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Depressive symptoms in the Belgian population: disentangling age and cohort effects

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Abstract
Objective Although the association between age and depression has been previously demonstrated, uncertainty remains because of the confounding relationship existing between age and cohort. A study by Yang (J Health Soc Behav 48(1):16, 2007) has evidenced important cohort effects and age-by-cohort interactions in depressive symptoms among US citizens. A crucial limitation, however, is that this study confines itself to elderly population. The objective of the present study is to bring further clarification to the association between age, cohort membership and depressive symptoms, by analyzing a sample with a wider age range.
Methods The Panel Study of Belgian Households is a prospective longitudinal survey, following adults ages 25–74, annually from 1992 to 2002. Missing data were replaced using multiple imputation, allowing for a complete dataset ($N = 7{,}000$) at each wave. Respondents were classified into one of five birth cohorts: 1918–1927; 1928–1937; 1938–1947; 1948–1957; 1958–1967. Frequency of depressive symptoms was reported using a modified version of the Health and Daily Living form. Growth curve modeling was used to determine the effect of age and cohort on depression trajectory.
Results All cohorts differed significantly from one another, with recent cohorts always obtaining the highest mean HDL-depression score. The intensity of depressive symptoms increases linearly with age, but significant age-by-cohorts interactions were detected, indicating that the relationship between age and depression varies across cohorts. No evidence of a WW2 effect was found.
Conclusion The association between age and depression has to take cohort membership into account. Cohort replacement effects explain the increase in depression in Belgium.

Keywords Depressive symptoms · Age effects · Cohort effects · Growth curve modelling · Belgium

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Introduction

Major depression, characterized notably by symptoms of sadness, depressed mood, loss of interest and irritability is an important factor of disability [1]. It is also one of the most prevalent mental disorders worldwide [2, 3], with an estimated 12-month prevalence similar in both developed (5.5%) and developing countries (5.9%) [4].

Depressive symptoms among adolescents and adults were reported to increase over time [5–7], but their trajectory over the life-course is still unclear. Although the association between age and depression was previously studied, uncertainty remains because of the confounding relationship existing between age and cohort [8–10]. Yang [11] made an attempt to solve this issue, and evidenced important cohort effects and age-by-cohort interactions in depressive symptoms among US citizens. A crucial

limitation, however, is that Yang's [11] study confines itself to an elderly population (over the age of 65).

The objective of the present study is to bring further clarification to the association between age, cohort membership and depressive symptoms. To disentangle age and cohort effects, we analyze data from a longitudinal study among the Belgian population, covering a wide age range (from 25 to 74 years old). Respondents ($N = 7{,}000$), grouped into five birth cohorts covering more than half of the twentieth century, were surveyed annually over 11 years. Growth curve modeling was used to study cohort differences on the age-trajectory in depression. Missing data were replaced using multiple imputation.

Age and depression

Depression is expected to vary throughout the life-course of individuals, as ageing is characterized by important biological, psychological, and social changes. According to Mirowsky and Ross [12] five hypotheses (age as maturity, as decline, as stage, as survival, and as historical trend) could explain either the increase or the decrease of depression as people grow older. Other explanations for the age differences in depression are selective mortality and migration, changes in diagnostic criteria, in professional attitude, and in the cultural meaning of depression [13].

Previous research on the association between age and depression shows contradictory patterns [13, 14]. A review of epidemiological studies on general populations published prior to 2000 concludes that most studies, having controlled for some risk factors (civil, employment, and socio-economic statuses, level of education, physical disabilities, etc.), reported a decline in depression with ageing [14]. There is also more recent empirical evidence confirming this trend [15–17], but only in developed countries [4]. Inversely, depression was observed to increase over the life-course. Some studies have identified a U-shaped trajectory over the life-course, with the lowest score in middle age, and the highest in older age [12]. Others have observed a linear increase of mean depressive symptoms score and of the prevalence of depression with ageing in a general population (20–89 years old) [18], even after controlling for confounding factors including physical illness [19].

These contradictory findings may reflect major limitations of previous research, notably their cross-sectional nature and the neglect of cohort effects. In cross-sectional studies, differences in depression related to age are often wrongly interpreted as caused by the process of ageing, while they should rather be related to cohort differences in ageing [9].

Cohort membership, age, and depression

While age effects refer to changes that result from the biological, psychological or social components of age, and which transcend the cohort membership or the historical period, cohort effects refer to changes that are associated with being a member of a particular group, and that persist despite the ageing of its members and the historical period. A cohort is a group of individuals sharing an initial event together, the most common being a birth year [10]. Each cohort is unique and differs from the others, notably in its demographic composition, attitudes, values, beliefs, and behaviors. Cohorts might even differ in the way they age [8, 10]. In this perspective, each cohort differs by the age at which their members enter a social role, accomplish some key life-course events (diploma, marriage, parenthood, etc.), and experience specific historical events.

The cohort differentiation process results from the fact that each cohort experienced specific historic conditions during the formative period of their life. Important changes in society, notably in education, politics, life conditions, socialization, norms, and technological innovation played a crucial role in this regard [8, 10]. Inter-cohort differences towards depression are expected, because of social transformations that have shaped the twentieth century [20], notably with regard to the occurrence of individualism [21].

Evidence from accelerated longitudinal studies, in which age and cohort effects were disentangled, underline the importance of cohort membership in the problematic of depression. Despite some exceptions [22, 23], the succession of cohorts is associated with a higher level of depressive symptoms [6, 11, 20, 24, 25], a greater prevalence of depression [13, 26, 27], and a lower health status related notably to depression [28]. In other words, the net cohort effect reveals that recent cohorts have a greater level of depression than previous cohorts.

