PUBLIC HEALTH

Emergence of the obesity epidemic preceding the presumed obesogenic transformation of the society

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The obesity epidemic, evolving in many countries since the 1970s, has been attributed to the widespread contemporary so-called obesogenic transformation of the societies, but what preceded the epidemic? Using quantile regression, we studied the trends by year of birth in the percentile distribution of body mass index (BMI = weight/height^2) of 320,962 Danish school children, born from 1930 to 1976, and of 205,153 Danish young conscripts, born from 1939 to 1959. The overall trend of the percentiles of the BMI distributions were found to be linear across the years of birth. While the percentiles below the 75th were almost stable, those above showed a steadily steeper rise the more extreme the percentile among both school children and young men is. These changes, indicating the emergence of the obesity epidemic, preceded the presumed obesogenic transformation of the society by several decades and imply that other, so far unknown, factors have been involved.

INTRODUCTION

Since the 1970s, the prevalence of obesity has risen steadily in many parts of the world to a level that has justified denoting it an obesity pandemic (*1*–*3*). It has been attributed to a concomitantly ongoing so-called obesogenic transformation of the societies, where food environment and opportunities for reduced physical activity have favored the development of whole-body positive energy balance (*1*–*4*). However, the development of obesity is a slow process that reflects a preceding, on average, miniscule, unobservable daily increment in growth of the adipose compartment. With adiposity being measured by the body mass index {BMI = weight (kg)/[height (m)]^2} and obesity in adults usually being defined as BMI ≥ 30.0 kg/m^2 (*1*) [in children, by analogous criteria (*5*)], the obesity epidemic reflects an increasing proportion of individuals exceeding this threshold. The changes in the BMI distribution may have occurred long before the epidemic took off in the 1970s.

Previous studies of the well-defined unselected population of school children and young adult men in the Copenhagen area, for which unique data are available back to the interwar period (*6*, *7*), have shown that a small rise in prevalence of obesity in children and young adults began already among those born during the Second World War (*8*–*11*).

In the present study, we took advantage of the opportunity of having access to these data to investigate the trends in the BMI distribution in this population among the girls and boys at school ages (7, 10, and 13 years) and young men (conscripts around age 19) through the decades before it became manifest in rapidly rising prevalence of obesity after the 1970s (*11*).

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RESULTS

The prevalence of obesity in the children and the young men was very low but rose similarly and slightly by year of birth (for girls born from 1930 to 1970, at age 7, 0.32 to 0.94%; at age 10, 0.25 to 0.28%; and at age 13, 0.33 to 0.57%; for boys born from 1930 to 1970, at age 7, 0.28 to 0.80%; at age 10, 0.18 to 1.13%; and at age 13, 0.08 to 1.33%; and for the young men born from 1939 to 1959, 0.32 to 1.05%.

We use quantile regression to model distributional shifts in BMI percentiles by year of birth for all groups and to quantify how these shifts differ for different parts of the distribution (*12*–*14*). The observed percentiles 0.1, 0.25, 0.5, 0.75, 0.9, 0.95, and 0.99 are shown in Fig. 1 for the young men and in fig. S1 for the children.

We first estimated both linear and nonlinear quantiles for these percentiles, shown in Fig. 2 for the young men and in fig. S2 for the children. We found that the overall results were well compatible with the linear models that these form the basis for the reported results. Thereafter, we fitted and reported linear quantile regression models for each percentile from the 1st to 99th.

The estimated slopes from the linear quantile regression models for each percentile are plotted for the children in Fig. 3 and for the young men in Fig. 4. Each dot is the estimated slope (with 95% confidence intervals) defined as changes in the particular BMI percentile per year of birth through the period of the study; dots below, at and above 0.00 kg/m^2 per year, indicate declining, stable, and increasing percentiles by advancing years of birth. All groups show the same distinct pattern; slopes were around 0 from the bottom percentiles to approximately the 75th percentile, above which there was a steadily greater slope.

