+
Calvin et al (2011)	+	+	+	+	-	-	0	-
Choi et al (2008) (a)	+	+	+	+	N/A	0	+	+
Choi et al (2008) (b)	+	+	+	+	-	0	+	-
Frijling et al (2004)	+	+	0	+	+	+	+	+
Hoffmann & Del Mar (2012)	+	0	0	+	0	+	0	-
Homko et al (2010)	+	+	+	0	N/A	-	-	-
Kausar et al (2013)	+	+	+	+	N/A	0	0	0
Martell-Claros et al (2011)	+	+	+	+	+	+	+	+
Merz et al (2002)	+	+	-	-	N/A	-	-	-

Table C.2 Continued: Study quality: studies assessed using an adapted version of the QAT*.

Portnoy et al (2014) (a)	+	+	+	+	+	-	0	-
Portnoy et al (2014) (b)	+	+	+	+	N/A	-	0	-
Portnoy et al (2014) (c)	+	+	+	+	+	-	0	-
Saver et al (2014)	+	+	+	-	+	-	-	-
Walker et al (2007)	+	+	+	+	N/A	0	0	0
Welschen et al (2012)	+	+	+	+	+	0	+	+
Sum good (%)	14/14 (100%)	12/14 (85%)	11/14 (78%)	11/14 (78%)	8/11 (73%)	4/15 (27%)	4/16 (25%)	3/14 (21%)
Sum fair (%)	0/14 (0%)	2/14 (15%)	2/14 (15%)	1/14 (7%)	0/11 (0%)	5/15 (33%)	7/16 (44%)	3/14 (21%)
Sum poor (%)	0/14 (0%)	0/14 (0%)	1/14 (7%)	2/14 (15%)	3/11 (27%)	6/15 (40%)	5/16 (31%)	8/14 (58%)

Note (a): '+': Good; '0': Fair; '-': Poor; 'N/A': Not applicable.

Note (b): Study research: 'Was the research question or objective in this paper clearly stated?'; Study population: 'Was the study population clearly specified and defined?'; Inclusion criteria: 'Were all subjects selected or recruited from the same or similar populations? Were inclusion and exclusion criteria for being in the study prespecified and applied uniformly to all participants?'; Data collection: perceived risks: 'Were the perceived risks adequately measured?'; Data collection: actual risks: 'Were the actual risks adequately estimated?'; Quantitative data: 'Were quantitative estimates provided?'; Analysis: 'Was the data analysis rigorous?'

*: Because they do not apply to the studies included in the review, the items specific to the assessment of cohort studies were removed (items numbered 3, 5-10, 12-14 in the original QAT). There is no consequence on the rating system, as scores are cumulative and all items have the same weight.

D

Ethics documentation (elicitation study)

NHS
Health Research Authority
North East - Tyne & Wear South Research Ethics Committee

North East REC Office
Room 001
Jarrow Business Centre
Rolling Mill Road
Jarrow
Tyne & Wear
NE32 3DT

Tel: 0207 104 8118

Please note: This is the favourable opinion of the REC only and does not allow you to start your study at NHS sites in England until you receive HRA Approval

15 June 2016

Mr Thomas Rouyard
DPhil Student
University of Oxford
HERC, Nuffield Department of Population Health
Old Road Campus
OX3 7LF

Dear Mr Rouyard

Study title:	**Measuring risk preferences in the health domain**
REC reference:	**16/NE/0203**
IRAS project ID:	**202510**

The Proportionate Review Sub-committee of the North East - Tyne & Wear South Research Ethics Committee reviewed the above application on 09 June 2016.

We plan to publish your research summary wording for the above study on the HRA website, together with your contact details. Publication will be no earlier than three months from the date of this favourable opinion letter. The expectation is that this information will be published for all studies that receive an ethical opinion but should you wish to provide a substitute contact point, wish to make a request to defer, or require further information, please contact the REC Assistant Miss Kerry Dunbar, nrescommittee.northeast-tyneandwearsouth@nhs.net

Under very limited circumstances (e.g. for student research which has received an unfavourable opinion), it may be possible to grant an exemption to the publication of the study.

Ethical opinion

On behalf of the Committee, the sub-committee gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation, subject to the conditions specified below.

Conditions of the favourable opinion

The REC favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements. Each NHS organisation must confirm through the signing of agreements and/or other documents that it has given permission for the research to proceed (except where explicitly specified otherwise).

Guidance on applying for HRA Approval (England)/ NHS permission for research is available in the Integrated Research Application System, www.hra.nhs.uk or at http://www.rdforum.nhs.uk.

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of management permissions from host organisations.

Registration of Clinical Trials

All clinical trials (defined as the first four categories on the IRAS filter page) must be registered on a publically accessible database. This should be before the first participant is recruited but no later than 6 weeks after recruitment of the first participant.

There is no requirement to separately notify the REC but you should do so at the earliest opportunity e.g. when submitting an amendment. We will audit the registration details as part of the annual progress reporting process.

To ensure transparency in research, we strongly recommend that all research is registered but for non-clinical trials this is not currently mandatory.

If a sponsor wishes to request a deferral for study registration within the required timeframe, they should contact hra.studyregistration@nhs.net. The expectation is that all clinical trials will be

registered, however, in exceptional circumstances non registration may be permissible with prior agreement from the HRA. Guidance on where to register is provided on the HRA website.

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion").

Summary of discussion at the meeting

Informed consent process and the adequacy and completeness of participant information

Members stated that documentation needed to state whether the study touched on sensitive areas or not and if decided that it did, the plan if someone gets upset must be submitted.

*You replied that you did not expect the study to touch upon any sensitive areas. The questionnaire had been designed in collaboration with two patient representatives (affiliated to the NIHR Oxford-CLAHRC) and no issues had been raised on this matter. No concern had been expressed regarding a possibility that some of the questions may cause anxiety. Moreover, the exact same questionnaire was currently being run with a sample of healthy people drawn from the general population (control group). You had received ethics approval from the University of Oxford's Inter-Divisional Research Ethics Committee (IDREC) for conducting this study (reference number **R44523/RE001**).*

So far in running this questionnaire, there were no issues that had been encountered with any of the participants. However, in the unlikely event of a participant experiencing distress, he/she would be strongly encouraged to express his/her feelings to the leader of the study (Thomas Rouyard). You who would be present continually throughout the questionnaire as a support for the participant, would in turn advise the participant's GP about the issue. An opportunity would then be arranged for the participant to discuss their questions/concerns with their GP. This was stated, as a precautionary measure, in the Patient Information Sheet, which had been modified accordingly

> *"If any of the questions cause you distress, please inform the study researcher (Thomas Rouyard). You will then be given the opportunity of discussing any concerns with your doctor."*

Members stated that the Participant Information Sheet was very complicated in places and requested this be revised to make it easier to understand for participants.

You replied that the Patient Information Sheet had been simplified while retaining all relevant information. The new version had been modified in conjunction with a patient representative (affiliated to the NIHR Oxford-CLAHRC) who had approved its readability.

Members stated that withdrawal was possible at any point however this was not the case once the survey had been anonymously submitted. Members requested tracked changes.

You replied that since answers would be recorded anonymously, participants would no longer be able to withdraw from the study. Both the Patient Information Sheet and the Research Protocol had been modified accordingly.

Members asked for clarification on the process of consenting to the study, i.e. where they would fill in the survey monkey and where the control group data fitted in.

The applicant replied with the following:

Informing eligible participants
After receiving preliminary information about the study from Dr Richard Baskerville during their regular clinical appointment (at 27 Beaumont St Practice, OX1 2NR, Oxford), eligible patients who showed interest in participating in the study would be given a Patient Information Sheet. Dr Baskerville would answer potential questions related to the Participant Information Sheet. Patients willing to participate in the study after reading the Patient Information Sheet would let Dr Baskerville know and research visits would be set up to coincide with patients' appointments.

Informed consent
Participants interested in participating in the study would be welcomed by Thomas Rouyard (at 27 Beaumont St Practice, OX1 2NR, Oxford) and would be presented with the online tool (Survey Monkey questionnaire). It would be made clear to the participants that their consent for participating in the study would be implied by filling out this questionnaire. According to the UK data archive, "for data gathered during quantitative surveys (questionnaires), where no personal data are collected or where personal identifiers are removed from the data, obtaining written consent may not be required".

Survey Monkey questionnaire
The study would take place at 27 Beaumont St Practice, OX1 2NR, Oxford and would be led in a confidential area of the GP practice; only the participant and you would have access to the screen of the computer on which the survey would be run.

You would be continually present as a support for the participant, throughout the questionnaire. You would make sure that both the instructions and the questions were well understood. This was a key aspect of the procedure: no participant would be asked to read the instructions or answer the questions on their own. The written instructions would be accompanied by clear, additional verbal explanations. The verbal explanations would be tailored according to the initial level of understanding of each participant. This procedure was similar to those applied in comparable studies on risk preferences.

Control group data
The objective of the research was to identify specific trends (or biases) in the way people with type 2 diabetes make choices when facing health risks (we talk about risk preferences). In order to identify significant differences, in terms of risk preferences, between patients and healthy people, there was a need to run the exact same questionnaire on a sample of healthy people drawn from the general population (comparable in terms of age). In March 2016 ethics approval from the University of Oxford's IDREC was received for conducting this study on healthy people (no NHS patients or NHS sites involved; reference number ***R44523/RE001****).*

Currently in the process of recruiting these healthy participants (10 people have filled out the questionnaire so far). From experience, filling out the questionnaire with healthy participants takes around 20 minutes on average, including going through the instructions (both written

instructions and oral explanations) and answering the questions. There was no reason to expect that this time would be different with patients.

The Research Protocol had been modified in accordance with these clarifications.

Members were concerned that the tool was quite confusing and stated that it took a lot of reading to understand it. Members queried whether the documents could be made more user friendly.

You replied that the tool was as user friendly as possible as it had been one of the major concerns during the design of the study. Unfortunately, there was a trade-off between simplifying the hypothetical scenarios and obtaining reliable, accurate information. Making the scenarios simpler, and thus reducing the amount of information provided to understand the questions, would come at the cost of measuring imprecise risk preferences. Therefore in order to reduce the confusion and the cognitive weight of the questionnaire, the Survey Monkey tool had been developed and validated in collaboration with patient representatives (affiliated to the NIHR Oxford-CLAHRC). It was taken into account the remarks from both patients and lay persons when designing it and had already made numerous modifications to make it as user friendly as possible. This had notably led to the display of coloured faces to describe the different levels of quality of life (no other similar study in the literature had presented participants with such displays).
Despite the apparent complexity of the scenarios, people turn out to understand quite easily and quickly the types of choices that were asked. Although some people may have needed more verbal explanation than others, all participants had been able to answer the questions in the end. Although participants were faced with quite a lot of information to process in order to understand the hypothetical scenarios, once these scenarios had been understood, questions (choices) were repetitive and not very cognitively demanding. This was why the role of the researcher, as a permanent support throughout the study, was crucial. You explained that you were confident that patients would not have more difficulty than healthy people in understanding the instructions and answering the questions. Finally, modifying the questionnaire for the patient sample alone would come at a heavy price, in terms of comparability between the two samples, and would bias the analysis.

The Sub Committee was satisfied with the responses given to the issues raised.

Approved documents

The documents reviewed and approved were:

Document	*Version*	*Date*
Evidence of Sponsor insurance or indemnity (non NHS Sponsors only) [Sponsor insurance or indemnity]	1	01 August 2015
IRAS Application Form [IRAS_Form_02062016]		02 June 2016
IRAS Application Form XML file [IRAS_Form_02062016]		02 June 2016
IRAS Checklist XML [Checklist_02062016]		02 June 2016
IRAS Checklist XML [Checklist_14062016]		14 June 2016
Letter from funder [Funder letter]	1	23 May 2014
Letter from sponsor [Signed sponsorship letter]	1	12 May 2016
Other [HRA statement of activities]	4	30 March 2016
Other [CV Leal]	2	23 May 2016

Other [Response to Queries]		14 June 2016
Other [Oxford IDREC approval letter (R44523/RE001)]	1	16 March 2016
Other [Responses to queries raised by the PR sub-committee]	1	14 June 2016
Participant information sheet (PIS) [Patient Information Leaflet]	3	14 June 2016
Research protocol or project proposal [Research Protocol]	2	14 June 2016
Summary CV for student [CV CI]	1	21 March 2016
Summary CV for supervisor (student research) [CV Gray]	2	20 May 2016
Validated questionnaire [Questionnaire]	2	21 March 2016

Membership of the Proportionate Review Sub-Committee

The members of the Sub-Committee who took part in the review are listed on the attached sheet.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements

The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The HRA website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

User Feedback

The Health Research Authority is continually striving to provide a high quality service to all applicants and sponsors. You are invited to give your view of the service you have received and the application procedure. If you wish to make your views known please use the feedback form available on the HRA website:
http://www.hra.nhs.uk/about-the-hra/governance/quality-assurance/

HRA Training

We are pleased to welcome researchers and R&D staff at our training days – see details at http://www.hra.nhs.uk/hra-training/

With the Committee's best wishes for the success of this project.

16/NE/0203	**Please quote this number on all correspondence**

Yours sincerely

pp



Mr Paddy Stevenson
Chair

Email: nrescommittee.northeast-tyneandwearsouth@nhs.net

Enclosures: *List of names and professions of members who took part in the review*

"After ethical review – guidance for researchers"

Copy to: *Ms Heather House, Oxford University Hospitals NHS Trust*

North East - Tyne & Wear South Research Ethics Committee

Attendance at PRS Sub-Committee of the REC meeting on 09 June 2016 via correspondence.

