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The Contribution of Health Behaviors to Depression Risk Across Birth Cohorts

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Background: More recent birth cohorts are at a higher depression risk than cohorts born in the early 20th century. We aimed to investigate to what extent changes in alcohol consumption, smoking, physical activity, and obesity contribute to these birth cohort variations.

Methods: We analyzed panel data from US adults born 1916–1966 enrolled in the Health and Retirement Study (N = 163,760 person-years). We performed a counterfactual decomposition analysis by combining age-period-cohort models with g-computation. We thereby compared the predicted probability of elevated depressive symptoms (CES-D 8 score ≥3) in the natural course to a counterfactual scenario where all birth cohorts had the health behaviors of the 1945 birth cohort. We stratified analyses by sex and race-ethnicity.

Results: We estimated that depression risk of the 1916–1949 and 1950–1966 birth cohort would be on average 2.0% (–2.3 to –1.7) and 0.5% (–0.9 to –0.1) higher with the alcohol consumption levels of the 1945 cohort. In the counterfactual with the 1945 BMI distribution, depression risk is on average 2.1% (1.8 to 2.4) higher for the 1916–1940 cohorts and 1.8% (–2.2 to –1.5) lower for the 1950–1966 cohorts. We find no cohort variations in depression risk for smoking and physical activity. The contribution of alcohol is more pronounced for Whites than for other race-ethnicity groups, and the contribution of BMI more pronounced for women than for men.

Conclusion: Increased obesity levels were associated with exacerbated depression risk in recent birth cohorts in the United States, while drinking patterns only played a minor role.

Keywords: Depression; Health behavior; Body mass index; Birth cohort; Age-period-cohort analysis; Decomposition; Causal inference; Parametric g-formula

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INTRODUCTION

Depression is a common mental disorder and major cause of disease burden worldwide. In the United States specifically, it is estimated that about 5% of the population suffers from major depression,[1] with females disproportionally affected.[2] Depression is prevalent across all ages[1] but is particularly high in older adults due to an accumulation of risk factors, such as multimorbidity, cognitive decline, and loneliness.[3] Depression poses a major public health concern due to the high cost of illness and affects all aspects of an individual's life, including productivity, work performance, and social engagement.[4] Additionally, depression is associated with an increased mortality risk, largely due to an increased risk of suicide.[5]

In population health, it is observed that some generations are healthier than others, independent of age and time period.[6–9] The causes of generational differences vary, ranging from early life exposure to health determinants, such as famine, to overall variations in health behavior over the life course (e.g., smoking prevalence).[6,7,10] Understanding of birth cohort differences is crucial in assisting health policy making and predicting trends for future generations.[11]

Regarding depression, more recent birth cohorts have higher levels of depression, psychological distress, and worse mental health compared with birth cohorts born earlier in the 20th century.[12–18] Although some studies identify an overall increase in depression prevalence across birth cohorts,[13–16] others find an increase in depression only for birth cohorts born during or after 1935–1945.[12,18]

These cohort variations in depression risk could be partly explained by cohort differences in health behavior-related determinants of depression.[12,14,19] Indeed, more recent birth cohorts are more likely to be obese,[20] have higher physical activity levels,[21] and have decreased alcohol

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The Health and Retirement Study data is accessible at (http://hrsonline.isr.umich.edu/). The R code for reproducing our analysis can be requested from the corresponding author.

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consumption- and smoking-related mortality than earlier born cohorts.[6,22] Alcohol abuse, smoking, and obesity are associated with increased depression risk, whereas physical activity is in the protective direction.[23–26]

The aim of this study was to assess to which extent changes in health behavior-related determinants of depression, that is, alcohol consumption, smoking, physical activity, and obesity, explain the link between birth cohort and depression risk.

DATA AND METHODS

Data Source

For our analysis, we used the 2016 RAND HRS Longitudinal File of the Health and Retirement Study (HRS). The HRS is a nationally representative longitudinal survey based in the United States and started in 1992 with biannual follow-up interviews ever since. It comprises data on over 37,000 adults over the age of 50 years and their spouses.[27] The HRS data is sponsored by the National Institute on Aging (grant number U01AG009740) and is conducted by the University of Michigan. Ethical approval was obtained from the University of Michigan Institutional Review Board.

We excluded observations from wave 1, as reporting of the outcome measure changed from wave 2 onwards (eFigure 1; http://links.lww.com/EDE/B951). Proxy respondents were excluded because the outcome measure was not administered.[28] We furthermore excluded observations with an age below 50 and above 80 years due to data scarcity at the extremes of age. After exclusion of nonrespondents and ineligible respondents, we identified missing observations for 6.2% of the outcome depressive symptoms, 0% of the key covariate birth year, 0.5–8.8% of the health-behavior variables and for up to 0.1% of the confounders (sex, race-ethnicity, education). The majority of missingness of health-behavior variables is due to alcohol consumption, which was not measured in wave 2. Exclusion of wave 2 resulted in missingness of 0.2–1.5% for health behavior variables. We therefore assumed missingness at random and performed a complete-case analysis for 34,542 persons and 163,760 person-years.