For both girls and boys of age 7 years, the slopes of all percentiles were slightly above 0, whereas the lower percentiles were slightly below 0 for girls and boys of age 10 and 13 and for the young men, indicating a slight tendency for the lower BMI percentiles to decrease over time. The percentiles where the slopes were at exactly 0 were increasing by advancing age; for girls and boys at age 10, it was below the median BMI percentile; for boys at age 13, it was around the median; and for girls at age 13 and the young men, it was above the median. Taking the median BMI as an indicator of the overall position of the BMI distribution, it has thus moved

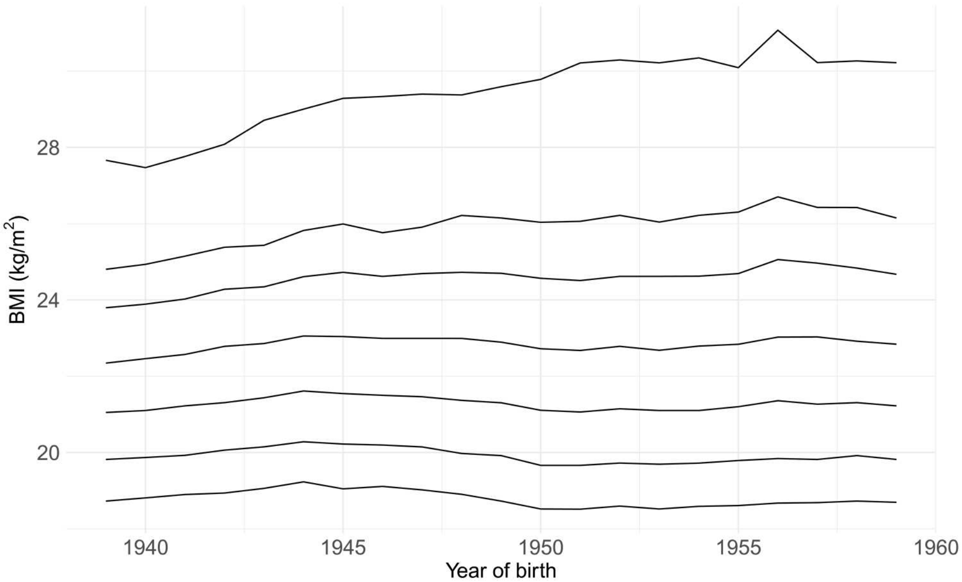

Fig. 1. Changes by year of birth of observed BMI percentiles in young men. Selected BMI percentiles (0.1, 0.25, 0.5, 0.75, 0.9, 0.95, and 0.99) for young men born from 1939 to 1959 and examined at the draft boards in the Copenhagen region at age 18 to 26 years.

slightly upward over time for the girls and boys at age 7 and less so at age 10 and was stable for boys at age 13 but tended move slightly downward for girls at age 13 and the young men. Overall, the results indicate that the central parts of the BMI distributions were mostly stable, while the right-sided skewness was increased.

DISCUSSION

This study revealed that continuous steady increases since the interwar period in the upper percentiles of the BMI distribution preceded the obesity epidemic, with an almost similar pattern in the children and the young men. The linearly, steeply increasing percentiles occurring only in the upper quartile range was greater the more extreme the percentiles are. The overall position of the distributions, as indicated by the medians, showed only minor changes, slight increases for the youngest children and slight decreases for the older children and the young men. Unexpectedly, there was minor downward tendency of the lowest BMI percentiles among the older children and the young men. Thus, the very slight increases in the prevalence of obesity among both the school children and the young men during these years were not attributable to an overall increase in BMI in the population but to a selective increase in the highest percentiles, exceeding the defined thresholds for obesity.

Owing to the mandatory health examinations, the study populations encompassed almost all individuals in the respective background populations (*6*, *7*). While the findings undoubtedly are valid for this particular population, we see no reason why the essence of the results should not be applicable to other populations before the emergence of the obesity epidemic, whether inside or outside Denmark. However, proving this will be difficult, if at all possible, because of lack of suitable data elsewhere covering the period of time before the obesity epidemic.