Committee Members:

Name	*Profession*	*Present*	*Notes*
Ms Sam Barron	Clinical Lead, Speech and Language Therapist	Yes	
Mrs Debra J Lett	Manager Newcastle Brain Tissue Resource	Yes	
Mr Paddy Stevenson (Chair)	Research Operations Manager	Yes	

Also in attendance:

Name	*Position (or reason for attending)*
Miss Kerry Dunbar	REC Assistant

MEDICAL SCIENCES
INTER-DIVISIONAL RESEARCH ETHICS COMMITTEE

Research Services, University of Oxford, Wellington Square, Oxford, OX1 2JD
Tel: +44(0)1865 616577 Fax: +44(0)1865 280467
ethics@medsci.ox.ac.uk

CONFIDENTIAL

Ref: R44523/RE001

Mr Thomas Rouyard
Health Economics Research Centre
Nuffield Department of Population Health
University of Oxford
Old Road Campus
Oxford

16th March 2016

Dear Thomas

Research Ethics Approval - CUREC 1

Project title: Measuring risk attitudes in the health domain

The above application has been considered on behalf of the Medical Sciences Inter-divisional Research Ethics Committee (IDREC) in accordance with the procedures laid down by the University for Ethical Approval of all research involving human participants.

I am pleased to inform you that, on the basis of the information provided to the IDREC, the proposed research has been judged as meeting appropriate ethical standards, and approval has been granted for a period of **6 months**, commencing on **16th March 2016**. The reference number for this project is **R44523/RE001**.

This is **subject to:**

a) **your following the BPS guidelines for online research**

b) **it is your responsibility to comply with the requirements for administering any tests or questionnaires and if in doubt to contact the publisher of those tests or questionnaires.**

c) **if new staff are engaged MS IDREC should be informed of their names, status and ethics training.**

Please may I remind you that your project may be reviewed at some stage during an annual audit of projects.

Amendments

Should there be any subsequent changes to the project, which raise ethical issues not covered in the original application, you should submit details to the IDREC for consideration and approval.

Please do not hesitate to contact me if you have any queries.

Yours Sincerely

H. Barnby-Porritt

Dr. Helen Barnby-Porritt
Research Ethics Manager, Medical Sciences

NHS
National Institute for Health Research

Clinical Research Network
Thames Valley and South Midlands

Block 8
Nuffield Orthopaedic Centre
Windmill Road
Headington
Oxford, OX3 7LD
Email: studysupport.crnthamesvalley@nihr.ac.uk
Web: www.crn.nihr.ac.uk/thamesvalley

Mr Thomas Rouyard
107 Hurst Street
Oxford
OX4 1HE

9 December 2016

Dear Thomas,

Letter of Access for research

Study: Measuring risk preferences in the health domain
IRAS number: 202510

This letter of access should be presented to the independent contractor when negotiating access to conduct your research as confirmed in the Letter of HRA Approval dated 23 August 2016.

This letter of access is effective from 9 December 2016 and ends on 8 December 2019 unless terminated earlier in accordance with the clauses below.

Please note that you cannot commence your research until the Principal Investigator for the research project has received a signed Statement of Activities for HRA Approval from each participating GP Practice.

This letter of access is only applicable to the primary care NHS organisations within the following areas:

• Formerly Oxfordshire PCT (27 Beaumont Street Practice)

Yours sincerely

VClatworthy

Vicki Clatworthy
Research Facilitator

cc: HR Department of Substantive Employer
Enclosed: Guidance for Independent Contractors receiving this Letter of Access

E

Instructions for participants (elicitation study)

GENERAL INSTRUCTIONS

Welcome to this experiment on making decisions about your health! We are going to show you some hypothetical situations in which you will be asked to make choices involving your future length of life and quality of life. Remember, **there are no right or wrong answers**, we are simply interested in your preferences!

The experiment has three tasks. Before each task, we will give you some instructions to read.

In all the tasks, years of life will be represented by the following symbols:



A green face means a year lived with perfect quality of life (on a scale from 0% to 100%, you would rate your quality of life as **100%**).



A yellow face means a year lived with good quality of life (on a scale from 0% to 100%, you would rate your quality of life as **75%**).



A red face means a year lived with moderate quality of life (on a scale from 0% to 100%, you would rate your quality of life as **50%**).

Please take your time and consider each question carefully. There is no rush and most people manage to finish all three tasks within 20 minutes. If you have any questions or remarks, don't hesitate to ask.

Thank you!

TASK 1

Imagine that you expect to live another **20 years** with perfect quality of life (**100%**):



After that, you will get a fatal disease that causes an immediate, painless death.

Luckily, a drug has recently been invented which controls the disease and extends the length of your life. You have to take the drug only once and it has no side effects. However, it is not certain that the drug is going to work well. Its effect will depend on your lifestyle once you get the disease.

You have two options:

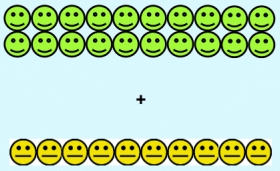
- **Option A: You adopt a recommended lifestyle**

You will need to follow a diet, to exercise regularly, to see the doctor regularly, and to take daily medications.

This change in behaviour will create some constraints in your daily life. For example, you may have to eat less of some foods you like, or experience mild to moderate side effects associated with your medications. As a result, imagine that your quality of life drops from 100% to **75%**. However, by making these efforts, the drug works well and you live **another 10 years** without experiencing any complication. Then you die from an immediate, painless death.

Option A: Adopt a recommended lifestyle

Gain 10 years with a quality of life of 75%



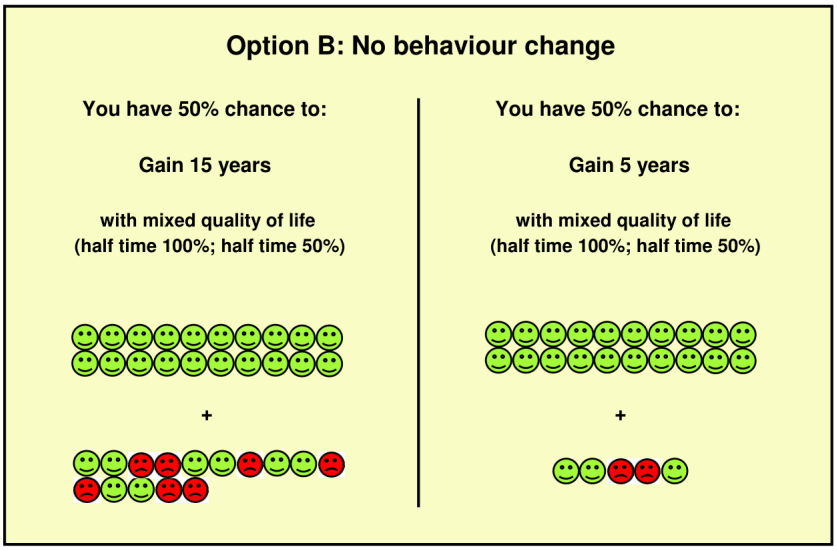
- **Option B: You DO NOT change your behaviour**

You decide to live as you were living before getting the disease. However, you get a complication that is going to impact your quality of life.

Half of the time you will live with perfect quality of life (**100%**); but the other half of the time your quality of life will be moderate (**50%**). For example, you might experience mild to moderate pain, tiredness, or difficulties in performing daily tasks. You do not know which years will be lived with perfect quality of life and which years will not.

Moreover, it is not certain that the drug will work well. **Two things may happen:**

- You have a 1 in 2 chance that the drug works. In that case, you live **another 15 years**. Then you die from an immediate, painless death.
- But you also have a 1 in 2 chance that the drug does not work. If that happens, you only live **another 5 years**. Then you die from an immediate, painless death.



Options A and B are summarised in the table below:

Option A: Recommended lifestyle	Option B: No behaviour change	
Gain for certain:	**50% chance to gain:**	**50% chance to gain:**
		
+	+	+
		
10 years	**15 years**	**5 years**
with a quality of life of 75%	**with mixed quality of life (half time 100%; half time 50%)**	**with mixed quality of life (half time 100%; half time 50%)**

In the following questions, we will ask you to choose one of the two options, A or B, according to your preferences. The situations will be of the same type, but the lengths of life will be changed.

TASK 2

Imagine that you expect to live another **20 years** with perfect quality of life (**100%**). After that, you will die from an immediate, painless death.

However, now, you get a disease that is going to impact both your length of life and your quality of life.

Luckily, this disease can be controlled. However, controlling the disease requires some change in your behaviour. You will need to follow a diet, to exercise regularly, to see the doctor regularly, and to take daily medications. Adopting this recommended lifestyle will reduce the impact of the disease.

You have two options:

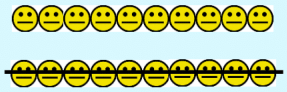
- **Option A: You adopt a recommended lifestyle**

As in the previous task, this change in behaviour creates some constraints in your daily life. For example, you may have to eat less of some foods you like, or experience mild to moderate side effects associated with your medications. As a result, your quality of life drops from 100% to **75%**. However, by making these efforts, your length of life will not be less than 10 years (that is, **you would not lose more than 10 years** of the 20 years you are expecting to live).

You live 10 years, for certain, with moderate quality of life (**75%**).

Option A: Adopt a recommended lifestyle

Lose 10 years which have a quality of life of 75%



- **Option B: You DO NOT change your behaviour**

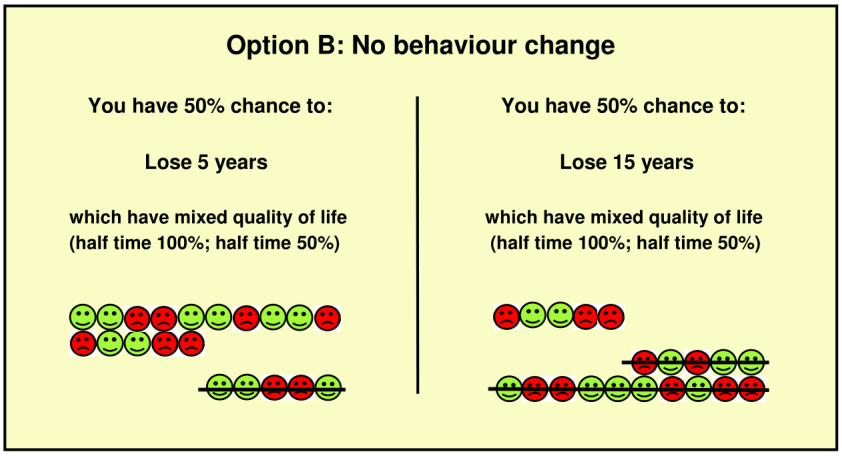
You decide to live as you were living before getting the disease. However, you get a complication that is going to impact your quality of life.

Half of the time you will live with perfect quality of life (**100%**); but the other half of the time your quality of life will be moderate (**50%**). For example, you might experience mild to moderate pain, tiredness, or difficulties in performing daily tasks. You do not know which years will be lived with perfect quality of life and which years will not.

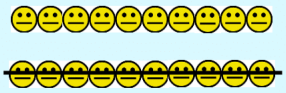
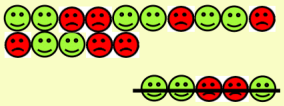
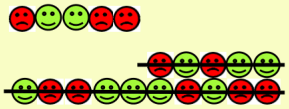
Moreover, your length of life might be less than 10 years (**you might lose more than 10 years** of the 20 years you are expecting to live). **Two things may happen:**

- You have a 1 in 2 chance to **lose 5 years of life**. You live 15 years with mixed quality of life (half of the time with perfect quality of life, half of the time with moderate quality of life). Then you die from an immediate, painless death.

- But you also have a 1 in 2 chance to **lose 15 years of life.** You live only 5 years with mixed quality of life (half of the time with perfect quality of life, half of the time with moderate quality of life). Then you die from an immediate, painless death.



Options A and B are summarised in the table below:

Option A: **Recommended lifestyle**	**Option B:** **No behaviour change**	
Lose for certain:	**50% chance to lose:**	**50% chance to lose:**
		
10 years **with a quality of life of 75%**	**5 years** **with mixed quality of life (half time 100%; half time 50%)**	**15 years** **with mixed quality of life (half time 100%; half time 50%)**

In the following questions, we will ask you to choose one of the two options, A or B, according to your preferences. The situations will be of the same type, but the lengths of life will be changed.

TASK 3

Imagine that you live with a disease which, to be controlled, requires a change in your behaviour (you need to follow a diet, to exercise regularly, to see the doctor regularly, and to take daily medications). As a result, your quality of life is **75%** (for example, you may have to eat less of some foods you like, or experience mild to moderate side effects associated with your medications). You have **20 remaining years** to live in that state (after that, you die from an immediate, painless death):



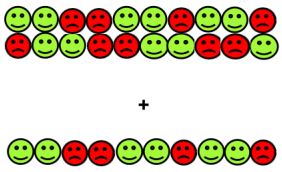
Imagine that not respecting that daily behaviour would cause an immediate, painless death.

Luckily, a new drug has recently been invented which can do something about this. **The drug allows you to live without changing your lifestyle.** You have to take the drug only once and it has no side effects.

