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RESEARCH ARTICLE

Medical progress, relaxed natural selection, and adolescent obesity: implications for global health

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ABSTRACT

Objective: To examine the role of relaxed natural selection, measured using the Henneberg Index (I_{bs}), in influencing adolescent obesity prevalence across 191 countries.
Methods: Population-level variables, including adolescent obesity prevalence, I_{bs} (Henneberg Index), GDP PPP, urbanization, and calorie intake, were obtained from United Nations sources. The relationship between the Henneberg Index and adolescent obesity was analyzed using curvilinear and linear regression models with raw and log-transformed data to address non-homoscedasticity. Regional correlations were explored by grouping countries.
Results: A significant correlation (r~0.5) between the Henneberg Index and adolescent obesity was found and remained consistent through third-order polynomial regression and partial correlations after adjusting for GDP PPP, urbanization, and calorie intake. The correlation was stronger in developing countries compared to developed ones. Stepwise multiple regression analysis identified the Henneberg Index as the second most significant predictor of adolescent obesity, following GDP PPP. Calorie intake did not significantly predict adolescent obesity in the models.
Conclusions: Reduced natural selection, facilitated by medical practices allowing individuals with obesity-linked traits to reproduce, may contribute to the population-level accumulation of these traits, increasing adolescent obesity. These findings underscore the need to consider evolutionary and genetic factors alongside environmental and socioeconomic determinants in developing obesity prevention strategies.

PLAIN LANGUAGE SUMMARY

This study explores how improvements in healthcare and living conditions might contribute to rising obesity rates among adolescents worldwide. As modern medicine helps more people survive and have children, natural selection—the process through which harmful genes are gradually reduced in a population—has become weaker. We measured this reduction using the Henneberg Index, which shows how many people in a population live through their full reproductive years.

By analyzing data from 191 countries, we found that nations with higher Henneberg Index values, meaning lower natural selection, also have higher rates of adolescent obesity. This relationship remained significant even after accounting for calorie intake, economic development, and urbanization. The findings suggest that genetic factors linked to obesity may be accumulating over generations as a result of medical progress. Recognizing this long-term effect could help improve future obesity prevention strategies that address both biological and environmental factors.

ARTICLE HIGHLIGHTS

- The Henneberg Index (Biological State Index) quantifies the degree of reduced natural selection in human populations.
- This study examined the association between the Henneberg Index and adolescent obesity across 191 countries.
- Both curvilinear and linear analyses revealed significant positive correlations between the Index and obesity prevalence.
- The association remained significant after adjusting for calorie intake, GDP, and urbanization.

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- The relationship was stronger in developing than developed countries, indicating greater variability in natural selection and obesity patterns.
- Findings suggest that reduced natural selection may contribute to obesity prevalence alongside socioeconomic and environmental drivers.

1. Introduction

Obesity is a chronic, multifactorial, and largely preventable condition characterized by excessive or abnormal fat accumulation that poses significant health risks [1]. As a complex phenotype, it arises from dynamic interactions between environmental exposures and genetic predispositions, leading to substantial variability in susceptibility across individuals and populations [2–4]. It arises from a complex interplay between genetic predispositions and environmental exposures, contributing to a broad spectrum of non-communicable diseases (NCDs) such as diabetes, cardiovascular diseases, and certain cancers. Although obesity and its related NCDs are considered preventable [5–7], global efforts to curb their prevalence have met limited success. Despite numerous interventions—ranging from behavioral and dietary programs to public health campaigns—obesity rates continue to rise worldwide [8,9]. This persistent increase underscores obesity as a major public health challenge, prompting calls for population-based strategies that address "obesogenic" environments [10–13].

While environmental and lifestyle factors such as diet and physical inactivity are critical, genetic susceptibility also plays a substantial role. Evidence from family and twin studies suggests that genetic factors explain approximately 30–70% of the variance in body mass index (BMI) [14,15]. At the population level, genetic background is estimated to account for 30–50% of cross-national variation in obesity prevalence [16]. These findings highlight the importance of considering evolutionary and genetic mechanisms when interpreting contemporary obesity trends—particularly the interplay between relaxed natural selection and the rising prevalence of obesity.

The Henneberg Index, also known as the Biological State Index (Ibs), provides a quantitative measure of the extent to which natural selection operates within a population. In this study, the Index is calculated using age-specific fertility and mortality data from the beginning of the 21st century United Nations demographic datasets, which reflect survival through the reproductive period (15–50 years). This demographic basis aligns with established methods for estimating the current degree of natural selection in human populations.

Originally proposed by Henneberg (1975), I_{bs} is calculated using the following formula [17–20]:

$$Henneberg\ index\,/\,Biological\ State\ Index\left(I_{bs}\right)=1-\sum_{x=0}^{x=\omega}d_x s_x$$

Where

d_x = the frequency of deaths at age x

s_x =the probability of not having completed fertility at age x

ω: the age at death of the oldest member of the group

A higher Henneberg Index value indicates reduced opportunity for natural selection to act through differential mortality, allowing a greater proportion of individuals to survive to and through their entire reproductive period (15–50 years) and fully realize their fertility [17,18]. At the population level, the Index represents the fraction of individuals who successfully pass on their genetic material to the next generation. A value approaching 1 signifies a population in which advanced medical and social conditions have largely neutralized natural selection, while a value near 0 reflects populations still strongly constrained by selection pressures that limit reproductive success [19].