The findings raise the question whether the process of selectively greater increases in the upper percentiles continued during the subsequent development of the obesity epidemic and whether it also took place among adults. Whereas we have no currently available analyses pertaining to the Danish population, detailed analyses of American data indicate that this has been going on also during the more recent years of much higher prevalence of obesity [supplementary appendix 2 in (*15*)].

On the other hand, our findings are apparently in contrast to the findings from a recent global analysis of the later phase of the obesity epidemic (*4*). This study investigated how much changes in age-standardized mean BMI explains changes in the prevalence of underweight, obesity, and severe obesity in different regions during the period from 1985 to 2016 using the data from 2033 population-based studies of 132.6 million participants with measurements of height and weight at ages 20 to 79 years. They found great heterogeneity across the world, but, in general, the changes in the prevalence of underweight and obesity and, to a lesser extent, severe obesity were largely driven by shifts in the mean values of the distributions of BMI, with smaller contributions from changes in the SD of the distributions. However, the section of their analyses that comes closest to our analyses is the results for the younger populations in the parts of the world that, during this period, still had a very low prevalence of obesity, particularly in Asia [figure 5 in (*4*)]. It seems as if the change in SDs in these populations has been more important than change in the mean values. In addition, the general, relatively greater contribution of SDs to the increases in prevalence of severe obesity seems to reflect the same phenomenon that we have observed.

Our findings indicate that the processes that induced the epidemic likely have been active much earlier and have grown steadily, affecting primarily the individuals with BMI in the upper quartile of the distribution and more so the further up in percentiles. The emergence and continued development of the epidemic in this population can then be described as the increasing percentiles crossing the fixed but arbitrary thresholds for obesity. The later global study indicates that this process continued and included an increasingly broader part of the BMI distribution (*4*). Rather than asking what

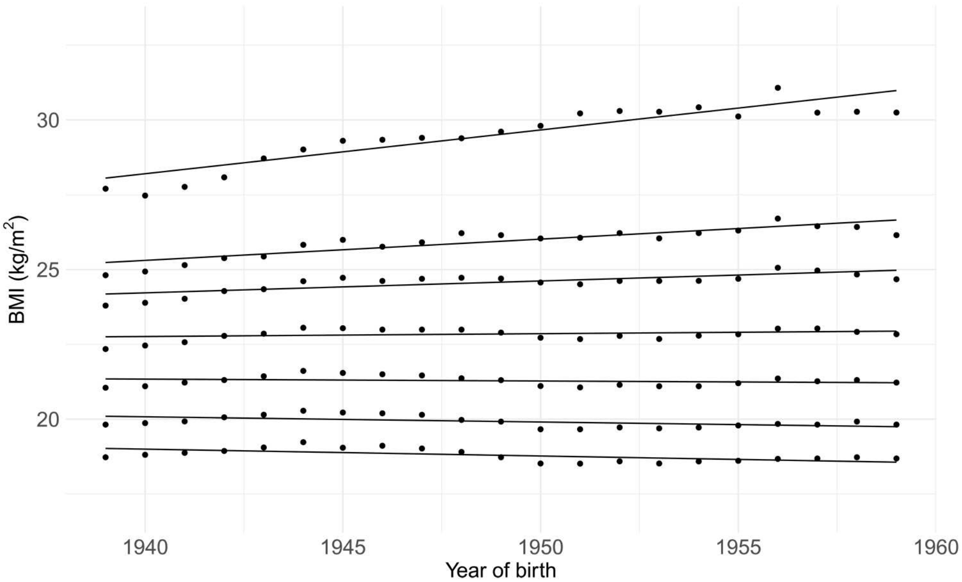

Fig. 2. Changes by year of birth of quantile regression estimates of BMI percentiles in young men. Selected quantile regression–based estimated linear (lines) and nonlinear (dots) BMI percentiles (0.1, 0.25, 0.5, 0.75, 0.9, 0.95, and 0.99) for young men born from 1939 to 1959 and examined at the draft boards in the Copenhagen region at age 18 to 26 years.