However, the drug will impact your quality of life. Half of the time you will live with perfect quality of life (**100%**); but the other half of the time your quality of life will be moderate (**50%**). For example, you might experience mild to moderate pain, tiredness, or difficulties in performing daily tasks. You do not know which years will be lived with perfect quality of life and which years will not.

Moreover, the drug will impact your length of life. **Two things may happen:**

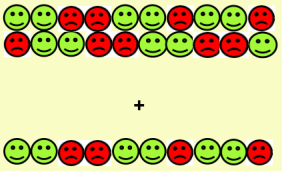
- You have a 1 in 2 chance to **gain 10 years of life**. You live 30 years with mixed quality of life (half time with perfect quality of life, half time with moderate quality of life). Then you die from an immediate, painless death:



- But you also have a 1 in 2 chance to **lose 10 years of life.** You live only 10 years with mixed quality of life (half time with perfect quality of life, half time with moderate quality of life).Then you die from an immediate, painless death.



Whether the drug increases or decreases your length of life depends solely on chance and, hence, has nothing to do with your personal features. As for any other person, you have exactly 1 out of 2 chances that one of the two situations happens.

Option A: **DO NOT take the drug**	**Option B:** **Take the drug**	
Gain nothing and lose nothing:	**50% chance to GAIN** **10 years:**	**50% chance to LOSE** **10 years:**
		
20 years **with a quality of life of 75%**	**30 years** **with mixed quality of life** **(half time 100%; half time 50%)**	**10 years** **with mixed quality of life** **(half time 100%; half time 50%)**

In the following questions, we will ask you to choose one of the two options, A or B, according to your preferences. The situations will be of the same type, but the lengths of life will be changed.

F

Distributions of individual parameters

Figure F.1: Cumulative distribution functions (CDF) of individual utility parameters for gains (black line) and losses (blue line).

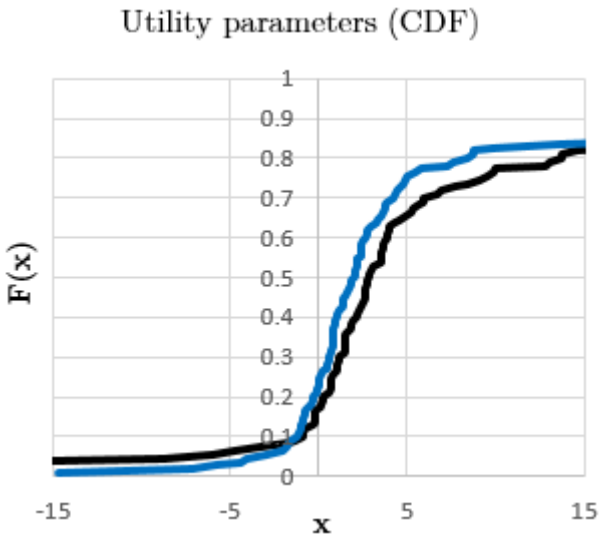
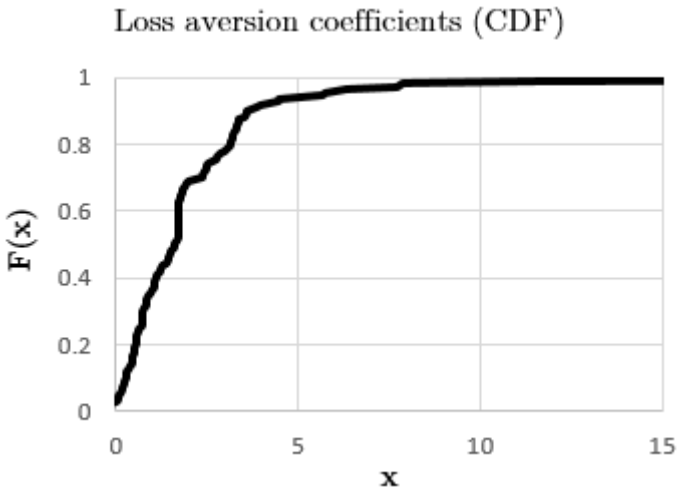


Figure F.2: Cumulative distribution functions (CDF) of individual loss aversion parameters.



G
Additional analyses

Table G.1: Robustness analysis: estimation results for the utility parameters for gains and losses and for loss aversion (allowing for discounting of the 15 years till onset of the behavioural change).

		γ	ω^+	δ	ω^-	λ
T2DM group (n=52)	Median (IQ1-IQ3)	8.89 (2.44-23.23)	0.48 (0.48-0.49)	5.35 (1.76-17.26)	0.48 (0.44-0.50)	2.04 (0.96-10.91)
Control group (n=58)	Median (IQ1-IQ3)	6.51 (1.15-16.04)	0.49 (0.48-0.50)	2.99 (-0.05-8.57)	0.49 (0.45-0.53)	1.75 (0.91-4.27)
Total (n=110)	Median (IQ1-IQ3)	7.18 (1.71-20.76)	0.49 (0.48-0.50)	4.07 (0.95-11.30)	0.49 (0.45-0.51)	1.80 (0.91-5.52)

Note: The median coefficient was significantly different from 0 for gains (λ=1.80; p<0.01) and losses (δ=4.07; p<0.01). However, they were not significantly different from each other (p=0.60). Probability weighting was significant in both the gain and the loss domains (ω^+=0.49; p<0.01 and ω^-=0.49; p<0.01). The median coefficient for loss aversion was significantly different from 1 (λ=1.80; p<0.01).

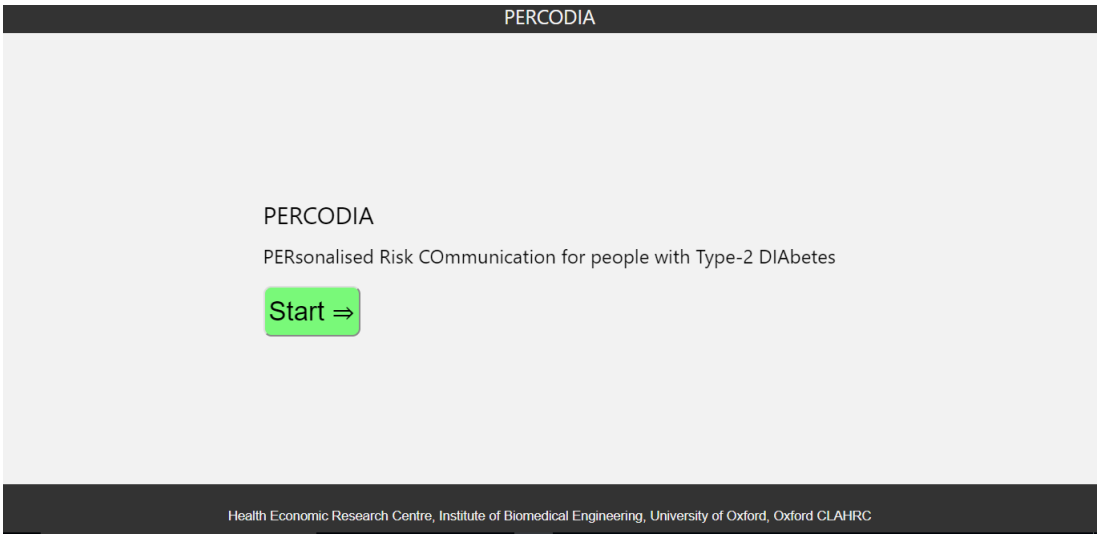
Table G.2: Robustness analysis combining gain and loss prospects (i.e. assuming only a kink at the reference point but no differential utility functions for gains and losses), and allowing : estimation results for the utility parameters for gains and losses and for loss aversion (allowing for discounting of the 15 years till onset of the behavioural change.

		γ	ω	λ
T2DM group (n=52)	Median (IQ1-IQ3)	7.21 (1.77-21.69)	0.49 (0.48-0.50)	1.62 (1.08-6.30)
Control group (n=58)	Median (IQ1-IQ3)	4.37 (0.37-12.56)	0.49 (0.48-0.51)	1.62 (1.06-5.86)
Total (n=110)	Median (IQ1-IQ3)	5.45 (1.32-16.38)	0.49 (0.48-0.50)	1.57 (1.07-6.01)

H

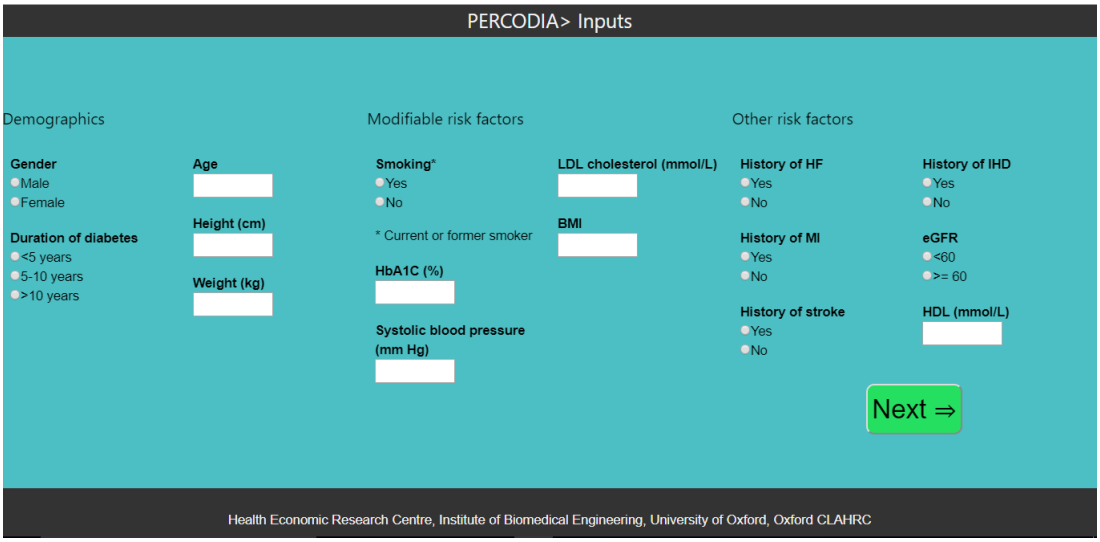
PERCODIA: user guide

Page 1 - Welcome page



1. This is the welcome page. Click on **START** to reach the inputs page.

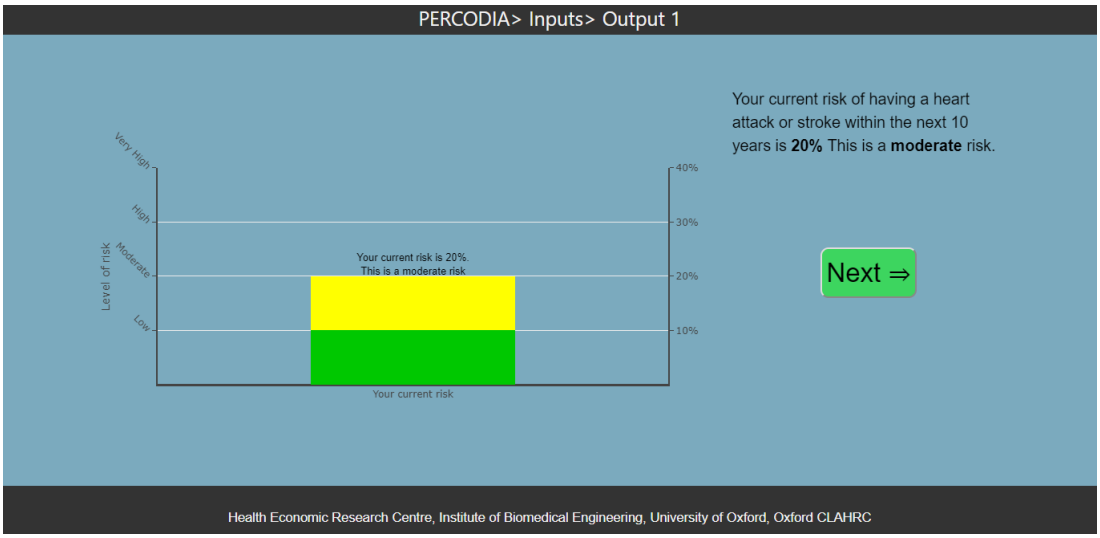
Page 2 - Inputs page



1. Please inform each section carefully. BMI is automatically calculated by informing height and weight.

2. Once all sections have been completed, click on **NEXT** to reach the first output page (absolute 10-year CV risk). **BE CAREFUL! From now on, you will not be able to go back to a previous page.** Therefore, please make sure to spend as much time as needed on each output page. If you really need to go back to one of the previous pages, you will need to reload PERCODIA, which will lead you back to the welcome page.