The Henneberg Index, therefore quantifies the probability of gene transmission within a population. Modern medical practice and improved living conditions have substantially weakened the process of natural selection by reducing mortality associated with genetic or health-related disadvantages—a phenomenon termed reduced natural selection [20,21]. Under normal evolutionary balance, the constant introduction of new mutations in the human genome [22] is counteracted by stabilizing selection that prevents the widespread persistence of deleterious alleles. However, as selection pressures diminish, this equilibrium shifts toward accumulation of disadvantageous genetic variants, a process proposed as a contributing factor to the global rise in obesity [7,19,23].

As medical advances enable increasing numbers of individuals—regardless of genetic predispositions affecting fertility or survival—to reproduce, obesity-related genes that might once have been selected against can persist and propagate across generations [4,19,24–26]. This mechanism suggests that the rising prevalence of obesity may partly reflect a long-term genetic accumulation process, superimposed on environmental and behavioral risk factors. The Henneberg Index has thus been employed as an indicator of reduced natural selection and the potential accumulation of deleterious gene variants at the population level, having demonstrated positive correlations with diseases such as cancer [27], dementia [28], type 1 diabetes [20], and adult obesity [7,23].

In this study, the Henneberg Index is used as a proxy for the relaxation of natural selection resulting from medical and public health progress. A higher Index value reflects weaker selective pressures and a greater likelihood of the persistence of obesity-associated genetic traits [29–34]. The study aims to test whether populations with higher Henneberg Index values exhibit higher adolescent obesity prevalence, even after accounting for key socioeconomic and environmental covariates such as energy intake, GDP, and urbanization. Understanding this evolutionary dimension may enrich obesity research by revealing how the genetic consequences of improved survival and fertility intersect with modern environments. This perspective provides a novel explanatory framework for global obesity trends and may inform more sustainable, multi-dimensional strategies to mitigate obesity risk in future generations.

2. Methods

2.1. Data collection and selection

This study examined the role of the Henneberg Index (Biological State Index) in influencing global variation in adolescent obesity prevalence using a cross-sectional ecological design. Three analytical objectives guided the investigation:

1. To determine the overall correlation between the Henneberg Index and adolescent obesity at global and regional (country-group) levels without controlling for confounders;
2. To assess the independent relationship between the Henneberg Index and adolescent obesity after adjusting for major contextual variables—calorie intake, GDP, and urbanization; and
3. To compare the relative predictive strength of the Henneberg Index with these economic and environmental factors in explaining variation in adolescent obesity.

Data for five key variables were obtained from established international sources. The Henneberg Index was derived from previously published datasets [7,19,20,23,27,28]. It indicates the opportunity for selection in the first decade of the 21st century, thus close to the date of birth of current adolescents. Adolescent obesity prevalence—defined as the percentage of individuals aged 10–19 years with a body mass index (BMI) ≥ +2 standard deviations above the median—was sourced from the World Health Organization (WHO) Global Health Observatory (2022) [35]. The average per capita daily calorie intake was obtained from the Food and Agriculture Organization (FAO), based on the 2019–2021 mean [36]. Gross domestic product (GDP) per capita at purchasing power parity (PPP) and urbanization rates (percentage of population residing in urban areas) were extracted from The World Bank (2021) [37–40].

All variables were analyzed in their raw form (non-log-transformed) for the initial correlation analyses. The study included 191 countries, though the number contributing to each variable varied slightly due to differences in data availability across United Nations agencies. Each country was treated as a single analytical unit, reflecting a macro-epidemiological approach to investigating population-level relationships.

Economic and environmental indicators—namely GDP, calorie intake, and urbanization—were included as potential confounding factors to control for contextual influences. Economic development is often linked to greater availability of calorie-dense foods and reduced physical activity opportunities [39,40], while urbanization can exert complex effects: improving access to healthcare and education but simultaneously promoting sedentary lifestyles and unhealthy dietary behaviors [2,18,41–43].

Economic and environmental variables (GDP (PPP), calorie intake, and urbanization) were selected as confounders because they influence both components of the Henneberg Index (mortality and fertility patterns) and established determinants of obesity prevalence. These variables capture the major socioeconomic and environmental conditions shaping population survival, dietary energy availability, and lifestyle-related obesity risks. Their appropriateness was confirmed empirically: in the entire regression model, GDP and urbanization significantly predicted adolescent obesity, and the full model explained 62.1% of its variance. The unadjusted model showed that the Henneberg Index alone explained 19.98% of the variance, indicating that the chosen confounders effectively accounted for shared influences while leaving a substantial independent contribution attributable to reduced natural selection.

2.2. Data robusticity check

Potential multicollinearity among the five variables was assessed using the Variance Inflation Factor (VIF) and tolerance statistics in SPSS version 31. The diagnostic results indicated that all tolerance values were above 0.20 and all VIFs were below 5 [44], confirming the absence of multicollinearity issues [44]. Detailed results are presented in Supplementary File 1.