Outcome

Information on depressive symptoms was assessed with the eight-item Center for Epidemiological Studies—Depression scale (CES-D 8). The CES-D 8 includes dichotomous questions on six negative and two positive items and results in a score from 0 to 8 with a higher score indicating higher depressive symptomatology.[28] We used a CES-D score of ≥3 as an indicator for elevated depressive symptoms.[28] The CES-D 8 was validated in older adults in the United States.[29]

Measurement of Exposure, Health Behavior, and Confounders

Information on the exposure birth year, age, health behaviors (alcohol consumption, smoking, physical activity, height, and weight) and confounders (sex, race-ethnicity, education level) was collected through face-to-face or telephone interviews. Body mass index (BMI) was calculated based on self-reported height and weight of the respondents (weight(kg)/(height(m))^2) and categorized into underweight (<18 kg/m²), normal weight (18–<25 kg/m²), overweight (25–<30 kg/m²), or obese (≥ 30 kg/m²). We categorized smokers as current smoker or nonsmoker. Alcohol consumption was reported in drinks per day and categorized into nondrinker, moderate drinker (1 drink/day for females, 1–2 drinks/day for males), heavy drinker (2–3 drinks/day for females, 3–4 drinks/day for males), or excessive drinker (≥4 drinks/day for females, ≥5 drinks/day for males). We defined physical activity as performing vigorous physical activity three or more times per week (yes/no). Education level was categorized into less than high-school degree, general education diploma, high-school graduate, some college, and college and above.

Statistical Analysis

We performed our analyses in the total sample and stratified by sex (male/female) and by race-ethnicity (White/Black/Hispanic). To account for the oversampling of Hispanics and Blacks, we applied poststratification weights provided by the HRS in descriptive graphs.[30]

To determine the presence of birth cohort patterns in depression, we used an age-period-cohort (APC) model to investigate the associations between birth cohort and depression. We specified the following logistic regression model to estimate the probability of elevated depressive symptoms (depr) as a function of age, period, and cohort:

$$Logit\,(depr) = ns\,(age, a) + ns\,(period, p) + ns\,(cohort, c) \quad (Eq.\ 1)$$

where ns refers to a natural cubic spline function, which allows for nonlinear patterns of age, period, and cohort. One characteristic of APC models is the identification problem, which describes the collinearity between the age, period, and birth cohort dimension ($Age = Period - Cohort$). We addressed this problem with the Carstensen approach[31] and detrended the period dimension by replacing the part of the design matrix corresponding to period with a matrix with columns orthogonal to the intercept and period drift column.[31] The reference group was defined as age 50, 1996 for calendar year and 1945 for birth cohort.

To address our main objective of analyzing the contribution of health behaviors to depression risk across birth cohorts, we performed a counterfactual decomposition. The assumed directed acyclic graph can be found in Figure 1. We performed the g-computation based causal decomposition for every health behavior (mediator), using an approach that is described in detail by Sudharsanan & Bijlsma.[32]

First, we specified the probability of elevated depressive symptoms as our measure of interest, and the relative difference between each birth cohort and the birth cohort with the

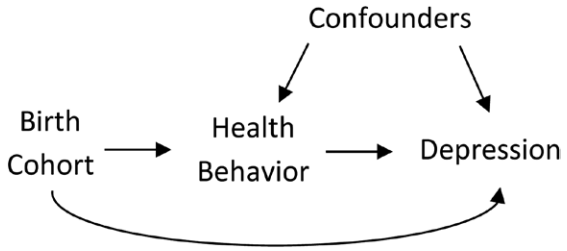

FIGURE 1. Assumed causal directed acyclic graph. We note that birth cohorts are nonmanipulable; health behaviors and the outcome are clustered within birth cohorts.

lowest probability of depressive symptoms in our sample, that is, the 1945 birth cohort, as our contrast.

Second, we fitted a logistic regression model with the probability of depressive symptoms as a function of age, period, birth cohort and the each of the health behaviors. Additionally, we modeled each respective health behavior as a function of age, period, and cohort. The model specifications of age, period and cohort were identical to the APC model specifications in *Equation 1* for both the outcome and mediator model. We used logistic regression for binary mediators and multinomial logistic regression for categorical mediators. Both the outcome and the mediator models were adjusted for education, sex and race-ethnicity in the total sample, education and race-ethnicity in sex strata and education and sex in race-ethnicity strata.