Page 3 - Output page: 10-year CV risk (1/2)



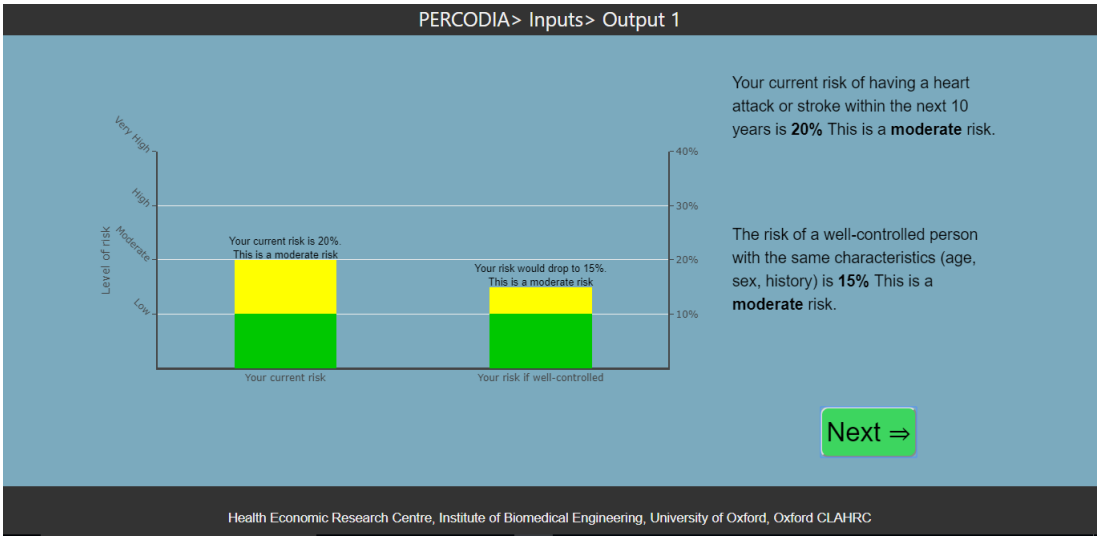
1. This is the first part of the first output page, which displays the personalised absolute 10-year CV risk:

- Simple, coloured bar graph: green (low risk, 0-10%), yellow (moderate risk, 10-20%), red (high risk, >20%).
- Summary statement: e.g. 'Your current risk of having a heart attack or stroke within the next 10 years is 20%. This is a moderate risk.'

2. This risk information must be accompanied by additional explanations about the causes and consequences of the risk, as well as with advice about how to reduce the risk.

3. Click on **NEXT** to reach the second part of the first output page (comparative 10-year CV risk).

Page 4 - Output page: 10-year CV risk (2/2)



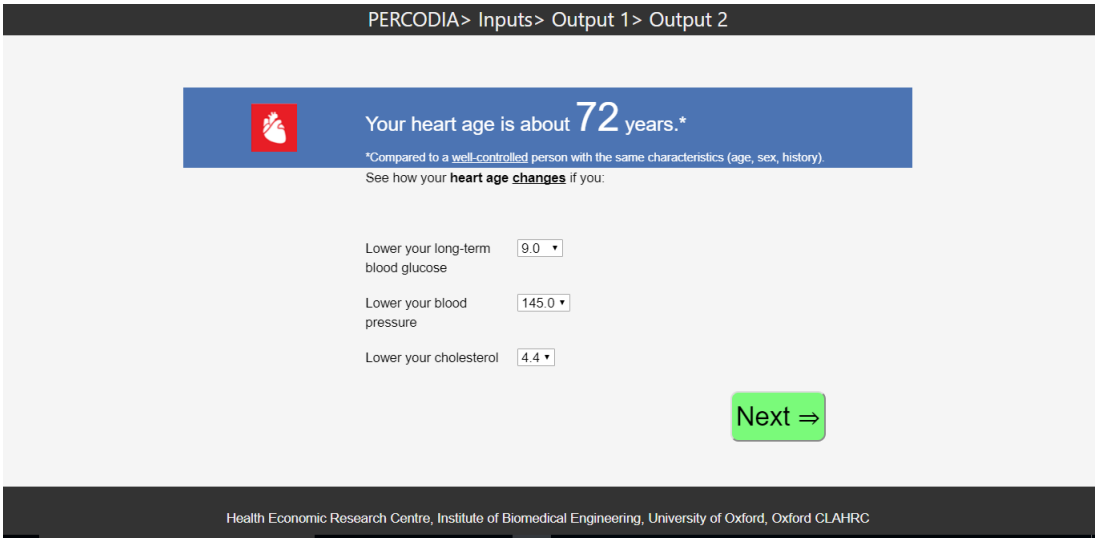
1. This is the second part of the first output page, which displays the personalised comparative 10-year CV risk:

   - The bar graph on the left is the same as in the previous page.
   - The bar graph on the right shows the risk of an individual with the same characteristics (demographics, history of events) who would be well-controlled (i.e. whose modifiable risk factors are within normal ranges). A second summary statement appears.

2. This risk information must be accompanied by additional explanations about the causes and consequences of the risk, as well as with advice about how to reduce the risk (e.g. 'The risk of a person like you (same age, same sex, and same medical history) who is well-controlled is 15%. This risk is moderate. This would be your level of risk if you were well-controlled. You can achieve this by eating healthier / doing more physical exercise / taking your medications...').

3. Click on **NEXT** to reach the second output page (heart age).

Page 5 - Output page: effective heart age



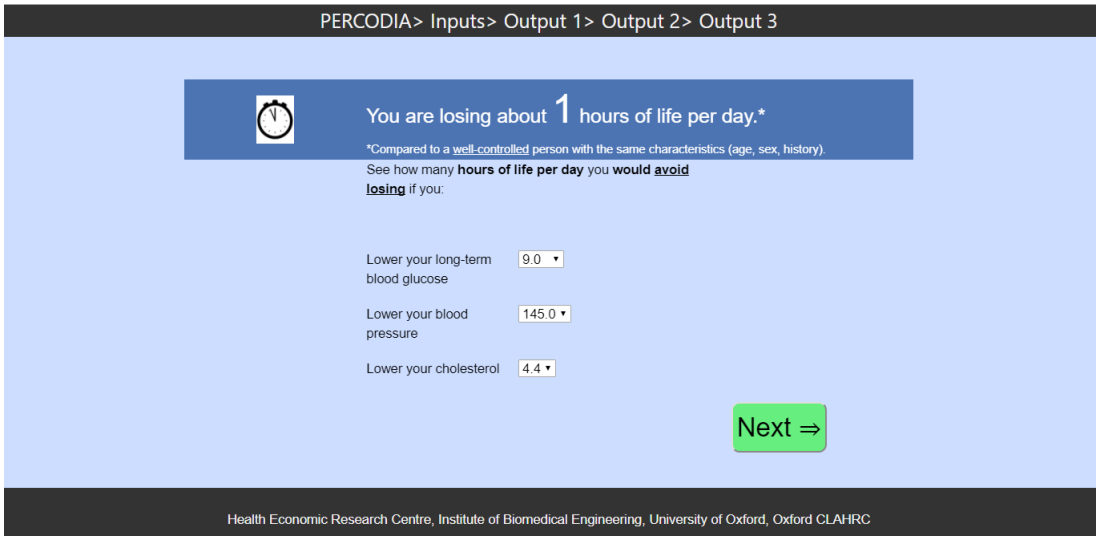
1. This is the second output page, which converts the personalised 10-year CV risk into an effective heart age.

2. This risk information must be accompanied by additional explanations (e.g. 'This is the same risk as the one we calculated before, but expressed in a different way. According to your level of risk, it is like your heart is 72 years old. Putting differently, this means that your level of risk is the same than someone who is 72 and whose diabetes is well-controlled.').

3. It is possible to see **how the heart age varies when the three modifiable risk factors (HbA1c, SBP, and LDL) change**. This allows to show the positive [negative] impact of better [worse] self-management behaviour (e.g. 'Let's see how lowering your long-term blood glucose from 8.0% to 7.0% would impact your heart age. By doing so, your heart age would drop to [...]').

4. Click on **NEXT** to reach the last output page (loss of daily life time).

Page 6 - Output page: change in life expectancy



1. This is the last output page, which calculates the loss of life time, expressed as the number of hours lost per day as compared to a well-controlled individual with the same characteristics.

2. This risk information must be accompanied by additional explanations (e.g. 'Let's see how your risk factors impact your life expectancy. As compared to a person just like you who is well-controlled, you are currently losing two hours of life time per day').

3. Similarly to the heart age, it is possible to see **how variations in the three modifiable risk factors impact this outcome**. This allows to show the positive [negative] impact of better [worse] self-management behaviour (e.g. '**You would avoid losing 1 hour of life per day** if your blood pressure would go from 160 mmHg to 130 mm Hg.').

4. Click on **NEXT** to reach the final page of PERCODIA, which allows the user to print out the results.

I

Development of two models to forecast the risk information of interest in a limited amount of time. Methods and Stata outputs.

The two models have been built following the approach recommended by Royston *et al.* (2009).

I.1 Model 1: 10-year CV risk forecast

Step 1 - Selecting clinically relevant candidate predictors for possible inclusion in the model.

In agreement with the GP involved in the development of the tool, I chose 14 candidate predictors that had been reported to be prognostic of 10-year CV risks in diabetes populations: sex, age, duration of diabetes, BMI, HbA1c, SBP, LDL, HDL, eGFR, and history of MI, stroke, IHD and HF.

Step 2 - Handling data related to pre-selected predictors.

As data used to estimate the two models came from an artificial dataset specifically created for this purpose, there were no missing data issues to handle. Due to the high number of individuals used to cover all value ranges (n=1,920,000), models for males and females had to be run separately with Stata software package. Given the nature of the dependent variable, I used binomial generalised linear models (GLM) with a logistic link to fit the data.[1] When the dependent variable took a value higher than 1 in the dataset (10-year CV risk was defined as the risk of having MI or stroke, i.e. the sum of two risk probabilities comprised between 0 and 1), it was rounded down to 1.

Step 3 - Selecting the predictors to be included in the final model by following a backward elimination approach.

The model was first tested with all the candidate variables (full model, see Tables I.1 and I.2). Due to the difficulty of obtaining certain data in primary care, three continuous variables needed to be converted into categorical variables to improve the usability of the tool in practice: duration of diabetes (see Tables I.3 and I.4)

[1] Generalised linear models are an extension of linear regression models that allow the dependent variable to be non-normal.

and eGFR (see Tables I.5 and I.6). The predictive accuracy of the model (assessed by comparing Akaike criteria) did not improve by dropping any of the variables included in model (3).

Step 4 - Modelling continuous predictors.
The predictive accuracy of the model improved when adding the square of the age variable in the model (see Tables I.7 and I.8). Model (4) is the model that was ultimately selected.

Step 5 - Assessing the model's performance.
The selected model had a high goodness of fit (see Hosmer-Lemeshow test results in 4.4.3). Adding the squared age variable did not modify the mean absolute error (MAE) of the model (see Tables I.9 and I.10) but slightly increased its MSE (mean squared error) (see Tables I.11 and I.12).

Table I.1: Model 1 (males): GLM estimation of the model (1).

```
. glm cases age smoke duration BMI HDL LDL BP HbAlc eGFR preIHD preHF prestroke preMI, family(binomi
> al total) link(logit)

Iteration 0:   log likelihood =  -15562947
Iteration 1:   log likelihood =  -15378813
Iteration 2:   log likelihood =  -15378717
Iteration 3:   log likelihood =  -15378717

Generalized linear models                         No. of obs      =    960,000
Optimization     : ML                             Residual df     =    959,986
                                                  Scale parameter =          1
Deviance         =  24147240.67                   (1/df) Deviance =   25.15374
Pearson          =  24834651.73                   (1/df) Pearson  =   25.86981

Variance function: V(u) = u*(1-u/total)           [Binomial]
Link function    : g(u) = ln(u/(total-u))         [Logit]

                                                  AIC             =   32.03902
Log likelihood   = -15378717.16                   BIC             =   1.09e+07
```

cases	OIM Coef.	Std. Err.	z	P>|z|	[95% Conf.	Interval]
age	.0364158	5.65e-06	6446.69	0.000	.0364047	.0364268
smoke	.263277	.0001544	1705.12	0.000	.2629744	.2635797
duration	-.0203612	.0000189	-1077.82	0.000	-.0203982	-.0203242
BMI	.0125666	.0000106	1189.42	0.000	.0125459	.0125873
HDL	-.3302025	.0001156	-2855.52	0.000	-.3304291	-.3299758
LDL	.2242009	.0000552	4058.16	0.000	.2240926	.2243091
BP	.0076523	3.65e-06	2099.02	0.000	.0076452	.0076595
HbAlc	.0932144	.0000338	2754.04	0.000	.0931481	.0932807
eGFR	-.0029745	3.85e-06	-771.77	0.000	-.0029821	-.002967
preIHD	.7419833	.0002503	2963.92	0.000	.7414927	.742474
preHF	.3571379	.0002578	1385.57	0.000	.3566327	.3576431
prestroke	.478513	.000255	1876.54	0.000	.4780133	.4790128
preMI	.5398929	.0002538	2127.62	0.000	.5393956	.5403903
_cons	-5.916264	.000852	-6944.25	0.000	-5.917934	-5.914594

Table I.2: Model 1 (females): GLM estimation of the model (1).

```
.
. glm cases age smoke duration BMI HDL LDL BP HbAlc eGFR preIHD preHF prestroke preMI, family(binomi
> al total) link(logit)

Iteration 0:   log likelihood =  -15421043
Iteration 1:   log likelihood =  -15269337
Iteration 2:   log likelihood =  -15269267
Iteration 3:   log likelihood =  -15269267

Generalized linear models                         No. of obs      =    960,000
Optimization     : ML                             Residual df     =    959,986
                                                  Scale parameter =          1
Deviance         =  23964493.82                   (1/df) Deviance =   24.96338
Pearson          =  24878945.43                   (1/df) Pearson  =   25.91595

Variance function: V(u) = u*(1-u/total)           [Binomial]
Link function    : g(u) = ln(u/(total-u))         [Logit]

                                                  AIC             =     31.811
Log likelihood   =  -15269267.2                   BIC             =   1.07e+07
```

cases	Coef.	OIM Std. Err.	z	P>|z|	[95% Conf.	Interval]
age	.0301479	5.71e-06	5275.59	0.000	.0301367	.0301591
smoke	.2875723	.0001578	1822.30	0.000	.287263	.2878816
duration	.0128231	.0000193	664.98	0.000	.0127853	.0128609
BMI	.0119201	.0000108	1104.75	0.000	.011899	.0119412
HDL	-.025093	.0001173	-213.89	0.000	-.025323	-.0248631
LDL	.1563758	.000056	2790.04	0.000	.156266	.1564857
BP	.0069266	3.72e-06	1861.22	0.000	.0069193	.0069339
HbAlc	.0672353	.0000345	1950.72	0.000	.0671678	.0673029
eGFR	-.0149685	3.98e-06	-3760.13	0.000	-.0149763	-.0149607
preIHD	.7414826	.0002573	2882.05	0.000	.7409783	.7419868
preHF	.4245794	.0002644	1605.87	0.000	.4240612	.4250976
prestroke	.3858484	.0002655	1453.51	0.000	.3853281	.3863687
preMI	.6140413	.0002598	2363.47	0.000	.6135321	.6145505
_cons	-5.13203	.0008584	-5978.66	0.000	-5.133712	-5.130348

