General discussion

The aim of this thesis was to explore the importance of cultural capital in the understanding of socioeconomic inequalities in health-related behaviors. In this final chapter the main findings will be summarized and the research questions will be answered, before reflecting on the limitations and methodological considerations of these studies. Subsequently, the interpretation of the findings will be discussed. This chapters ends with implications for future research, and implications for policies and interventions that aim to tackle socioeconomic inequalities in health-related behaviors, and ultimately socioeconomic inequalities in health.

MAIN FINDINGS AND ANSWER TO RESEARCH QUESTIONS

1) To what extent do material risk factors and health-related behaviors contribute to socioeconomic inequalities in mortality?

Chapter 2 examined the contribution of material and behavioral risk factors, measured three times during adulthood, to educational and occupational inequalities in mortality. Moreover, analyses with multiple measurements of the risk factors were compared to analyses in which only the baseline measurements of the material and behavioral risk factors were used. The findings showed that the contribution of behavioral risk factors to socioeconomic inequalities in mortality was greater in time-varying models compared to time-fixed models, whereas the contribution of material risk factors was smaller in time-varying models. Behavioral and material risk factors combined explained about 75% of the educational gradient in mortality, for both men and women, in both time-fixed and time-varying models. Behavioral and material risk factors combined fully explained the occupational gradient in mortality for men in time-varying models and slightly less in time-fixed models, whereas they explained only half of the occupational gradient for women in both time-fixed and time-varying models.

We observed that the socioeconomic gradient in both material and behavioral risk factors increased over time, whereas associations between behavioral factors and mortality were larger in time-varying models and associations between material factors and mortality were smaller in time-varying models. These results indicate that differences in the relative importance of material and behavioral risk factors for socioeconomic inequalities in mortality between time-fixed and time-varying models were mostly driven by differences in the estimated associations between risk factors and mortality, and not by differences in the socioeconomic gradients over time (as for both material and behavioral risk factors the gradient increased).

The underlying mechanisms that could contribute to the differences between time-fixed and time-varying models were further explored in chapter 3. This chapter

systematically listed the underlying mechanisms that may cause differences between time-varying and time-fixed models in studies investigating explanations for socioeconomic inequalities in mortality. We argued that differences between time-varying and time-fixed models could result from changing socioeconomic gradients in risk factors (e.g. due to age or period effects) or due to changing associations between risk factors and mortality. A clear advantage of time-varying models is that they reduce misclassification by capturing actual changes in risk factors over time. For example, if a substantial proportion of participants quit smoking during follow-up, they will still contribute to the 'exposed' group in time-fixed models, whereas they will be re-classified as 'non-exposed' in time-varying models (as long as one of the repeated measures was taken after smoking cessation). Incorporating changes in risk factors becomes even more important if the association between risk factors and mortality also changes over time (e.g. risk ratios may change due to better treatment or prevention). However, time-varying models may also introduce bias if they are not correctly specified. First, the time lapse that is needed for changes in risk factors to affect mortality should be accurately modelled and could differ between risk factors (and between causes of death). For example, if the mortality risk of participants who quit smoking during follow-up does not decrease to a mortality risk similar to other 'non-exposed' participants, the risk ratio of smoking will not be accurately estimated. Note however, that this risk ratio may still be more accurate than the risk ratio in time-fixed models. Second, if changes in risk factors are associated with changes in confounders, and these time-varying confounders are not accurately modelled, time-varying models will introduce bias.

2) Is early-life environment related to health-related outcomes in later life?

Chapter 4 used a large US-based cohort study to examine the effects of childhood rural residence on health and health-related behaviors in adulthood, accounting for adult rural residence. Health effects of early-life environment extend far beyond the effects of growing up in a rural or non-rural community, which prohibits extrapolation outside the context of the current study. However, since the health effects of early-life environment are key in the theoretical background of this thesis, it is worthwhile to include a chapter that explores this relationship. Moreover, the extensive data available in REGARDS made it possible to thoroughly investigate the effect of childhood rural residence on health-related outcomes in adulthood, adopting newly developed mediation methods that are highly relevant for research on socioeconomic inequalities in health. Nevertheless, the findings from this study should be interpreted in the context of rural versus non-rural residence in the US, and seen as one case-study within the overall aim of investigating health effects of early-life environments.

The results of this study showed mixed effects of living in a rural community in childhood on health-related outcomes in adulthood, and also mixed effects according to race. Childhood rurality was associated with indicators of better health in some dimensions, such as more physical activity, but also with indicators of worse health in others, such as adoption of a Southern diet (characterized by fried food, processed meats, and sugar-sweetened beverages). Moreover, childhood rurality was associated with obesity, the adoption of a Southern diet and fair/poor self-rated health among white participants (but not among black participants) who remained in rural communities in adulthood, whereas no such association was observed among white participants who migrated to non-rural communities. This effect was largest for the general measure of self-rated health: the effect of childhood rurality was beneficial if the individual migrated to a non-rural community in adulthood, but disadvantageous if the individual remained in a rural community in adulthood.

3) To what extent do inequalities in cultural capital contribute to socioeconomic inequalities in health-related behaviors?

Chapter 5 explored to what extent cultural capital contributed to educational inequalities in food consumption using a recently developed questionnaire that specifically measures food choice related cultural capital (more details on the development of the questionnaire and proof of concept can be found in Kamphuis et al. [1]). This study found that cultural capital was related to healthy food consumption, and that the associations between cultural capital and the consumption of healthy food products were stronger than the associations between economic and social capital and the food outcomes. In addition, the study showed that cultural capital contributed greatly to educational inequalities in healthy food consumption, over and above economic and social capital.

Chapters 6 and 7 explored to what extent social distinction contributed to socioeconomic inequalities in health-related behaviors. In both studies, cultural participation was used as an indicator of social distinction, because we deemed it to be the best available indicator of social distinction based on a recent review on measures of cultural capital [1]. Chapter 6 explored the association between cultural participation and the consumption of 'superfoods'. The study showed that there was a strong dose-response relationship between cultural participation and the consumption of spelt products, quinoa, and goji berries, chia seeds or wheatgrass, even after controlling for education and income. Furthermore, the study found that the initial associations between education and income and the consumption of superfoods were greatly attenuated after controlling for cultural participation. In chapter 7 we found similar results for more 'common' health behaviors, i.e. fruit and vegetable consumption, leisure time walking and cycling and sports participation. This study also observed strong associations be-

tween cultural participation and the health-related behaviors, even after controlling for education and income. Similarly, the associations between education and income and the health-related behaviors greatly attenuated after controlling for cultural participation. In combination, chapters 6 and 7 showed that social distinction may be an important determinant of health-related behaviors, and may contribute to socioeconomic inequalities in physical activity and dietary patterns. Interestingly, by showing similar patterns for both contemporary 'distinctive' foods (i.e. 'superfoods'), and more general health-related behaviors (i.e. fruit and vegetable consumption, leisure time walking and cycling and sports participation), this diptych was able to provide stronger evidence for the hypothesis that social distinction is an important determinant of health-related behaviors.

4) Via which pathways is high cultural capital related to health-related behaviors?

Chapter 8 explored potential pathways that could explain how high cultural capital is related to lower BMI. We theoretically identified four dispositions, theorized to act as cultural signifiers in higher social strata, that could explain how cultural capital is related to a lower BMI. To measure these dispositions, we developed new survey items and included them in our interview survey. Confirmatory factor analysis showed that the survey items measured six different constructs: 'asceticism', 'general refinement', 'food refinement', 'non-sweet taste', 'diversity', and 'reflexivity'. However, only asceticism, general refinement, food refinement and reflexivity could be reliably measured. These four dispositions were all associated with (at least one form of) cultural capital, but only asceticism, general refinement and reflexivity were also associated with lower BMI. Asceticism, general refinement and reflexivity together explained 55% of the association between embodied/objectified cultural capital and BMI, and 36% of the association between institutionalized cultural capital (i.e. education) and BMI. Mediation effects were especially pronounced for reflexivity, which explained 40% of the association between embodied/objectified cultural capital and BMI, and 32% of the association between institutionalized cultural capital and BMI. These findings indicate that participants with high levels of cultural capital were more likely to have dispositions that encourage asceticism, reflexivity and a preference for refinement, which contributed to lower BMI.

METHODOLOGICAL CONSIDERATIONS

There are several methodological considerations to keep in mind when interpreting the results from this thesis. Most have already been discussed in the relevant chapters, but

some overarching limitations will be addressed in the following paragraph. These are related to the study design and the limitations of the statistical analyses.

The GLOBE-study

Most chapters used data from the GLOBE-study. The GLOBE-study is a longitudinal cohort study situated in the city of Eindhoven in The Netherlands [2, 3]. For this thesis, two new data collections were executed. First, in 2014, data were collected by a postal survey (N=4,851, response=45.5%), and second, in 2016, a subsample of the survey respondents was interviewed (N=597, response=50%). In addition, data from earlies waves of the GLOBE-study were used in chapters 2 and 5.

The use of observational data from the GLOBE-study has some important limitations. First, all measures used were self-reported, which may result in measurement error and misclassification. Measurement error and misclassification pose a threat to the validity of the results in this thesis, but because they are also well-known and extensively described in the literature, this paragraph will elaborate more on other limitations. Second, the observational nature of the GLOBE-study is by definition not exogenous, and may suffer from unobserved confounding (e.g. by biological, psychological or cognitive factors). Third, in most chapters, cross-sectional data were used because measures on cultural capital were only measured once. Cross-sectional data are not able to infer causality and limit investigating the temporal ordering of the measures used. Moreover, cross-sectional data exclude the possibility of studying changes over time, and increase the potential for unobserved confounding. Such limitations however, are common for quantitative research investigating socioeconomic inequalities in health-related behaviors. Moreover, our studies were among the first to examine the relation of cultural capital to socioeconomic inequalities in health-related behaviors, which required data that are not routinely collected by other (longitudinal) studies.

