Obesity Prevention/Management

Modelling obesity outcomes: reducing obesity risk in adulthood may have greater impact than reducing obesity prevalence in childhood

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Summary

A common policy response to the rise in obesity prevalence is to undertake interventions in childhood, but it is an open question whether this is more effective than reducing the risk of becoming obese during adulthood. In this paper, we model the effect on health outcomes of (i) reducing the prevalence of obesity when entering adulthood; (ii) reducing the risk of becoming obese throughout adult life; and (iii) combinations of both approaches. We found that, while all approaches reduce the prevalence of chronic diseases and improve life expectancy, a given percentage reduction in obesity prevalence achieved during childhood had a smaller effect than the same percentage reduction in the risk of becoming obese applied throughout adulthood. A small increase in the probability of becoming obese during adulthood offsets a substantial reduction in prevalence of overweight/obesity achieved during childhood, with the gains from a 50% reduction in child obesity prevalence offset by a 10% increase in the probability of becoming obese in adulthood. We conclude that both policy approaches can improve the health profile throughout the life course of a cohort, but they are not equivalent, and a large reduction in child obesity prevalence may be reversed by a small increase in the risk of becoming overweight or obese in adulthood.

Keywords: Children, cohort, modelling, obesity.

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Introduction

The prevalence of overweight and obesity has increased sharply among many populations since the mid-1980s. Recent estimates suggest that over 50% of US and western European adults are overweight, rising to over 70% in some population groups, and that some 20% of children are overweight, rising to over 30% in some countries (1–3). These figures represent a doubling or even tripling of the prevalence levels in several regions within three decades, and the steep increase is leading to significant consequences for the health of affected individuals, along with a rising burden on health services, social support and economic productivity (4,5).

Monitoring the changes in the prevalence of obesity and modelling the future occurrence of both obesity and its effects on chronic disease and life expectancy is essential for the management of the problem. Computer modelling is widely used to understand and predict behaviour in complex systems (6); nevertheless, the use of such models in the field of obesity prevention is still in a nascent stage (7). In particular, computer modelling may be able to assess the relative effects of different types of interventions and provide guidance to policymakers on the most appropriate, effective or least costly strategies for dealing with obesity or preventing its increase when an array of policies exist (8).

A wide range of policy options for preventing obesity, especially in the earlier years of life, have been proposed by expert groups and multilateral agencies (9,10). Examples for the reduction of obesity among children include a ban on TV advertising of fatty, sugary foods; improved nutrition in school food services; intensive education programmes in nutrition and physical activity; better environments for play and active transport. Measures proposed for reducing obesity among adults and the population in general include some of those given, along with work-place programmes for exercise; work-place meals service improvement; traffic light labelling of food packaging; menu labelling at catering outlets; taxes on soft drinks; and health insurance schemes to cover counselling for obesity prevention (5,11).

The evidence base for the effectiveness and cost-effectiveness of such interventions is poorly developed. Modelling the likely impact of interventions on the development of obesity is needed (11), and the present paper explores this issue using an established risk factor focused modelling tool DYNAMO-HIA (Dynamic Modelling for Health Impact Assessment). This modelling tool has been used to analyse the effects of changes in smoking behaviour (12) and alcohol consumption in the European Union (EU) (13,14). In this application, we focus on the life course of a population cohort, in order to compare the effects of two types of interventions:

1. Interventions which reduce the number of overweight/obese individuals in a cohort that enters adulthood. This models the cumulative effect of interventions to reduce obesity prevalence in childhood;

2. Interventions which alter the probability of a given individual becoming overweight/obese during his or her adult life. This models the sum of the obesogenic influences in an environment acting throughout adulthood.

Materials and method

For this paper, we simulated a birth cohort of males over their life course and quantified the effects of changes in either (i) the prevalence of overweight and obesity at age 18 years, or (ii) the probability of becoming overweight or obese during the life course after the age of 18 years, and (iii) combinations of both (i) and (ii).