Reporting the results of epidemiological studies on temporal trends in depression, Klerman and Weissman [25] noted a distinction between cohorts born before or after the Second World War (WW2). Findings from the US, Canada, Sweden, Germany, New Zealand and Belgium revealed an increase in the prevalence of major depression in cohorts of people born after the war [24, 25]. This "age of melancholia" [13] was also confirmed by studies using small samples limited to mothers [28, 29]. Although the impacts of WW2 on society and on cohort experience are well documented [30], its effects on depression were not confirmed by all studies. Instead, and contrary to the cohort succession's findings, Robert et al. [23] observed that older cohorts, especially those born before the turn of the twentieth century, had a higher prevalence of depression

than younger cohorts. However, this study is limited by the birth year of the recent cohorts (no later than 1949).

To our knowledge, the results of the few studies that have investigated the association between age and depression, net of cohort effects, are contradictory. It had first been found that the prevalence [23] and the intensity [11, 20] of depressive symptoms increase with age. However, in the latter studies, the direction of the trajectory became negative, when health status (mainly chronic illness and physical disabilities) was controlled for [11, 20]. This led the intensity of depressive symptoms to decrease with age.

The absence of a clear pattern also emerges when observing age-by-cohort interactions. The inclusion of distinct measurements and different birth cohorts lead to diverse trajectories in depression over the life-course. As an example, while the prevalence of depression was reported to increase with age for all cohorts [25, 27], the intensity of depressive symptoms decreased throughout the life-course [11, 20], but not for all cohorts [29]. Inter-cohort differences are also observed in the speed of the trajectory: some recent cohorts were characterized as having either a slow [11], or a fast decline [20, 29] over age, but the older one experienced a stability or an increase in depression over the life-course [29]. Furthermore, some evidence points toward a decrease in the age of onset of depression with the succession of cohorts [13, 25, 27].

Hypotheses

Based on previous research, we have the following expectations with respect to the disentangling of age and cohort effects on the intensity of depressive symptoms:

1. A net age effect characterized by an increase in the intensity of depressive symptoms over the life-course. Thus, people are expected to have a higher intensity of depression as they grow older. However, controlling for health status and the presence of physical illness could change the direction of the relationship.
2. A net cohort effect characterized by differences between cohorts in respect of the intensity and frequency of depressive symptoms. Specifically, we expect the most recent cohort to show the highest depressive symptoms score, and a distinction between cohorts born before or after WW2, with depressive symptoms being higher for the cohort of individuals born after the war.
3. An interaction between age and cohort, characterized by heterogeneous patterns of life-course trajectory per cohort. It appears difficult to make a more precise assumption about the direction of the effects, as no pattern emerges from previous studies.

Methods

Sample

The Panel Study of Belgian Households (PSBH) is a prospective longitudinal survey designed to follow households and their members, covering a wide range of socio-economic, demographic and family sociological themes (education, work, relationships, health, marriage, divorce, finances, etc). The original sample is composed of 8,741 adults aged 16 and above, coming from 4,438 Belgian households selected via stratified multistage area probability sampling. The PSBH's participants were followed on a yearly basis between 1992 and 2002, for a total of 11 waves [7].

Two specific age groups were left out of this analysis. Respondents under 25 years of age at baseline ($N = 1{,}152$) were excluded, to enhance sample's homogeneity in respect of life circumstances. Younger people had a larger probability of being students, being unemployed or having odd-jobs, and living with their parents. The greater diversity of this age group could create bias in the outcome and in covariates. Also, persons older than 74 in 1992 ($N = 587$) were excluded to limit mortality drop-out and bias resulting from difficulties in measuring depressive symptoms in old age [14, 31]. Two additional respondents had to be excluded, as information on age and birth year was missing. The resulting research sample is composed of 7,000 adults (3,392 men and 3,608 women) aged between 25 and 74 years of age in 1992.

The phenomenon of attrition is almost inevitable in longitudinal studies, and the PSBH is no exception. Among the 7,000 respondents surveyed at baseline, only 2,509 completed all 11 waves of data collection (Table 1). The other participants dropped out, sometimes permanently, sometimes for one or a few waves only. Using logistic regression (Table 2), drop-out was found to occur most frequently among men [odds ratio (OR) 1.25], older persons (OR 1.01), persons who lived alone (OR 1.20), were unmarried (OR 1.25), or were unemployed (OR 1.45). Furthermore, respondents with a higher educational degree (OR 0.93) and a higher household income (OR 0.81) were less likely to leave the study. Previous research has shown that, with the exception of being male, all these conditions are positively related with depressive symptoms [32]. Consequently, our results may underestimate the mean score of depressive symptoms. However, additional analyses showed no difference in mean depression score at baseline for drop-out and complete wave response participants. Besides attrition, the presence of selective item non-response on the depression items (Table 1) could also affect the results. Logistic regression reveals that missing values on depression items were more common among

Table 1 Number of participants, dropouts and missing depression items at each wave (%) (non-imputed)