Chapters 6, 7 and 8 used data from the 2014 and 2016 data collection of the GLOBE study. Whereas participation rates in the first waves of GLOBE in the 1990's reached levels over 70%, the 2014 data collection of GLOBE had a participation rate of only 45.5%. Participation rates of population studies have steadily decreased in the last decade, which is of increasing concern to the validity and generalizability of these studies. In order to assess the magnitude of potential selection bias within our cross-sectional sample, prevalence rates of several health-related behaviors (e.g. smoking, sports participation, obesity) were compared to those of the target population (i.e. the city of Eindhoven). These results showed similar prevalence rates within our study compared to prevalence rates from 'official' municipality statistics. However, the comparison data stem from the health monitor of the Dutch municipality health services, which of course suffers from similar caveats, i.e. data are also acquired by observational studies that may be biased. For example, the most deprived groups (i.e. the unemployed, substance users, ethnic

minority groups) are likely underrepresented in both studies. However, to the extent that this selection bias would have affected the studies in this thesis, observed results are likely underestimated, not overestimated. Nevertheless, generalization to these groups is limited.

Whereas selection bias is often acknowledged as a problem for representativeness and prevalence estimates, it is less often considered as a problem for effect estimates. However, selection can also induce 'collider bias' (sometimes called 'endogenous selection bias' [4]), which can lead to biased effect estimates [5-7]. This bias is caused by conditioning (e.g., controlling, stratifying, selecting) on a variable that is a common outcome of the exposure (or a variable associated with the exposure) and the outcome (or a variable associated with the outcome). Conditioning on these so-called 'collider' variables (which may even temporally precede both exposure and outcome) may distort the relationship between exposure and outcome. For example, the 'paradoxical' finding that maternal smoking appears protective for infant mortality among low birth weight infants, is induced by conditioning on a collider (i.e. low birth weight): smokers are more likely to have low birth weight infants, but low birth weight among infants that are not born to smokers is caused by something even worse (e.g. birth defects) [8]. Although there are more examples where collider bias is a serious problem [6, 9], recent simulations studies have shown that the magnitude of collider bias due to selection or censoring in prospective cohort studies is relatively modest [10-12]. This suggests that selection and attrition of GLOBE-participants may not have severely biased the observed associations. However, almost all analyses in this thesis conditioned on several other variables that were recognized as potential confounders. Although potential collider biases are expected to be smaller than biases resulting from omitting a confounder [11], it is still important to recognize that both confounding bias and (endogenous) selection bias may have affected the results.

Multiple imputation

Most chapters used multiple imputation techniques to account for missingness. In chapter 2, in addition to missingness on measured covariates, multiple imputation was also used to account for loss to follow-up, which means that we imputed all measurements for participants that were still alive but no longer participating in the GLOBE-study. By using multiple imputations, we assume that both missingness and loss to follow-up are 'missing at random', i.e. missingness and loss to follow-up can be accurately predicted by the variables included in the multiple imputation model [13-16]. Although a range of covariates were included in the multiple imputation model, to the extent that missingness or dropout was related to unmeasured factors (e.g. psychological characteristics or disorders, cognitive abilities), we were not able to adjust for this selection effect. Similarly, in other chapters that used multiple imputation to account for missing data,

it is assumed that missingness is random given observed covariates. Although it is not possible to verify whether this assumptions is accurate, the method is still preferred over other techniques, because most methods (e.g. 'complete case analyses') are only valid if missing data are 'missing completely at random' [13-16].

Cox proportional hazards regression and time-varying covariates

Chapter 2 made use of Cox proportional hazards regression in order to examine the contribution of behavioral and material risk factors to socioeconomic inequalities in mortality. In this chapter we adopted similar methods as previous studies, by using multiple measurements of the mediating risk factors and modelling them as time-varying covariates [17-20], rather than assuming that risk factors remain stable over the life-course. This method reclassified participants at every assessment in order to take changes in the status of risk factors into account (e.g. from current smoker to former smoker). However, the method may still not be completely accurate in modelling the actual changes in the hazards of participants [21]. First, correct specification of the etiological period is important. Participants were reclassified every 6-7 years, which may not correspond to the etiological period of all risk factors. Second, exposure to unfavorable material or behavioral risk factors in early adulthood may be associated with a lasting effect on mortality even if participants are unexposed in later years [22]. Such 'sensitive periods' are missed in the time-varying models. Moreover, to the extent that these sensitive periods have already occurred before the age of 25 years, they were completely missed in this study. Third, the method does not take an accumulation of exposure to a risk factor into account (e.g. those who are exposed only once contribute evenly to the estimations as those that are exposed multiple times) [22]. Fourth, interactions between risk factors are not taken into account. If exposure to multiple risk factors increases the risk of mortality more than the sum of the individual risks, this is missed in the current analysis. Fifth, using time-varying covariates also introduces the potential for time-varying confounders [23-25]. Specifically, in our study, the estimated associations between time-varying physical activity and mortality may have been overestimated due to confounding. Note however, that except for the latter, similar limitations hold for the time-fixed estimations. Moreover, by using this method, we were able to compare our results to previous studies that also adopted these methods.

Mediation methods

Several chapters in this thesis made use of mediation analysis. Mediation analysis is popular in studies examining explanations for socioeconomic inequalities in health and health-related behaviors, because it can be used to identify important risk factors that are on the causal pathway between socioeconomic position and the health-related outcomes, which could possibly be targeted by policies in order to reduce these health

inequalities. Mediation analysis is used to assess the extent to which the effect of socioeconomic position on the health outcomes is explained by the hypothesized mediators. Mediation analysis allows researchers to decompose the total effect of an exposure on an outcome (e.g. the effect of socioeconomic position on health-related behaviors) into a direct effect (unexplained by the mediators) and an indirect effect (explained by the mediators). The traditional approach to mediation analysis is a four-step approach that consists of three regression models [26, 27]. First, the total effect of the exposure on the outcome is estimated by regressing the outcome on the exposure (i.e. without adjustment for the mediators). Second, the effect of the exposure on the mediator is estimated by regressing the mediators on the exposure. Third, the effect of the mediators on the outcome conditional on the exposure is estimated by regressing the outcome on the mediators and the exposure. Fourth, the estimated effect of the exposure on the outcome, while adjusting for the mediators (i.e. the direct effect, which is estimated with the regression analysis in step three) is compared to the estimated total effect from the first step. Within this last step, a percentage mediated is usually calculated as the percent excess risk (on the relative scale) explained by the mediators. This percentage mediated is calculated by dividing the difference between the total risk (total effect from step one) and the direct risk (direct effect from step 3) by the total excess risk (total effect from step one – 1). This approach was also used in chapter 2 of this thesis.

The decomposition of a total effect into a direct and indirect effect as described above has recently been criticized, because of the limitations and implicit assumptions of this mediation method [27-30]. There are four important (unverifiable) assumptions that are required for effect decomposition: 1) no uncontrolled exposure-outcome confounding, 2) no uncontrolled mediator-outcome confounding, 3) no uncontrolled exposure-mediator confounding, and 4) no measured or unmeasured mediator-outcome confounder that is affected by the exposure. In addition, the traditional method also assumes that there is no interaction between exposure and mediator on the outcome. The main critique given to the traditional mediation method revolves around two of these potential biases that were never explicitly mentioned in the literature describing this method: a) mediator-outcome confounding, and b) exposure-mediator interaction. First, whereas the potential for bias arising from unmeasured exposure-outcome confounding is usually acknowledged, the potential for bias arising from unmeasured mediator-outcome confounding is often not explicitly acknowledged. However, conditioning on the mediator to estimate the direct effect of the exposure on the outcome (third regression analysis described above) induces a bias if there are unmeasured confounders of the mediator-outcome relationship. For example, in chapter two, the association between behavioral factors and mortality may have been confounded by biological risk factors (e.g. addiction susceptibility), psychological characteristics (e.g. personality) or psychosocial risk factors (e.g. stress). Not adequately adjusting for these potential confounders,

could therefore have biased our estimates. Second, if the direct effect of the exposure on the outcome varies across levels of the mediator (i.e. in the presence of exposure-mediator interaction), the difference between the direct effect and total effect will not only be caused by 'mediation' but also by 'moderation', which cannot be disentangled by this mediation method [28, 29].

The first bias (i.e. mediator-outcome confounding) can in principle be handled by the traditional method (i.e. accurate adjustment for all confounders), but this does require that all potential confounders have been measured. This requirement however, applies both to traditional methods and to newly developed methods (which are discussed below). In the presence of *unmeasured* confounders, no method will be able to observe unbiased effect estimates. In these circumstances, sensitivity analysis can be used to examine the potential severity of the bias [29]. An important limitation of the traditional methods however, is that it is only able to accurately adjust for *measured* confounders of the mediator-outcome relationship, if these confounders are not affected by the exposure (e.g. if they are not on the causal pathway from exposure to mediator/outcome) [23, 24, 28, 29]. If the mediator-outcome confounders *are* affected by the exposure, then conditioning on these confounders will also eliminate part of the direct effect of the exposure on the outcome, which will also results in biased effect estimates. For example, in chapter 4, the effect of the mediator (adult rural residence) on the (health-related) outcomes was confounded by education, while education itself was affected by the exposure (childhood rural residence). Conditioning on education (which is done in traditional mediation techniques) was therefore not possible, because it would have biased the effect estimates. The second bias (i.e. exposure-mediator interaction) also cannot be addressed by traditional mediation methods and requires the adoption of other techniques.