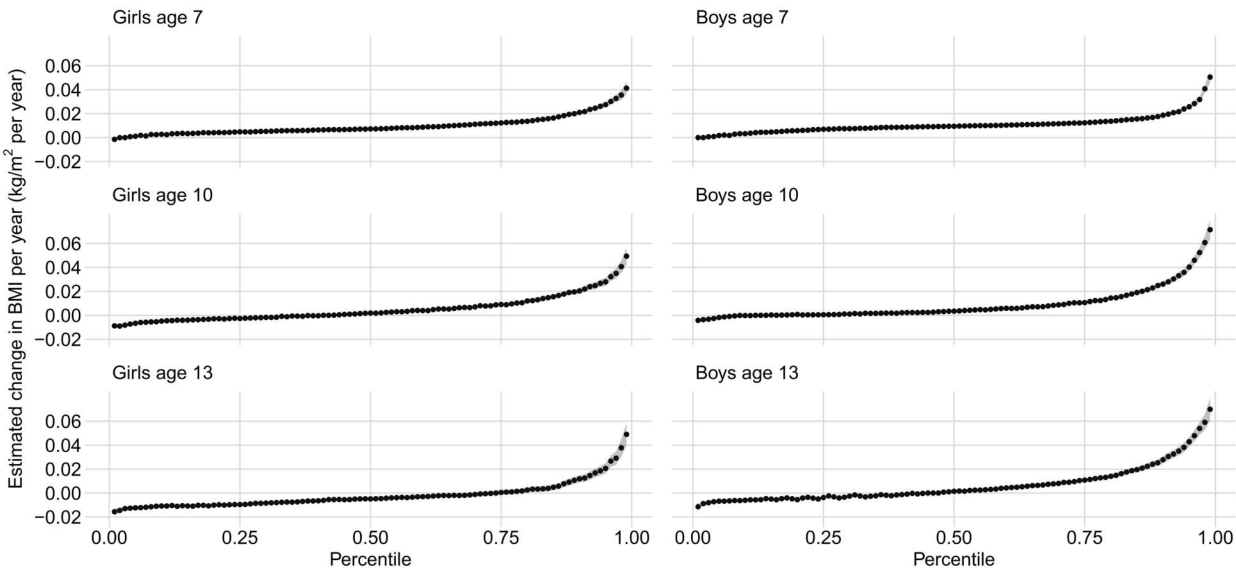

Fig. 3. Quantile regression–based slopes of each linear BMI percentile per year of birth among children. Estimated slopes of linear changes in BMI percentiles of girls and boys at ages 7, 10, and 13 years by year of birth from 1930 to 1976. Slopes (kg/m^2 per year) are on the *y* axis for each percentile from the 1st to the 99th of the BMI distributions (with gray zones around each dot indicating 95 percent confidence intervals, only visible at the highest percentiles).

caused the obesity epidemic, measured by numbers of people passing above the threshold, it may be necessary to ask what caused the much earlier percentile changes to identify the roots of the obesity epidemic.

The results of the present study are based on the assumption of linearity of the changes over time in the percentiles, which may be easier challenged in the extremes of the distribution, where the basis in the data is weaker. Previous analyses of the prevalence of obesity suggested a nonlinear trend with a sharp increase in the year of births from the early 1940s to the early 1950s (*9*, *11*). It is possible that a minor latent subpopulation of individuals with a strong genetic predisposition to obesity (*16*) throughout the study period has constituted a constant small group with more severe obesity. Combining this with the steady linear increases in the upper percentiles may lead to the observed pattern of early nonlinear acceleration of the prevalence of obesity.