The Kolmogorov–Smirnov and Shapiro–Wilk tests were performed using SPSS version 28 to assess normality. Both tests indicated that none of the five variables followed a normal distribution ($p < 0.05$), as detailed in Supplementary File 1. Therefore, appropriate statistical approaches were applied to accommodate non-normal distributions in subsequent analyses.

2.3. Data analyses considering the abnormal distributions of variables

Given the non-normal distribution of variables, both parametric and non-parametric approaches were employed to ensure analytical robustness. The relationships between the Henneberg Index and adolescent obesity were examined using linear (Pearson's r) and non-linear (curvilinear regression) models [7,45]. This dual-method strategy allowed for cross-validation of results and identification of potential non-linear trends. Statistical significance was defined at three levels: $p < 0.05$, $p < 0.01$, and $p < 0.001$.

2.4. Non-linear and linear correlation analyses

Curvilinear regression models (polynomial best fits) were first applied to capture potential non-linear associations between the Henneberg Index and adolescent obesity prevalence. Linear relationships were then examined using Pearson's correlation coefficients, complemented by Spearman's rho analyses to confirm consistency across non-parametric methods. Comparative results of both models are presented in the main text, with full details available in Supplementary File 2.

2.5. Regional correlation analyses

To explore potential geographic and socioeconomic variation, analyses were stratified by continent, income level, and cultural grouping [7,20]. This allowed examination of differences in the magnitude and direction of correlations between the Henneberg Index and adolescent obesity across diverse contexts. These subgroup analyses provided further insight into how demographic transition, economic development [41], and healthcare access may shape the genetic–environmental interplay underlying adolescent obesity patterns worldwide.

3. Results

3.1. Global association (curvilinear, raw data)

A global scatterplot of adolescent obesity prevalence against the Henneberg Index revealed a significant positive curvilinear relationship (polynomial best fit), with $r = 0.4907$ and $R^2 = 0.2408$ ($n = 190$, $p < 0.001$). This indicates that the Henneberg Index accounts for approximately 24% of the between-country variation in adolescent obesity prevalence (Figure 1; Table 1).

3.2. Robustness to confounding (curvilinear residualisation, raw data)

To evaluate the independence of the association from major covariates, adolescent obesity was sequentially standardized for potential confounders: calorie intake (1st order), calorie intake plus GDP (2nd order), and calorie intake plus GDP and urbanization (3rd order). The Henneberg Index remained significantly correlated with adolescent obesity at each stage, although the strength of association progressively declined:

- 1st order: $r = 0.3483$, $R^2 = 0.1213$ ($n = 183$, $p < 0.001$)
- 2nd order: $r = 0.1855$ ($n = 183$, $p < 0.05$)
- 3rd order: $r = 0.1916$ ($n = 183$, $p < 0.05$)

A comparison of the unadjusted and third-order adjusted correlations using Fisher's *r*-to-*z* transformation confirmed a significant reduction in strength ($z = 3.28$, $p < 0.01$), indicating that while socioeconomic and

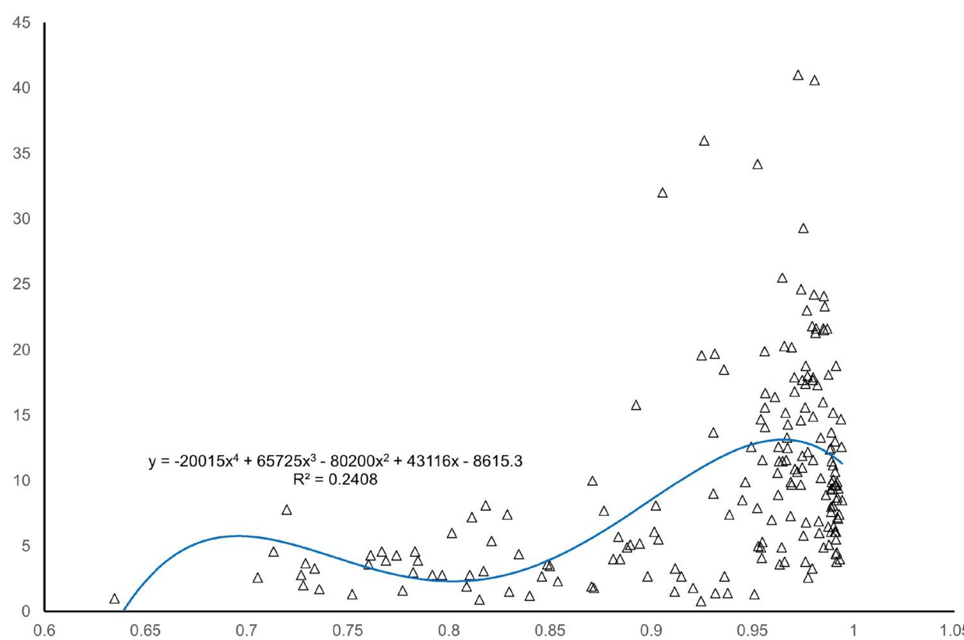

Figure 1. Relationship between population-level reduction in natural selection (Henneberg Index) and adolescent obesity prevalence across 191 countries.
Data definition and source: Henneberg Index (Biological State Index) from You & Henneberg [27]; adolescent obesity prevalence (% of individuals aged 10–19 years with BMI > +2 SD above median) from WHO Global Health Observatory (2022). All data analyzed in raw form.