As a result of the limitations of the traditional mediation methods, a new mediation methodology has rapidly developed, often referred to as 'causal mediation analysis' [23, 28, 29]. This methodology is based on the potential outcomes framework and the seminal works of Pearl and Robins & Greenland [31, 32]. Chapter 4 makes use of these new mediation methods, because of the presence of exposure-mediator interaction and exposure-induced mediator-outcome confounders. The formulas used in this chapter are derived from counterfactual outcomes, which, under specific assumptions, allow for a causal interpretation of the estimated effects. However, the assumptions that are needed to make an actual causal interpretation with these methods are very strong. For example, in chapter 4, interpreting our observed effect estimates as causal is theoretically possible under the assumptions of no unmeasured confounding of the exposure-outcome and mediator-outcome relationships (in addition to the other assumptions, e.g. an unbiased study design and correct specification of the statistical models). However, even though we had an unusually comprehensive range of observed

confounders, it may still be implausible to assume that rural residents and non-rural residents are actually exchangeable given these observed covariates. Nevertheless, although a causal interpretation remains difficult, to actually nullify the magnitude of some of the observed effects, the confounding effects would have to be so large, that it is far more likely that the observed associations do actually exist.

Different strokes for different chapters

Since chapter 2 showed that time-fixed and time-varying models may differ, this has implications for the results of the other chapters that could not apply time-varying models due to a lack of longitudinal data. First, it is possible that the level of cultural capital of our participants changed over time and by measuring it just once we may have missed these changes (although cultural capital is arguably less dynamic in adults than for example health behaviors). Whether this would have resulted in greater or smaller effect estimates is hard to tell, given the diverging results of material and behavioral factors in chapter 2. Another limitation is that our measures of cultural capital are relatively limited. Cultural capital encompasses more than the measures used in this thesis, and because cultural capital accumulates over the life-course, it arguably requires data over the entire life-course (including parental history). Second, whereas chapter 2 showed that health-related behaviors change over the life-course, subsequent chapters only used a single measurement of the health-related behaviors as an outcome. This 'snapshot' of behavioral patterns over the life-course warrants caution when interpreting the results of this thesis. Clearly, more research is needed to examine the effect of cultural capital on the adoption of and changes in health-related behaviors. The results of this thesis should be considered as an exploration of the potential of cultural capital as an (increasingly) important determinant of socioeconomic inequalities in health-related behaviors.

Another discrepancy between chapters is the use of different mediation methods. The use of 'causal mediation analysis' was pursued in chapter 4, whereas other chapters adopted 'traditional' mediation methods. As described earlier, there is increasing attention for causal inference methodology in observational research and the newly developed mediation methods are highly relevant for research on health inequalities. In line with these developments, chapter 4 was used to explore and adopt these methods. However, although these newly developed methods offer solutions to many analytical issues that cannot be handled by conventional regression methods (i.e. controlling for time-varying confounding), not all of these methods can always be applied. For example, in chapter 2 we tried to adjust for time-varying confounding with inverse probability weights, but a violation of the positivity assumption obstructed this approach [33]. Further, in chapter 8 we did not need to adopt causal mediation techniques, because without the presence of non-linearities or interaction effects, the conventional mediation method is equivalent

to the causal mediation formulas from Valeri & VanderWeele (which are generalizations of the product method) [28, 34].

INTERPRETATION OF THE FINDINGS

The contribution of material risk factors and health-related behaviors to socioeconomic inequalities in mortality

In most studies that have examined explanations for socioeconomic inequalities in health, intermediary risk factors (e.g. material risk factors and health-related behaviors) were measured once (often at baseline) and subsequently linked to mortality or health after a certain period of follow-up time [35-37]. These studies assumed that risk factors remained fairly stable over the life-course and that the initial baseline measurement was a good indicator of life-long exposure. Clearly, this approach overlooks the dynamic nature of these risk factors, because risk factors change over the life-course, especially in studies with a long period of follow-up time between measuring risk factors and outcome.

The longitudinal GLOBE-study offered a great opportunity to study the extent to which material and behavioral risk factors, measured three times during adulthood, contributed to socioeconomic inequalities in mortality. Our results suggest that the contribution of health-related behaviors to socioeconomic inequalities in mortality is likely greater than previously thought, which corresponds with findings in the UK [20], US [17, 38] and Norway [18], but not with findings in France [19]. However, previous studies have done little to actually investigate what mechanisms contribute to the differences between time-fixed and time-varying models. The main rationale for these studies seem to be that socioeconomic inequalities have increased over time, and these increasing gradients may result in a greater contribution of health-related behaviors to socioeconomic inequalities in mortality. However, our results show that the increasing contribution of health-related behaviors to socioeconomic inequalities in mortality seems to be mostly driven by changing associations between the time-varying health behaviors and mortality. Moreover, the mechanisms underlying these changing associations may vary between risk factors. In our study, the most pronounced differences between time-fixed and time-varying health-related behaviors were found for leisure time physical activity and sports participation. For these health behaviors, it is likely that multiple measurements decreased misclassification and provided a better estimate of the true effect of leisure time physical activity on mortality. In addition, an age effect cannot be excluded. As participants age, leisure time physical activity may be increasingly important because work-related and transport-related physical activity decreases.

The smaller contribution of material factors in time-varying models (even though socioeconomic inequalities in material factors increased over time) is more puzzling and we admit that we are still not able to fully explain these results. First, it seems unlikely that the differences between time-fixed and time-varying models are caused by unmeasured time-varying confounding by health status, because changes in material factors were not associated with changes in health status. Although other potential unmeasured confounders may have affected the results, we were unable to identify any obvious confounders that could explain these findings. Second, there is a possibility that misspecification of the follow-up period contributed to the results. Mortality risks of exposure to adverse material conditions may only manifest after a time period that exceeds the 6-7 years between follow-up measurements in our time-varying models. However, using a follow-up time of less than 7 years is not uncommon in research investigating the contribution of material factors to socioeconomic inequalities in mortality [35, 37]. Third, it may be that our measures (i.e. financial difficulties, housing tenure and type of health insurance) do not adequately capture the proposed adverse material conditions of the materialist explanation, which may have resulted in more misclassification in time-varying models and driven the results towards the null. Fourth, the results could also reflect actual changes in the effect estimates. For example, it may be that exposure to adverse material conditions is more strongly related to mortality when experienced at younger ages (e.g. a sensitive/critical period effect) or in an earlier period in our study (i.e. the early 1990s). Further exploration of these age and period effects, and similar research in other contexts, is needed to better understand our findings.

Overall, we observed that behavioral factors explained about half of the excess mortality risk of lower socioeconomic groups (not taking material factors into account). This matches with previous studies that have estimated the contribution of repeatedly measured health behaviors to socioeconomic inequalities in all-cause mortality [17-20, 38]. These studies have used education, occupation and income as indicators of socioeconomic position and – although estimates varied between 17% and 75% depending on the context – most studies observed that the excess risk of lower socioeconomic groups was reduced by about half after adjustment for health-related behaviors. However, it is important to note that part of the effect of behavioral factors on mortality may be attributed to material factors, since unhealthy behaviors may be induced by adverse material conditions. Previous research within the GLOBE-study has tried to disentangle these direct and indirect contributions of material risk factors to socioeconomic inequalities in mortality [35, 37]. It did so by separately estimating the direct contribution of material factors (not through behavioral risk factors), the indirect contribution of material risk factors via behavioral factors, and the independent contribution of behavioral factors (not affected by material factors). The independent contribution of behavioral factors was determined as the percentage reduction of the model including both be-

havioral and material factors minus the percentage reduction of the model including only material factors. Using this method in our study would result in an 'independent' contribution of repeatedly measured health behaviors to respectively the educational and occupational gradient in mortality of 19% and 18% for men and 39% and 22% for women. Subsequently, the 'indirect' effect of material factors through health behaviors, was measured by subtracting the independent contribution of behavioral factors from the total contribution of health behaviors. Using this method would result in an 'indirect' contribution of repeatedly measured material factors of 29% and 30% for men and 12% and 18% for women, respectively. Finally, the 'direct' effect of material factors was calculated by subtracting the 'indirect' contribution of the material factors from the total contribution of the material factors. Using this method would result in a 'direct' contribution of material factors of 27% and 35% for men and 25% and 8% for women, respectively. These results show that even though the contribution of material factors may be lower than previously thought, adverse material conditions still impact strongly on health disparities, partly via health-related behaviors.

Early-life environment and health-related outcomes in later life

In the US, place of residence has been shown to be an important determinant of health inequalities [39-46]. US research has also shown tremendous differences between rural residents and non-rural residents in several health and health behavior outcomes [47-60]. Hartley (2004) even argued that the patterning of risky behavior in contemporary rural areas is suggestive of a "rural culture" as an important social determinant of health [61]. Exploring the effects of childhood rural residence on adult health and health-related behaviors therefore offered the opportunity to investigate whether early-life environments have long lasting effects on health-related behaviors of adults (albeit in the US context). Data from the REGARDS study included extensive information on childhood circumstances as well as health and health behaviors outcomes in adulthood [62], which made it possible to thoroughly examine effects of early-life environment on later life outcomes, adopting newly developed mediation methods to investigate the marginal health effects of childhood rural residence and evaluate whether these effects differ depending on place of adult residence.