Epidemiological evidence

The DYNAMO-HIA project (2007–2010) compiled a publicly available data set with risk factor and disease information for a large set of EU countries.[1] For the present analysis, we have used age- and sex-specific UK data for all-cause mortality and for nine major chronic diseases: ischaemic heart disease (IHD), diabetes, chronic obstructive pulmonary disease (COPD), stroke and cancers of the lung, breast, colon, oral-cavity and oesophagus. Each disease is characterized by age- and sex-specific incidence, prevalence and excess mortality. For stroke and IHD, excess mortality is a combination of two factors: (i) an age- and sex-dependent increase in individual mortality when having those diseases and (ii) an acute but temporarily increased mortality when contracting the disease, to reflect the higher mortality from those diseases at the time of incidence. We also included the increased relative risk of IHD and stroke incidence when having diabetes. Where appropriate, missing data have been back-calculated using the DISMOD II software, utilizing the mathematical relationship between incidence, prevalence and excess mortality rate for chronic diseases within a given population (15). In addition, DISMOD II was used to ensure smooth and internally consistent data. Lastly, we have estimated the age- and sex-specific relative risks connecting body mass index (BMI) to disease incidence and all-cause mortality, using published sources. Relative risk of disease for BMIs of 25 kg m^{-2} and above were estimated for the DYNAMO-HIA project and can be found at http://www.iaso.org/policy/healthimpactobesity/. Further details of the project methodology, processes and full reports are available at http://www.dynamo-hia.eu/.

Dynamic modelling

Within the DYNAMO-HIA consortium, we designed and implemented a publicly available software tool that quantifies the effect of changing risk factor exposure on population health. DYNAMO-HIA was specifically developed to quantify the effects of changes in risk factor prevalence on different health outcomes, including prevalence of specific diseases, overall mortality and summary measures of population health including life expectancy and disease-free life expectancy (DFLE). DYNAMO-HIA uses real-life population data and hence can account for the population-specific incidence, prevalence and mortality profile of relevant diseases.

The detailed methodology is described elsewhere (16,17). DYNAMO-HIA synthesizes data according to the causal epidemiological pathway, linking risk-factor

[1]For details of the DYNAMO-HIA project, please refer to the reports available online at http://www.dynamo-hia.eu.

exposure through relative risks of incidence of associated diseases and death, to prevalence of diseases, mortality and summary measures of population health, taking into account competing risks. The model uses relative risks by risk factor class, i.e. incidence in exposed risk factor classes are a multiple of the incidence in the non-exposed. A change in risk factor exposure thus changes disease incidence and in turn disease prevalence and mortality later in life. The effect of the risk factor change on mortality through diseases that are not explicitly included in the model, i.e. other-cause mortality, is taken into account through the relative risk for total, i.e. all-cause, mortality.

Reference scenario

For the specification of a scenario, DYNAMO-HIA requires information on the starting BMI prevalence and how the BMI prevalence will change over time specified by a set of transition probabilities. For every new year, DYNAMO-HIA applies to the simulated cohort a probability determining the proportion of individuals that will stay in their current BMI category or will move to another BMI category, e.g. how many will keep their normal weight or will become overweight or obese in the next year. DYNAMO-HIA aims to compare different scenarios, and this requires a reference scenario which specifies how we expect the birth cohort to develop over time, given the obesity policy measures that are already in place. This is sometimes also called the 'business-as-usual' scenario. In this paper, we use prevalence projections for the male population of England as provided by National Heart Forum to model the development of the birth cohort (18). The DYNAMO-HIA software then estimates the net-transition probabilities necessary to replicate the prevalence at each age using the approach recommended by van de Kassteele *et al.* (19).

The baseline disease incidence and mortality risk for healthy individuals with normal weight is that of the currently observed conditions, which in this application refer to the current conditions prevailing in the UK. Hence, the DYNAMO-HIA projection does not take into account any past or future trends or changes in disease incidence or mortality unconnected with obesity. Moreover, the changes in health status of the reference cohort over the life course in the DYNAMO-HIA projection reported in this paper are completely driven by the change in BMI prevalence using the age- and sex-specific disease and mortality risk observed under current conditions.