Table I.3: Model 1 (males): GLM estimation of the model (2).

```
. glm cases age smoke i.durcat BMI HDL LDL BP HbAlc eGFR preIHD preHF prestroke preMI, family(binomi
> al total) link(logit)

Iteration 0:   log likelihood =  -15534236
Iteration 1:   log likelihood =  -15349983
Iteration 2:   log likelihood =  -15349888
Iteration 3:   log likelihood =  -15349888

Generalized linear models                         No. of obs      =    960,000
Optimization     : ML                             Residual df     =    959,985
                                                  Scale parameter =          1
Deviance         =  24089581.67                   (1/df) Deviance =   25.09371
Pearson          =   24788190.6                   (1/df) Pearson  =   25.82144

Variance function: V(u) = u*(1-u/total)           [Binomial]
Link function    : g(u) = ln(u/(total-u))         [Logit]

                                                  AIC             =   31.97896
Log likelihood   = -15349887.66                   BIC             =   1.09e+07
```

cases	Coef.	OIM Std. Err.	z	P>|z|	[95% Conf.	Interval]
age	.036418	5.65e-06	6446.86	0.000	.0364069	.036429
smoke	.2632933	.0001544	1705.18	0.000	.2629907	.263596
durcat						
1	-.0631476	.0001866	-338.34	0.000	-.0635134	-.0627818
2	-.2046726	.0001895	-1080.31	0.000	-.2050439	-.2043012
BMI	.0125674	.0000106	1189.46	0.000	.0125467	.0125881
HDL	-.3302229	.0001156	-2855.61	0.000	-.3304495	-.3299962
LDL	.2242146	.0000552	4058.27	0.000	.2241064	.2243229
BP	.0076528	3.65e-06	2099.09	0.000	.0076457	.00766
HbAlc	.0932202	.0000338	2754.12	0.000	.0931538	.0932865
eGFR	-.0029747	3.85e-06	-771.80	0.000	-.0029823	-.0029672
preIHD	.7420279	.0002503	2964.01	0.000	.7415372	.7425185
preHF	.3571575	.0002578	1385.60	0.000	.3566523	.3576627
prestroke	.4785401	.000255	1876.59	0.000	.4780403	.4790399
preMI	.5399239	.0002538	2127.68	0.000	.5394265	.5404212
_cons	-5.929194	.0008538	-6944.31	0.000	-5.930868	-5.927521

Table I.4: Model 1 (females): GLM estimation of the model (2).

```
. glm cases age smoke i.durcat BMI HDL LDL BP HbAlc eGFR preIHD preHF prestroke preMI, family(binomi
> al total) link(logit)

Iteration 0:   log likelihood =  -15269641
Iteration 1:   log likelihood =  -15118872
Iteration 2:   log likelihood =  -15118803
Iteration 3:   log likelihood =  -15118803

Generalized linear models                         No. of obs      =    960,000
Optimization     : ML                             Residual df     =    959,985
                                                  Scale parameter =          1
Deviance         =   23663565.8                   (1/df) Deviance =   24.64993
Pearson          =  24612265.65                   (1/df) Pearson  =   25.63818

Variance function: V(u) = u*(1-u/total)           [Binomial]
Link function    : g(u) = ln(u/(total-u))         [Logit]

                                                  AIC             =   31.49754
Log likelihood   = -15118803.19                   BIC             =   1.04e+07
```

cases	Coef.	OIM Std. Err.	z	P>|z|	[95% Conf.	Interval]
age	.030158	5.72e-06	5276.38	0.000	.0301468	.0301692
smoke	.28767	.0001578	1822.61	0.000	.2873607	.2879794
durcat						
1	.1562172	.0001937	806.47	0.000	.1558375	.1565968
2	.130057	.0001942	669.58	0.000	.1296764	.1304377
BMI	.0119242	.0000108	1104.94	0.000	.011903	.0119453
HDL	-.0251016	.0001173	-213.93	0.000	-.0253316	-.0248716
LDL	.1564289	.0000561	2790.50	0.000	.156319	.1565387
BP	.0069289	3.72e-06	1861.53	0.000	.0069216	.0069362
HbAlc	.0672582	.0000345	1951.05	0.000	.0671906	.0673257
eGFR	-.0149735	3.98e-06	-3760.74	0.000	-.0149814	-.0149657
preIHD	.7417273	.0002573	2882.52	0.000	.7412229	.7422316
preHF	.4247116	.0002644	1606.12	0.000	.4241933	.4252299
prestroke	.3859677	.0002655	1453.74	0.000	.3854473	.3864881
preMI	.6142395	.0002598	2363.85	0.000	.6137302	.6147488
_cons	-5.165126	.000861	-5999.30	0.000	-5.166813	-5.163438

Table I.5: Model 1 (males): GLM estimation of the model (3).

```
. glm cases age smoke i.durcat BMI HDL LDL BP HbA1c i.eGFRcat preIHD preHF prestroke preMI, family(b
> inomial total) link(logit)

Iteration 0:   log likelihood =  -15534236
Iteration 1:   log likelihood =  -15349983
Iteration 2:   log likelihood =  -15349888
Iteration 3:   log likelihood =  -15349888

Generalized linear models                         No. of obs      =    960,000
Optimization     : ML                             Residual df     =    959,985
                                                  Scale parameter =          1
Deviance         =  24089581.67                   (1/df) Deviance =   25.09371
Pearson          =   24788190.6                   (1/df) Pearson  =   25.82144

Variance function: V(u) = u*(1-u/total)           [Binomial]
Link function    : g(u) = ln(u/(total-u))         [Logit]

                                                  AIC             =   31.97896
Log likelihood   = -15349887.66                   BIC             =   1.09e+07
```

cases	OIM Coef.	Std. Err.	z	P>|z|	[95% Conf.	Interval]
age	.036418	5.65e-06	6446.86	0.000	.0364069	.036429
smoke	.2632933	.0001544	1705.18	0.000	.2629907	.263596
durcat						
1	-.0631476	.0001866	-338.34	0.000	-.0635134	-.0627818
2	-.2046726	.0001895	-1080.31	0.000	-.2050439	-.2043012
BMI	.0125674	.0000106	1189.46	0.000	.0125467	.0125881
HDL	-.3302229	.0001156	-2855.61	0.000	-.3304495	-.3299962
LDL	.2242146	.0000552	4058.27	0.000	.2241064	.2243229
BP	.0076528	3.65e-06	2099.09	0.000	.0076457	.00766
HbA1c	.0932202	.0000338	2754.12	0.000	.0931538	.0932865
1.eGFRcat	.1189886	.0001542	771.80	0.000	.1186864	.1192908
preIHD	.7420279	.0002503	2964.01	0.000	.7415372	.7425185
preHF	.3571575	.0002578	1385.60	0.000	.3566523	.3576627
prestroke	.4785401	.000255	1876.59	0.000	.4780403	.4790399
preMI	.5399239	.0002538	2127.68	0.000	.5394265	.5404212
_cons	-6.137424	.0008388	-7316.54	0.000	-6.139068	-6.13578

Table I.6: Model 1 (females): GLM estimation of the model (3).

```
. glm cases age smoke i.durcat BMI HDL LDL BP HbAlc i.eGFRcat preIHD preHF prestroke preMI, family(b
> inomial total) link(logit)

Iteration 0:   log likelihood =  -15269641
Iteration 1:   log likelihood =  -15118872
Iteration 2:   log likelihood =  -15118803
Iteration 3:   log likelihood =  -15118803

Generalized linear models                         No. of obs      =    960,000
Optimization     : ML                             Residual df     =    959,985
                                                  Scale parameter =          1
Deviance         =   23663565.8                   (1/df) Deviance =   24.64993
Pearson          =  24612265.65                   (1/df) Pearson  =   25.63818

Variance function: V(u) = u*(1-u/total)           [Binomial]
Link function    : g(u) = ln(u/(total-u))         [Logit]

                                                  AIC             =   31.49754
Log likelihood   = -15118803.19                   BIC             =   1.04e+07
```

cases	Coef.	OIM Std. Err.	z	P>|z|	[95% Conf.	Interval]
age	.030158	5.72e-06	5276.38	0.000	.0301468	.0301692
smoke	.28767	.0001578	1822.61	0.000	.2873607	.2879794
durcat						
1	.1562172	.0001937	806.47	0.000	.1558375	.1565968
2	.130057	.0001942	669.58	0.000	.1296764	.1304377
BMI	.0119242	.0000108	1104.94	0.000	.011903	.0119453
HDL	-.0251016	.0001173	-213.93	0.000	-.0253316	-.0248716
LDL	.1564289	.0000561	2790.50	0.000	.156319	.1565387
BP	.0069289	3.72e-06	1861.53	0.000	.0069216	.0069362
HbAlc	.0672582	.0000345	1951.05	0.000	.0671906	.0673257
1.eGFRcat	.598942	.0001593	3760.74	0.000	.5986298	.5992541
preIHD	.7417273	.0002573	2882.52	0.000	.7412229	.7422316
preHF	.4247116	.0002644	1606.12	0.000	.4241933	.4252299
prestroke	.3859677	.0002655	1453.74	0.000	.3854473	.3864881
preMI	.6142395	.0002598	2363.85	0.000	.6137302	.6147488
_cons	-6.213274	.0008583	-7238.89	0.000	-6.214956	-6.211592

Table I.7: Model 1 (males): GLM estimation of the model (4).

```
. glm cases age agesq smoke i.durcat BMI HDL LDL BP HbAlc i.eGFRcat preIHD preHF prestroke preMI, fa
> mily(binomial total) link(logit)

Iteration 0:   log likelihood =  -15510945
Iteration 1:   log likelihood =  -15317747
Iteration 2:   log likelihood =  -15317625
Iteration 3:   log likelihood =  -15317625

Generalized linear models                         No. of obs      =    960,000
Optimization     : ML                             Residual df     =    959,984
                                                  Scale parameter =          1
Deviance         =   24025056.6                   (1/df) Deviance =   25.02652
Pearson          =  24831876.24                   (1/df) Pearson  =   25.86697

Variance function: V(u) = u*(1-u/total)           [Binomial]
Link function    : g(u) = ln(u/(total-u))         [Logit]

                                                  AIC             =   31.91175
Log likelihood   = -15317625.13                   BIC             =   1.08e+07
```

cases	OIM Coef.	Std. Err.	z	P>|z|	[95% Conf.	Interval]
age	.0498325	.0000532	937.22	0.000	.0497283	.0499368
agesq	-.0001195	4.71e-07	-253.84	0.000	-.0001204	-.0001185
smoke	.2632447	.0001544	1705.03	0.000	.2629421	.2635474
durcat						
1	-.0631385	.0001866	-338.31	0.000	-.0635043	-.0627727
2	-.2046342	.0001894	-1080.21	0.000	-.2050055	-.2042629
BMI	.0125651	.0000106	1189.36	0.000	.0125444	.0125858
HDL	-.3301594	.0001156	-2855.34	0.000	-.330386	-.3299327
LDL	.2241678	.0000552	4057.87	0.000	.2240595	.224276
BP	.0076514	3.65e-06	2098.90	0.000	.0076443	.0076586
HbAlc	.0932025	.0000338	2753.86	0.000	.0931361	.0932688
1.eGFRcat	.1189674	.0001542	771.73	0.000	.1186653	.1192695
preIHD	.7418594	.0002503	2963.71	0.000	.7413688	.74235
preHF	.3570365	.0002577	1385.37	0.000	.3565314	.3575416
prestroke	.478393	.000255	1876.31	0.000	.4778933	.4788927
preMI	.5397672	.0002537	2127.38	0.000	.5392699	.5402645
_cons	-6.490549	.0016267	-3989.99	0.000	-6.493738	-6.487361

Table I.8: Model 1 (females): GLM estimation of the model (4).

```
. glm cases age agesq smoke i.durcat BMI HDL LDL BP HbAlc i.eGFRcat preIHD preHF prestroke preMI, fa
> mily(binomial total) link(logit)

Iteration 0:   log likelihood =  -15219733
Iteration 1:   log likelihood =  -15059144
Iteration 2:   log likelihood =  -15059052
Iteration 3:   log likelihood =  -15059052

Generalized linear models                         No. of obs      =    960,000
Optimization     : ML                             Residual df     =    959,984
                                                  Scale parameter =          1
Deviance         =  23544064.15                   (1/df) Deviance =   24.52548
Pearson          =  24632195.45                   (1/df) Pearson  =   25.65896

Variance function: V(u) = u*(1-u/total)           [Binomial]
Link function    : g(u) = ln(u/(total-u))         [Logit]

                                                  AIC             =   31.37306
Log likelihood   = -15059052.36                   BIC             =   1.03e+07
```

cases	Coef.	OIM Std. Err.	z	P>|z|	[95% Conf.	Interval]
age	.0487134	.0000541	901.09	0.000	.0486074	.0488193
agesq	-.0001655	4.79e-07	-345.35	0.000	-.0001664	-.0001646
smoke	.2876283	.0001578	1822.48	0.000	.287319	.2879377
durcat						
1	.1561943	.0001937	806.41	0.000	.1558146	.1565739
2	.130037	.0001942	669.53	0.000	.1296563	.1304177
BMI	.0119225	.0000108	1104.87	0.000	.0119014	.0119437
HDL	-.0250982	.0001173	-213.92	0.000	-.0253282	-.0248682
LDL	.1564053	.0000561	2790.30	0.000	.1562954	.1565151
BP	.0069279	3.72e-06	1861.40	0.000	.0069206	.0069352
HbAlc	.0672486	.0000345	1950.91	0.000	.0671811	.0673162
1.eGFRcat	.5988326	.0001592	3760.48	0.000	.5985205	.5991447
preIHD	.7415858	.0002573	2882.29	0.000	.7410815	.7420901
preHF	.4245943	.0002644	1605.90	0.000	.4240761	.4251125
prestroke	.3858576	.0002655	1453.54	0.000	.3853374	.3863779
preMI	.6140994	.0002598	2363.60	0.000	.6135901	.6146086
_cons	-6.700894	.0016555	-4047.72	0.000	-6.704139	-6.69765

Table I.9: Model 1: mean absolute errors (males).