Wave	*N*	Women	Dropout		Missing depression items			
			Cumulative	Women	None	1–3	4–12	13 (all)
1992	7,000	3,608 (51.5)	–	–	6,595 (94.2)	320 (4.6)	26 (0.4)	59 (0.8)
1993	5,856	3,038 (51.9)	1,144 (16.3)	(49.8)	5,474 (93.5)	229 (3.9)	10 (0.2)	143 (2.4)
1994	5,186	2,716 (52.4)	1,814 (25.9)	(49.2)	4,936 (95.2)	203 (3.9)	12 (0.2)	35 (0.7)
1995	4,868	2,565 (52.7)	2,132 (30.5)	(48.9)	4,649 (95.5)	158 (3.2)	13 (0.3)	48 (1.0)
1996	4,565	2,418 (53.0)	2,435 (34.8)	(48.9)	4,361 (95.5)	156 (3.4)	9 (0.2)	39 (0.9)
1997	4,201	2,237 (53.2)	2,799 (40.0)	(49.0)	4,010 (95.5)	121 (2.9)	11 (0.3)	59 (1.4)
1998	3,849	2,062 (53.6)	3,151 (45.0)	(49.1)	3,674 (95.5)	122 (3.2)	8 (0.2)	45 (1.2)
1999	3,576	1,924 (53.8)	3,424 (48.9)	(49.2)	3,426 (95.8)	105 (2.9)	3 (0.1)	42 (1.2)
2000	3,316	1,790 (54.0)	3,684 (52.6)	(49.3)	3,183 (96.0)	93 (2.8)	1 (0.0)	39 (1.2)
2001	3,006	1,628 (54.2)	3,994 (57.1)	(49.6)	2,893 (96.2)	75 (2.5)	3 (0.1)	35 (1.2)
2002	2,852[a]	1,546 (54.2)	4,148 (59.3)	(49.7)	2,752 (96.5)	57 (2.0)	4 (0.1)	39 (1.4)

[a] 2,509 participants were present at all 11-waves. The others dropped out, sometimes permanently, sometimes for one or a few waves only

men (OR 1.30), older persons (OR 1.03), and those with lower educational attainment (OR 1.09).

These findings indicate clearly that the missingness is not completely at random, and thus cannot be ignored without threatening the validity of the results. Among the numerous solutions used to deal with missing data [33, 34], we chose multiple imputation (MI) to tackle this problem [35, 36]. MI is a simulation technique in which missing values are replaced by m independent draws from the distribution of the variable that is being imputed, conditional on the observed data. The m imputed datasets are analyzed separately, and the results are combined into a single set of parameter estimates using Rubin's rules [35]. The strength of MI is that it allows for random variations between the imputed datasets, and preserves the characteristics of the data [33]. Thanks to the MI procedure, it is possible to analyze a complete dataset ($N = 7{,}000$) at each wave.

Because the amount of missings is substantial in this study, we opted for a relatively large number of imputations ($m = 10$) [37]. In order to keep the complexity of the imputation model within the limits of reason [33], we imputed the score of the depression scale rather than the 13 individual depression items[1] (find more information on the depression scale below). The imputation model included the available depression scores at all available time points, as well as the variables that were included in the final growth model (Table 4), specifically the cohort variables, gender, depression score, and socio-demographic variables (education, civil status, living status, income, and employment) at each wave.

Multiple imputation assumes that the data is missing at random (MAR), i.e. that the missingness can be predicted by the observed data. This is a much less restrictive assumption than missing completely at random (MCAR) [38]. Unfortunately, the MAR assumption is impossible to test, and we acknowledge the possibility that the data is missing not at random (MNAR).[2] However, since previous studies have shown that MI is less sensitive to the missing data mechanisms and that results obtained by means of MI are quite robust even under MNAR conditions, we decided to apply MI [33, 39, 40].

Depressive symptoms

Depression was measured using the Health and Daily Living form (HDL) depression scale [41]. Participants were asked to report the frequency of occurrence for each of the 17 depression symptoms within three months preceding the survey. The respondents were offered a five-point scale to formulate their answer (0 never, 1 rarely, 2 sometimes, 3 regularly, 4 often).

Our study employed a revised version of the HDL-depression scale, which includes only 13 items (feeling

[1] Imputation models including 143 depression variables (13 items measured at 11 time points) become practically impossible to estimate.

[2] Selection models (simultaneously modeling the dependent variable and the missingness process) or pattern-mixture models (PMM) (distinguishing respondents by their missingness pattern) were developed to deal with data where the missingness process is not at random (MNAR). These models require a rigorous and relatively complex sensitivity analysis [38], as their results can be highly dependent on untestable assumptions about the missingness model (for selection models) or the choice for so-called identifying restrictions (for PMM).

Table 2 Socio-demographic and clinical characteristics at baseline for dropout and complete-waves response participants (bivariate logistic regressions)

	Complete 11 waves *N* (%)	Dropouts[a] *N* (%)	Sig. (Wald; *df*; *p*)	Odds ratio (Exp *B*)
Men vs.	1,127 (44.9)	2,265 (50.4)	19.6; $df = 1$; $p = 0.000$	1.25
Women	1,382 (55.1)	2,226 (49.6)		
Not living with partner vs.	466 (18.6)	964 (21.5)	8.5; $df = 1$; $p = 0.004$	1.20
Living with a partner	2,043 (81.4)	3,518 (78.5)		
Not employed vs.	973 (38.8)	2,145 (47.9)	53.8; $df = 1$; $p = 0.000$	1.45
Employed	1,533 (61.2)	2,329 (52.1)		
Not married vs.	611 (24.4)	1,284 (28.6)	15.0; $df = 1$; $p = 0.000$	1.25
Married	1,898 (75.6)	3,198 (71.4)		
	Mean (SD)	Mean (SD)		
Age	44.7 (13.5)	47.3 (14.4)	52.0; $df = 1$; $p = 0.000$	1.01
Education	3.3 (2.7)	2.7 (2.7)	59.9; $df = 1$; $p = 0.000$	0.93
Household income	1.0 (0.5)	0.9 (0.5)	19.3; $df = 1$; $p = 0.000$	0.81
Depression score 1	13.0 (8.6)	13.2 (9.1)	1.5; $df = 1$; $p = 0.219$	–
Depression score 2	13.9 (8.4)	14.1 (9.1)	1.1; $df = 1$; $p = 0.303$	–
Depression score 3	13.7 (8.4)	14.1 (9.1)	3.1; $df = 1$; $p = 0.076$	–
Depression score 4	13.7 (8.2)	14.5 (9.3)	9.0; $df = 1$; $p = 0.003$	1.01
Depression score 5	14.0 (8.7)	14.9 (9.3)	9.8; $df = 1$; $p = 0.002$	1.01
Depression score 6	14.5 (8.5)	15.4 (9.7)	10.0; $df = 1$; $p = 0.002$	1.01
Depression score 7	14.3 (8.9)	15.2 (9.7)	8.7; $df = 1$; $p = 0.003$	1.01
Depression score 8	14.8 (9.0)	15.0 (9.6)	0.6; $df = 1$; $p = 0.425$	–
Depression score 9	14.7 (9.0)	15.7 (10.2)	5.6; $df = 1$; $p = 0.017$	1.01
Depression score 10	14.6 (9.0)	15.7 (9.9)	5.7; $df = 1$; $p = 0.017$	1.01
Depression score 11	14.7 (9.0)			
N	2,509	4,491		7,000