Intervention scenarios

In the intervention scenarios, we explore the effects of two kinds of stylized types of intervention to reduce the life course prevalence of overweight/obesity and compare the resulting health outcomes with the reference scenario ('business-as-usual').

The first set of intervention scenarios represents interventions designed to reduce the relative prevalence of overweight/obesity at the end of childhood, which is modelled in this paper as the cumulative effect on prevalence of all childhood interventions up to age 18 years, and expressed as a reduction by a given percentage of the proportion of the population at age 18 years classified as overweight or obese. In the modelled scenarios, no account is taken of previous history of overweight or obesity, and all individuals in a BMI category at age 18 years are treated as being equally likely to remain in that category or to change their category during subsequent years.

The second set of intervention scenarios represents interventions that would result in a lifelong change of behaviour after entering adulthood (starting from age 18 years) by changing at every age the probability of becoming overweight when being normal weight, and becoming obese when being overweight, as compared to the reference scenario, by a given percentage. For this, we use the net-transition probabilities calculated for the reference scenario and change these by a given percentage for every age above age 18 years. For example, if the reference scenario shows that from age 50 years to age 51 years 100 normal-weight individuals become overweight, then a reduction in the transition probability by 10% means that only 90 normal-weight individuals become overweight.

First, we applied and compared both types of interventions separately, i.e. (i) we reduced the prevalence of overweight/obesity at age 18 years or (ii) we reduced the transition probability of becoming overweight/obese at every age after 18 years. Then, we applied combinations of both interventions, i.e. (iii) simultaneously reducing the prevalence of overweight/obesity at age 18 years and reducing the transition probability, as most real-life scenarios contain elements of both types of interventions. Finally, we compared the relative effects of both kinds of interventions by offsetting child prevalence against the transition probabilities, i.e. (iv) we ran scenarios where we reduced the youth prevalence at age 18 years but increased the transition probabilities, both by varying levels. This was designed to indicate to what extent the gains made in childhood are reduced by an increase in the adult risk of becoming overweight or obese.

Results

Body mass index outcomes

Figure 1 shows the development of the share of normal weight (left hand column) and obese (right hand column) in the population, according to our model. The proportion of