The results of this study suggest that early-life environments have, to some extent, long lasting health effects, and perhaps more strikingly, that these effects depend on later life environment. This could suggest an accumulation effect (continued exposure to rural environments contribute to worse health and a higher prevalence of unhealthy behavior), which would indicate that health behavior preferences accumulate during lifelong socialization. However, no data were available on actual behavioral patterns of REGARDS participants during childhood, which limits inferences on the extent to which childhood behavioral patterns and preferences contributed to the observed results. In

addition, the most pronounced results were found for self-rated health, with lower estimated effect sizes for health-related behaviors, which suggests that social and economic disadvantages strongly impede on the health of rural US populations over and above increasing unhealthy behavior (i.e. smoking, physical activity and dietary patterns). Indeed, recent studies have found marked increases in suicide, accidental poisoning and liver disease in rural communities [63], suggesting that rising morbidity and mortality rates in some geographic areas of the United States reflect an increase in "deaths of despair" of rural populations [63-65]. Similar trends in mortality have not been found in other high-income countries [66], and suggest that there may be strong differences in the determinants of health inequalities between the US and Europe. For example, more generous social security systems, better access to (affordable) health care, and a lower availability and use of painkillers have likely contributed to lower socioeconomic inequalities in health in European countries compared to the US [65, 66]. Lastly, the observed interaction effects of living in a rural community in childhood and adulthood on the health outcomes may have also been affected by compositional changes (i.e. residents with characteristics related to better health have migrated to non-rural areas, whereas residents with characteristics related to worse health have remained in rural communities). Although we controlled for a range of potential confounders (i.e. education, childhood health and family finances), if there are other important characteristics related to migration patterns and health outcomes that were not included in the analysis, these may have affected the results.

Cultural capital and socioeconomic inequalities in health-related behaviors

Studies investigating socioeconomic inequalities in health-related behaviors have increasingly investigated whether environmental determinants are able to explain the social gradient in healthy dietary patterns, physical activity and overweight/obesity [67-77]. The most commonly investigated environments are the built environment (e.g. availability of facilities, walkability), the economic environment (e.g. costs and affordability) and the social environment (e.g. social support). The least often studied environment however, is the cultural environment. The results from this thesis suggest that this may have been an oversight from previous studies.

Although the studies in this thesis should be seen as exploratory studies into the role of cultural capital in the explanation of socioeconomic inequalities in health-related behaviors, our findings suggest that cultural capital may indeed partly explain why higher socioeconomic groups often adopt healthier dietary patterns, engage in more leisure time physical activity and are less often overweight or obese than lower socioeconomic groups. Cultural capital seems to provide higher socioeconomic groups with resources that increase the likelihood that they will adopt and maintain a more healthy lifestyle. Moreover, cultural capital may be a crucial resource to resist the temptations

of contemporary obesogenic environments that are characterized by an abundance of sugar, fat and sedentary activities [78, 79]. Indeed, a recent study from the UK found that lower educated groups were more vulnerable to unhealthy neighborhood fast-food outlet exposure than higher educated groups [80], which may be partly explained by differences in cultural capital.

Our findings support the hypothesis that higher socioeconomic groups engage in healthy behavior as a way to distinguish themselves from lower socioeconomic groups. Moreover, it may be that in contemporary societies healthiness and thinness are increasingly pursued as objects of cultivation for their own sake. This is likely reinforced by the fact that a healthy lifestyle is demanding: unhealthy behavior is often easy, whereas it requires effort and restrain to live healthily. Furthermore, globalization and commercialization have increased overall wealth, mass production and mass marketing, which has reduced the barriers to engage in "conspicuous consumption" [81, 82]. As a result, higher socioeconomic groups may more often adopt healthy lifestyles as a marker of distinction. This seems to be partly explained by a higher tendency towards reflexivity among higher socioeconomic groups. The embodiment of reflexivity as a cultural signifier among high cultural capital groups likely enforces a continuous self-reflection and monitoring of their (healthy) choices and actions.

The findings from this thesis are in line with other studies that have found that cultural capital is related to healthier dietary patterns [83-87], more physical activity [88, 89], differential sports preferences [89-92], and lower body weight [86, 88, 93]. Cultural capital however, may also be relevant for other health behaviors, such as smoking and alcohol intake [94, 95]. Moreover, cultural capital may be highly relevant for other health-related outcomes. For example, Shim (2010) argued that cultural capital is critical to effectively engage and communicate with clinical providers, and an important determinant of inequalities in treatment and care [96, 97]. This suggests that cultural capital may be a key determinant of social inequalities in health through multiple pathways, which may become increasingly important in societies where advances in biomedical knowledge and technology allow for an ever increasing possibility to postpone the onset of disease and mortality [98].

Reconsidering culture and socioeconomic inequalities in health

Studies on 'culture' are often received with suspicion and accused of "blaming the victim". This response can be traced back to work that emerged around the notion of a "culture of poverty", which argued that sustained poverty generated a set of cultural attitudes and practices, that would perpetuate over time even in the underlying structural conditions that gave rise to these practices were changed [99]. Although there were (and are) good reasons to be hesitant about studies that suggest a form of "pathology" as a determinant of social inequalities, cultural capital conceives of culture in a substantially different way

[100]. In an editorial in the *Annals of the American Academy of Political and Social Science*, Small, Harding & Lamont [100] list several reasons why culture should be studied by those interested in social inequality and poverty, not only because of scholarly motivations, but also because of policy motivations. Culture is part of the political and public discourse, and shapes policy decisions and policy makers. This is true for policies on health and health behaviors as much as it is for policies on poverty. Neglecting culture may therefore not only limited explanations for socioeconomic inequalities in health, but also lead to bad policy [100].

The primary reason for scholars to oppose studying 'culture' as a determinant of socioeconomic inequalities in health, is that it supposedly steers away from the material inequalities that are believed to be the actual root causes of socioeconomic inequalities in health. This discourse suggests that culture cannot give an independent explanation for socioeconomic inequalities in health, but merely reflects underlying inequalities in material factors. However, framing socioeconomic inequalities in health-related behaviors as a mere consequence of inequalities in material conditions implies that if these material conditions would be removed nothing would hold the individual back from engaging in healthier behavior. Adopting a perspective that takes the sociocultural environment into account, suggests that health-related behaviors are more than just a set of behaviors practiced and controlled by the individual, and part of a broader sociocultural context that affects health [101]. Moreover, allowing additional explanations in studies on socioeconomic inequalities in health also acknowledges that material conditions are simply not able to fully account for the distribution of health [102-104]. This thesis merely suggests that one of these alternative explanations may be found in differences in cultural capital. Everything we do has social meaning, health-related behaviors are clearly not excluded. Why should research on socioeconomic inequalities in health and health-related behaviors shy away from these explanations? *"[H]uman action is both constrained and enabled by the meaning people give to their actions, these dynamics should become central to our understanding of the production and reproduction of poverty and social inequality."* (*Small, Harding & Lamont, 2010, p.23)*

IMPLICATIONS FOR FUTURE RESEARCH

Studies that investigate the contribution of the (socio-)cultural environment to socioeconomic inequalities in health-related behaviors are scarce. This lack of research is likely reinforced by the difficulty of systematically measuring 'culture'. Cultural capital however, provides a more rigorous framework that can be used to measure and capture cultural differences [1, 105-107]. Moreover, an explanation of socioeconomic inequalities in health-related behaviors that includes cultural capital as a key determinant has some

advantages that other explanations in social epidemiology lack. More research however, is needed to examine to what extent cultural capital is able to provide an explanation for the prevalence of and trends in socioeconomic inequalities in health-related behaviors.

First, the theory of cultural capital states that health-related behaviors are likely a reflection of socially distinct lifestyles and not necessarily linked to economic resources [107-109], which doesn't contradict with the clustering of expensive unhealthy behaviors and the lower prevalence of low-cost healthy behaviors among lower socioeconomic groups. Indeed, the findings in this thesis also suggest that health-related behaviors are not mainly driven by economic capital, but by cultural capital. However, more research is needed to corroborate whether the results from this thesis are also observed in other contexts. Moreover, cultural capital encompasses more than the measures used in our studies [95, 106]. Additional research should explore whether other and more elaborate measures of cultural capital are also associated with health-related behaviors.

Second, cultural capital is transmitted from generation to generation [110], which signifies that it is not merely a determinant of individual health behaviors, but also a key determinant in the reproduction of health inequalities across generations [105]. Indeed, previous research in The Netherlands has found that higher parental cultural capital is associated with more institutional cultural capital (i.e. higher educational attainment) and more embodied cultural capital in later life [110]. If higher parental cultural capital also results in the embodiment of healthier practices and healthier behaviors, it may have long lasting effects on health-related behaviors in later life, net of one's own socioeconomic position. Research among children also supports this hypothesis. For example, higher parental cultural capital seems to be associated with healthier dietary patterns [84], lower BMI [111] and less smoking [112] among children. However, these are among the first studies that have investigated these intergenerational effects, and future research into this area is warranted.

Third, cultural capital may help to explain trends in health-related behaviors over time [113-116]. Those with a high level of cultural capital may adopt novel lifestyles in order to distinguish themselves from other social groups. These novelties usually diffuse to lower status groups over time [117], after which it loses it distinctiveness and high status groups change their lifestyles. Time trends in smoking and obesity follow similar patterns and may be explained by such status distinctions [118, 119]. More research on trends in health-related behaviors over time could provide a much needed historical perspective on the role of cultural capital as a potential determinant of health inequalities.

Fourth, other researchers have emphasized that the interplay between different types of capital (i.e. economic capital, social capital and cultural capital) is key to understanding health inequalities [83, 120]. Although these studies have thus far provided limited evidence for this perspective, the interplay between the different types of capital is a

key concept in the theory of cultural capital and warrants additional exploration [95, 105, 106].

Fifth, an additional avenue of research is to examine the role of 'habitus' for adopting, maintaining and changing health-related behaviors. As argued by Bourdieu, habitus results from lifelong socialization and mostly operates unconsciously through internalized preferences and tastes [95, 121]. Exploring the effects of the habitus therefore requires research designs that are better able to elicit the automated responses of the habitus [122]. Potential methods that are better suited to explore the role of the habitus include the use of Implicit Association Tests – specifically designed to elicit unconscious attitudes of subjects [123] – or prototype perceptions – social images of typical persons engaging in certain behaviors [124].