[a] This category includes respondents that have dropped-out sometimes permanently, sometimes for one or a few waves only

depressed, feeling tired, feeling irritable, feeling pessimistic, feeling guilty, not feeling good, having gloomy thoughts, having strange thoughts, loss of appetite, sleep disorder, difficulty in sitting still, lack of concentration, need for confirmation). The four remaining items were rejected for methodological reasons. "Have you suffered from crying easily" was abandoned because this symptom is more prevalent in women [42, 43], and could overestimate depressive symptoms for this group. "Did you have any physical symptoms of disease" was not included because of its lack of validity as an explicit predictor of depression. Finally, items related to suicide and death had to be left out, as from the third to the fourth wave of data collection, the item's structure had changed from one question including the two symptoms, to two distinct questions; one for each symptom. This is undoubtedly a limit, as an indicator of severe depression should include information concerning thinking about death/suicide. Our depression scale may thus be less able to separate respondents with more severe levels of depression from those with less severe psychological distress. Nevertheless, previous research has evidenced that the reliability and validity of this revised HDL-depression scale is satisfactory [7, 43–45].

The depression score was obtained by adding up the scores for the 13 items. As a result, the HDL-depression scale ranges from 0 to 52, where higher scores indicate a greater severity of depressive symptoms.

As shown in Table 1, the vast majority of the respondents (>93% at each wave) has completed all 13 depression items. Item non-response is limited: 2–5% of the remaining respondents had one to three missing depression items, a low proportion (<0.5%) had four to 12 missing items and finally 2.5% had missing items on all 13 depression questions. For respondents with 3 or less missing depression items, mean substitution was used (the missing value was replaced by the respondent's mean on the available depression items). If 4 or more missings were present, the HDL-depression score was not computed, but instead imputed by means of the MI procedure described above. Despite its limitation of dropping available

Table 3 Characteristics of the cohorts at the first data-collection wave (1992) (non-imputed data)

	Post-WW2		WW2	Pre-WW2		Total
	1958–1967	1948–1957	1938–1947	1928–1937	1918–1927	
N	1,905	1,638	1,240	1,178	1,039	7,000
Women (%)	983 (51.6)	838 (51.2)	623 (50.2)	609 (51.7)	555 (53.4)	3,608 (51.5)
Age (Md)	25–34 (30)	35–44 (40)	45–54 (49)	55–64 (59)	65–74 (69)	25–74 (49)
Depression score (SD)	14.06 (8.7)	13.71 (9.1)	13.65 (9.3)	11.98 (8.7)	11.18 (8.5)	13.13 (8.9)
Married (%)	1,257 (66.1)	1,264 (77.2)	976 (78.9)	902 (76.6)	697 (67.1)	5,096 (72.9)
Single/widowed (%)	528 (27.8)	155 (9.5)	99 (8.0)	172 (14.6)	303 (29.2)	1,257 (18.0)
Divorce/separated (%)	116 (6.1)	218 (13.3)	162 (13.1)	103 (8.8)	39 (3.8)	638 (9.1)
Living with partner (%)	1,485 (78.1)	1,380 (84.3)	1,044 (84.4)	935 (79.4)	717 (69.0)	5,561 (79.5)
Employed (%)	1,482 (78.0)	1,236 (75.7)	808 (65.4)	297 (25.3)	39 (3.8)	3,862 (55.3)
Monthly income € (SD)	951 (450)	955 (479)	1,026 (590)	957 (591)	909 (571)	963 (528)
Education (SD)	3.86 (2.6)	3.39 (2.8)	2.88 (2.7)	1.89 (2.3)	1.68 (2.2)	2.92 (2.7)

information for some respondents, the cutoff point was selected at 4 missing items on the 13 available, as it corresponds to a high proportion of missing data [40, 46].

Age and cohorts

The age of participants is expressed in years and goes from 25 to 74 years old at baseline, and from 35 to 84 for the last wave. The variable age was centered on the grand median, corresponding to 49 years old.[3]

Participants were classified into five birth cohorts: 1918–1927, 1928–1937, 1938–1947, 1948–1957, and 1958–1967. Thus, two cohorts were born before WW2, one during, and two after. This categorization in 10-year groups has the advantage of creating groups with a sufficient sample size (Table 3) to obtain reliable estimates.

Gender and time-varying covariates

In order to get a clear view on existing age and cohort effects, it is important to control for a number of potentially confounding variables. First, gender is known to be an important predictor of depressive symptoms and of major depression episodes, with a greater prevalence among women [42, 45]. This remains true even after controlling for gender bias in measurement [48]. This variable was coded 0 for men and 1 for women.