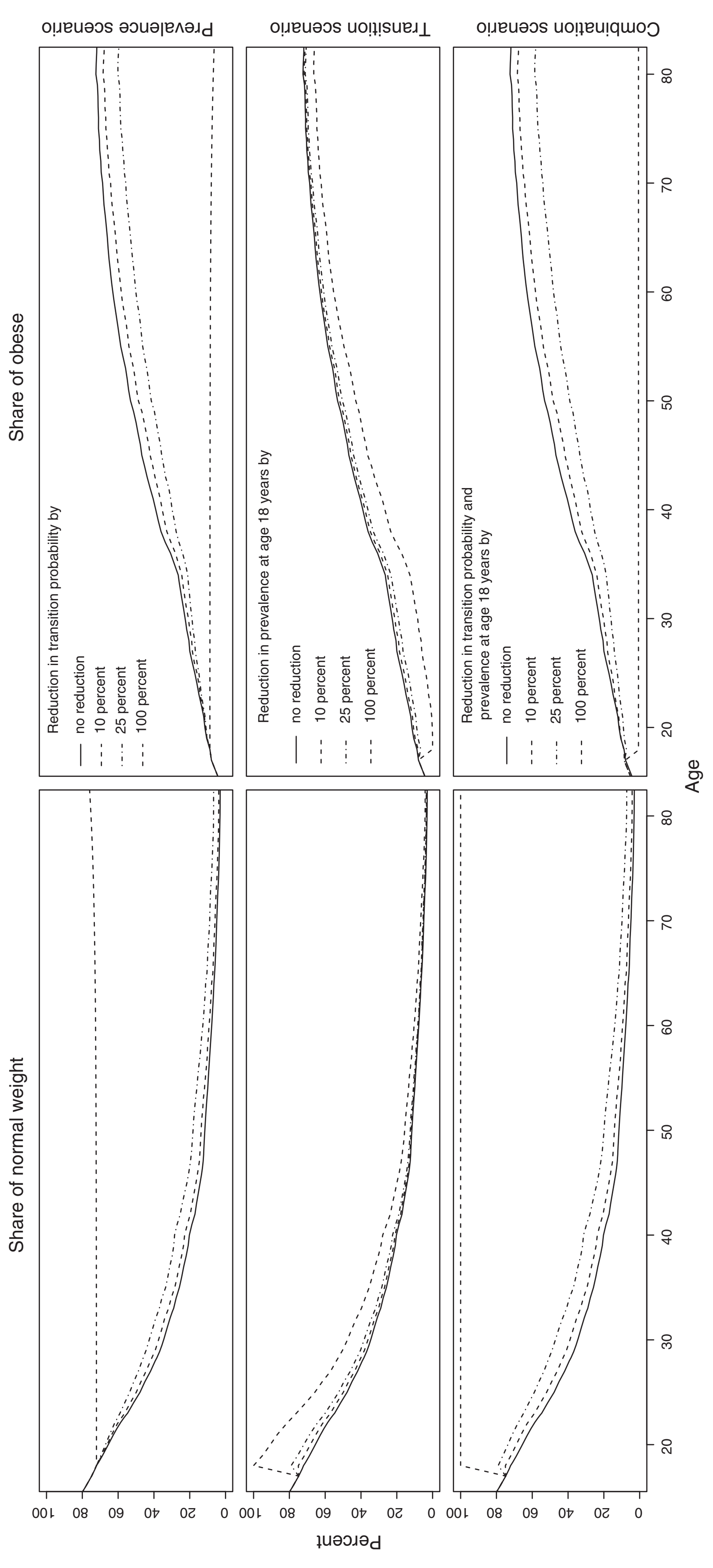

Figure 1 Proportion of population normal weight and obese for different scenarios.

adults who are overweight but not obese is not shown, for ease of presentation. The solid black line is always the reference scenario, i.e. the prevalence of normal weight and obesity in the cohort in the business-as-usual scenario.

The top row shows scenarios where only the transition probabilities of becoming overweight/obese after age 18 years are being changed. The most extreme reduction in transition probability, i.e. by 100%, leads to a 'frozen' cohort where every member keeps the BMI he has at age 18 years throughout life. This can be seen by the horizontal extensions from the prevalence at age 18 years. The other two, more realistic reductions in the transition probability of becoming overweight/obese through the life course, i.e. a reduction by 10 and 25%, visibly lead to an increase in the share of normal weight as compared to the reference scenario and conversely, a reduction in the share of obese.

The middle row shows the effect of reducing prevalence at age 18 years. A reduction in prevalence by 100% leads to a cohort that has no overweight/obese individuals at age 18 years, while the subsequent lifelong age-specific risks of becoming overweight or obese remain unchanged. All three magnitudes, i.e. a reduction by 10, 25 and 100% in prevalence at age 18 years, show a life-long reduction of obesity compared to the reference scenario.

The bottom row shows combinations of both approaches. The most extreme scenario, a reduction of prevalence of overweight/obesity at age 18 years by 100% and a reduction of the probability of becoming overweight/obese throughout adulthood by 100%, leads to cohort where everybody at age 18 years is normal weight and remains normal weight throughout the life course.

Comparing the two types of interventions, a percentage reduction in the probability of becoming overweight/obese during adulthood has a larger effect on the lifelong reduction of obesity than an equally sized percentage reduction of prevalence at age 18 years. This is best demonstrated by the scenario in which the transition probabilities (to overweight/obesity) are reduced by 100%, freezing everybody in their respective weight category at each 18 years. However, both a reduction in the probability of becoming overweight/obese during adulthood and a reduction of prevalence at age 18 years have a favourable effect on the share of overweight and obese. Even a small reduction in youth prevalence at age 18 years of 10% leads to a measurable lifelong reduction in the share of people who become obese, compared to the reference scenario.