Table 1. Global curvilinear and linear correlations between the henneberg Index (i_{bs}) and adolescent obesity prevalence.

Analysis type	Adjustment (confounder control)	Regression equation / model	Correlation coefficient (r)	R^2 (%)	Sample (*n*)
Curvilinear regression (raw data, Excel®)	None	$y = -20015x^4 + 65725x^3 - 80200x^2 + 43116x - 8615.3$	**0.4907*** **	**24.08**	190
	Controlled for calorie intake (1st order)	$y = -6E{-}06x^2 + 0.0367x - 47.821$	0.3483***	12.13	183
	Controlled for calorie intake + GDP (2nd order)	$y = -1111.1x^3 + 2805.3x^2 - 2329.3x + 634.56$	0.1855*	3.44	183
	Controlled for calorie intake + GDP + urbanization (3rd order)	$y = -1372.5x^3 + 3431.4x^2 - 2824.4x + 764.09$	0.1916*	3.67	183
Linear correlation (SPSS v31)	None	Pearson's r	**0.447*** **	19.98	190
	Controlled for calorie intake	Partial r	0.389***	—	178
	Controlled for calorie intake + GDP	Partial r	0.391***	—	173
	Controlled for calorie intake + GDP + urbanization	Partial r	0.363***	—	173

Data definitions and sources: Henneberg Index (Biological State Index) from You & Henneberg (2018); adolescent obesity prevalence (% of adolescents aged 10–19 years with BMI > +2 SD above median) from WHO Global Health Observatory (2022); calorie intake (daily per-capita energy intake, averaged 2019–2021) from FAO; GDP (PPP per capita) and urbanization (% population in urban areas) from the World Bank (2021). All variables were analyzed in raw form for curvilinear models and log-transformed for residual and correlation analyses. Significance levels: $p < 0.05$ (*), $p < 0.01$ (**), $p < 0.001$ (***).

environmental adjustments attenuate the relationship, the Henneberg Index retains a statistically independent association with adolescent obesity (Table 1).

3.3. Linear correlation analyses

Two complementary linear approaches were used to verify the robustness of the relationship between the Henneberg Index and adolescent obesity.

The global Pearson correlation yielded r = 0.447 ($n = 190$, $p < 0.001$), explaining 19.98% of the variance in adolescent obesity. After sequential adjustment for calorie intake (r = 0.389, $n = 178$), calorie intake plus GDP (r = 0.391, $n = 173$), and calorie intake plus GDP and urbanization (r = 0.363, $n = 173$), the association remained statistically significant (all $p < 0.001$). Although attenuated, the reduction from the unadjusted correlation (r = 0.447) to the fully adjusted value (r = 0.363) was not statistically significant (z = 0.96, $p = 0.337$), demonstrating that the relationship is not driven solely by shared socioeconomic or environmental factors. These results confirm that reduced natural selection, as indexed by the Henneberg Index, contributes independently to adolescent obesity beyond major contextual determinants.

3.4. Log-transformed bivariate correlations (robustness check)

To improve homoscedasticity, all variables were log-transformed and reanalyzed using both Pearson and Spearman methods. The results remained consistent, with a bivariate Pearson correlation of $r = 0.698$ and Spearman's ρ = 0.595 (both $p < 0.001$; SF 2), confirming a strong and monotonic positive relationship between reduced natural selection and adolescent obesity. These findings demonstrate that the observed correlations are robust to data transformation and not driven by heteroscedasticity or outliers.

3.5. Regional analyses

Country-group analyses revealed moderate-to-strong positive correlations between the Henneberg Index and adolescent obesity across most regional, economic, and cultural classifications (Table 2). In alignment with WHO reports indicating that the majority of pediatric obesity occurs in developing countries, the association was substantially stronger in developing, low- and middle-income, and African and Latin American regions. Conversely, the relationship was weaker in high-income and European subgroups, where genetic variability is lower and healthcare systems tend to be more uniform. Fisher's *r*-to-*z* transformations confirmed that these differences were statistically significant in both the curvilinear ($|z| \approx 2.58$, $p < 0.01$) and linear analyses ($|z| \approx 3.26$, $p < 0.01$). Collectively, these patterns indicate greater cross-national variability in the relaxation of natural selection—and its association with adolescent obesity—within developing regions undergoing rapid demographic and epidemiological transitions.

3.6. Multivariable models

In the enter regression models, GDP (β ≈ 0.529, $p < 0.001$) and urbanization (β ≈ 0.183, $p < 0.05$) were significant predictors when the Henneberg Index was excluded. When the Henneberg Index was added, it emerged as an additional strong independent predictor (β ≈ 0.392, $p < 0.001$), and together these variables explained 62.1% of the variance in adolescent obesity. Calorie intake remained negligible across all models.