The other control variables were collected annually and are time-varying, meaning that their value can change over time for the same individual. Controlling for these risk factors is important, as their distribution could be age-dependent, and thus could mask potential age effects [14]. Among them, being married, being employed [7, 12], and having high socio-economic status [49], are all conditions associated with a lower level of depressive symptoms. Participants were classified as either employed (1) or unemployed (0). Civil status was separated into: single-widowed, divorced-separated, and married (reference category). We also included a variable that assesses the effect of living alone (0) versus living with a partner (1) [7]. Educational attainment consisted of 10 categories (from primary school to post-university), with higher values indicating higher educational attainment at the time of the survey. The variable was centered on the grand mean, which corresponds to a Belgian professional high school diploma (12 years of education). The household income measure available in the PSBH is the sum of net salary, social allocations, and other revenues. It was adjusted for household size (the first adult counts for 1 point, the others for 0.7, and each child for 0.5) and for inflation (using the consumer price index with 1996 as a base year) and centered on the grand mean, which corresponds to an income of €990 per month. A higher score relates to a higher household income.

Analyses

Growth curve modeling [50, 51] was used to determine the effects of age and cohort on the depressive score trajectory. The growth curve model allows us to estimate simultaneously how depression scores evolve over the life-course (as a function of age), and how certain covariates affect the growth pattern. In other words, this technique simultaneously assesses the within-individual change over time, and the inter-individual differences in patterns of change [51]. In the multilevel approach to growth curves, the differentiation between within- and between-individual

[3] Age must be measured on the same scale for respondents from different cohorts, as we want to estimate pure cohort effects, i.e. under control for the differences in the age composition of the cohorts [47:405]. Thus, grand-median centering of the age variable is preferred over cohort-median centering.

changes is obtained by introducing two levels of analysis with their own random components. One of the principal advantages of this approach is that it is appropriate for dealing with the statistical dependency of observations (several measurements per individual), which is inherent in longitudinal data [52].

More detailed model specifications are represented by the following growth curve equation (Eq. 1), using Singer and Willett's notation [51]. In our models, cohort membership is seen as the main predictor of inter-individual differences in intercept as well as in the rate of growth of the HDL-depression score. Cohort membership is introduced as 4 dummy variables. The cohort of individuals born during WW2 (1938–1947: C3_3847) serves as a reference cohort.

$$\begin{aligned} Y_{ij} = & [\gamma_{00} + \gamma_{01}(C1\_5867) + \gamma_{02}(C2\_4857) \\ & + \gamma_{03}(C4\_2837) + \gamma_{04}(C5\_1827) \\ & + ((\gamma_{10} + \gamma_{11}(C1\_5867) + \gamma_{12}(C2\_4857) \\ & + \gamma_{13}(C4\_2837) + \gamma_{14}(C5\_1827)) \times \mathrm{Agec}_{ij})] \\ & + [\zeta_{0i} + \zeta_{1i}(\mathrm{Agec}_{ij}) + \varepsilon_{ij}] \end{aligned} \quad (1)$$

Assuming that $\varepsilon_{ij} \sim N(0, \sigma_\varepsilon^2)$ and

$$\begin{bmatrix} \zeta_{0i} \\ \zeta_{1i} \end{bmatrix} \sim N\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_0^2 & \sigma_{01} \\ \sigma_{10} & \sigma_1^2 \end{bmatrix} \right)$$

where Y_{ij} represents the estimated depressive symptoms (HDL) score for individual i at time j; intercept γ_{00} represents the mean HDL-depression score for the reference cohort at median age; γ_{01} to γ_{04} are the estimated differences in mean HDL-depression score between the respective cohorts and the reference cohort; γ_{10} represents the mean growth rate in HDL-depression score of the reference cohort; γ_{11} to γ_{14} are the estimated differences in the mean growth rate between the respective cohorts and the reference cohort; ζ_{0i} and ζ_{1i} are the Level-2 residuals.

We have already mentioned that the sample was drawn by means of a two-stage procedure: (1) households were selected and (2) within the household, individuals were selected. Besides the measurement within-individual and the individual level, our dataset also contains a third level; the household level. However, because the household is less relevant to our specific research questions, and in order to reduce the complexity of the model, we decided not to model this level explicitly. As the average number of individuals per household is very small (1.7), neglecting this dependency between members of the same household can only have minor statistical consequences. Nevertheless, we use robust standard errors, i.e. the so-called "sandwich estimator" [38]; to correct for eventual bias in standard errors.

All analyses were carried out using SAS 9.2, specifically the procedures "MI" for the multiple imputation, "MIXED" for the estimation of the growth curve models, and "MIANALYZE" for combining the results from the multiple imputed datasets.

Results

Table 4 summarizes the four models that were estimated block-by-block to determine the effect of age and cohort on depressive symptoms.

MODEL 1: empty model

An unconditional model (Model 1), used to decompose the outcome variance between levels of analysis [50, 51], shows that 61% (intraclass correlation coefficient $\rho = 0.61$) of the total variation in depressive symptoms is situated at the between-individual level, while 39% is within-individual change. Differences between individuals are, on average, somewhat larger than changes that individuals go through during their life-course. This within-individual variance could nevertheless be underestimated, as the estimation is limited to an 11-year period, and not to the entire life. These results essentially confirm the need for a multilevel analysis. The estimated intercept shows that the overall mean HDL-depression score in the dataset equals 14 points.