Health outcomes

Table 1 shows cohort life expectancy and DFLE, i.e. life years without a chronic disease, for several scenarios. In the reference scenario (without any interventions to lower the adult transition probabilities or childhood prevalence), the life expectancy is 75.7 years (DFLE: 66.9 years). In the most optimistic scenario, i.e. when both the risk of adult transition to overweight/obesity is lowered by 100% and the prevalence of overweight/obesity at age 18 years is set to zero so that everybody in the cohort is normal weight and stays normal weight throughout adulthood, the resulting life expectancy rises to 78.7 years (DFLE: 71.9 years). Table 1 also shows that reductions in the lifelong transition probabilities to overweight/obesity yield larger improvements in disease reduction, total life expectancy, and disease-free life expectancy compared to the corresponding percentage reduction in overweight/obesity prevalence at age 18 years. A reduction of the transition probabilities by 100%, i.e. the maximum intervention, would yield a cohort life expectancy of 78.0 years (DFLE: 70.8 years) compared to a life expectancy of 76.0 years (DFLE: 67.4) for a 100% reduction of overweight/obesity prevalence at age 18 years.

Table 2 shows the disease prevalence at ages 40, 60 and 80 years for selected diseases. Comparing the most extreme combination of both types of interventions, i.e. all are normal weight and stay normal weight, with the reference scenario shows that at age 80, the reference scenario has a fourfold higher prevalence of diabetes. Similarly, at age 60, the reference scenario has a near-double IHD prevalence compared to the most extreme intervention.

Table 1 Cohort life expectancy (LE) and disease-free life expectancy (DFLE) in years at birth

		Reduction in the probability of becoming overweight or obese throughout the life course after age 18 years compared to the reference cohort							
		0%		10%		25%		100%	
		LE	DFLE	LE	DFLE	LE	DFLE	LE	DFLE
Reduction in prevalence of overweight and obesity at age 18 years compared to the reference cohort	0%	75.69	66.88	75.81	67.11	76.04	67.52	78.02	70.78
	10%	75.72	66.94	75.84	67.16	76.08	67.59	78.08	70.88
	25%	75.75	67.00	75.88	67.24	76.13	67.69	78.19	71.06
	100%	75.95	67.37	76.11	67.66	76.4	68.18	78.71	71.94

Table 2 Prevalence (%) of selected diseases at ages 40 years, 60 years and 80 years

Disease		Colorectal cancer			Diabetes			Stroke			Ischaemic heart disease		
Age		40 years	60 years	80 years	40 years	60 years	80 years	40 years	60 years	80 years	40 years	60 years	80 years
Percent reduction in													
adult transition probabilities	prevalence at age 18 years												
0	0	0.03	0.48	3.50	0.61	8.01	16.84	0.11	2.49	13.66	0.23	8.05	27.08
0	10	0.03	0.48	3.49	0.59	7.93	16.71	0.11	2.48	13.63	0.22	8.01	26.98
0	25	0.03	0.48	3.49	0.58	7.81	16.55	0.10	2.47	13.58	0.22	7.95	26.85
0	100	0.02	0.47	3.46	0.49	7.22	15.74	0.10	2.41	13.36	0.21	7.64	26.20
10	0	0.03	0.48	3.49	0.59	7.93	16.71	0.11	2.48	13.63	0.22	8.01	26.98
25	0	0.03	0.48	3.49	0.58	7.81	16.55	0.10	2.47	13.58	0.22	7.95	26.85
100	0	0.02	0.47	3.46	0.49	7.22	15.74	0.10	2.41	13.36	0.21	7.64	26.20
100	100	0.02	0.38	2.71	0.21	2.22	4.66	0.08	1.79	9.70	0.15	4.69	16.52

Table 3 Expected years with at least one chronic disease (ischaemic heart disease, diabetes, COPD, stroke, lung-, breast-, colon-, oral-cavity- and/or oesophagal-cancer)

		Increase in the probability of becoming overweight or obese throughout the life course after age 18 years compared to the reference cohort				
		10%	20%	30%	40%	50%
Reduction in prevalence of overweight and obesity at age 18 years compared to the reference cohort	0%	8.90	8.98	9.06	9.15	9.20
	10%	8.88	8.96	9.05	9.13	9.19
	25%	8.84	8.93	9.02	9.10	9.17
	50%	***8.80***	8.89	8.98	9.07	9.14
	100%	***8.69***	***8.78***	8.89	8.98	9.05

Values in bold italics are as good or as better than the reference scenario of 8.81 years.