Stepwise regression further supported these findings: introducing the Henneberg Index increased the adjusted R^2 from 0.544 to 0.621 ($\Delta R^2 = 0.077$), indicating that the Index explains an additional 7.7% of variance beyond economic and environmental covariates (Table 3).

Overall, across both modeling approaches, the Henneberg Index consistently emerged as one of the strongest independent predictors of adolescent obesity at the population level. These results suggest that genetic accumulation under relaxed natural selection contributes meaningfully to global variation in adolescent obesity, complementing established socioeconomic and environmental determinants.

Taken together, the above findings demonstrate that while economic development and urbanization contribute substantially to adolescent obesity, the Henneberg Index—reflecting reduced natural selection—adds

Table 2. Regional curvilinear and linear correlations between the henneberg Index (ibs) and adolescent obesity prevalence across country groupings.

Country grouping	Sample size (n)	Best-fit equation (curvilinear regression)	Curvilinear r	Linear pearson's r	Significance (*p*)
UN classification					
Developed	45	$y = 2975.1x^2 - 5713.9x + 2749.4$	0.177	0.077	ns
Developing	145	$y = 298.36x^2 - 456.71x + 177.26$	**0.560**	**0.572**	$p < 0.001$
Fisher's r-to-z (Developing vs. Developed)	—	—	z = 2.58	z = 3.26	$p < 0.01$
World Bank income classification					
Low income	29	$y = 0.0159e^{6.7399}x$	0.578	0.566	$p < 0.01$
Lower-middle income	48	$y = -1149x^3 + 3162.7x^2 - 2840.1x + 839.28$	0.562	0.546	$p < 0.001$
Upper-middle income	51	$y = 10.588x^{4.6643}$	0.522	0.516	$p < 0.001$
High income	61	$y = -10724x^3 + 27832x^2 - 23850x + 6750.4$	0.451	0.255	$p < 0.05$
WHO regional classification					
Africa (AFRO)	47	$y = -597.59x^3 + 1603.9x^2 - 1370.1x + 379.44$	**0.627**	**0.612**	$p < 0.001$
Americas (AMRO)	34	$y = 108.54x^2 - 130.45x + 40.04$	0.518	0.567	$p < 0.001$
Eastern Mediterranean (EMRO)	21	$y = -2190.2x^3 + 5900.2x^2 - 5230.5x + 1533.2$	0.400	0.342	$p < 0.05$
Europe (EURO)	50	$y = -14410x^3 + 39232x^2 - 35462x + 10648$	0.473	0.391	$p < 0.01$
South-East Asia (SEARO)	11	$y = 3078.3x^3 - 7794x^2 + 6564.7x - 1834.7$	0.705	0.506	$p < 0.01$
Western Pacific (WPRO)	27	$y = -2363.7x^3 + 6162.5x^2 - 5277.1x + 1491.2$	0.335	0.325	$p < 0.01$
Cultural/economic groupings					
Asia Cooperation Dialogue (ACD)	30	$y = 347.34x^2 - 570.8x + 237.64$	0.480	0.469	$p < 0.01$
Asia-Pacific Economic Cooperation (APEC)	18	$y = -1167.6x^3 + 3329.9x^2 - 3079.5x + 930.72$	0.638	0.554	$p < 0.05$
Arab World	21	$y = -3800.8x^3 + 10334x^2 - 9293.4x + 2771$	0.463	0.442	$p < 0.05$
English-speaking countries	54	$y = -5689.3x^3 + 14736x^2 - 12579x + 3546.5$	0.517	0.503	$p < 0.001$
European Economic Area (EEA)	29	$y = -40275x^3 + 111338x^2 - 102396x + 31338$	0.480	0.228	$p < 0.01$
European Union (EU)	27	$y = -40396x^3 + 111522x^2 - 102432x + 31310$	0.486	0.124	$p < 0.05$
Latin America (LA)	22	$y = 2844.7x^3 - 7606.5x^2 + 6808.2x - 2029.8$	**0.690**	**0.677**	$p < 0.001$
Latin America & Caribbean (LAC)	32	$y = -5927.2x^3 + 15670x^2 - 13698x + 3968.3$	0.501	0.476	$p < 0.01$
OECD	36	$y = -5218.9x^3 + 13692x^2 - 11847x + 3383.1$	0.494	0.394	$p < 0.05$
Southern African Development Community (SADC)	16	$y = 3732.9x^3 - 9218.7x^2 + 7571.5x - 2064.5$	**0.881**	**0.677**	$p < 0.001$

Data definitions and sources: Henneberg Index (Biological State Index) from You & Henneberg (2018); adolescent obesity prevalence from WHO Global Health Observatory (2022). Curvilinear analyses used raw data; linear correlations (Pearson's *r* and Spearman's ρ) were performed on log-transformed data to enhance homoscedasticity.

a distinct genetic-demographic dimension, warranting further discussion of its biological and societal implications.

4. Discussion

From an environmental perspective, it is well-established that the global increase in obesity prevalence is a largely preventable health condition; however, prevention efforts have yet to succeed. Analyzing global data on the relationship between reduced natural selection and adolescent obesity prevalence, while accounting for the confounding effects of calorie intake, GDP, and urbanization, suggests that reduced natural selection, due to current medical practice, may be a significant determinant of adolescent obesity. This effect appears resistant to prevention through public health interventions.