Sixth, an important question is *how* does cultural capital affect different health behaviors [125]. As argued in chapter 8, the relationship between classical measures of cultural capital (e.g. 'highbrow' cultural activities) and health-related behaviors does not reflect an actual causal mechanism. However, identification of the causal mechanisms (i.e. cultural signifiers that vary across social groups) is critical for a more rigorous test of the importance of cultural capital for socioeconomic inequalities in health. Future research should further explore potential mechanisms that may explain the relationship between cultural capital and different types of health-related behaviors. Qualitative research designs may be key to identifying those mechanism, which could then perhaps be further explored in quantitative research with more potential for generalization.

Seventh, in order to provide more evidence for an actual causal effect of cultural capital on health-related behaviors, studies should also use different designs or techniques that are better suited for causal inference. For example, the use of twin studies, relating cultural capital to health-related behaviors of twins who grew up in environments with different levels of cultural capital, would be able to effectively control for genetic factors, which would provide stronger evidence that an observed association represents an actual causal effect.

Lastly, the relational positioning of health behaviors in social space is an important aspect of the work of Bourdieu and only sparsely explored in other studies [126, 127]. Using multiple correspondence analysis techniques – which visually maps to what extent categories of included variables are related to each other – may provide useful insights into the relationship between several forms of capital and health-related behaviors. In addition, qualitative research designs hold the promise to provide greater insight and in-depth understanding of the mechanisms that drive health-related behaviors in different social contexts. Although these methods are much less common in social epidemiologic research, other disciplines (e.g. anthropology, sociology) do have rich tradition of qualitative research. More collaboration and exchange between disciplines

will likely increase our collective imagination and provide a richer understanding of the drivers of health inequalities.

The quest for causality and theoretical depth

The lack of success in the field of social epidemiology (i.e. a failure to reduce health inequalities) has let social epidemiologist to ponder what could be improved [98, 128-141]. Two important problems are often mentioned, sometimes concurrently: 1) lack of evidence on causal relations, and 2) lack of adequate theoretical models. Although both are often acknowledged to be important, they are currently also often in odds with each other. The first problem refers to inconclusive evidence and potential biases that arise from using observational studies. These issues have led to the rise of the field of causal inference, which has come up with formal definitions and rules that allow for causal inference on the basis of observational data [142-146]. The second problem has led to the adoption of socioecological and life-course models, with increasing attention for environmental determinants and differential effects of exposures across the life-course [22, 98, 104, 140, 147-150].

These two emergent paradigms are often in conflict with each other [151, 152], because the prevailing statistical techniques in the social sciences are not yet able to unite them. Whereas the causal inference paradigm is formal and requires well-defined interventions (consistency assumption), the paradigm concerned with theoretical substance is often not able to refuse and sacrifice the rich detail of social life that is needed to come to an adequate understanding of socioeconomic inequalities in health. However, at the core, both paradigms are concerned with the same questions: *whether* – and to what extent – X affects Y and *why* or *how* X affects Y. To be able to make any scientific claims about these questions, causality should take priority. However, to have any significance, these questions should be answered in reference to the real-world, which is not always reducible to well-defined interventions. This means that the causal processes under study will often need to be contextualized with theories that will inevitable not adhere to the rules of the causal inference paradigm. A major challenge will be to successfully integrate these two paradigms, because both are needed to make any significant progress in explaining and reducing health inequalities.

IMPLICATIONS FOR POLICY AND INTERVENTIONS

Socioeconomic inequalities in health-related behaviors are of interest to social epidemiologists first and foremost because they contribute to socioeconomic inequalities in health. Such inequalities in health are deemed unfair, at least to the extent that they are related to societal and environmental processes that are beyond the control of individu-

als, and should therefore be reduced. Moreover, increasing the health status of lower socioeconomic groups to that of their more advantaged counterparts would generate a substantial improvement in population health and reduction in health care costs, which benefits the whole of society. However, in order to effectively reduce socioeconomic inequalities in health, it is important to identify mechanisms that contribute to these inequalities and that provide entry-points for policies and interventions to improve the health of lower socioeconomic groups. This is where the attention for socioeconomic inequalities in health-related behaviors has emerged: health-related behaviors have been shown to contribute to socioeconomic inequalities in health and could therefore be targeted as a way to reduce health inequalities. To aid in this endeavor, this last chapter is devoted to the implications of our findings for policies and interventions that aim to reduce socioeconomic inequalities health-related behaviors and health.

Culturally adaptive interventions

The findings in this thesis suggest that cultural inequalities may be more relevant for health-related behaviors than economic inequalities, at least in the Dutch context. This suggests that adopting healthy behavioral patterns will be much more difficult for lower socioeconomic groups, because it will conflict more with their identity and that of their socio-cultural environment. This cultural context should be given a more prominent role in interventions. Whereas interventions targeting racial/ethnic minority groups do sometimes adopt culturally tailored/targeted interventions [153-159], this is almost never done outside the realm of racial/ethnic differences. However, as is shown in this thesis, culture is much more than race or ethnicity, and behavioral interventions should also be tailored to the sociocultural context of lower socioeconomic groups to improve their effectiveness. An important question is how to do so. This is not a question that should – or can – be answered by (public health) professionals, but should be informed by the ideas and perceptions of the target groups themselves. It seems clear that health-related behaviors do not have the same meaning in lower socioeconomic groups as they have in higher socioeconomic groups. Interventions should therefore be better aligned to what is considered meaningful and practicable in different socio-cultural environments [101, 111]. For example, among groups with a high level of institutional distrust (which is related to low cultural capital), governmental health campaigns emphasizing the health consequences of certain behaviors (e.g. smoking, alcohol consumption, the consumption of ultra-processed foods) are likely ineffective. However, if such a message would be transferred through (culturally) significant peers, it may prove to be more effective. A similar strategy was adopted in the ASSIST trail, where a peer-led intervention in secondary schools in the UK was found to be effective in preventing smoking among adolescents [160].

As the twig is bent, so is the tree inclined

Socialization is key in the adoption of cultural capital and the embodiment of norms and preferences. Socialization therefore likely plays a strong role in the adoption of health-related behaviors [105, 107, 161]. Interventions focusing on young children and their parents may therefore be most successful, as incorporating healthy habits then becomes part of the socialization process. For example, helping parents with young children to adopt childrearing practices that will stimulate the development of healthy eating practices can be seen as a promising strategy. However, as previously described, it is important that such an intervention is aligned with the socio-cultural environment of the parents, and doesn't 'force' practices that are orthogonal to those of the target group. Schools also provide a promising setting to stimulate healthy behavior, for example by providing healthy meals to pupils, restricting unhealthy products in canteens and vending machines, and by increasing time for physical education. However, it is key that such interventions and policies focus on continuity and long term effects (e.g. by making it part of the school's curriculum), because it takes time to accept and internalize new practices and that is best accomplished by continued exposure. Although such interventions do already exist [162], there is an urgent need to expand these initiatives in order to tackle the vicious cycle of social and health-related problems that persist from generation to generation.

Target unhealthy production, not unhealthy consumption

The importance of cultural capital in generating and maintaining socioeconomic inequalities in health-related behaviors also suggests that changing this distribution will not be easily accomplished. However, there are options available that bypass this problem entirely and that have the greatest potential to improve the health of all population groups: reducing exposure to unhealthy products for the entire population. Research also shows that structural interventions – interventions that remove individual agency and render the healthier option the only option – are most likely to reduce socioeconomic inequalities in health [163, 164]. For example, smoke-free legislation, restrictions on the amount of sugar or salt in food and drink products, or removal of vending machines with unhealthy snacks from schools or workplaces, bypasses that individuals have to make the healthy choice themselves. These structural policies have a far greater chance of reducing health inequalities than interventions that rely solely on the conscious action (i.e. agency) of the individual. The findings of this thesis also support this perspective, because it suggests that due to differences in cultural capital, lower socioeconomic groups will have lower engagement with and more negative reactions to health behavior interventions that merely targets individual choices (e.g. interventions relying on information and education). Unfortunately, contemporary governments seem to be very reluctant to intervene in unhealthy production [150, 163,

164]. This is not just a matter of political ideology (i.e. political ideologies that prefer less governmental regulations), but rather a striking gap between regulations for products that have the most severe health consequences (and high economic value) and regulations for other consumption products. As a consequence, most public health initiatives are focused on unhealthy consumption. However, it should be a core task of public health professionals to continuously remind our legislators that they have the obligation to protect the health of the population. There is a far greater likelihood to tackle health inequalities if effort is given to necessitate structural interventions, rather than investing increasing amounts of time and money in short-term interventions that aim to change health behaviors one person at a time.

REFERENCES

[1] Kamphuis CB, Jansen T, Mackenbach JP, van Lenthe FJ. Bourdieu's Cultural Capital in Relation to Food Choices: A Systematic Review of Cultural Capital Indicators and an Empirical Proof of Concept. PLoS One. 2015;10:e0130695.

[2] Mackenbach JP, van de Mheen H, Stronks K. A prospective cohort study investigating the explanation of socio-economic inequalities in health in The Netherlands. Soc Sci Med. 1994;38:299-308.

[3] van Lenthe FJ, Kamphuis CB, Beenackers MA, Jansen T, Looman CW, Nusselder WJ, et al. Cohort Profile: Understanding socioeconomic inequalities in health and health behaviours: the GLOBE study. Int J Epidemiol. 2014;43:721-30.

[4] Elwert F, Winship C. Endogenous selection bias: The problem of conditioning on a collider variable. Annual Review of Sociology. 2014;40:31-53.