Deteriorating transition probabilities during adulthood

Changes in the environment which lead to increases in the risk of obesity – for example, economic stresses on healthy food supplies or increases in sedentary behaviour – may offset the gains made through childhood interventions. Tables 3 and 4 show, respectively, the expected number of years with chronic disease and the total life expectancy for different combinations of percentage reductions in overweight/obesity prevalence at age 18 years set against specified *increases* in life-long transition probabilities to overweight/obesity. In virtually all cases, a relatively small increase in the transition probability to overweight/obesity in adulthood completely offsets the gains made by large reductions in childhood overweigh/obesity prevalence. The tables show that gains following a 50 or 100% reduction in prevalence at age 18 years are fully offset by an increase in adult transition probabilities of 10 or 20%, respectively.

Discussion

According to our model, both a reduction in the prevalence of overweight and obesity at age 18 years and a reduction in the lifelong transition probability of becoming overweight and obese among adults have long-term effects on the health status of the cohort, even for seemingly modest reductions of 10% in each case. This finding is in line with Wang *et al.* (4) who demonstrated long-term health benefits from a modest BMI reduction at baseline. However, our findings clearly demonstrate that a given percentage reduction in the transition probabilities during adulthood has a consistently stronger effect than an equally sized percentage reduction in prevalence at age 18 years.

Although we use similar data and types of hypothetical interventions as the Foresight Obesity project (FOP) (20), our results are not directly comparable. The FOP models the two types of interventions separately for the whole English population for approximately 50 years. This makes

Table 4 Cohort life expectancy (years)

		Increase in the probability of becoming overweight or obese throughout the life course after age 18 years compared to the reference cohort				
		10%	20%	30%	40%	50%
Reduction in prevalence of overweight and obesity at age 18 years compared to the reference cohort	0%	75.57	75.48	75.37	75.28	75.22
	10%	75.60	75.50	75.39	75.30	75.23
	25%	75.64	75.53	75.42	75.33	75.26
	50%	***75.69***	75.58	75.47	75.37	75.30
	100%	***75.82***	***75.70***	75.58	75.47	75.39

Values in bold italics are as good or as better than the reference scenario of 75.69 years.

it difficult to assess to what extent their findings are influenced by the underlying age composition of the population. As we model the lifelong experience of a single cohort using several combinations of the two types of interventions, our findings should be more generalizable.

It should be noted that although both types of interventions result in a lower share of the population with obesity and a higher share with normal weight, the share of overweight non-obese is more complex. It increases for both strategies as compared to the reference scenario, although the pattern varies by age, with the prevalence of overweight lower as compared to the reference scenario below the age of 40 years, while above this age the share of overweight is higher than in the reference scenario (data not shown). This pattern is a direct consequence of higher absolute numbers of people moving from normal weight to overweight compared to those moving from overweight to obesity leading to a gradual 'pooling' of individuals in the middle category.

Our analysis indicates that interventions aiming at long-term behavioural change over an adult lifetime may be superior to child-focused interventions. Furthermore, the results indicate that substantial reductions in child obesity can be offset comparatively swiftly if long-term adult behaviour is unchanged or deteriorates. However, the lesser effects of childhood intervention should not be interpreted as a disqualification of the value of childhood interventions as such, but should raise questions about childhood interventions that have no lasting effect over the life course. We are not arguing for a disinvestment in child obesity programmes but rather an investment in programmes that have long-term, sustainable effects, and help the individual resist the obesogenic environments they may face throughout life. Equally, there is now a strong case to be made for interventions that change the exposure to obesogenic environments for people of all ages.

Limitations

All models are simplified versions of reality and the DYNAMO-HIA tool is no exception. But a modelling tool provides a means of integrating the knowledge base from a variety of diverse fields such as statistics, epidemiology, biology, economics, and sociology (7,21).