MODEL 2: age trajectories in depression

In the second model, individual depression trajectories over the life-course are modeled by introducing a variable age. Additional analysis (not shown here) identified a quadratic function as the best functional form for the association between age and depression. The statistically significant estimated parameters imply that the mean HDL-depression score initially rises with age, but that the rate of growth is reduced as the individual gets older. Judging by the size of the standardized parameters, the effects of age are quite modest (<0.10). Yet, we should keep in mind that the effects of the variable 'age' shown here are not pure age effects, but are instead confounded with cohort effects. Since this longitudinal study comprises only 11 years, there exists a strong relation between age and cohort membership.

Model 2 also contains a significant random slope for the first-order age effect meaning that the rate of change varies significantly across individuals. We decided not to include a random slope for the quadratic age effect, as this led to estimation problems for the variance-covariance matrix of the random components. These estimation problems indicate over-specification of the variance

Table 4 Estimates of growth curve models of depressive symptoms ($N = 7{,}000$) (imputed data)

	Model 1		Model 2			Model 3			Model 4		
Fixed effects											
Initial status											
Intercept y_{00}	14.33	***	14.732	***		14.104	***		13.65	***	
C1 1958–1967						4.274	***	[0.208]	4.366	***	[0.212]
C2 1948–1957						1.985	***	[0.092]	1.930	***	[0.089]
C3 1938–1947						REF			REF		
C4 1928–1937						−2.595	***	[−0.106]	−2.808	***	[−0.115]
C5 1918–1927						−5.130	***	[−0.199]	−5.489	***	[−0.213]
Woman									3.360	***	[0.183]
Education									−0.084	*	[−0.025]
Married									REF		
Single/widowed									0.248		[0.010]
Divorced/separated									0.349		[0.012]
Partner									−1.042	**	[−0.047]
Monthly income €									−0.163		[−0.009]
Employed									−0.609	***	[−0.034]
Growth rate											
Agec y_{10}			0.049	***	[0.077]	0.078	*	[0.123]	0.054		[0.085]
Agec2 y_{20}			−0.001	**	[0.026]						
Agec × C1_5867						0.137	***	[0.099]	0.165	***	[0.119]
Agec × C2_4857						0.092	**	[0.028]	0.105	**	[0.032]
Agec × C3_3847						REF			REF		
Agec × C4_2837						−0.003		[−0.002]	−0.003		[−0.002]
Agec × C5_1827						0.063		[0.063]	0.065		[0.065]
Variance components (random effects)											
Level 1: within-person	32.462	***	30.455	***		30.289	***		30.315	***	
Level 2: in initial status	51.447	***	50.638	***		47.533	***		43.739	***	
Level 2: in linear growth			0.095	***		0.094	***		0.087	***	
Proportion of variance explained											
Within-person	n.a.		2.007	6.2%		2.173	6.7%		2.147	6.6%	
In initial status	n.a.		0.809	1.6%		3.914	7.6%		7.708	15.0%	
In linear growth	n.a.		n.a.			0.001	1.1%		0.008	8.4%	

Unstandardized estimates [standardized estimates]

The goodness of fit statistics, employed for determining parsimony of the different models, could not be used here because of the multiple imputation process. First, deviance statistics are not provided with the SAS 9.2 MIANALYZE procedure. Second, as the estimated coefficients correspond to the mean of coefficients from all the imputed datasets, the number one rule for applying the goodness of fit "ensure the dataset has remained the same across models" [51:119] is not respected

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

structure, and most probably result in the finding that the age trajectory per cohort is not significantly curved—see model 3.

By introducing age in Model 2, the within-person variance decreased substantially (by 6.2% compared to Model 1). This illustrates the relevance of age in understanding within-individual changes in depressive symptoms. The between-individual variance has also slightly decreased, by 1.6%.

MODEL 3: bringing in cohort membership

The third model disentangles age and cohort effects, and specifically explores whether differences exist between

cohorts (a) in the intensity and frequency of depressive symptoms, and (b) in the age trajectories.

Main cohort effect

The intercept of Model 3 (14.10) corresponds to the mean HDL-depression score of the reference cohort (born between 1938 and 1947) at the overall median age of 49. The main cohort effects refer to deviations from this reference cohort. The younger cohorts show a significantly higher intensity of depressive symptoms. Cohorts 1 (1958–1967) and 2 (1948–1957) have an estimated HDL-depression score of 18.38 and 16.09 points, respectively. For the older cohorts, the main cohort effect is negative, indicating a lower intensity of depressive symptoms. Estimated HDL-depression scores are 11.51 for Cohort 4 (1928–1937) and 8.97 for Cohort 5 (1918–1927). Thus, the cohort main effects are clearly patterned with a steady increase in depressive score with the succession of cohorts. All cohorts' mean depressive symptoms scores differed significantly from one another, with recent cohorts always having the highest mean HDL-depression score.

Some caution is needed when interpreting these cohort main effects. The estimated parameters refer to differences between respondents from various cohorts at a specific age (see Footnote 3). Since age was centered on the grand median, in this model the age of comparison is 49. As a result of the accelerated longitudinal design, however, only cohorts 2 and 3 contain respondents that were surveyed at this specific age. For the other cohorts, cohort comparisons are based on extrapolations of age trends beyond the observed age range. Furthermore, as shown in the next section, we find evidence of age by cohort interactions, which mean that the sizes of cohort differences can vary over the life-course. As a result, our conclusions on the existence of cohort main effects could depend on the specific age at which cohorts are compared.

To deal with these limitations, and in order to strengthen our conclusions on the existence of cohort differences, additional contrast tests (not shown) were performed. For every cohort, we tested whether the HDL-depression score at their specific median age differed significantly from the previous (older) and the next (younger) cohort. In this way, it is possible to estimate cohort differences without having to rely on extrapolations. All additionally estimated cohort differences were found to be strongly significant ($p < 0.001$), except for one. Taking the age of 74 as a reference point, the difference between cohort 4 and 5 is only border-significant ($p = 0.0551$). Also in these additional tests, it is found that younger cohorts score systematically higher on the HDL-depression scale, which largely confirms the presence of important and clearly patterned cohort main effects.