Third, we formed our natural course (nc) and counterfactual (cf) pseudo-populations. Mediator values were simulated by randomly sampling from either a binomial distribution or a multinomial distribution, depending on the mediator type, based on the predictions of the specified mediator model. We allowed the confounder distribution to vary between birth cohorts. We then predicted the probability of elevated depressive symptoms for each birth cohort based on the health behavior (mediator) and confounder distribution of each birth cohort, using the following formula:

$$Depr_{nc}: E\left[depr^{B}\left(M \sim f_{M}^{B}\right)\right] = \sum_{C}\sum_{M \in f_{M}^{B}} E[depr|M=m, C=c, B] * P[M=m|C=c, B] * P[C=c, B] \quad (Eq.2)$$

where $depr^{B}$ refers to the predicted probability of elevated depressive symptoms for each birth cohort (B), M indicates the mediator (alcohol consumption, smoking, physical activity, or BMI) with f_{M}^{B} representing the distribution of mediator values by birth cohort, and C marks the confounding factors (sex, race-ethnicity, education).

In the natural course scenario, each cohort gets its own observed health behaviors. However, in the counterfactual scenario, each cohort's health behaviors are taken from the 1945 birth cohort. The confounder distribution is not changed in the counterfactual:

$$Depr_{cf}: E\left[depr^{B}\left(M \sim f_{M}^{B_{1945}}\right)\right] = \sum_{C}\sum_{M \in f_{M}^{B_{1945}}} E[depr \mid M=m, C=c, B] * P[M=m \mid C=c, B_{1945}] * P[C=c, B] \quad (Eq.3)$$

We analyzed the impact of the counterfactual by calculating the relative difference in predicted probabilities as $\frac{Depr_{cf}}{Depr_{nc}} - 1$. We furthermore calculated the contribution of each mediator to the difference with respect to the 1945 birth cohort as $1 - \frac{Depr_{cf} - Depr_{1945}}{Depr_{nc} - Depr_{1945}}$.

The contribution can be interpreted as "How much does each health behavior attribute to a change in the probability of elevated depressive symptoms if every cohort had the health behavior distribution of the 1945 cohort?"

We sampled from probability distributions, which results in Monte Carlo error. We performed Monte Carlo error reduction by repeating the simulation steps 50 times and averaging over their estimates. Furthermore, we performed 499 bootstrap iterations to compute 95% confidence intervals (see eAppendix section 4.1; http://links.lww.com/EDE/B951). We calculated the mean relative difference and contribution aggregated by birth cohorts using inverse variance weighting.[33]

RESULTS

Sample Characteristics

Participants are on average 65 ± 8 years old. Most participants are female (58%), White (77%), nondrinkers (65%), and nonsmokers (84%). About half (45%) report vigorous physical activity and about one-third (32%) of participants are obese. Females have a higher percentage of elevated depressive symptoms than males. Both sexes are comparable in their age and birth cohort but differ in their health behavior distributions (Table). Characteristics by race-ethnicity can be found in eAppendix section 1; http://links.lww.com/EDE/B951.

Birth Cohort Patterns in Elevated Depressive Symptoms

Figure 2 shows the prevalence of elevated depressive symptoms by birth year for 10-year age groups. We find an overall decrease in the prevalence for cohorts 1916–1946 within age groups 61–70 and 71–80, with a more pronounced decrease in ages 71–80. In contrast, within age group 50–60, we find an increase in the prevalence of elevated depressive symptoms from 1932 to 1966. In subgroup analysis, we find a higher prevalence of elevated depressive symptoms across most cohorts in females, Hispanics, and Blacks, compared with males and Whites respectively. We furthermore observe a narrowing of the sex gap in depression prevalence across birth cohorts (Figure 3). Descriptive plots by race-ethnicity can be found in eAppendix section 1; http://links.lww.com/EDE/B951.

We predicted the birth cohort patterns based on the specified APC model and find similar cohort patterns as in descriptive Figure 2. In short, individuals born before 1920