Variable	Obs	Mean	Std. Dev.	Min	Max
mae1	960,000	15.20544	8.66101	1.199056	44.97596
mae2	960,000	15.20544	8.661022	1.201546	44.97626
mae3	960,000	15.20544	8.661022	1.201546	44.97626
mae4	960,000	15.20544	8.661317	1.205545	44.97691

Table I.10: Model 1: mean absolute errors (females).

Variable	Obs	Mean	Std. Dev.	Min	Max
mae1	960,000	16.46348	9.187246	1.286836	46.74169
mae2	960,000	16.46348	9.187301	1.272617	46.74268
mae3	960,000	16.46348	9.187301	1.272617	46.74268
mae4	960,000	16.46348	9.187696	1.279223	46.74397

Table I.11: Model 1: mean squared errors (males).

Variable	Obs	Mean	Std. Dev.	Min	Max
mse1	960,000	306.2184	328.0403	1.437736	2022.837
mse2	960,000	306.2186	328.0404	1.443712	2022.864
mse3	960,000	306.2186	328.0404	1.443712	2022.864
mse4	960,000	306.2237	328.0925	1.453338	2022.922

Table I.12: Model 1: mean squared errors (females).

Variable	Obs	Mean	Std. Dev.	Min	Max
mse1	960,000	355.4514	369.6068	1.655947	2184.786
mse2	960,000	355.4524	369.6077	1.619553	2184.878
mse3	960,000	355.4524	369.6077	1.619553	2184.878
mse4	960,000	355.4597	369.679	1.636412	2184.999

I.2 Model 2: life expectancy forecast

Step 1 - Selecting clinically relevant candidate predictors for possible inclusion in the model.

In agreement with the GP involved in the development of the tool, I chose 14 candidate predictors that had been reported to be prognostic of 10-year CV risks in diabetes populations: gender, age, duration of diabetes, BMI, HbA1c, SBP, LDL, HDL, eGFR, and history of MI, stroke, IHD and HF.

Step 2 - Handling data related to pre-selected predictors.

As data used to estimate the two models came from an artificial dataset specifically created for this purpose, there was no missing data issues to handle. Due to the high number of individuals used to cover all value ranges (n=1,920,000), models for males and males had to be run separately with Stata software package. Given the nature of the dependent variable, I used linear regression models to fit the data (ordinary least squares (OLS) regressions).

Step 3 - Selecting the predictors to be included in the final model by following a backward elimination approach.

The model was first tested with all the candidate variables (full model, see Tables I.13 and I.14). Due to the difficulty of obtaining certain data in primary care, three continuous variables needed to be converted into categorical variables to improve the usability of the tool in practice: duration of diabetes (see Tables I.15 and I.16) and eGFR (see Tables I.17 and I.18). At the cost of the model's goodness of fit, and in agreement with the GP involved in the development of the tool, I decided to drop the BMI variable. The reason is that the model predicted a positive correlation between BMI and life expectancy, most likely due to the presence of comorbidities (e.g. cancer) in individuals with low BMI in the initial UKPDS data. As the objective of the tool is to encourage self-management behaviour in T2DM populations, which often includes weight loss, it was hardly conceivable to communicate improved outcomes associated with weight gain. The predictive

accuracy of the model (assessed by comparing R^2) did not improve by dropping any additional variable included in the model.

Step 4 - Modelling continuous predictors.
The predictive accuracy of the model improved when adding the square of the age variable in the model (see Tables I.19 and I.20). Model (4) is the model that was ultimately selected.

Step 5 - Assessing the model's performance.
The selected model had a high goodness of fit (see also additional test results in 4.4.3), although dropping the BMI variable slightly increased the MAE (see Tables I.21 and I.22) and the MSE (see Tables I.23 and I.24).

Table I.13: Model 2 (males): OLS estimation of the model (1).

```
. reg lifeexp age smoke duration BMI HDL LDL BP HbAlc eGFR preIHD preHF prestroke preMI, robust

Linear regression                               Number of obs     =    960,000
                                                F(13, 959986)     >  99999.00
                                                Prob > F          =     0.0000
                                                R-squared         =     0.9102
                                                Root MSE          =     2.568

------------------------------------------------------------------------------
             |               Robust
    lifeexp |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |  -.5096879   .0002174 -2344.56   0.000    -.510114   -.5092618
       smoke |  -2.252514    .005242  -429.71   0.000    -2.262798   -2.242229
    duration |    .009514   .0006429    14.80   0.000     .0082539    .010774
         BMI |   .2198509   .0003697   594.68   0.000     .2191263    .2205755
         HDL |   .0905717   .0039522    22.92   0.000     .0828256    .0983178
         LDL |  -.8381202   .0018862  -444.33   0.000    -.8418171   -.8344232
          BP |  -.0259213    .000124  -209.00   0.000    -.0261644   -.0256782
       HbAlc |  -.2146731   .0011457  -187.37   0.000    -.2169187   -.2124275
       eGFR |   .0769197    .000131   586.95   0.000     .0766629    .0771766
     preIHD |  -2.543653   .0092951  -273.65   0.000    -2.561871   -2.525434
      preHF |   -7.10723   .0086032  -826.11   0.000    -7.124092   -7.090368
  prestroke |  -6.367932   .0084969  -749.44   0.000    -6.384585   -6.351278
      preMI |  -4.326489   .0086525  -500.03   0.000    -4.343447    -4.30953
      _cons |   46.55231   .0295676  1574.44   0.000     46.49435    46.61026
------------------------------------------------------------------------------
```

Table I.14: Model 2 (females): OLS estimation of the model (1).

```
. reg lifeexp age smoke i.durcat BMI HDL LDL BP HbAlc eGFR preIHD preHF prestroke preMI, robust

Linear regression                               Number of obs     =    960,000
                                                F(14, 959985)     >  99999.00
                                                Prob > F          =     0.0000
                                                R-squared         =     0.9214
                                                Root MSE          =     2.5538

------------------------------------------------------------------------------
             |               Robust
    lifeexp |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |  -.5473417   .0002113 -2589.76   0.000    -.5477556   -.5469278
       smoke |  -2.541354   .0052129  -487.51   0.000    -2.551571   -2.531137
             |
     durcat |
          1  |  -.2014344   .0063279   -31.83   0.000    -.2138368   -.1890319
          2  |  -.1535028   .0063862   -24.04   0.000    -.1660195   -.1409861
             |
         BMI |   .2169916   .0003712   584.65   0.000     .2162642    .2177191
         HDL |  -.5007188   .0039228  -127.64   0.000    -.5084073   -.4930303
         LDL |  -.6372884   .0018695  -340.88   0.000    -.6409526   -.6336242
          BP |  -.0228253   .0001234  -184.98   0.000    -.0230672   -.0225835
       HbAlc |  -.1790657   .0011391  -157.20   0.000    -.1812982   -.1768331
       eGFR |   .0759943   .0001303   583.13   0.000     .0757388    .0762497
     preIHD |  -4.208463   .0091019  -462.37   0.000    -4.226303   -4.190624
      preHF |  -8.524912   .0087366  -975.77   0.000    -8.542036   -8.507789
  prestroke |  -7.365436   .0086281  -853.65   0.000    -7.382346   -7.348525
      preMI |  -5.122575   .0086211  -594.19   0.000    -5.139472   -5.105678
      _cons |   50.69736   .0295396  1716.25   0.000     50.63946    50.75526
------------------------------------------------------------------------------
```

Table I.15: Model 2 (males): OLS estimation of the model (2).

```
.
. reg lifeexp age smoke i.durcat BMI HDL LDL BP HbAlc eGFR preIHD preHF prestroke preMI, robust

Linear regression                               Number of obs     =    960,000
                                                F(14, 959985)     >  99999.00
                                                Prob > F          =     0.0000
                                                R-squared         =     0.9102
                                                Root MSE          =     2.5679

------------------------------------------------------------------------------
             |               Robust
    lifeexp |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |  -.5096879   .0002174 -2344.64   0.000    -.5101139   -.5092618
       smoke |  -2.252514   .0052417  -429.73   0.000    -2.262787    -2.24224
             |
      durcat |
          1  |  -.0093677   .0063368    -1.48   0.139    -.0217875    .0030522
          2  |   .0951396   .0064286    14.80   0.000     .0825397    .1077395
             |
         BMI |   .2198509   .0003697   594.70   0.000     .2191263    .2205754
         HDL |   .0905717    .003952    22.92   0.000      .082826    .0983174
         LDL |  -.8381202   .0018861  -444.36   0.000    -.8418017   -.8344234
          BP |  -.0259213    .000124  -209.01   0.000    -.0261644   -.0256782
       HbAlc |  -.2146731   .0011457  -187.38   0.000    -.2169185   -.2124277
       eGFR  |   .0769197    .000131   586.98   0.000     .0766629    .0771766
     preIHD  |  -2.543653   .0092949  -273.66   0.000     -2.56187   -2.525435
      preHF  |   -7.10723   .0086029  -826.14   0.000    -7.124092   -7.090369
  prestroke  |  -6.367932   .0084963  -749.49   0.000    -6.384584   -6.351279
      preMI  |  -4.326489   .0086519  -500.06   0.000    -4.343446   -4.309531
       _cons |   46.57128   .0296194  1572.32   0.000     46.51323    46.62934
------------------------------------------------------------------------------
```

Table I.16: Model 2 (females): OLS estimation of the model (2).

```
. reg lifeexp age smoke i.durcat BMI HDL LDL BP HbAlc eGFR preIHD preHF prestroke preMI, robust

Linear regression                               Number of obs     =    960,000
                                                F(14, 959985)     >  99999.00
                                                Prob > F          =     0.0000
                                                R-squared         =     0.9214
                                                Root MSE          =     2.5538

------------------------------------------------------------------------------
             |               Robust
    lifeexp |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |  -.5473417   .0002113 -2589.76   0.000     -.547756   -.5469275
       smoke |  -2.541354   .0052129  -487.51   0.000    -2.551571   -2.531137
             |
      durcat |
          1  |  -.2014344   .0063279   -31.83   0.000    -.2138368   -.1890319
          2  |  -.1535028   .0063862   -24.04   0.000    -.1660195   -.1409861
             |
         BMI |   .2169916   .0003712   584.65   0.000     .2162642    .2177191
         HDL |  -.5007188   .0039228  -127.64   0.000    -.5084073   -.4930303
         LDL |  -.6372884   .0018695  -340.88   0.000    -.6409526   -.6336242
          BP |  -.0228253   .0001234  -184.98   0.000    -.0230672   -.0225835
       HbAlc |  -.1790657   .0011391  -157.20   0.000    -.1812982   -.1768331
       eGFR  |   .0759943   .0001303   583.13   0.000     .0757388    .0762497
     preIHD  |  -4.208463   .0091019  -462.37   0.000    -4.226303   -4.190624
      preHF  |  -8.524912   .0087366  -975.77   0.000    -8.542036   -8.507789
  prestroke  |  -7.365436   .0086281  -853.65   0.000    -7.382346   -7.348525
      preMI  |  -5.122575   .0086211  -594.19   0.000    -5.139472   -5.105678
       _cons |   50.69736   .0295396  1716.25   0.000     50.63946    50.75526
------------------------------------------------------------------------------
```