Table 3. Multiple linear regression analyses describing the predictive effects of the henneberg Index (i_{bs}) and confounding variables on adolescent obesity prevalence.

Model type	Predictor variable(s)	Standardized β	Significance (*p*)	Adjusted R^2	Model interpretation
Enter model – Henneberg Index not included	Calorie intake	0.568	0.450 (ns)	0.544	Economic (GDP) and environmental (urbanization) factors significantly predict adolescent obesity when genetic influence (ibs) is excluded.
	GDP (PPP per capita)	0.529	<0.001	—	
	Urbanization	0.183	<0.050	—	
Enter model – Henneberg Index included	Henneberg Index (Ibs)	0.392	<0.001	**0.621**	Addition of the henneberg index improves model fit (ΔR^2 = 0.077) and remains a strong independent predictor alongside GDP and urbanization.
	Calorie intake	− 0.020	0.851 (ns)	—	
	GDP (PPP per capita)	0.307	<0.001	—	
	Urbanization	0.184	<0.010	—	
Stepwise model – Henneberg Index not included	GDP (PPP per capita)	—	<0.001	0.235	GDP remains the sole significant predictor; urbanization and calorie intake are not selected.
	Urbanization	—	Insignificant	—	
	Calorie intake	—	Insignificant	—	
Stepwise model – Henneberg Index included	Henneberg Index (I_{bs})	—	<0.001	**0.321**	The henneberg index enters first as the strongest predictor, followed by urbanization (ΔR^2 = + 0.017).
	Urbanization	—	<0.050	**0.338**	
	GDP (PPP per capita)	—	Insignificant	—	
	Calorie intake	—	Insignificant	—	

Data definitions and sources: Henneberg Index (Biological State Index) from You & Henneberg (2018); adolescent obesity prevalence (% of adolescents aged 10–19 years with BMI > +2 SD above median) from WHO (2022); calorie intake from FAO (2019–2021 average); GDP (PPP per capita) and urbanization (% urban population) from the World Bank (2021). All data were log-transformed for regression analyses.

Before evaluating the public health implications of this work, it is important to acknowledge the study's key limitations and the distinction between individual- and population-level determinants of health. This distinction is essential for interpreting our findings within an appropriate epidemiological framework [34].

Firstly, the study examines population-level patterns in adolescent obesity. While a significant association was observed between reduced natural selection and obesity prevalence, the findings do not imply individual-level causality. The concept of an "obesity-associated genetic background" refers to population-level tendencies rather than specific genes, and evidence for precise obesity-related genetic markers remains limited and complex.

Secondly, the assumption that elevated body fatness is deleterious before reproductive age is not universally supported. Reduced natural selection may allow a wider range of genetic variants—including those potentially linked to obesity—to persist across generations.

Thirdly, this study highlights an unintended consequence of medical progress. The interpretation of our findings requires caution, as they challenge the commonly held belief that higher levels of medical advancement always produce uniformly healthier populations.

Although major socioeconomic and environmental confounders were controlled for, residual or unmeasured confounding cannot be fully ruled out, as is inherent in ecological analyses. Factors such as lifestyle variations, differences in healthcare access, or population-specific genetic structures may influence both the Henneberg Index and adolescent obesity. Therefore, the results should be interpreted as evidence of an independent association rather than definitive causal inference.

Despite these limitations, reduced natural selection emerges as a meaningful factor in the global rise of adolescent obesity. The Henneberg Index shows a strong and statistically significant association with adolescent obesity, independent of environmental variables. When combined with GDP and urbanization, the correlation between calorie intake and adolescent obesity disappears, indicating that average national calorie intake does not exert an independent effect. The association is notably stronger in developing countries, likely due to greater variation in both obesity levels and natural selection pressures.

Natural selection is a multi-generational process through which human populations adapt to their environments via the combined effects of differential fertility and mortality [42]. Individuals who are better suited to their environment tend to survive longer, which increases their likelihood of completing reproduction and producing more offspring. Acting alone or together, fertility and mortality determine the strength of natural selection in a population and shape its overall reproductive fitness [43]. The proportion of individuals who survive through the full reproductive period, quantified by the Henneberg Index (Ibs), is determined by age-specific fertility and mortality rates [19].

Under natural conditions, individuals whose genetic background is poorly suited to their environment are less likely to survive to complete reproduction, and therefore contribute less to the gene pool. Modern medical practice has altered this process by reducing premature mortality, allowing individuals with unfavorable or previously disadvantageous genetic backgrounds to survive and reproduce. These individuals effectively become adapted to an environment that includes medical support. Historically, carriers of metabolic or obesity-related genetic disadvantages were less likely to reach reproductive age, which restricted the transmission of such variants. Improvements in healthcare, sanitation, nutrition, and living conditions have substantially increased survival before and during the reproductive period. As a result, selective forces that once constrained these variants have weakened, allowing them to persist and accumulate across successive generations. The rising Henneberg Index reflects this long-term relaxation of natural selection and provides a plausible evolutionary mechanism for the increasing population-level burden of obesity-related genetic susceptibility. When individuals with these genetic backgrounds reproduce, they pass these predispositions on to their offspring [23]. Because protecting human life is an ethical necessity, this relaxation of natural selection cannot and should not be reversed by limiting medical care.