TABLE. Sample Characteristics of the Total Sample

	Total	Females	Males
N, person-years	16,376	95,451	68,309
Outcome			
Elevated depressive symptoms, yes, N(%)	35,788 (22)	23,907 (25)	11,881 (17)
Exposure			
Age, mean (SD)	65 (8)	65 (8)	65 (8)
Period, mean (SD)	2006 (6)	2006 (6)	2006 (6)
Birth cohort, mean (SD)	1942 (10)	1942 (10)	1941 (10)
Mediators			
BMI, N (%)			
Underweight	1,455 (1)	1,166 (1)	289 (0.4)
Normal	46,892 (29)	30,572 (32)	16,320 (24)
Overweight	62,484 (38)	31,515 (33)	30,969 (45)
Obese	52,929 (32)	32,198 (34)	20,731 (30)
Alcohol consumption, N (%)			
Nondrinker	105,684 (65)	68,333 (72)	37,351 (55)
Moderate drinker	34,865 (21)	14,307 (15)	20,558 (30)
Heavy drinker	18,693 (11)	11,449 (12)	7,244 (11)
Excessive drinker	4,518 (3)	1,362 (1)	3,156 (5)
Smoking, no, N(%)	137,277 (84)	80,755 (85)	56,522 (83)
Vigorous physical activity, yes N (%)	74,389 (45)	38,002 (40)	36,387 (53)
Confounders			
Sex, female, N(%)	95,451 (58)	–	–
Race-ethnicity, N(%)			
White	125,446 (77)	72,032 (75)	53,414 (79)
Hispanic	5,699 (4)	3,181 (3)	2,518 (4)
Black	28,047 (17)	17,672 (19)	10,375 (15)
Other	4,568 (3)	2,566 (3)	2,002 (3)
Education level, N (%)			
Limited high-school	33,160 (20)	19,873 (21)	13,287 (19)
GED	8,155 (5)	4,342 (5)	3,813 (6)
High-school graduate	49,373 (30)	31,164 (33)	18,209 (27)
Some college	37,911 (23)	22,559 (24)	15,352 (22)
College and above	35,161 (22)	17,513 (18)	17,648 (26)

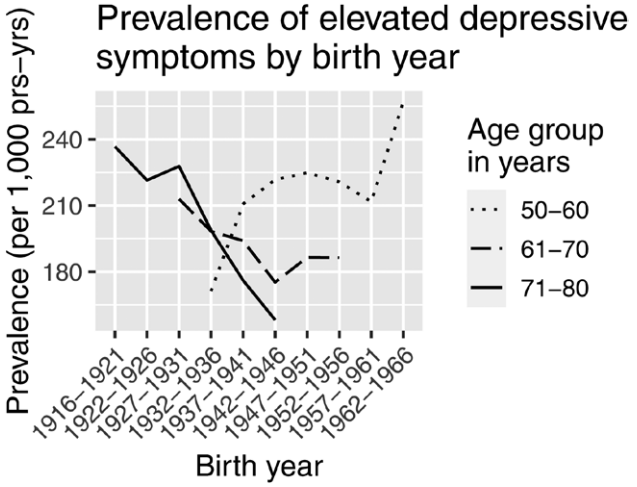

FIGURE 2. Prevalence of elevated depressive symptoms per 1,000 person-years by 5-year birth year groups for 10-year age groups. Cohorts with n < 5 were excluded.

and after 1950 show a higher probability of elevated depressive symptoms compared with birth cohorts born between those years, when age and period are held constant. The description of age and period effects, and subgroup analysis by sex and race-ethnicity can be found in eAppendix section 1; http://links.lww.com/EDE/B951.

Counterfactual Decomposition of Health Behavior-related Determinants of Depression

Our mediator models closely approximate the empirically observed health behavior patterns across cohorts. In the natural course (standardized for age and period), over cohorts, alcohol consumption and BMI increases, and smoking and physical activity show only small variations. For the counterfactual, we assign the health behavior distribution of the 1945 cohort to every cohort and set the prevalence of heavy and excess drinking to 15%, smoking prevalence to 26%, prevalence of no vigorous physical activity to 44% and obesity

prevalence to 28% (eAppendix section 2; http://links.lww.com/EDE/B951).

Figure 4 shows the relative difference in the probability of elevated depressive symptoms between the counterfactual and natural course scenario for all health behavior factors.

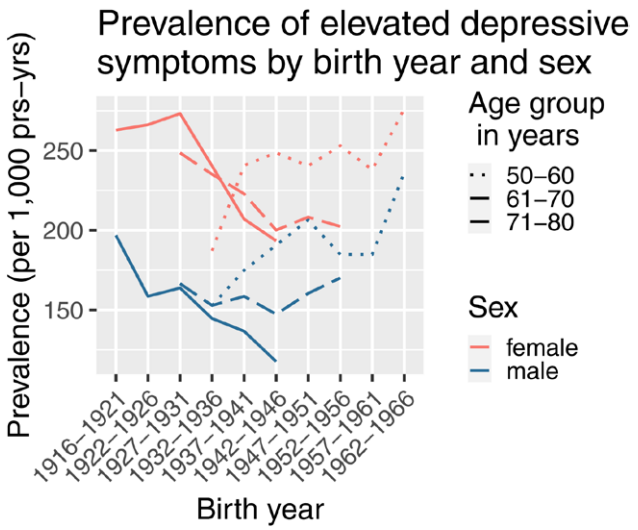

FIGURE 3. Prevalence of elevated depressive symptoms per 1,000 person-years by 5-year birth year groups and sex for 10-year age groups. Cohorts with n < 5 were excluded.