Neither the present findings nor those of later upward changes in the central position of the BMI distribution in the global analysis

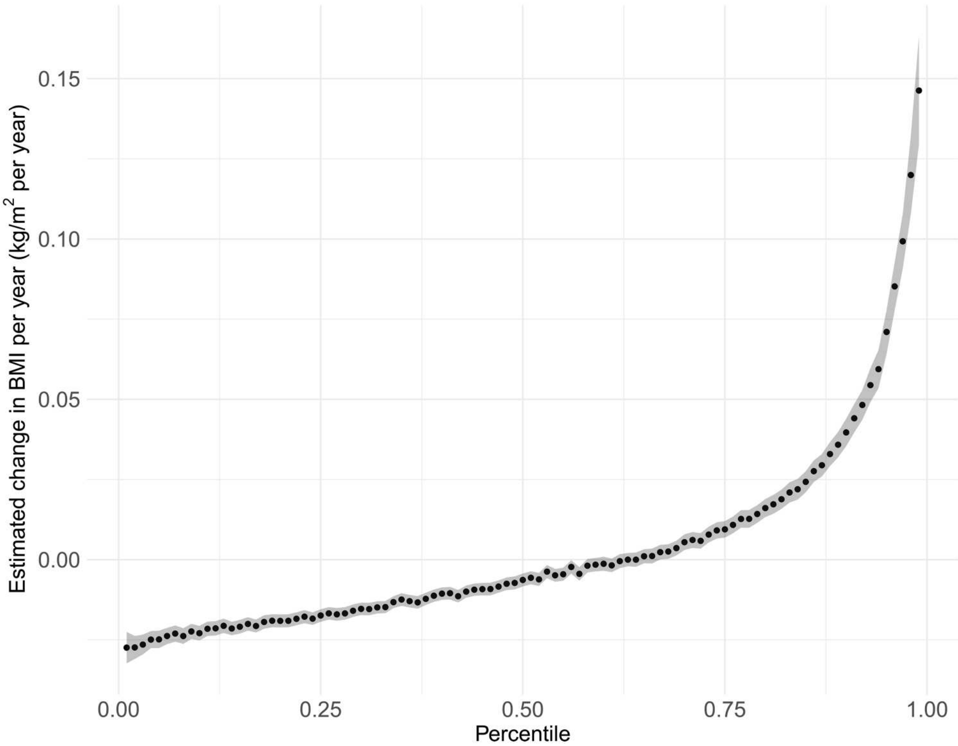

Fig. 4. Quantile regression–based slopes of each linear BMI percentile per year of birth among young men. Estimated slopes of linear changes in BMI percentiles of young men around age 19 by year of birth from 1939 to 1959. Slopes (kg/m^2 per year) are on the *y* axis for each percentile from the 1st to the 99th of the BMI distributions (with gray zones around each dot indicating 95 percent confidence intervals).

(*4*) may be compatible with a previously suggested simple two-component distribution with stable positions but changing sizes (*17*). The findings from genome-wide association studies of BMI, indicating that thousands of genetic variants across the genome are associated with small differences in BMI, also suggest it to be considered as a continuous trait (*16*).

It remains an open question what the exposures have been that have elicited the observed changes preceding the emergence of the obesity epidemic. The currently prevailing concept of the so-called obesogenic environment and corresponding obesogenic behaviors imply a key role to the abundance and consumption of various types of junk food (cheap, palatable, energy-dense, with high sugar and/or fat content, salty, nutrient-poor, highly processed) and sugar-sweetened beverages in combination with availability of devices in daily life during both work and leisure time increasing the opportunities to a sedentary life (cars, TV, computers, automated working procedures, etc.) and reducing needs of all types of physical activity (*2*). While these societal changes may support the continuation of the obesity epidemic, it is obviously not this sort of changes that were introduced when the percentile changes began before the Second World War in Denmark. The post-war economic growth, taking off during the 1950s (*18*), may be considered a facilitator and hence indicator of the obesogenic transformation of the society.