Country-specific improvements in medical provision and broader societal conditions influence the fertility and mortality patterns that determine the extent of reduced natural selection [17,18]. Although healthcare systems play a central role, enhancements in sanitation, nutrition, and living standards also increase survival to reproductive completion. The current findings should therefore be interpreted within the combined influence of medical and non-medical contributors to reduced natural selection, which together can facilitate the accumulation of obesity-related genetic backgrounds at the population level.

Rapid advances in healthcare over the past two centuries, particularly in developed countries, have substantially reduced premature mortality [19,46,47]. Historical demographic evidence indicates that the Henneberg Index has increased steadily during this period, corresponding to approximately six to seven generations [19]. Earlier generations experienced substantially higher mortality before completing reproduction, so individuals with metabolic or health-related disadvantages contributed less to the gene pool. As survival improved, these individuals increasingly reached reproductive age and transmitted their genes. This long-term upward shift in I_{bs} provides the evolutionary context in which obesity-related variants have persisted and accumulated in modern populations.

A simple population genetics model offers support for this interpretation. Using conservative assumptions, such as recessive obesity-related alleles, low penetrance of ten percent, and mutation rates 10^{-4} to 10^{-6}, and incorporating approximately one thousand one hundred obesity-associated loci identified by genome-wide association studies [46,48], the model demonstrates that reductions in the strength of natural selection, expressed as equal to one minus I_{bs}, can produce measurable increases in the prevalence of obesity-linked genotypes across generations. Historical increases in I_{bs} from approximately 0.60 to approximately 0.99 support the likelihood that relaxed selection enables these variants to persist and accumulate, producing a phenotypic effect even at low penetrance. Although detailed modeling of environmentally mediated changes in gene expression is beyond the scope of this study, these findings highlight the importance of future research that integrates genomic, environmental, and epigenetic data. Environmental exposures, including diet, physical activity, and early-life conditions, may also influence gene expression through epigenetic mechanisms and may further shape obesity susceptibility over time.

Although relaxed natural selection has been operating for multiple generations, direct genomic evidence of cohort-level differences in obesity-related allele frequencies remains limited. Existing genome-wide association studies are predominantly cross-sectional and do not include longitudinal birth-cohort samples that span the period following the Second World War. As a result, temporal shifts in genetic susceptibility to obesity cannot yet be directly evaluated at the genomic level. Future analyses that incorporate multi-cohort genomic data would be valuable for assessing whether relaxed natural selection has produced measurable genetic change in recent decades. Nevertheless, because the Henneberg Index reflects multi-generational demographic processes rather than short-term allele frequency fluctuations, it remains a meaningful population-level indicator in the absence of cohort-based genomic data.

During this period, various heritable disease phenotypes may have been influenced by different degrees of reduced natural selection, depending on the types and levels of healthcare services available [43,47,49]. The stronger relaxation of natural selection in developed countries may partly explain why the burden of obesity is more prominent in these regions [27]. The effects of reduced natural selection on observable heritable traits have been documented for several conditions, including deformed nasal septa [50], type 1 diabetes [20], adult obesity [7,23,51], cancer [27], dementia [28] and lacrimal bone defects [52].

Obesity as a phenotype is determined by genetic background, environmental factors and their interaction. Adolescents have less environmental exposure than adults because of their young age. Compared to adult obesity, adolescent obesity should be determined more by the genetic background than environmental factors. Logically, the genetic risk factor for obesity measured by reduced natural selection (Henneberg Index), should be more associated with adolescent than adult obesity. This, however, may depend on how strongly environmental factors influence obesity. The correlation coefficient between adult obesity and the Henneberg Index reported in a previous study ($r = 0.6066$) [23] was compared with the adolescent obesity–Henneberg Index correlation identified in the present study ($r = 0.4907$). The difference between the two coefficients did not reach statistical significance based on Fisher's r-to-z transformation, indicating no significant difference in the strength of association between the two age groups. The possible other reasons for the insignificant difference may be different definitions of obesity in adolescents (BMI ≥ +2 standard deviations above the median) and adults (BMI≥ 30 kg/m^2) [35,53] and limited sample sizes due to the number of countries in the world that have full sets of information.

In parallel to the increase of obesity prevalence, the underweight prevalence is increasing as well because reduced natural selection also promotes more population with underweight health conditions for participating in reproduction and passing their body weight associated genetic background [23,50,54]. This issue should have attracted attention from the healthcare system as well. However, few studies were focused on increasing underweight prevalence. The underlying reason may be that slim body shape (low body mass) has been considered a desirable feature [23]. Due to the lack of underweight data at the population level, this hypothesis cannot be tested, but it is worth studying further.

In the past several decades, most of the populations in the world have tried to control "obesogenic" environments to reduce the obesity pandemic [11,12]. However, unfortunately, no country has reported that its strategies have been successful [13]. This may be due to overlooking reduced natural selection which has driven the accumulation of genetic background countering effects of environmental reductions of obesity [19].