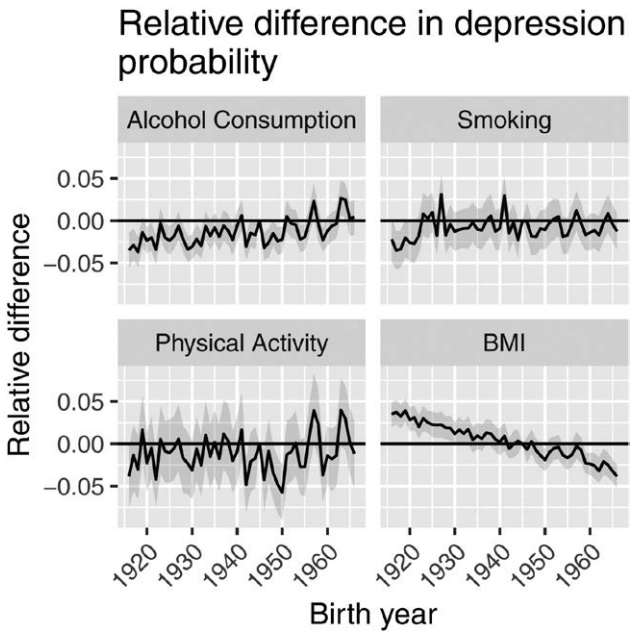

FIGURE 4. Relative difference (95% CI) between the counterfactual and natural course estimates of probability of elevated depressive symptoms by birth cohort for each health behavior factor. Relative differences are calculated as counterfactual/natural course-1. Positive/negative values indicate that if the cohort had the behavioral profile of the 1945 cohort, their risk of depression would have been higher/lower.

After the alcohol consumption distribution is set to that of birth cohort 1945, we find an average reduction in elevated depressive symptoms of 2.0% (–2.3 to –1.7) for birth cohorts 1916–1949 and 0.5% (–0.9 to –0.1) for cohorts 1950–1966. For physical activity and smoking, we do not find cohort variations in the relative differences in depression risk. After we counterfactually set all cohorts to have the BMI distribution of the 1945 cohort, the probability of elevated depressive symptoms increases on average by 2.1% (1.8 to 2.4) for cohorts born before 1940 and decreases on average by 1.8% (–2.2 to –1.5) for cohorts born 1945–1966.

To assess what fraction of cohort variations in depression risk are explained by health behavior, we calculated the contribution (eAppendix Section 2; http://links.lww.com/EDE/B951). Alcohol consumption contributes on average 7.5% (7.5 to 7.6) to the probability of elevated depressive symptoms in cohorts born 1916–1949 and 1.9% (1.8 to 2.0) in cohorts born 1950–1966. We do not identify cohort patterns in the contribution of smoking and physical activity. BMI contributes on average –7.7% (–7.8 to –7.6) to depression risk for cohorts born before 1940 and 5.5% (5.4 to 5.5) for cohorts born after 1948.

Subgroup Analysis

The subgroup analysis revealed sex-specific differences for BMI only (Figure 5; eAppendix Section 2; http://links.lww.com/EDE/B951). After counterfactually setting BMI to the 1945 distribution, the probability of elevated depressive symptoms increases for females, but not males, born before 1940 by an average of 3.5% (3.1 to 3.8) and decreases for female cohorts born after 1950 by 2.4% (–2.8 to –2.0).

For females born before 1940 and after 1950, we estimate average contributions of BMI of –12% (–12 to –11) and 6.8% (6.7 to 6.9), respectively. We find no contributions in males.

The relative difference of alcohol consumption, physical activity and BMI differs by race–ethnicity (eAppendix section 2.1; http://links.lww.com/EDE/B951). For alcohol consumption and BMI, adopting the counterfactual health behavior distributions results in a larger change in elevated depressive symptoms in Whites, compared with Hispanics and Blacks across all cohorts. In Hispanics born 1916–1935, adopting the 1945 physical activity distribution decreases the probability of elevated depressive symptoms on average by 9.8% (–11 to –9.0).

For alcohol consumption, we estimate larger contributions to depression risk in Whites, followed by Blacks and Hispanics across all cohorts (eAppendix Section 2; http://links.lww.com/EDE/B951).

Sensitivity Analysis

We repeated the analysis for smoking as a categorical variable (never, former, or current smoker), which did not meaningfully affect the decomposition results (eAppendix Section 4.2; http://links.lww.com/EDE/B951).