[5] Cole SR, Platt RW, Schisterman EF, Chu H, Westreich D, Richardson D, et al. Illustrating bias due to conditioning on a collider. Int J Epidemiol. 2010;39:417-20.

[6] Munafo MR, Tilling K, Taylor AE, Evans DM, Davey Smith G. Collider scope: when selection bias can substantially influence observed associations. Int J Epidemiol. 2018;47:226-35.

[7] Hernan MA, Hernandez-Diaz S, Robins JM. A structural approach to selection bias. Epidemiology. 2004;15:615-25.

[8] Hernandez-Diaz S, Schisterman EF, Hernan MA. The birth weight "paradox" uncovered? Am J Epidemiol. 2006;164:1115-20.

[9] Stovitz SD, Banack HR, Kaufman JS. Paediatric obesity appears to lower the risk of diabetes if selection bias is ignored. J Epidemiol Community Health. 2018;72:302-8.

[10] Whitcomb BW, McArdle PF. Collider-stratification Bias Due to Censoring in Prospective Cohort Studies. Epidemiology. 2016;27:e4-5.

[11] Greenland S. Quantifying biases in causal models: classical confounding vs collider-stratification bias. Epidemiology. 2003;14:300-6.

[12] Glymour MM, Vittinghoff E. Commentary: selection bias as an explanation for the obesity paradox: just because it's possible doesn't mean it's plausible. Epidemiology. 2014;25:4-6.

[13] Goldstein H. Handling attrition and non-response in longitudinal data. Longitudinal and Life Course Studies. 2009;1.

[14] Harel O, Mitchell EM, Perkins NJ, Cole SR, Tchetgen Tchetgen EJ, Sun B, et al. Multiple Imputation for Incomplete Data in Epidemiologic Studies. Am J Epidemiol. 2018;187:576-84.

[15] Pedersen AB, Mikkelsen EM, Cronin-Fenton D, Kristensen NR, Pham TM, Pedersen L, et al. Missing data and multiple imputation in clinical epidemiological research. Clin Epidemiol. 2017;9:157-66.

[16] Sterne JA, White IR, Carlin JB, Spratt M, Royston P, Kenward MG, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. BMJ. 2009;338:b2393.

[17] Mehta NK, House JS, Elliott MR. Dynamics of health behaviours and socioeconomic differences in mortality in the USA. J Epidemiol Community Health. 2015;69:416-22.

[18] Skalicka V, Ringdal K, Witvliet MI. Socioeconomic inequalities in mortality and repeated measurement of explanatory risk factors in a 25 years follow-up. PLoS One. 2015;10:e0124690.

[19] Stringhini S, Dugravot A, Shipley M, Goldberg M, Zins M, Kivimaki M, et al. Health behaviours, socioeconomic status, and mortality: further analyses of the British Whitehall II and the French GAZEL prospective cohorts. PLoS Med. 2011;8:e1000419.

[20] Stringhini S, Sabia S, Shipley M, Brunner E, Nabi H, Kivimaki M, et al. Association of socioeconomic position with health behaviors and mortality. JAMA. 2010;303:1159-66.

[21] Fisher LD, Lin DY. Time-dependent covariates in the Cox proportional-hazards regression model. Annu Rev Public Health. 1999;20:145-57.
[22] Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. Int J Epidemiol. 2002;31:285-93.
[23] VanderWeele TJ. Marginal structural models for the estimation of direct and indirect effects. Epidemiology. 2009;20:18-26.
[24] Robins JM, Hernan MA, Brumback B. Marginal structural models and causal inference in epidemiology. Epidemiology. 2000;11:550-60.
[25] Mansournia MA, Etminan M, Danaei G, Kaufman JS, Collins G. Handling time varying confounding in observational research. BMJ. 2017;359:j4587.
[26] Baron RM, Kenny DA. The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. Journal of personality and social psychology. 1986;51:1173.
[27] Richiardi L, Bellocco R, Zugna D. Mediation analysis in epidemiology: methods, interpretation and bias. Int J Epidemiol. 2013;42:1511-9.
[28] Valeri L, Vanderweele TJ. Mediation analysis allowing for exposure-mediator interactions and causal interpretation: theoretical assumptions and implementation with SAS and SPSS macros. Psychol Methods. 2013;18:137-50.
[29] VanderWeele T. Explanation in causal inference: methods for mediation and interaction: Oxford University Press; 2015.
[30] VanderWeele TJ, Vansteelandt S. Conceptual issues concerning mediation, interventions and composition. Statistics and its Interface. 2009;2:457-68.
[31] Pearl J. Direct and indirect effects. Proceedings of the seventeenth conference on uncertainty in artificial intelligence: Morgan Kaufmann Publishers Inc.; 2001. p. 411-20.
[32] Robins JM, Greenland S. Identifiability and exchangeability for direct and indirect effects. Epidemiology. 1992:143-55.
[33] Cole SR, Hernan MA. Constructing inverse probability weights for marginal structural models. Am J Epidemiol. 2008;168:656-64.
[34] Naimi AI, Schnitzer ME, Moodie EE, Bodnar LM. Mediation Analysis for Health Disparities Research. Am J Epidemiol. 2016;184:315-24.
[35] Schrijvers CT, Stronks K, van de Mheen HD, Mackenbach JP. Explaining educational differences in mortality: the role of behavioral and material factors. Am J Public Health. 1999;89:535-40.
[36] Skalicka V, van Lenthe F, Bambra C, Krokstad S, Mackenbach J. Material, psychosocial, behavioural and biomedical factors in the explanation of relative socio-economic inequalities in mortality: evidence from the HUNT study. Int J Epidemiol. 2009;38:1272-84.
[37] van Oort FV, van Lenthe FJ, Mackenbach JP. Material, psychosocial, and behavioural factors in the explanation of educational inequalities in mortality in The Netherlands. J Epidemiol Community Health. 2005;59:214-20.
[38] Nandi A, Glymour MM, Subramanian SV. Association among socioeconomic status, health behaviors, and all-cause mortality in the United States. Epidemiology. 2014;25:170-7.
[39] Dwyer-Lindgren L, Bertozzi-Villa A, Stubbs RW, Morozoff C, Shirude S, Unutzer J, et al. Trends and Patterns of Geographic Variation in Mortality From Substance Use Disorders and Intentional Injuries Among US Counties, 1980-2014. JAMA. 2018;319:1013-23.
[40] Dwyer-Lindgren L, Mackenbach JP, van Lenthe FJ, Mokdad AH. Self-reported general health, physical distress, mental distress, and activity limitation by US county, 1995-2012. Popul Health Metr. 2017;15:16.

[41] Dwyer-Lindgren L, Bertozzi-Villa A, Stubbs RW, Morozoff C, Mackenbach JP, van Lenthe FJ, et al. Inequalities in Life Expectancy Among US Counties, 1980 to 2014: Temporal Trends and Key Drivers. JAMA Intern Med. 2017;177:1003-11.

[42] Dwyer-Lindgren L, Bertozzi-Villa A, Stubbs RW, Morozoff C, Kutz MJ, Huynh C, et al. US County-Level Trends in Mortality Rates for Major Causes of Death, 1980-2014. JAMA. 2016;316:2385-401.

[43] Dwyer-Lindgren L, Mokdad AH, Srebotnjak T, Flaxman AD, Hansen GM, Murray CJ. Cigarette smoking prevalence in US counties: 1996-2012. Popul Health Metr. 2014;12:5.

[44] Dwyer-Lindgren L, Freedman G, Engell RE, Fleming TD, Lim SS, Murray CJ, et al. Prevalence of physical activity and obesity in US counties, 2001-2011: a road map for action. Popul Health Metr. 2013;11:7.

[45] Howard G, Howard VJ, Katholi C, Oli MK, Huston S. Decline in US stroke mortality: an analysis of temporal patterns by sex, race, and geographic region. Stroke. 2001;32:2213-20.

[46] Casper M, Kramer MR, Quick H, Schieb LJ, Vaughan AS, Greer S. Changes in the Geographic Patterns of Heart Disease Mortality in the United States: 1973 to 2010. Circulation. 2016;133:1171-80.

[47] Befort CA, Nazir N, Perri MG. Prevalence of obesity among adults from rural and urban areas of the United States: findings from NHANES (2005-2008). J Rural Health. 2012;28:392-7.

[48] Doescher MP, Jackson JE, Jerant A, Gary Hart L. Prevalence and trends in smoking: a national rural study. J Rural Health. 2006;22:112-8.

[49] Eberhardt MS, Freid VM, Harper S, Ingram DD, Makuc DM, Pamuk E, et al. Health, United States, 2001; with Urban and rural health chartbook. 2001.

[50] Howard G, Kleindorfer DO, Cushman M, Long DL, Jasne A, Judd SE, et al. Contributors to the Excess Stroke Mortality in Rural Areas in the United States. Stroke. 2017;48:1773-8.

[51] Lutfiyya MN, Chang LF, Lipsky MS. A cross-sectional study of US rural adults' consumption of fruits and vegetables: do they consume at least five servings daily? BMC Public Health. 2012;12:280.

[52] Martin SL, Kirkner GJ, Mayo K, Matthews CE, Durstine JL, Hebert JR. Urban, rural, and regional variations in physical activity. J Rural Health. 2005;21:239-44.

[53] Matthews KA, Croft JB, Liu Y, Lu H, Kanny D, Wheaton AG, et al. Health-Related Behaviors by Urban-Rural County Classification - United States, 2013. MMWR Surveill Summ. 2017;66:1-8.

[54] Meit M, Knudson A, Gilbert T, Yu AT, Tanenbaum E, Ormson E, et al. The 2014 update of the rural-urban chartbook. Rural Health Reform Policy Research Center2014.