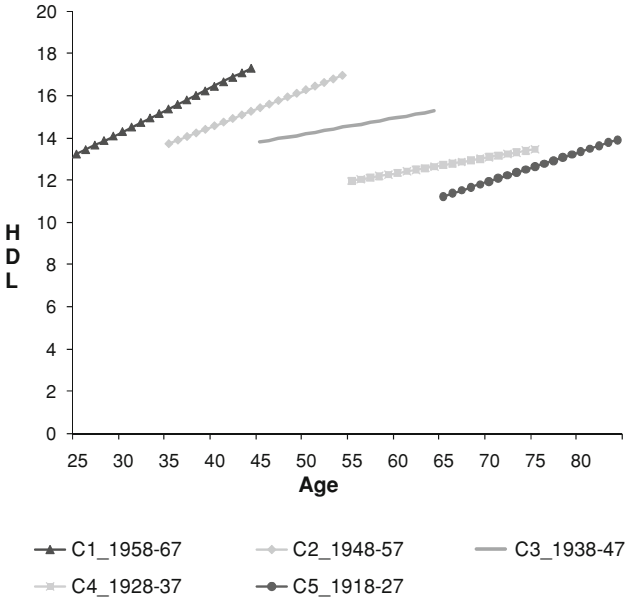

Fig. 1 HDL-depression score trajectory by age and cohort (based on Model 3's estimates)

Age-by-cohort interactions

Model 3 also contains age-by-cohort interactions. These interactions indicated that the life-course trajectories of depressive symptoms are dependent on cohort membership. Figure 1 summarizes the age-by-cohort interactions, as well as the cohort main effect on HDL-depression scores (based on Model 3 estimates).

In Model 2, a quadratic age effect was introduced. Additional tests, however, made clear that significant curvature is not present within the cohorts. Probably as a result of the limited time-span, cohort-specific age trajectories in depression turned out to be linear (for a similar finding, see Raudenbush and Chan [47]). Consequently, we decided to drop the quadratic term from the analyses.

The results of Model 3 show that the cohorts under study differ significantly in respect of the strength of the linear age effect. Contrast testing (not shown) was performed, firstly to assess the statistical significance of each age-by-cohort estimate, and secondly to determine which estimates differed significantly from the others.

For Cohort 1, the linear age effect equals 0.215, which is not statistically different to the linear trajectory over age for cohorts 2 and 5. Consequently, even if Cohort 1 has the highest rate of growth over age (0.215), it is comparable to that of Cohort 2 (0.170) and of Cohort 5 (0.141). In contrast, Cohorts 3 (0.078) and 4 (0.075) have the lowest increase over age, indicating that depressive symptoms score increase over age, but less rapidly than other cohorts.

Everything considered, cohort membership is an important predictor of between-individual differences in

mean HDL-depression score and in mean rate of growth. The introduction of cohorts in Model 3 results in an explanation for 6% of the between-individual variance in the initial status (of a total of almost 8% combined with age). Furthermore, cohort membership also explained 1.1% of the between-individual variance in the growth rate of depressive symptoms (Model 2 vs. Model 3).

MODEL 4: control variables

Model 4 shows that age and cohort effects remain largely unchanged after controlling for gender, education, civil status, living status, income, and employment.

As expected, gender is an important predictor of depressive symptoms, and women show a significantly higher HDL-depression score than men. Comparing the random part of Model 3 to a subsequent model introducing gender as the only covariate (models not shown), we estimated a decrease of 6.2% in the amount of variance between individuals for the intercept. No gender interaction either with cohort, age, or age-by-cohort was found. Thus gender's main effect on depressive symptoms explains a great deal of differences between individuals.

Education has a small, but significant, effect on the depressive symptoms score. On average, higher educational attainment increases the probability of having a lower HDL-depression score. Being employed and living with a partner statistically reduced the HDL-depression score of an individual. Civil status and monthly income have no effect on mean HDL-depression scores. The comparison of the size of the standardized parameters indicates the effects of the cohort are greater than the ones associated with the control variables, including gender.

Throughout the analyses, we were more successful in explaining the between-individual variance, than the within-individual variance in HDL-depression scores. The comparison between Model 1 and Model 4, indicates a decrease of 15% in the variance of the intercept between-individual, and one of 8.4% in the variance of the growth rate between-individual, while we explained only 6.6% of the within-individual variance in HDL-depression score. Under the assumption that changes in social conditions are adequately captured by the models, this conclusion confirms the structural effects of conditions on mental health.

Discussion

Using a representative imputed sample of 7,000 Belgian adults, ages 25–74, surveyed annually from 1992 to 2002, our results confirmed the existence of differences between birth cohorts in the intensity and frequency of depressive symptoms, and the age trajectories. We also described a linear increase in the intensity of depressive symptoms over the life-course. However, we could not corroborate the existence of specific patterns in the intensity, or in the life-course trajectory, of depressive symptoms for cohorts born after WW2.

Importance of the succession of cohorts

The respondents were grouped into five birth cohorts (1918–1927, 1928–1937, 1938–1947, 1948–1957, 1958–1967), covering more than half of the twentieth century. The intensity of depressive symptoms score increases progressively with the succession of cohorts, leading the most recent cohorts to have the highest score. Moreover, all cohorts' mean depressive symptoms score differed significantly from one another. This strong relationship between cohort membership and depressive symptoms was similar for men and women, and remained after controlling for education, civil status, living status, income, and employment.