We assessed the sensitivity of our decomposition results to the underlying APC model constraints by calculating the relative

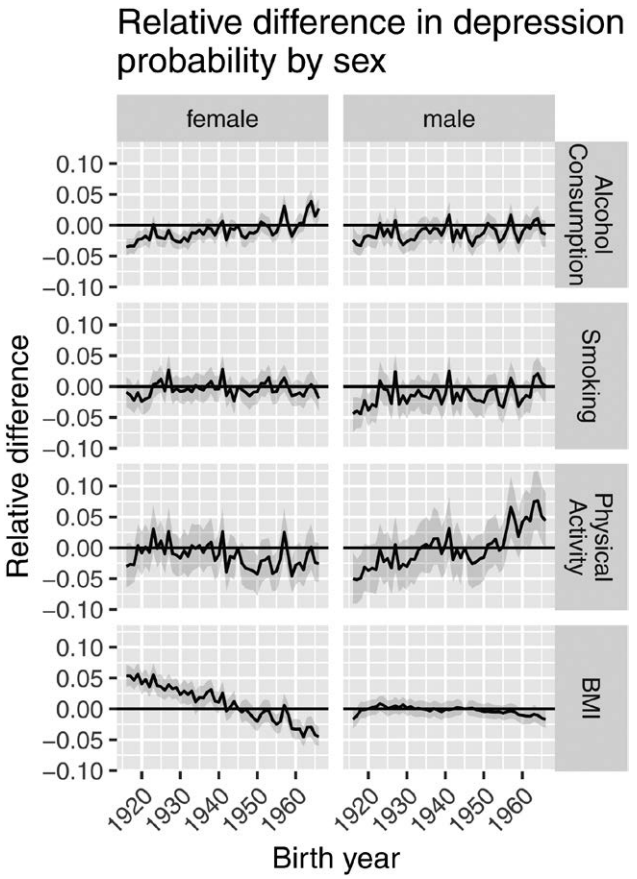

FIGURE 5. Relative difference (95% CI) between the counterfactual and natural course estimates of probability of elevated depressive symptoms by birth cohort by sex for each health behavior factor. Relative differences are calculated as counterfactual/natural course-1. Positive values indicate that the counterfactual increases depression risk and negative values indicate that the counterfactual decreases depression risk.

difference for three sets of model constraints: (1) Drift (the linear time trend) is assigned to period (instead of the cohort dimension); (2) Drift is assigned to period and the reference is set to the 1996 period instead of 1945 cohort; (3) Drift is assigned to cohort and the reference is set to the 1996 period. For BMI, model constraint (2) results in a relative difference that follows the same linear downward trend as the results in Figure 4. For model constraints (1) and (3), however, we do not find a linear downward trend of BMI. We reflect on this in the discussion. The counterfactual decomposition of alcohol consumption, smoking or physical activity is not sensitive to changes in model constraints. Results from the sensitivity analysis can be found in eAppendix Section 3; http://links.lww.com/EDE/B951.

DISCUSSION

Summary of Results

The aim of this study was to investigate to what extent birth cohort differences in depression risk can be explained by health behavior with the use of a combination of age-period-cohort analysis and counterfactual decomposition analysis. Depression risk in recent birth cohorts would have been lower for females and Whites if obesity prevalence had not increased past 1945. In contrast, alcohol consumption was a more important risk factor for depression in cohorts born before 1950 than in more recent birth cohorts. We estimated alcohol consumption to be a stronger contributor to depression risk in Whites compared with Hispanics and Blacks across all cohorts. We did not find evidence for contributions of smoking and physical activity.

The Link Between Birth Cohort, Health Behavior, and Depression

For BMI, we estimate that recent cohorts benefit from the counterfactual scenario with an average decrease in the probability of elevated depressive symptoms of 2% after the prevalence of obesity is reduced by approximately 7%-points and reassigned to normal weight and overweight. In cohorts born before 1940, depression risk increases on average by 2.1% after the prevalence of normal weight is reduced by 10%-points and reassigned to obesity. These results are consistent with the hypothesis that obesity in particular is a driver of depression risk, with higher depression in more obese populations.

Sex-stratified analyses show that the contribution of obesity to depression risk is strong for women but negligible for men across most birth cohorts. This is likely due to cohort patterns of obesity being more pronounced in females[20,34] and that obesity increases depression risk for females but not for males.[35,36] Females experience more weight stigmatization than males across various life domains which in turn affects their mental health negatively.[37,38] Therefore, it is worth considering that the mental health of women and girls may have particularly suffered from the increase in obesity levels in the United States.