Table I.17: Model 2 (males): OLS estimation of the model (3).

```
. reg lifeexp age smoke i.durcat BMI HDL LDL BP HbAlc i.eGFRcat preIHD preHF prestroke preMI, robust

Linear regression                               Number of obs     =    960,000
                                                F(14, 959985)     >  99999.00
                                                Prob > F          =     0.0000
                                                R-squared         =     0.9102
                                                Root MSE          =     2.5679

------------------------------------------------------------------------------
             |               Robust
    lifeexp |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |  -.5096879   .0002174 -2344.64   0.000    -.5101139   -.5092618
       smoke |  -2.252514   .0052417  -429.73   0.000    -2.262787   -2.24224
             |
      durcat |
          1  |  -.0093677   .0063368    -1.48   0.139    -.0217875    .0030522
          2  |   .0951396   .0064286    14.80   0.000     .0825397    .1077395
             |
         BMI |   .2198509   .0003697   594.70   0.000     .2191263    .2205754
         HDL |   .0905717    .003952    22.92   0.000      .082826    .0983174
         LDL |  -.8381202   .0018861  -444.36   0.000    -.8418017   -.8344234
          BP |  -.0259213    .000124  -209.01   0.000    -.0261644   -.0256782
       HbAlc |  -.2146731   .0011457  -187.38   0.000    -.2169185   -.2124277
  1.eGFRcat  |  -3.076789   .0052417  -586.98   0.000    -3.087062   -3.066515
     preIHD  |  -2.543653   .0092949  -273.66   0.000     -2.56187   -2.525435
      preHF  |   -7.10723   .0086029  -826.14   0.000    -7.124092   -7.090369
  prestroke  |  -6.367932   .0084963  -749.49   0.000    -6.384584   -6.351279
      preMI  |  -4.326489   .0086519  -500.06   0.000    -4.343446   -4.309531
       _cons |   51.95567    .031174  1666.64   0.000     51.89457    52.01677
------------------------------------------------------------------------------
```

Table I.18: Model 2 (females): OLS estimation of the model (3).

```
. reg lifeexp age smoke i.durcat BMIcat HDL LDL BP HbAlc i.eGFRcat preIHD preHF prestroke preMI, rob
> ust

Linear regression                               Number of obs     =    960,000
                                                F(14, 959985)     >  99999.00
                                                Prob > F          =     0.0000
                                                R-squared         =     0.9050
                                                Root MSE          =     2.8063

------------------------------------------------------------------------------
             |               Robust
    lifeexp |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |  -.5473417   .0002281 -2400.64   0.000    -.5477886   -.5468949
       smoke |  -2.541354   .0057283  -443.65   0.000    -2.552581   -2.530127
             |
      durcat |
          1  |  -.2014344   .0069537   -28.97   0.000    -.2150633   -.1878054
          2  |  -.1535028   .0070222   -21.86   0.000     -.167266   -.1397396
             |
      BMIcat |   1.297138   .0032827   395.14   0.000     1.290704    1.303572
         HDL |  -.5007188   .0043051  -116.31   0.000    -.5091567   -.4922809
         LDL |  -.6372884   .0020446  -311.69   0.000    -.6412958   -.6332811
          BP |  -.0228253   .0001355  -168.50   0.000    -.0230908   -.0225598
       HbAlc |  -.1790657   .0012509  -143.14   0.000    -.1815175   -.1766139
  1.eGFRcat  |   -3.03977   .0057283  -530.66   0.000    -3.050998   -3.028543
     preIHD  |  -4.208463   .0095859  -439.03   0.000    -4.227251   -4.189675
      preHF  |  -8.524912   .0091151  -935.25   0.000    -8.542777   -8.507047
  prestroke  |  -7.365436   .0090328  -815.41   0.000     -7.38314   -7.347732
      preMI  |  -5.122575   .0090764  -564.38   0.000    -5.140365   -5.104786
       _cons |   61.06673   .0312077  1956.78   0.000     61.00556    61.1279
------------------------------------------------------------------------------
```

Table I.19: Model 2 (males): OLS estimation of the model (4).

```
. reg lifeexp age agesq smoke i.durcat HDL LDL BP HbAlc i.eGFRcat preIHD preHF prestroke preMI, robu
> st

Linear regression                               Number of obs     =    960,000
                                                F(14, 959985)     >  99999.00
                                                Prob > F          =     0.0000
                                                R-squared         =     0.8833
                                                Root MSE          =      2.927

------------------------------------------------------------------------------
             |               Robust
    lifeexp |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
------------+----------------------------------------------------------------
         age |  -1.020303   .002156  -473.25   0.000    -1.024528   -1.016077
       agesq |    .004642   .0000186   249.12   0.000     .0046054    .0046785
       smoke |  -2.252514   .0059748  -377.00   0.000    -2.264224   -2.240803
             |
      durcat |
           1 |  -.0093677   .0071867    -1.30   0.192    -.0234534    .004718
           2 |   .0951396   .0073322    12.98   0.000     .0807688    .1095105
             |
         HDL |   .0905717   .0044996    20.13   0.000     .0817527    .0993907
         LDL |  -.8381202   .0021371  -392.18   0.000    -.8423088   -.8339315
          BP |  -.0259213   .0001411  -183.68   0.000    -.0261979   -.0256447
       HbAlc |  -.2146731   .0013041  -164.62   0.000     -.217229   -.2121172
  1.eGFRcat  |  -3.076789   .0059748  -514.96   0.000    -3.088499   -3.065079
     preIHD  |  -2.543653    .010142  -250.80   0.000     -2.56353   -2.523775
      preHF  |   -7.10723   .0092418  -769.03   0.000    -7.125344   -7.089117
  prestroke  |  -6.367932   .0090844  -700.97   0.000    -6.385737   -6.350126
      preMI  |  -4.326489   .0093916  -460.68   0.000    -4.344896   -4.308081
       _cons |   71.17116   .0661581  1075.77   0.000     71.04149    71.30083
------------------------------------------------------------------------------
```

Table I.20: Model 2 (females): OLS estimation of the model (4).

```
. reg lifeexp age agesq smoke i.durcat HDL LDL BP HbAlc i.eGFRcat preIHD preHF prestroke preMI, robu
> st

Linear regression                               Number of obs     =    960,000
                                                F(14, 959985)     >  99999.00
                                                Prob > F          =     0.0000
                                                R-squared         =     0.9001
                                                Root MSE          =     2.8786

------------------------------------------------------------------------------
             |               Robust
    lifeexp |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
------------+----------------------------------------------------------------
         age |  -1.114943   .0020977  -531.50   0.000    -1.119055   -1.110832
       agesq |     .00516   .0000182   283.41   0.000     .0051243    .0051957
       smoke |  -2.541354   .005876  -432.50   0.000    -2.552871   -2.529837
             |
      durcat |
           1 |  -.2014344   .0071092   -28.33   0.000    -.2153681   -.1875006
           2 |  -.1535028   .0072109   -21.29   0.000    -.1676359   -.1393698
             |
         HDL |  -.5007188   .0044181  -113.33   0.000    -.5093782   -.4920594
         LDL |  -.6372884   .002091   -304.77   0.000    -.6413868   -.6331901
          BP |  -.0228253   .0001389  -164.34   0.000    -.0230975   -.0225531
       HbAlc |  -.1790657   .0012829  -139.57   0.000    -.1815802   -.1765511
  1.eGFRcat  |   -3.03977   .005876  -517.32   0.000    -3.051287   -3.028254
     preIHD  |  -4.208463   .0099879  -438.94   0.000    -4.227255   -4.189671
      preHF  |  -8.524912   .0090851  -938.34   0.000    -8.542719   -8.507106
  prestroke  |  -7.365436   .008989  -819.38   0.000    -7.383054   -7.347817
      preMI  |  -5.122575   .0091485  -559.94   0.000    -5.140506   -5.104644
       _cons |   76.61662   .0642238  1192.96   0.000     76.49074    76.7425
------------------------------------------------------------------------------
```

Table I.21: Model 2: mean absolute errors (males).

Variable	Obs	Mean	Std. Dev.	Min	Max
mae1	960,000	1.996629	1.614977	1.41e-06	10.90082
mae2	960,000	1.996485	1.614931	9.99e-08	10.88185
mae3	960,000	1.996485	1.614931	9.99e-08	10.88185
mae4	960,000	2.257593	1.862969	1.65e-07	12.89831

Table I.22: Model 2: mean absolute errors (females).

Variable	Obs	Mean	Std. Dev.	Min	Max
mae1	960,000	2.01809	1.566044	1.71e-06	10.84149
mae2	960,000	2.017431	1.565789	6.06e-06	10.84228
mae3	960,000	2.017431	1.565789	6.06e-06	10.84228
mae4	960,000	2.233286	1.816262	3.11e-06	11.8131

Table I.23: Model 2: mean squared errors (males).

Variable	Obs	Mean	Std. Dev.	Min	Max
mse1	960,000	6.594674	10.65265	2.00e-12	118.828
mse2	960,000	6.593953	10.6525	9.99e-15	118.4146
mse3	960,000	6.593953	10.6525	9.99e-15	118.4146
mse4	960,000	8.567378	13.42706	2.72e-14	166.3665

Table I.24: Model 2: mean squared errors (females).

Variable	Obs	Mean	Std. Dev.	Min	Max
mse1	960,000	6.525175	9.845296	2.91e-12	117.538
mse2	960,000	6.521721	9.84292	3.68e-11	117.5551
mse3	960,000	6.521721	9.84292	3.68e-11	117.5551
mse4	960,000	8.286374	12.57658	9.67e-12	139.5493

J

Characteristics of the artificial cohort population used to estimate the two forecasting models

Table J.1: Demographic variables.

Variable	Category or value range (step)
Sex	Male; Female
Ethnicity	White
Age (years)	35-75 (10)

Table J.2: Risk factor variables.

Variable	Category or value range (step)
Duration of T2DM (years)	0-10 (5)
Smoking status	Yes (current smoker); No (none or past smoker)
BMI	18-38 (7)
HbA1c (%)	5-12 (1)
SBP (mm Hg)	110-170 (15)
LDL (mmol/L)	1-5 (1)
HDL (mmol/L)	0.4-2.2(0.6)
eGFR (mL/min/1.73m^2)	30; 70
History of MI	Yes; No
History of IHD	Yes; No
History of HF	Yes; No
History of stroke	Yes; No

Table J.3: Input variables required by the UKPDS-OM but not included in the forecasting models.

Variable	Category or value range (step)
AF	Yes; No
PVD	Yes; No
Albuminuria	Yes (micro- or microalbuminuria); No
Heart rate (bpm)	84.2
WBC ($\times 10^9$/L)	6.9
Haemoglobin (g/dL)	14.0
History of amputation	Yes; No
History of blindness	Yes; No
History of renal failure	Yes; No
History of ulcer	Yes; No

For each individual of the cohort, all risk estimates were calculated yearly for a 70-year period (the maximum possible number of years to be simulated per subject), holding all risk factors constant over this time path. This was necessary to ensure accurate life expectancy estimates were calculated. As recommended, the number of internal loops (Monte-Carlo trials) per subject was set to 5,000 in order to reduce first order uncertainty (Monte Carlo Error - MCE).

K

Ethics documentation (pilot study)

NHS
Health Research Authority

Mr Thomas Rouyard
DPhil student
University of Oxford
HERC, Nuffield Department of Population Health
Old Road Campus, Headington
Oxford
OX3 7LF

Email: hra.approval@nhs.net

10 May 2017

Dear Mr Thomas Rouyard

Letter of HRA Approval

Study title:	**Type-2 diabetes: risk perceptions and self-management behaviour.**
IRAS project ID:	**210623**
REC reference:	**17/NW/0267**
Sponsor	**University of Oxford**

I am pleased to confirm that **HRA Approval** has been given for the above referenced study, on the basis described in the application form, protocol, supporting documentation and any clarifications noted in this letter.

Participation of NHS Organisations in England

The sponsor should now provide a copy of this letter to all participating NHS organisations in England.

Appendix B provides important information for sponsors and participating NHS organisations in England for arranging and confirming capacity and capability. **Please read *Appendix B* carefully**, in particular the following sections:

- *Participating NHS organisations in England* – this clarifies the types of participating organisations in the study and whether or not all organisations will be undertaking the same activities
- *Confirmation of capacity and capability* - this confirms whether or not each type of participating NHS organisation in England is expected to give formal confirmation of capacity and capability. Where formal confirmation is not expected, the section also provides details on the time limit given to participating organisations to opt out of the study, or request additional time, before their participation is assumed.
- *Allocation of responsibilities and rights are agreed and documented (4.1 of HRA assessment criteria)* - this provides detail on the form of agreement to be used in the study to confirm capacity and capability, where applicable.

Further information on funding, HR processes, and compliance with HRA criteria and standards is also provided.

It is critical that you involve both the research management function (e.g. R&D office) supporting each organisation and the local research team (where there is one) in setting up your study. Contact details and further information about working with the research management function for each organisation can be accessed from www.hra.nhs.uk/hra-approval.

Appendices

The HRA Approval letter contains the following appendices:

- A – List of documents reviewed during HRA assessment
- B – Summary of HRA assessment

After HRA Approval

The document *"After Ethical Review – guidance for sponsors and investigators",* issued with your REC favourable opinion, gives detailed guidance on reporting expectations for studies, including:

- Registration of research
- Notifying amendments
- Notifying the end of the study

The HRA website also provides guidance on these topics, and is updated in the light of changes in reporting expectations or procedures.

In addition to the guidance in the above, please note the following:

- HRA Approval applies for the duration of your REC favourable opinion, unless otherwise notified in writing by the HRA.
- Substantial amendments should be submitted directly to the Research Ethics Committee, as detailed in the *After Ethical Review* document. Non-substantial amendments should be submitted for review by the HRA using the form provided on the HRA website, and emailed to hra.amendments@nhs.net.
- The HRA will categorise amendments (substantial and non-substantial) and issue confirmation of continued HRA Approval. Further details can be found on the HRA website.