[55] Parks SE, Housemann RA, Brownson RC. Differential correlates of physical activity in urban and rural adults of various socioeconomic backgrounds in the United States. J Epidemiol Community Health. 2003;57:29-35.

[56] Patterson PD, Moore CG, Probst JC, Shinogle JA. Obesity and physical inactivity in rural America. J Rural Health. 2004;20:151-9.

[57] Roberts ME, Doogan NJ, Kurti AN, Redner R, Gaalema DE, Stanton CA, et al. Rural tobacco use across the United States: How rural and urban areas differ, broken down by census regions and divisions. Health Place. 2016;39:153-9.

[58] Singh GK, Siahpush M. Widening rural-urban disparities in all-cause mortality and mortality from major causes of death in the USA, 1969-2009. J Urban Health. 2014;91:272-92.

[59] Singh GK, Siahpush M. Widening rural-urban disparities in life expectancy, U.S., 1969-2009. Am J Prev Med. 2014;46:e19-29.

[60] Singh GK, Siahpush M, Williams SD. Changing urbanization patterns in US lung cancer mortality, 1950-2007. J Community Health. 2012;37:412-20.

[61] Hartley D. Rural health disparities, population health, and rural culture. Am J Public Health. 2004;94:1675-8.

[62] Howard VJ, Cushman M, Pulley L, Gomez CR, Go RC, Prineas RJ, et al. The reasons for geographic and racial differences in stroke study: objectives and design. Neuroepidemiology. 2005;25:135-43.

[63] Stein EM, Gennuso KP, Ugboaja DC, Remington PL. The Epidemic of Despair Among White Americans: Trends in the Leading Causes of Premature Death, 1999-2015. Am J Public Health. 2017;107:1541-7.

[64] Case A, Deaton A. Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century. Proc Natl Acad Sci U S A. 2015;112:15078-83.

[65] Case A, Deaton A. Mortality and morbidity in the 21(st) century. Brookings Pap Econ Act. 2017;2017:397-476.

[66] Mackenbach JP, Valverde JR, Artnik B, Bopp M, Bronnum-Hansen H, Deboosere P, et al. Trends in health inequalities in 27 European countries. Proc Natl Acad Sci U S A. 2018.

[67] Ball K, Crawford D, Mishra G. Socio-economic inequalities in women's fruit and vegetable intakes: a multilevel study of individual, social and environmental mediators. Public Health Nutr. 2006;9:623-30.

[68] Ball K, Lamb KE, Costa C, Cutumisu N, Ellaway A, Kamphuis CB, et al. Neighbourhood socioeconomic disadvantage and fruit and vegetable consumption: a seven countries comparison. Int J Behav Nutr Phys Act. 2015;12:68.

[69] Ball K, Timperio A, Salmon J, Giles-Corti B, Roberts R, Crawford D. Personal, social and environmental determinants of educational inequalities in walking: a multilevel study. J Epidemiol Community Health. 2007;61:108-14.

[70] Giskes K, Avendano M, Brug J, Kunst AE. A systematic review of studies on socioeconomic inequalities in dietary intakes associated with weight gain and overweight/obesity conducted among European adults. Obes Rev. 2010;11:413-29.

[71] Giskes K, Kamphuis CB, van Lenthe FJ, Kremers S, Droomers M, Brug J. A systematic review of associations between environmental factors, energy and fat intakes among adults: is there evidence for environments that encourage obesogenic dietary intakes? Public Health Nutr. 2007;10:1005-17.

[72] Giskes K, Turrell G, van Lenthe FJ, Brug J, Mackenbach JP. A multilevel study of socio-economic inequalities in food choice behaviour and dietary intake among the Dutch population: the GLOBE study. Public Health Nutr. 2006;9:75-83.

[73] Giskes K, van Lenthe FJ, Kamphuis CB, Huisman M, Brug J, Mackenbach JP. Household and food shopping environments: do they play a role in socioeconomic inequalities in fruit and vegetable consumption? A multilevel study among Dutch adults. J Epidemiol Community Health. 2009;63:113-20.

[74] Kamphuis CB, van Lenthe FJ, Giskes K, Brug J, Mackenbach JP. Perceived environmental determinants of physical activity and fruit and vegetable consumption among high and low socioeconomic groups in the Netherlands. Health Place. 2007;13:493-503.

[75] Kamphuis CB, Van Lenthe FJ, Giskes K, Huisman M, Brug J, Mackenbach JP. Socioeconomic status, environmental and individual factors, and sports participation. Med Sci Sports Exerc. 2008;40:71-81.

[76] Kamphuis CB, van Lenthe FJ, Giskes K, Huisman M, Brug J, Mackenbach JP. Socioeconomic differences in lack of recreational walking among older adults: the role of neighbourhood and individual factors. Int J Behav Nutr Phys Act. 2009;6:1.

[77] van Wijk DC, Oude Groeniger J, van Lenthe FJ, Kamphuis CB. The role of the built environment in explaining educational inequalities in walking and cycling among adults in the Netherlands. Int J Health Geogr. 2017;16:10.

[78] Swinburn BA, Egger G, Raza F. Dissecting obesogenic environments: the development and application of a framework for identifying and prioritizing environmental interventions for obesity. Prev Med. 1999;29:563-70.

[79] Swinburn BA, Sacks G, Hall KD, McPherson K, Finegood DT, Moodie ML, et al. The global obesity pandemic: shaped by global drivers and local environments. Lancet. 2011;378:804-14.

[80] Burgoine T, Forouhi NG, Griffin SJ, Brage S, Wareham NJ, Monsivais P. Does neighborhood fast-food outlet exposure amplify inequalities in diet and obesity? A cross-sectional study. Am J Clin Nutr. 2016;103:1540-7.

[81] Currid-Halkett E. The sum of small things: A theory of the aspirational class: Princeton University Press; 2017.

[82] Veblen T. The theory of the leisure class. New York: Macmillan; 1899.

[83] De Clercq B, Abel T, Moor I, Elgar FJ, Lievens J, Sioen I, et al. Social inequality in adolescents' healthy food intake: the interplay between economic, social and cultural capital. Eur J Public Health. 2017;27:279-86.

[84] Fismen AS, Samdal O, Torsheim T. Family affluence and cultural capital as indicators of social inequalities in adolescent's eating behaviours: a population-based survey. BMC Public Health. 2012;12:1036.

[85] Kraaykamp G. Cumulative advantages and inequality in lifestyle: A Dutch description of distinction in taste. 2002.

[86] McLaren L, Kuh D. Women's body dissatisfaction, social class, and social mobility. Soc Sci Med. 2004;58:1575-84.

[87] Sato PD, Gittelsohn J, Unsain RF, Roble OJ, Scagliusi FB. The use of Pierre Bourdieu's distinction concepts in scientific articles studying food and eating: A narrative review. Appetite. 2016;96:174-86.

[88] Christensen VT, Carpiano RM. Social class differences in BMI among Danish women: applying Cockerham's health lifestyles approach and Bourdieu's theory of lifestyle. Soc Sci Med. 2014;112:12-21.

[89] Wilson TC. The paradox of social class and sports involvement: The roles of cultural and economic capital. Int Rev Sociol Sport. 2002;37:5-16.

[90] Kraaykamp G, Oldenkamp M, Breedveld K. Starting a sport in the Netherlands: A life-course analysis of the effects of individual, parental and partner characteristics. Int Rev Sociol Sport. 2013;48:153-70.

[91] Scheerder J, Vanreusel B, Taks M, Renson R. Social sports stratification in Flanders 1969-1999: intergenerational reproduction of social inequalities? Int Rev Sociol Sport. 2002;37:219-45.

[92] Stempel C. Adult participation sports as cultural capital: A test of Bourdieu's theory of the field of sports. Int Rev Sociol Sport. 2005;40:411-32.

[93] Pampel FC. Does reading keep you thin? Leisure activities, cultural tastes, and body weight in comparative perspective. Sociology of Health & Illness. 2012;34:396-411.

[94] Pampel FC, Krueger PM, Denney JT. Socioeconomic Disparities in Health Behaviors. Annu Rev Sociol. 2010;36:349-70.

[95] Bourdieu P. Distinction: A social critique of the judgement of taste. Cambridge: Harvard University Press; 1984.