The sensitivity analysis in which we assigned drift to period rather than cohort showed that a large part of the trend in the contribution of BMI to depression risk is attributed to the overall time trend (eAppendix section 3; http://links.lww.com/EDE/B951). Previous research indicates that environments are becoming more obesogenic over time,[39] which is indicative of period effects. Reither et al.[34] on the other hand find cohort patterns independent of period and age, with recent cohorts being more susceptible to obesity. Hence, recent birth cohorts might be more strongly affected by increases in obesogenic forces than cohorts born earlier in the twentieth century. We interpret this as evidence for a combined effect of period and cohort. Regardless, the conclusion remains the same: over time, as the population became more obese, the prevalence of elevated depressive symptoms in recent birth cohorts is on average 5.5% attributable to this increase.

For alcohol consumption, we estimate that, in cohorts born before 1950, the counterfactual scenario decreases the

number of nondrinkers and excess drinkers on average by 4%-points and 0.3%-points, respectively, and assigns them to either moderate or heavy drinkers. This leads to an average decrease in the probability of elevated depressive symptoms of 2.0%. These results imply that lower percentages of nondrinkers in the population decrease depression risk. This might be partially explained by the fact that nondrinkers are more likely to be older and in turn suffer from chronic conditions or take medication that require them to abstain from alcohol.[40] Furthermore, nondrinkers are more likely to have a smaller social network,[40] which may in turn increase their depression risk.

For physical activity and smoking, we do not find a consistent mediating effect on birth cohort patterns of depression. These null findings are explained by the small variation of the mediator distribution between the estimated natural course and counterfactual scenario, rather than no effect of these mediators on depression risk (eAppendix Sections 2.2 and 2.3; http://links.lww.com/EDE/B951).

Though previous research suggests that lifestyle changes might explain increased depression risk in recent cohorts,[41] our study finds only small contributions of alcohol consumption, physical activity, smoking, and BMI. Another explanation for cohort variations in depression risk is that normal responses to sadness are more often misclassified as mental disorders in recent cohorts.[14,41] However, longitudinal studies do confirm a true rise in depression prevalence across time and cohort that cannot be attributed to differences in reporting only.[41] Instead, the increased depression risk in recent cohorts might be explained by the simultaneous rise in other noncommunicable diseases, leading to increased multimorbidity and changes in social milieu, leading to higher levels of competition, inequalities and loneliness.[41]

Age-Period-Cohort Model Results

Our analysis suggests that birth cohorts 1916–1920 and 1946–1966 experience an increased depression risk compared with birth cohorts 1921–1945, independent of age and period. These results are partly in line with current literature that found recent birth cohorts to experience a higher risk of depression.[12–18] In contrast, we also identified a higher depression risk in cohorts born in the early 20th century. Keyes et al.[19] identified a similar trend in cohort patterns of psychological distress in US adults for cohorts born 1912–1975, with increased psychological distress for most recent cohorts and cohorts born in the early twentieth century. Hence, our results are in line with the results by Keyes et al. for the cohorts available in our HRS sample (1916–1966).

The decline in depression risk for cohorts born 1916–1946 might be because the cohorts born 1916–1927 (part of the generation known as the "greatest generation") came of age during the great depression and constituted the main ages of military service during World War II. Cohorts 1928–1946 (also known as the "silent generation"), however, came of age in the post-World War II period.[42] Hence, the circumstances that the cohorts grew up in might explain why we see this decline in depression over cohorts 1916–1946 in age group 71–80.

Evaluation of Data and Methods

Our sample is drawn from the Health and Retirement study, which is representative of US older adults born 1916–1966.[27] Since the HRS is a longitudinal survey, we investigated the possibility of panel attrition and panel conditioning bias[43] and conclude that panel attrition might be present in the survey. More depressed individuals might be more likely to leave the study (eAppendix Section 4.3; http://links.lww.com/EDE/B951). We repeated the main analysis including only first observations of each study participant, to investigate how panel attrition could affect our conclusions. Even though the presence of panel attrition could potentially lead to an underestimation of depression prevalence within birth cohorts, we found that the main results are not meaningfully affected by panel attrition.

Our outcome variable measures the presence of elevated depressive symptoms, which can be interpreted as an increased risk for depression rather than a direct measure of major depressive disorder.[44] We may, therefore, overestimate the number of individuals suffering from a depressive disorder. However, populations with elevated depressive symptoms can be considered a target for primary prevention of depression.

In terms of mediators, self-reported measures of health behavior might suffer from misclassification bias, for example, due to social desirability of underreporting health behaviors that are perceived as unhealthy and overreporting behaviors that are perceived as healthy.[45,46] We performed a sensitivity analysis which shows that this misclassification bias does not affect our main results (eAppendix Section 3; http://links.lww.com/EDE/B951).