Our results confirm the conclusion of previous Belgian [24] and American studies, using very different samples [11, 20, 29] and different measures of depression [6, 26–28]. Similar effects of cohort succession were also identified for other mental disorders: younger cohorts showed a greater life-time prevalence of schizophrenia, bipolar disorder, panic disorder [27], and alcohol disorders [24] and a higher intensity of psychopathic deviation, paranoia, hypomania [6], and anxiety [53]. Our findings also explain the rise in depressive symptoms that was observed between 1992 and 1999 by Wauterickx and Bracke [7] using data from the same Belgian adult population (PSBH). Thus, the replacement of the population over time seems of special relevance to the explanation of trends in mental health.

As suggested by Ryder [10], cohort differentiation may reflect the transformations that have occurred in society. The constant increase in the level of depressive symptoms with each new cohort shows that the intensity of depression was observed not only for baby-boomers, but also for the cohorts born before and during WW2. For this reason, it is impossible for us to conclude that there is a special WW2 effect, as this would indicate the cohorts of individuals born after the war would exhibit a notably different pattern of depressive symptoms to those born before. The actual observations suggest a gradual, rather than sudden, appearance of social changes.

The ongoing occurrence of individualism in Western societies during the twentieth century could be of particular interest, especially since it was associated with lower health status and well-being [21]. Accordingly, we suggest that our younger cohorts may have been more exposed and socialized earlier in their life to individualism, via the new

norms of individuality [54], the rise of individual choice [55], the medicalization of unhappiness, and the transformation of family structure and relationships [56].

The growing importance given to responsibility, autonomy, and initiative [54], as well as to individual choices [55], led individuals to be responsible for their own success and happiness. As making the wrong choice could be seen as a personal failure, too much choice could be related to a decrease in life satisfaction and in happiness [55]. In this context, depression can thus be conceptualized as the "illness of responsibility" and may result from the individual's incapacity for action [54].

These new norms, in addition to the development of medicine, pharmaceutical innovations, and the importance accorded to health and well-being, encouraged the medicalization of unhappiness (or of sadness). Nowadays, the boundary between normal emotions, notably sadness, and medical symptoms seems blurred [57], whilst mood enhancement by pharmaceutical means is common [58]. Younger cohorts may thus focus on health and well-being more than previous cohorts, and thus be less tolerant to unhappiness and to sadness, more prone to use medical language to report their emotions and experiences, more willing to use medication, and may believe that the source of their problems lie in biological and psychological factors, rather than contextual ones.

The twentieth century was also characterized by an increasing level of modernization, which was correlated with a greater lifetime prevalence of depression, especially among young women [59]. A decline of traditional institutions like family, community, and religious groups [56], and a rise in the rate of divorce [60], single-parent families, and loneliness [56], i.e. a decrease in social relationships, social capital, and social networks, is associated with an increase in depression, anxiety, suicide, and unhappiness [53, 56].

Age effect on depressive symptoms

Age is an important predictor of psychological distress, specifically of depression. However, the association between ageing and depression has often led to confusing results, either because of the confounding of age and cohort effects, or because of the rarity of studies that have focused on net age effects. As with previous studies [11, 20, 23], we identified a linear and positive net age effect, suggesting the intensity of depressive symptoms increases with age. However, this relationship may be biased, because the presence of physical illnesses or chronic disorders was not controlled for, but could appear to change the initial trajectory [11, 20]. Cross-sectional studies nevertheless reported a "genuine" decline in depression over age, as it was reported that the association between physical illness and depression weakens in old age [4, 16].

Besides net age and cohort effects, the life-course trajectories of depressive symptoms are dependent on cohort membership, which corroborates the cohort differentiation in ageing [8, 10]. Our results revealed an increase in depressive symptoms over age for all cohorts, but different magnitude of growth for different cohorts. A steeper increase in depressive symptoms over age was noted for the two youngest (1958–1967, 1948–1957) and the oldest cohorts (1918–1927), indicating no pre/post WW2 effects. The two remaining cohorts, those that lived through the most dramatic experiences during their early years (the 1938–1947 cohort born during WW2, and the 1928–1937 cohort born during the Great depression), have a smoother increase in depressive symptoms over age. This latter pattern could result from a specific life-stage, during which, for a majority of people, the structural sources of distress (double burden, financial strain associated with mortgages, and insecurities related to career development) slowly dissolve, while the strains caused by old age (health problems, disability, loneliness) are yet to appear.

Strengths, limitations, and future researches

Although we covered a broader age range (25–74 years old) than previous studies, ours is still limited by the small age overlap between cohorts. This situation led, as previously mentioned, to the need to estimate mean depression score at a virtual age for some cohorts. Even if these statistical extrapolations are valid, the accuracy of the life-course estimates would increase if future longitudinal designs include cohorts with a greater age overlap.

Moreover, as with any longitudinal studies, we have to acknowledge the presence of missing data as a limitation to data analysis. Despite missing data were handled with a state-of-the-art technique, multiple imputation still has some limitations, namely that is assumes that the data is MAR. As it is impossible to test this assumption, future research could assess the robustness of our findings using a more complex missing data treatment that does not assume MAR, such as pattern-mixture modeling [38].

Even if the growth curve modeling makes possible the disentanglement of age and cohort effects, a period effect could be hidden. This effect refers to changes that are associated with a specific historical period, and that persist despite ageing and cohort membership. Known as the *Identification problem* [61], the confounding effects of age, period, and cohort come from the linear dependency that exists between these three factors: knowing two values automatically lead to the third one. Consequently, the non-inclusion of one of them could lead to error in the attribution of effect [61]. Nevertheless, period effects were

neither noted in our results nor expected, as a decade is a relatively small historical period over which to observe such trends, especially in the absence of sudden historical events.

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