We have analysed hypothetical policy interventions in which the effects are either assumed to stay constant (in the case of the reduction of lifelong probability) or only affecting prevalence when entering adulthood and not in any future behaviour (in the case of reducing the share of overweight and obese men at age 18 years). In real life, effects of policies are more difficult to disentangle, and successful policies targeting adolescents may reduce both the prevalence of obesity at age 18 years *and* the subsequent risk of becoming overweight and obesity during adulthood. For instance, childhood dietary interventions may change future dietary patterns during adulthood and even old age. However, reliable evidence for comparable real-life policies is lacking.

Equally, our assumption about the comparability of a percentage change in each of the interventions (e.g. a 10% change in prevalence at age 18 years compared to a 10% change in transition probabilities in adulthood) was made in the absence of any better approach. An intervention which reduces child obesity prevalence by 20% may in reality be more achievable or less costly than an intervention which reduces adult transition probabilities by only 1 or 2%, and therefore a cost-effectiveness approach could greatly assist in improving the modelling. Unfortunately, many childhood interventions fail to report on their costs or long-term sustained effects, despite the value of such information for policymaking.

DYNAMO-HIA compares the effects of intervention and policy approaches, i.e. it quantifies a reference scenario and one or more intervention scenarios. The goal is not to project future population health as such. For projecting future population health, accurate information on the future incidence, prevalence and excess mortality data of the diseases included in the model are needed, which are inherently uncertain. For the DYNAMO-HIA database, it was decided to include trend-free data partly estimated using DISMOD II software. Such trend-free data are used as a neutral option, because of the lack of reliable information on trends. In view of the intended use of DYNAMO-HIA – that is, comparing scenarios – this choice is not very significant as the same disease data are used both in the intervention and reference scenario(s). Therefore, we do not expect that this approach has an important effect on the findings of our study.

The prevalence of the reference scenario is based on a forecast for England, predicting a significant increase in levels of overweight/obesity over the next 40 years. This forecast might be overestimating or underestimating the future levels of overweight and obesity of the cohort. Since the Foresight analysis, there is some evidence of child obesity levels stabilizing or even falling in some age groups. This would affect the 'business as usual' scenario in absolute terms, but in the present analysis, we compare relative changes in our two-key intervention approaches, and therefore the ordering of the results will be largely unaffected by changes in the baseline scenario, although the absolute values of life years and prevalence levels would be affected.

It should be noted that in the present paper, we have analysed data for a male English population. For completeness, the modelling should also be undertaken for a female population, and perhaps for populations in other world regions. The risk profiles for obesity found in Europe and North America, and the patterns of age-related incidence levels found in male and female populations in these regions, are sufficiently similar that the authors would anticipate qualitatively similar outcomes, and that the policy implications – especially the relative importance of life-long exposure to obesogenic environments – would dominate the health outcomes in all cases.

Lastly, the model was designed to compare a basic scenario with potential changes. It was not designed to provide policymakers with specific predictions of future disease or life expectancy levels. However, the model does indicate the type of changes that policymakers should be considering if they wish to maximize the benefits of investments in interventions.

Conclusion

According to our model, relative reductions in the lifelong adult transition probabilities from normal weight to overweight and to obesity are consistently more effective than equally sized relative reductions in youth prevalence of overweight and obesity, both in terms of reducing the prevalence of overweight and obesity in the life course of a population cohort, and in terms of reducing the cohort's consequent experience of disease, and extending their life expectancy and disease-free life expectancy.

Childhood interventions to reduce the prevalence of overweight and obesity when entering adulthood can have a significant effect on adult health status, but it is clear that the greatest effect will be found if these childhood interventions are combined with life-long improvements in the adult transition probability to overweight and obesity. Childhood interventions should be evaluated in terms of how well they lead to sustained behaviour change which can reduce the risk of becoming overweight or obese in later adulthood.

In contrast, a relatively small increase in the risk of becoming overweight or obese in adulthood can fully offset the total elimination of overweight and obesity at age 18 years. Interventions that achieve a small but long-lasting effect in the likelihood of developing obesity in adulthood are likely to have a much greater potential to avert related diseases than short-term reductions in obesity prevalence, even if these short-term reductions are substantial.

Conflict of interest statement

No conflict of interest was declared.

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