[96] Dubbin LA, Chang JS, Shim JK. Cultural health capital and the interactional dynamics of patient-centered care. Soc Sci Med. 2013;93:113-20.
[97] Shim JK. Cultural health capital: A theoretical approach to understanding health care interactions and the dynamics of unequal treatment. J Health Soc Behav. 2010;51:1-15.
[98] Link BG. Epidemiological sociology and the social shaping of population health. J Health Soc Behav. 2008;49:367-84.
[99] Lewis O. La vida: A Puerto Rican family in the culture of poverty-San Juan and New York: Random House New York; 1966.
[100] Small ML, Harding DJ, Lamont M. Reconsidering culture and poverty. Sage Publications Sage CA: Los Angeles, CA; 2010.
[101] Frohlich KL, Corin E, Potvin L. A theoretical proposal for the relationship between context and disease. Sociology of health & illness. 2001;23:776-97.
[102] Phelan JC, Link BG, Tehranifar P. Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. J Health Soc Behav. 2010;51 Suppl:S28-40.
[103] Phelan JC, Link BG, Diez-Roux A, Kawachi I, Levin B. "Fundamental causes" of social inequalities in mortality: a test of the theory. J Health Soc Behav. 2004;45:265-85.
[104] Link BG, Phelan J. Social conditions as fundamental causes of disease. J Health Soc Behav. 1995;Spec No:80-94.
[105] Abel T. Cultural capital and social inequality in health. J Epidemiol Community Health. 2008;62:e13.
[106] Bourdieu P. The forms of capital. In: Richardson JG, editor. Handbook of Theory and Research for the Sociology of Education. Westport, CT: Greenwood Press; 1986. p. 241-58.
[107] Williams SJ. Theorising class, health and lifestyles: can Bourdieu help us? Sociology of health & illness. 1995;17:577-604.
[108] Cockerham WC. Health lifestyle theory and the convergence of agency and structure. J Health Soc Behav. 2005;46:51-67.
[109] Cockerham WC, Rutten A, Abel T. Conceptualizing contemporary health lifestyles: Moving beyond Weber. Sociol Quart. 1997;38:321-42.
[110] Kraaykamp G, van Eijck K. The Intergenerational Reproduction of Cultural Capital A Threefold Perspective. Soc Forces. 2010;89:209-31.
[111] Christensen VT. Does parental capital influence the prevalence of child overweight and parental perceptions of child weight-level? Soc Sci Med. 2011;72:469-77.
[112] Schori D, Hofmann K, Abel T. Social inequality and smoking in young Swiss men: intergenerational transmission of cultural capital and health orientation. Int J Public Health. 2014;59:261-70.
[113] Graham H. Smoking prevalence among women in the European community 1950–1990. Social science & medicine. 1996;43:243-54.
[114] Lopez AD, Collishaw NE, Piha T. A descriptive model of the cigarette epidemic in developed countries. Tobacco control. 1994;3:242.
[115] Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. Psychol Bull. 1989;105:260-75.
[116] Thun M, Peto R, Boreham J, Lopez AD. Stages of the cigarette epidemic on entering its second century. Tob Control. 2012;21:96-101.
[117] Rogers EM. Diffusion of innovations: Simon and Schuster; 2010.
[118] Pampel FC. Cigarette diffusion and sex differences in smoking. J Health Soc Behav. 2001;42:388-404.
[119] Mackenbach J, Roskam A. Gewichtige verschillen: sociale stratificatie en overgewicht. In: Dagevos H, Munichs G, editors. De obesogene samenleving. Amsterdam: University Press; 2007.

[120] Veenstra G, Abel T. Capital interplays and the self-rated health of young men: results from a cross-sectional study in Switzerland. Int J Equity Health. 2015;14:38.
[121] Bourdieu P. The logic of practice: Stanford University Press; 1990.
[122] Lamont M, Adler L, Park BY, Xiang X. Bridging cultural sociology and cognitive psychology in three contemporary research programmes. Nature Human Behaviour. 2017;1:866.
[123] Greenwald AG, McGhee DE, Schwartz JLK. Measuring individual differences in implicit cognition: The implicit association test. Journal of Personality and Social Psychology. 1998;74:1464-80.
[124] Gibbons FX, Gerrard M. Predicting Young-Adults Health Risk Behavior. Journal of Personality and Social Psychology. 1995;69:505-17.
[125] van der Waal J, de Koster W. Naar een analytische stratificatiesociologie. Over klasse, status en ongelijkheid. Sociologie. 2015;11:1-41.
[126] Veenstra G. Social space, social class and Bourdieu: health inequalities in British Columbia, Canada. Health Place. 2007;13:14-31.
[127] Burnett PJ, Veenstra G. Margins of freedom: a field-theoretic approach to class-based health dispositions and practices. Sociol Health Illn. 2017;39:1050-67.
[128] Harper S, Strumpf EC. Social epidemiology: questionable answers and answerable questions. Epidemiology. 2012;23:795-8.
[129] Galea S, Link BG. Six paths for the future of social epidemiology. Am J Epidemiol. 2013;178:843-9.
[130] Oakes JM. Invited commentary: Paths and pathologies of social epidemiology. Am J Epidemiol. 2013;178:850-1.
[131] Muntaner C. Invited commentary: On the future of social epidemiology--a case for scientific realism. Am J Epidemiol. 2013;178:852-7.
[132] Glymour MM, Osypuk TL, Rehkopf DH. Invited commentary: Off-roading with social epidemiology--exploration, causation, translation. Am J Epidemiol. 2013;178:858-63.
[133] Krieger N, Smith GD. Re: "Seeking causal explanations in social epidemiology". Am J Epidemiol. 2000;151:831-3.
[134] Kaufman JS, Cooper RS. Seeking causal explanations in social epidemiology. Am J Epidemiol. 1999;150:113-20.
[135] McMichael AJ. Prisoners of the proximate: loosening the constraints on epidemiology in an age of change. Am J Epidemiol. 1999;149:887-97.
[136] Krieger N. Epidemiology and the web of causation: has anyone seen the spider? Soc Sci Med. 1994;39:887-903.
[137] Galea S. An argument for a consequentialist epidemiology. Am J Epidemiol. 2013;178:1185-91.
[138] Kaplan GA. What's wrong with social epidemiology, and how can we make it better? Epidemiol Rev. 2004;26:124-35.
[139] Glymour MM, Rudolph KE. Causal inference challenges in social epidemiology: Bias, specificity, and imagination. Soc Sci Med. 2016;166:258-65.
[140] Glass TA, McAtee MJ. Behavioral science at the crossroads in public health: extending horizons, envisioning the future. Soc Sci Med. 2006;62:1650-71.
[141] Keyes KM, Galea S. Commentary: The Limits of Risk Factors Revisited: Is It Time for a Causal Architecture Approach? Epidemiology. 2017;28:1-5.
[142] Hernan MA, Robins JM. Instruments for causal inference: an epidemiologist's dream? Epidemiology. 2006;17:360-72.
[143] Hernan MA. A definition of causal effect for epidemiological research. J Epidemiol Community Health. 2004;58:265-71.

[144] Rehkopf DH, Glymour MM, Osypuk TL. The Consistency Assumption for Causal Inference in Social Epidemiology: When a Rose is Not a Rose. Curr Epidemiol Rep. 2016;3:63-71.
[145] Glymour MM, Spiegelman D. Evaluating Public Health Interventions: 5. Causal Inference in Public Health Research-Do Sex, Race, and Biological Factors Cause Health Outcomes? Am J Public Health. 2017;107:81-5.
[146] Glass TA, Goodman SN, Hernan MA, Samet JM. Causal inference in public health. Annu Rev Public Health. 2013;34:61-75.
[147] Lynch J, Smith GD. A life course approach to chronic disease epidemiology. Annu Rev Public Health. 2005;26:1-35.
[148] Fox J. A life course approach to chronic disease epidemiology. BMJ. 1998;317:421.
[149] Whitehead M, Dahlgren G. What can be done about inequalities in health? Lancet. 1991;338:1059-63.
[150] Braveman P, Egerter S, Williams DR. The social determinants of health: coming of age. Annu Rev Public Health. 2011;32:381-98.
[151] Pearce N, Vandenbroucke JP. Causation, mediation and explanation. Int J Epidemiol. 2016;45:1915-22.
[152] Vandenbroucke JP, Broadbent A, Pearce N. Causality and causal inference in epidemiology: the need for a pluralistic approach. Int J Epidemiol. 2016;45:1776-86.
[153] Mukherjea A, Modayil MV. Culturally specific tobacco use and South Asians in the United States: a review of the literature and promising strategies for intervention. Health Promot Pract. 2013;14:48S-60S.
[154] Joo JY. Effectiveness of culturally tailored diabetes interventions for Asian immigrants to the United States: a systematic review. Diabetes Educ. 2014;40:605-15.
[155] Katigbak C, Flaherty E, Chao YY, Nguyen T, Cheung D, Yiu-Cho Kwan R. A Systematic Review of Culturally Specific Interventions to Increase Physical Activity for Older Asian Americans. J Cardiovasc Nurs. 2018;33:313-21.
[156] Kong A, Tussing-Humphreys LM, Odoms-Young AM, Stolley MR, Fitzgibbon ML. Systematic review of behavioural interventions with culturally adapted strategies to improve diet and weight outcomes in African American women. Obes Rev. 2014;15 Suppl 4:62-92.
[157] Kong G, Singh N, Krishnan-Sarin S. A review of culturally targeted/tailored tobacco prevention and cessation interventions for minority adolescents. Nicotine Tob Res. 2012;14:1394-406.
[158] McCurley JL, Gutierrez AP, Gallo LC. Diabetes Prevention in U.S. Hispanic Adults: A Systematic Review of Culturally Tailored Interventions. Am J Prev Med. 2017;52:519-29.
[159] Zeh P, Sandhu HK, Cannaby AM, Sturt JA. The impact of culturally competent diabetes care interventions for improving diabetes-related outcomes in ethnic minority groups: a systematic review. Diabet Med. 2012;29:1237-52.
[160] Campbell R, Starkey F, Holliday J, Audrey S, Bloor M, Parry-Langdon N, et al. An informal school-based peer-led intervention for smoking prevention in adolescence (ASSIST): a cluster randomised trial. Lancet. 2008;371:1595-602.
[161] Singh-Manoux A, Marmot M. Role of socialization in explaining social inequalities in health. Soc Sci Med. 2005;60:2129-33.
[162] Willeboordse M, Jansen MW, van den Heijkant SN, Simons A, Winkens B, de Groot RH, et al. The Healthy Primary School of the Future: study protocol of a quasi-experimental study. BMC Public Health. 2016;16:639.

[163] Adams J, Mytton O, White M, Monsivais P. Why Are Some Population Interventions for Diet and Obesity More Equitable and Effective Than Others? The Role of Individual Agency. PLoS Med. 2016;13:e1001990.

[164] Backholer K, Beauchamp A, Ball K, Turrell G, Martin J, Woods J, et al. A framework for evaluating the impact of obesity prevention strategies on socioeconomic inequalities in weight. Am J Public Health. 2014;104:e43-50.