Scope

HRA Approval provides an approval for research involving patients or staff in NHS organisations in England.

If your study involves NHS organisations in other countries in the UK, please contact the relevant national coordinating functions for support and advice. Further information can be found at http://www.hra.nhs.uk/resources/applying-for-reviews/nhs-hsc-rd-review/.

If there are participating non-NHS organisations, local agreement should be obtained in accordance with the procedures of the local participating non-NHS organisation.

User Feedback

The Health Research Authority is continually striving to provide a high quality service to all applicants and sponsors. You are invited to give your view of the service you have received and the application

procedure. If you wish to make your views known please use the feedback form available on the HRA website: http://www.hra.nhs.uk/about-the-hra/governance/quality-assurance/.

HRA Training

We are pleased to welcome researchers and research management staff at our training days – see details at http://www.hra.nhs.uk/hra-training/

Your IRAS project ID is **210623**. Please quote this on all correspondence.

Yours sincerely

Joanna Strickland
Assessor

Email: hra.approval@nhs.net

Copy to: *Ms Heather House [sponsor contact] ctrg@admin.ox.ac.uk*
Ms Vicki Clatworthy, CRN Thames Valley and South Midlands [lead R&D contact] vicki.clatworthy@oxfordhealth.nhs.uk

Appendix A - List of Documents

The final document set assessed and approved by HRA Approval is listed below.

Document	*Version*	*Date*
Evidence of Sponsor insurance or indemnity (non NHS Sponsors only) [Sponsor insurance]	1	15 July 2016
IRAS Application Form [IRAS_Form_12042017]		12 April 2017
Letter from funder [Funding letter]	1	23 May 2014
Letter from sponsor [Sponsor letter]	1	12 April 2017
Other [Calculator of personalised risks (UKPDS-OM)_Manual]	2.0	11 May 2015
Other [Delegation log and signatures (sponsor template)]	1.0	07 April 2017
Other [GP site agreement]	1.0	10 April 2017
Other [The 8-Item Medication Adherence Scale]	1.0	07 April 2017
Other [Summary CV supervisor Leal]	1	23 May 2016
Other [SoE-dated]	1	10 May 2017
Other [SoA-amended-dated]	2	10 May 2017
Participant consent form [Consent form]	1.0	07 April 2017
Participant information sheet (PIS) [PIS]	1.0	07 April 2017
Research protocol or project proposal [Research Protocol]	1.0	07 April 2017
Summary CV for Chief Investigator (CI) [Summary CV CI]	1	15 March 2017
Summary CV for student [Summary CV student]	1	15 March 2017
Summary CV for supervisor (student research) [Summary CV supervisor Gray]	1	20 May 2016
Validated questionnaire [The Summary of Diabetes Self-Care Activities]	1.0	07 April 2017

IRAS project ID	210623

Appendix B - Summary of HRA Assessment

This appendix provides assurance to you, the sponsor and the NHS in England that the study, as reviewed for HRA Approval, is compliant with relevant standards. It also provides information and clarification, where appropriate, to participating NHS organisations in England to assist in assessing and arranging capacity and capability.

For information on how the sponsor should be working with participating NHS organisations in England, please refer to the, *participating NHS organisations*, *capacity and capability* and *Allocation of responsibilities and rights are agreed and documented (4.1 of HRA assessment criteria)* sections in this appendix.

The following person is the sponsor contact for the purpose of addressing participating organisation questions relating to the study:

Sponsor name: Heather House
Email: ctrg@admin.ox.ac.uk

HRA assessment criteria

Section	HRA Assessment Criteria	Compliant with Standards	Comments
1.1	IRAS application completed correctly	Yes	This study is linked to a previously approved study_REC reference 16/NE/0203.
2.1	Participant information/consent documents and consent process	Yes	No comments
3.1	Protocol assessment	Yes	No comments
4.1	Allocation of responsibilities and rights are agreed and documented	Yes	A sponsor GP site agreement will act as agreement of an NHS organisation to participate. A statement of activities and schedule of events has been supplied as information only.
4.2	Insurance/indemnity arrangements assessed	Yes	Where applicable, independent contractors (e.g. General Practitioners) should ensure that the professional indemnity provided by their medical defence organisation covers the

Section	HRA Assessment Criteria	Compliant with Standards	Comments
			activities expected of them for this research study
4.3	Financial arrangements assessed	Yes	There has been no application for external funding and no funding will be available to the NHS site.
5.1	Compliance with the Data Protection Act and data security issues assessed	Yes	No comments
5.2	CTIMPS – Arrangements for compliance with the Clinical Trials Regulations assessed	Not Applicable	No comments
5.3	Compliance with any applicable laws or regulations	Yes	No comments
6.1	NHS Research Ethics Committee favourable opinion received for applicable studies	Yes	REC favourable opinion was issued by North West Preston REC on 24 April 2017.
6.2	CTIMPS – Clinical Trials Authorisation (CTA) letter received	Not Applicable	No comments
6.3	Devices – MHRA notice of no objection received	Not Applicable	No comments
6.4	Other regulatory approvals and authorisations received	Not Applicable	No comments

Participating NHS Organisations in England

This provides detail on the types of participating NHS organisations in the study and a statement as to whether the activities at all organisations are the same or different.
There is one NHS site type acting as a full research site; performing the study activities described in the study protocol/schedule of events. The Chief Investigator or sponsor should share relevant study documents with participating NHS organisations in England in order to put arrangements in place to deliver the study. The documents should be sent to both the local study team, where applicable, and the office providing the research management function at the participating organisation. For NIHR CRN Portfolio studies, the Local LCRN contact should also be copied into this correspondence. For further guidance on working with

participating NHS organisations please see the HRA website.

If chief investigators, sponsors or principal investigators are asked to complete site level forms for participating NHS organisations in England which are not provided in IRAS or on the HRA website, the chief investigator, sponsor or principal investigator should notify the HRA immediately at hra.approval@nhs.net. The HRA will work with these organisations to achieve a consistent approach to information provision.

Confirmation of Capacity and Capability

This describes whether formal confirmation of capacity and capability is expected from participating NHS organisations in England.

Participating NHS organisations in England **will be expected to formally confirm their capacity and capability to host this research**.

- Following issue of this letter, participating NHS organisations in England may now confirm to the sponsor their capacity and capability to host this research, when ready to do so. How capacity and capacity will be confirmed is detailed in the *Allocation of responsibilities and rights are agreed and documented (4.1 of HRA assessment criteria)* section of this appendix.
- The Assessing, Arranging, and Confirming document on the HRA website provides further information for the sponsor and NHS organisations on assessing, arranging and confirming capacity and capability.

Principal Investigator Suitability

This confirms whether the sponsor position on whether a PI, LC or neither should be in place is correct for each type of participating NHS organisation in England and the minimum expectations for education, training and experience that PIs should meet (where applicable).

A local PI is expected to be in place at the NHS site to oversee the research activities and facilitate access for the external central research team to the GP practice where required.

The applicant has confirmed that study training on the protocol will be delivered to the GP practice and GCP training is expected of the local PI.

GCP training is not a generic training expectation, in line with the HRA statement on training expectations.

HR Good Practice Resource Pack Expectations

This confirms the HR Good Practice Resource Pack expectations for the study and the pre-engagement checks that should and should not be undertaken

As a non-commercial single site study taking place in the NHS it is unlikely that letters of access or honorary research contracts will be applicable, except where local staff employed by another Trust (or University) are involved (and then it is likely that arrangements are already in place). A Letter of Access based on standard DBS checks and occupational health clearance would be appropriate.

IRAS project ID	210623

Other Information to Aid Study Set-up

This details any other information that may be helpful to sponsors and participating NHS organisations in England to aid study set-up.
The applicant has indicated that they do not intend to apply for inclusion on the NIHR CRN Portfolio.

L

Questionnaires for participants (pilot study)

L.1 Primary outcomes

L.1.1 Satisfaction with the intervention

	Strongly disagree	Disagree	Undecided	Agree	Strongly agree
1. The doctor made me aware of the different options available to manage diabetes (lifestyle, treatments)					
2. The doctor gave me the chance to express my opinions about the different options (lifestyle, treatments)					
3. The doctor gave me the chance to ask for as much information as I needed about the different options (lifestyle, treatments)					
4. The doctor gave me enough information about the options available (lifestyle, treatments)					
5. The doctor gave enough explanation of the information about the options available (lifestyle, treatments)					
6. The information given to me was easy to understand					
7. I know the advantages of managing or not managing diabetes					
8. I know the disadvantages of managing or not managing diabetes					
9. The doctor gave me a chance to decide which option I thought was best for me					
10. The doctor gave me a chance to be involved in the decisions during the consultation					

L.1.2 Anxiety and worry

Please circle the most appropriate number. There are no right or wrong answer.

1. Thinking of my estimated risk of developing a heart attack or stroke in the next 10 years makes me feel:

   - **Anxious**: Not at all 1 2 3 4 5 6 7 Very much
   - **Worried**: Not at all 1 2 3 4 5 6 7 Very much

2. Thinking of my estimated heart age (as compared to a well-controlled person) makes me feel:

   - **Anxious:** Not at all 1 2 3 4 5 6 7 Very much
   - **Worried**: Not at all 1 2 3 4 5 6 7 Very much

3. Thinking of my estimated number of hours of life lost per day (as compared to a well-controlled person) makes me feel:

   - **Anxious:** Not at all 1 2 3 4 5 6 7 Very much
   - **Worried**: Not at all 1 2 3 4 5 6 7 Very much

L.1.3 General anxiety

Please tick the most appropriate box to indicate **how you feel right now, at this moment**. Do not spend too much time on any one statement but give the answer which seems to describe you present feelings best.

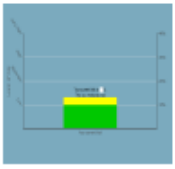
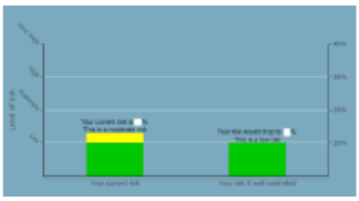
	NOT AT ALL	**Somewhat**	**Moderately**	**VERY MUCH**
1. I feel calm				
2. I am tense				
3. I feel upset				
4. I am relaxed				
5. I feel content				
6. I am worried				

L.2 Secondary outcomes

L.2.1 Recall of personalised risk information

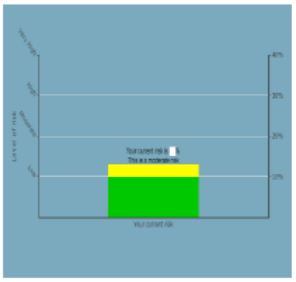
a Intervention group

The doctor has just calculated your personalised health risk estimates on his computer. Do you remember these numbers?

1. **Current risk** of developing a heart attack or stroke in the next 10 years: 	..... %
2. With perfectly controlled risk factors, **this risk can drop to**: 	..... %
3. **Heart age** (as compared to a perfectly controlled person): 	..... years
4. **Hours of life lost per day** (as compared to a perfectly controlled person): 	..... hours of life per day (that could be gained back)

b Control group

The doctor has just calculated your personalised health risk estimate on his computer. Do you remember this number?

Current risk of developing a heart attack or stroke in the next 10 years: 	..... %

L.2.2 Intentions to make lifestyle changes

For each statement, please tick the box that best describes **what you feel.**

	Totally DISAGREE	**Partially DISAGREE**	**Neither AGREE nor DISAGREE**	**Partially AGREE**	**Totally AGREE**
1. I **intend** to be more physically active within two months					
2. I **think that I will** be more physically active within two months					
3. I **intend** to eat healthier food within two months					
4. I **think that I will** eat healthier within two months					
Only if you are a smoker:					
5. I **intend** to stop smoking within a year					
6. I **think that I will** stop smoking within the next two months					

L.2.3 Self-management behaviour

The following questions ask you about your diabetes self-care activities during the past 7 days. **Please circle the right number of days for each question**. If you were sick during the past 7 days, please think back to the last 7 days that you were not sick.

Diet

1. On how many of the last SEVEN DAYS did you eat **five or more** servings of fruits and vegetables?

   0 1 2 3 4 5 6 7

2. On how many of the last SEVEN DAYS did you eat high fat foods (e.g. **red meat, full-fat dairy products, sweets, cakes**…)?

   0 1 2 3 4 5 6 7

Exercise

3. On how many of the last SEVEN DAYS did you participate in at least **30 minutes of continuous physical activity**? (Including walking).

   0 1 2 3 4 5 6 7

4. On how many of the last SEVEN DAYS did you participate in **specific exercise session** (such as swimming, walking, biking) other than what you do around the house or as part of your work?

   0 1 2 3 4 5 6 7

Blood Sugar Testing

5. On how many of the last SEVEN DAYS did you test your blood sugar?

0 1 2 3 4 5 6 7

Foot Care

6. On how many of the last SEVEN DAYS did you check your feet?

0 1 2 3 4 5 6 7

Smoking

7. Have you smoked a cigarette – even one puff – during the past SEVEN DAYS?

   Yes No

   If yes, how many cigarettes did you smoke on an average day? .....

Medications

8. On how many of the last SEVEN DAYS did you take your recommended insulin injections?

   I don't take insulin 0 1 2 3 4 5 6 7

9. On how many of the last SEVEN DAYS did you take your recommended number of diabetes pills?

   I don't take pills 0 1 2 3 4 5 6 7