Our APC model specification follows the Carstensen approach to address the identification problem. First, in this approach, the drift (the linear time trend) is assigned to either cohort or period, a choice that is not empirically testable. We include the drift into the cohort dimension with the rationale that depression is more likely to be an accumulation of experiences shared by birth cohorts over their development, rather than a period effect.[47,48] Second, our sensitivity analysis reveals that the results of the counterfactual decomposition of BMI cannot be accounted to nonlinear cohort or period effects, but rather to the drift. This highlights an important limitation of the APC model: The current approaches that address the identification problem can attribute nonlinear effects to each dimension, but not the total effects. Third, in our analysis, we aim to draw conclusions on population-level groups, that is, birth cohorts, though using individual-level longitudinal data. Therefore, to correct our standard errors,[49] we include a nonparametric bootstrap for clustered data, where observations are treated as clustered within

individuals.[50–52] As a sensitivity analysis, we also performed our analysis using a method that separates within-individual and between-individual heterogeneity[16] and conclude that our results are not strongly affected by it (eAppendix Section 3; http://links.lww.com/EDE/B951).

Our causal decomposition relies on three core assumptions: exchangeability (no unmeasured confounding), positivity, and consistency.[32] An advantage of causal decomposition is that only the mediator–outcome pathway (e.g., BMI-depression) can be confounded, but not the pathways from exposure to mediator or outcome because the exposure (birth cohort) is a group identifier.[32] Regarding our mediator–outcome pathway, unmeasured confounding might be present due to social or genetic factors. We controlled for education, which may not adjust for all possible confounders, resulting potentially in an overestimation of the contribution of health behavior to depression.

Positivity requires that it must be possible for individuals in all strata that are intervened on to be exposed or unexposed. A violation of random positivity occurs if, for example, no one is obese in some strata simply by chance, but not because it is impossible for individuals in those strata to be obese. We find a violation of random positivity in birth cohorts 1916–1919 and 1921 for physical activity in Hispanics due to limited sample size and assume that the effect is transportable from other cohorts to these cohorts. A deterministic violation of positivity occurs if, for example, in some birth cohorts, it is not possible for individuals to be obese. Such violations are unlikely as all birth cohorts should be able to experience all levels of the studied health behaviors.[53]

Consistency requires that the intervention be well-defined and that there is no interference between units.[54] Our BMI intervention cannot be classified as a well-defined intervention because BMI cannot be described as a treatment but rather as a summary outcome of many different treatments.[55] Hence, the BMI intervention could affect depression risk differently depending on how the change in BMI distribution takes place. Investigating these differential effects would add valuable information, but we believe that our conclusion that the effect of a BMI intervention on depression risk differs across birth cohorts would not be strongly affected. For alcohol consumption and physical activity, the effect of the intervention may differ if some cohorts have different compositions of those factors. For example, if some cohorts consume different type of alcohol or define the presence of vigorous physical activity levels differently than others. The duration of smoking and obesity may vary across cohorts,[56] thus potentially violating consistency.

Interference could be present for participants with partners: A health behavior intervention might motivate participants immediate surroundings to also change their health behavior; and for health behaviors with strong externalities, in particular smoking and alcohol consumption, reductions may have positive effects on partners' health.[57] We do not expect this to differ by birth cohorts and therefore do not expect to see strong consequences of interference in our analysis.

Another point of interest is the potential bidirectionality of the relationship between the mediators and the outcome.[25,58–61] For example, although alcohol consumption can be a cause of depression, depression may also cause increased alcohol consumption. We did not model this bidirectionality, which may result in an overestimation of the causal effect of the mediators on the outcome. Bidirectionality can be modeled with lag effects or the longitudinal g-formula. This would additionally allow to account for possible long-term effects of health behaviors on depression risk, though we argue that most of this long-term effect is captured by present health behavior. Although including lag effects would allow for a more accurate causal effect estimation, this would have resulted in exclusion of the oldest and youngest birth cohorts in our analysis.

CONCLUSION

To our knowledge, this is the first study to investigate possible causes of birth cohort variation with the use of models embedded in the causal inference (potential outcomes) framework, specifically g-computation. Recent birth cohorts are at a higher depression risk than cohorts born in the early twentieth century and we provide insight into reasons for these birth cohort variations: we estimate that BMI contributes on average 5.5% to depression risk in recent cohorts, whereas alcohol consumption is a more important risk factor in cohorts born before 1950, with average contributions of 7.5%. Our results highlight the possibility that obesity levels in recent female cohorts in particular might play a role in depression risk in the United States. Future research should investigate causes of generational differences in depression risk in cohorts born after 1966 and consider the bidirectionality between health behavior and depression.

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