The acceleration of the obesity epidemic has been stronger in rural and provincial areas than in densely populated urban areas, which was seen already in the beginning of the rise of prevalence in obesity in Danish young men during the 1960s (*9*, *10*) and demonstrated globally during the later phases after 1985 (*19*). Whereas abundance of food may be considered a permissive condition for the obesity epidemic to develop, the results indicate that other, so far unidentified, alterations in the environment driving the early changes in BMI distributions should be looked for. The particular pattern of percentile changes, also including the very small declines in percentiles in the lower end of the distribution, calls for a possible heterogeneity across the population in the time trends of the environmental determinants, whether in nutritional or non-nutritional factors. Obviously, this heterogeneity may apply as well between different populations, resulting in the reported differences seen in the global analysis (*4*). In view of this heterogeneity, we consider the current observation as a natural experiment in one population segment, focusing on the time sequence of the emergence of the obesity epidemic and the so-called obesogenic transformation of that society.

The predominance of changes in the upper percentiles may reflect a particular susceptibility due to genetic predisposition, epigenetic priming, or early life social environment (*16*, *20*). Undernutrition during pregnancy has been considered as a driver (*21*), but in Denmark, unlike in some of the neighboring countries, there were no indications of food scarcity during the Second World War (*22*). Because BMI by its construct is sensitive to height, note that there, during the study period, has been a seemingly steady parallel increase across the height percentiles as previously reported for the young men (*10*). This implies that the observed changes in BMI cannot be attributed to changes in height. However, a thorough analysis of the concomitant trends in weight and height may be warranted, especially among children in whom pubertal growth and adiposity appears related (*23*, *24*). Many other putative contributors to the obesity epidemic have been proposed (*21*, *25*, *26*), but none of them has so far been associated with the early changes before the epidemic became manifest.

MATERIALS AND METHODS

Study populations, design, and variables

The data on children used in this study are part of The Copenhagen School Health Records Register, which consists of information on 372,636 children attending school in the central municipality of Copenhagen (*6*). The register is based on children from the years of birth 1930 to 1983, for whom routine measurements of height and weight were carried out yearly from entering elementary school (around age 6 to 7) to leaving it (around age 13 to 14). To ensure a homogenous dataset across all cohorts, we limited the data to children aged 7 to 13. Since the annual measurements stopped in 1984 (and were thereafter including only the entry and exit measurements), we did not include measurements done after 1984 in the analysis, corresponding to years of birth after 1976. The children were measured in underwear only. The dataset used for the analyses consisted of 2,204,125 measurements from 320,962 children of which 160,035 were girls and 160,927 were boys.

The data on young men were extracted from the Danish Conscription Database, which included examinations of 728,160 men undergoing the mandatory conscription between the ages of 18 and 26 years from 1957 to 1984 across the entire country (*7*). To get almost complete cohorts by each year of birth and data from routine weighing, we restricted the study population to those examined in the greater Copenhagen region (including the Copenhagen municipality) and born in 1939 to 1959. The men were measured in underwear only. The study population included 205,153 men with a single measurement of height and weight.

The common variables for all analyses were sex, date of birth, measurement year and month, and BMI. The year of birth of each individual was extracted from the date of birth, and the age of the individual was calculated as the difference between the measurement year and the year of birth, resulting in an approximate age (rounded to the nearest whole number). For the young men, the majority were examined around age 19 (*7*), wherefore the age was disregarded in the further analyses. For the children, the presence of obesity was based on the criteria proposed by the International Obesity Task Force cutoffs for sex- and age-specific obesity (*5*), and for the men, the World Health Organization criterion of 30.0 kg/m^2 was applied (*1*).

Statistical analysis

The prevalence of obesity in the girls and boys and the young men was calculated for each year of birth as the percentage of all girls, boys, and young men whose measurements exceeded the defined thresholds for obesity. Empirical percentiles at 0.1, 0.25, 0.5, 0.75, 0.9, 0.95, and 0.99 were used to depict the overall dataset for girls and boys and for the young men, which show that the trend of the effects by year of birth on BMI was different for girls and boys and at the different ages, justifying separate analyses for these groups.

To quantify the changes of the percentiles in the BMI distribution by year of birth, we estimated the associations of the years of birth across each of the percentiles using quantile regression models for girls and boys within the age groups 7, 10, and 13 years and for the young men (*12*, *13*). The models were fitted using the "$quant_{reg}$" package in R version 3.6.1 (*14*). The method allowed us to fit a series of models capturing the relation to covariates (here year of birth) on development of individual quantiles and hence estimate different relations from the lowest through the highest quantiles.