Henneberg index is in significantly stronger correlation with adolescent obesity in developing countries than developed countries which is identified in both non-linear (curvilinear) and linear correlation. This is likely due to the fact that variability of both natural selection opportunity and obesity levels is greater in a group of countries with widely varied economic levels, while developed countries all have similar levels of opportunity for selection and obesity prevalence, thus smaller ranges of variation. The situation in developing countries illustrates well the role of relaxed selection in shaping obesity levels.

Interestingly, calorie intake did not show significant predicting effects on adolescent obesity when it was combined with GDP, urbanization and Henneberg index for observing their respective effects on adolescent obesity (enter and stepwise models). A previous study also reported that calorie intake did not correlate with late adolescent (conscript) obesity (18–20 years old) [23]. Calorie intake is conceptually associated with obesity due to its potential to cause an energy surplus if not offset by sufficient physical activity. Data on specific physical inactivity levels of adolescents by country were not available for this study. However, lower exercise levels can be gleaned from a common sedentary lifestyle proxy, urbanization. Urbanization had significant effects on adolescent obesity in this study.

This study offers new insights into the global rise of adolescent obesity by integrating evolutionary theory into contemporary health discourse. The findings suggest that the reduced opportunity for natural selection, resulting from medical and social progress, has allowed obesity-related genetic traits to persist and accumulate within populations. Over generations, this process may have contributed to the increasing prevalence of adolescent obesity worldwide. These results highlight the importance of addressing genetic and evolutionary factors, alongside environmental and behavioral determinants, in obesity prevention and management.

While it might be misinterpreted as an argument for eugenic interventions, this study firmly rejects such notions. Eugenics is both unethical and detrimental, as it undermines human rights and reduces genetic diversity [55,56]. Instead, emerging biomedical technologies such as genome editing hold potential for therapeutic innovation. For example, Glybera and Gendicine have been used clinically to treat lipoprotein lipase deficiency [57] and head and neck squamous cell carcinoma [58], respectively, demonstrating feasible gene-based interventions for metabolic conditions [59].

4.1. Public health implications

Reduced natural selection, driven by advances in medicine and social welfare that allow individuals with obesity-linked genetic traits to survive and reproduce, may be contributing to an increasing genetic predisposition to obesity. This recognition carries significant implications for public health. Conventional interventions focusing solely on diet, exercise, and lifestyle modification may not fully counteract inherited vulnerabilities. To achieve sustainable outcomes, public health frameworks should integrate evolutionary and genetic perspectives through:

1. Early-life metabolic interventions to reduce transgenerational transmission of risk factors.
2. Personalized prevention programs combining genetic screening with lifestyle guidance.
3. Public education campaigns acknowledging obesity as a condition influenced by both biology and behavior.

Recognizing the evolutionary dimension of obesity calls for a multidimensional strategy that unites environmental modification, genomic insight, and reproductive health awareness to strengthen long-term global obesity prevention.

5. Conclusions

Medical and social advancements may have reduced the strength of natural selection by lowering premature mortality and enabling more individuals to survive and reproduce. While obesity remains a complex, multifactorial condition with limited evidence for a single "obesity gene," the findings suggest that reduced natural selection has allowed obesity-related genetic traits to persist and accumulate across generations. Recognizing this evolutionary dimension highlights the potential for gene-based therapeutic approaches to complement existing strategies in addressing the global obesity challenge.

6. Public health implications

The findings reveal that reduced natural selection—driven by medical and social advances that allow individuals with obesity-linked genetic traits to survive and reproduce—may be contributing to a global accumulation of obesity susceptibility genes. This has important implications for public health policy and prevention strategies. Traditional interventions focusing solely on diet, physical activity, and environmental change may not fully address the biological underpinnings of obesity.

To mitigate this evolutionary contribution, health strategies should integrate genetic and evolutionary perspectives into population-level obesity prevention. This could include:

1. Early-life interventions targeting metabolic health to reduce transgenerational transmission of risk factors.
2. Personalized public health programs incorporating genetic screening and lifestyle counseling.
3. Public education highlighting that the obesity epidemic is not purely behavioral but also influenced by inherited vulnerability.

Recognizing the evolutionary dimension of obesity underscores the need for a multidimensional prevention approach—one that combines environmental modification with genomic and reproductive health awareness to achieve sustainable public health outcomes.

Author contributions

WY and MH contributed to the study design. Both authors conducted the data analysis and interpreted data analysis results. WY drafted the manuscript with the input from MH. Both authors read, edited and approved the final manuscript.

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Supplementary material

SF 1 Collinearity check among the variables.

SF 2 Pearson's r correlations (above the diagonal) and Spearman rho correlations (below the diagonal) between all variables.

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Data availability statement

All data used in this study are publicly available from international repositories. Adolescent obesity data were obtained from the WHO Global Health Observatory, calorie intake data from the FAO, and GDP and urbanization data from the World Bank. The Henneberg Index data were sourced from previously published datasets [7,23]. No new datasets were generated.

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