As shown in Fig. 1 and fig. S1, the distributional changes in BMI over time were not exactly linear. To model this nonlinearity and to argue whether an assumption of linearity might be reasonable, we fitted both nonlinear and linear quantile regression models.

Year of birth was centered at the median cohort when included in the models as a covariate. We chose the degrees of freedom to be the same for all combinations of percentiles in each group to make the results more easily comparable. We chose the number of degrees of freedom by first fitting models for each empirical percentile over year of birth. Each of these models was fitted with a cubic B-spline on year of birth and a varying number of degrees of freedom from 3 to 30. The result was 28 spline models for each combination of percentile within the groups. We then chose the number of degrees of freedom to be the number that minimized the Bayesian information criterion (BIC) most often for all these combinations. Five degrees of freedom minimized the BIC in 9 combinations, while 3 and 16 degrees of freedom minimized the BIC in 7 combinations. On the basis of this, we chose five degrees of freedom for fitting spline models.

The spline estimates for the earliest and latest year of births are, by construction, more uncertain than for other cohorts. We took no measures to account for this increased uncertainty, as the aim of using the nonlinear fits were to show the flexibility of the quantile regression methods and deviations from linearity. The estimates from the nonlinear model vary around the linear estimates. The 99th percentile has the most volatile estimates with large increases and decreases, likely because very few observations drive the extreme percentile, which implies that the fit of the nonlinear quantile regression models might show trends that are not present in the data. Overall, as a clear nonlinear change over time did not seem to be captured by the nonlinear models, we found that linear quantile regression models would produce an acceptable and most parsimonious fit to the data. The added benefit of the linear quantile regression models is that they produce easily interpretable estimates of the change in BMI over time.

Whereas it is possible to test both whether the year of birth trends, defined as the slopes from the linear quantile regression models, were equal to zero (flat slopes) and whether the trends were the same across the different percentiles (equal slopes), we have refrained from formal statistical hypothesis tests due to the very large size of the dataset. As apparent from the 95% confidence intervals of each estimated slope, testing will most likely result in a rejection of the null hypothesis even for minuscule effects and therefore provide no discernible evidence for or against the similarity of the trends.

Supplementary Materials

This PDF file includes:

Figs. S1 and S2

REFERENCES AND NOTES

1. "Obesity: Preventing and management of the global epidemic," *WHO Tech. Rep. Ser.* (no. 894) (1999).
2. B. A. Swinburn, G. Sacks, K. D. Hall, K. McPherson, D. T. Finegood, M. L. Moodie, S. L. Gortmaker, The global obesity pandemic: Shaped by global drivers and local environments. *Lancet* **378**, 804–814 (2011).
3. NCD Risk Factor Collaboration; (NCD-RisC), Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: A pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *Lancet* 390, 2627–2642 (2017).
4. NCD Risk Factor Collaboration (NCD-RisC), Heterogeneous contributions of change in population distribution of body mass index to change in obesity and underweight. *eLife* **10**, e60060 (2021).
5. T. J. Cole, M. C. Bellizzi, K. M. Flegal, W. H. Dietz, Establishing a standard definition for child overweight and obesity worldwide: International survey. *BMJ* **320**, 1240–1243 (2000).
6. J. L. Baker, L. W. Olsen, I. Andersen, S. Pearson, B. Hansen, T. I. A. Sørensen, Cohort profile: The copenhagen school health records register. *Int. J. Epidmiol.* **38**, 656–662 (2009).
7. G. T. Christensen, D. Molbo, L. H. Ängquist, E. L. Mortensen, K. Christensen, T. I. A. Sørensen, M. Osler, Cohort Profile: The Danish Conscription Database(DCD): A cohort of 728,160 men born from 1939 through 1959. *Int. J. Epidemiol.* 44, 432–440 (2015).
8. B. L. Thomsen, C. T. Ekstrøm, T. I. A. Sørensen, Development of the obesity epidemic in Denmark: Cohort, time and age effects among boys born 1930-1975. *Int. J. Obes. Relat. Metab. Disord.* **23**, 693–701 (1999).
9. S. Sonne-Holm, T. I. A. Sørensen, Post-war course of the prevalence of extreme overweight among Danish young men. *J. Chronic Dis.* 30, 351–358 (1977).
10. U. Christensen, S. Sonne-Holm, T. I. A. Sørensen, Constant median body mass index of Danish young men, 1943-1977. *Hum. Biol.* 53, 403–410 (1981).
11. L. W. Olsen, J. L. Baker, C. Holst, T. I. A. Sørensen, Birth cohort effect on the obesity epidemic in Denmark. *Epidemiology* 17, 292–295 (2006).
12. R. Koenker, K. F. Hallock, Quantile regression. *J. Econ. Perspect.* **15**, 143–156 (2001).
13. M. Geraci, Linear quantile mixed models: The lqmm package for Laplace quantile regression. *J. Statist. Software.* **57**, 1–29 (2014).
14. R. Koenker, Quantreg: Quantile Regression. R package version 5.51 (2019); https://CRAN.R-project.org/package=quantreg.
15. Z. J. Ward, M. W. Long, S. C. Resch, C. M. Giles, A. L. Cradock, S. L. Gortmaker, Simulation of growth trajectories of childhood obesity into adulthood. *N. Engl. J. Med.* **377**, 2145–2153 (2017).
16. R. F. J. Loos, G. S. H. Yeo, The genetics of obesity: From discovery to biology. *Nat. Rev. Genet.* **23**, 120–133 (2022).
17. R. A. Price, R. Ness, T. I. A. Sørensen, Changes in commingled body mass index distributions associated with secular trends in overweight among Danish young men. *Am. J. Epidemiol.* 133, 501–510 (1991).
18. J. Bua, L. W. Olsen, T. I. A. Sørensen, Secular trends in childhood obesity in Denmark during 50 years in relation to economic growth. *Obesity (Silver Spring)* **15**, 977–985 (2007).
19. NCD Risk Factor Collaboration (NCD-RisC), Rising rural body-mass index is the main driver of the global obesity epidemic in adults. *Nature* **569**, 260–264 (2019).
20. E. Hemmingsson, P. Nowicka, S. Ulijaszek, T. I. A. Sørensen, The social origins of obesity within and across generations. *Obes. Rev.* **24**, e13514 (2023).
21. R. A. H. Davis, E. P. Plaisance, D. B. Allison, Complementary hypotheses on contributors to the obesity epidemic. *Obesity (Silver Spring)* **26**, 17–21 (2018).
22. E. Angell-Andersen, S. Tretli, R. Bjerknes, T. Forsén, T. I. A. Sørensen, J. G. Eriksson, The association between nutritional conditions during World War II and childhood anthropometric variables in the Nordic countries. *Ann. Hum. Biol.* 31, 342–355 (2004).
23. L. Aksglaede, L. W. Olsen, T. I. A. Sørensen, A. Juul, Forty years trends in timing of pubertal growth spurt in 157,000 Danish school children. *PLOS ONE* **3**, e2728 (2008).
24. L. Aksglaede, A. Juul, L. W. Olsen, T. I. A. Sørensen, Age at puberty and the emerging obesity epidemic. *PLOS ONE* **4**, e8450 (2009).
25. T. I. A. Sørensen, B. Rokholm, T. A. Ajslev, "The History of the Obesity Epidemic in Denmark" in *Proceedings of the British Academy 174, Insecurity, inequality, and obesity in affluent societies* (The British Academy, 2012), pp. 161–178.
26. V. Catalán, I. Avilés-Olmos, A. Rodriquez, S. Becerril, J. A. Fernández-Fomoso, D. Kiortsis, P. Portincasa, J. Gómez-Ambrosi, G. Frühbeck, Time to consider the "exposome hypothesis" in the development of the obesity pandemic. *Nutrients* **14**, e1597 (2022).

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