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The stability and change of wellbeing across the lifespan: a longitudinal twin-sibling study

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Original Article

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Abstract

Background. Wellbeing is relatively stable over the life span. However, individuals differ in this stability and change. One explanation for these differences could be the influence of different genetic or environmental factors on wellbeing over time.

Methods. To investigate causes of stability and change of wellbeing across the lifespan, we used cohort-sequential data on wellbeing from twins and their siblings of the Netherlands Twin Register (NTR) (total $N = 46.885$, 56% females). We organized wellbeing data in multiple age groups, from childhood (age 5), to adolescence, up to old age (age 61+). Applying a longitudinal genetic simplex model, we investigated the phenotypic stability of wellbeing and continuity and change in genetic and environmental influences.

Results. Wellbeing peaked in childhood, decreased during adolescence, and stabilized during adulthood. In childhood and adolescence, around 40% of the individual differences was explained by genetic effects. The heritability decreased toward old adulthood (35–24%) and the contribution of unique environmental effects increased to 76%. Environmental innovation was found at every age, whereas genetic innovation was only observed during adolescence (10–18 years). In childhood and adulthood, the absence of genetic innovation indicates a stable underlying set of genes influencing wellbeing during these life phases.

Conclusion. These findings provide insights into the stability and change of wellbeing and the genetic and environmental influences across the lifespan. Genetic effects were mostly stable, except in adolescence, whereas the environmental innovation at every age suggests that changing environmental factors are a source of changes in individual differences in wellbeing over time.

Introduction

Wellbeing can be broadly defined as the subjective evaluation of feeling good and functioning well in life. Wellbeing is generally found to be moderately stable across different situations and across the lifespan in western populations, with average correlations ranging from 0.3 to 0.6 depending on the time interval (Anusic & Schimmack, 2016; Ehrhardt & Saris, 2000; Fujita & Diener, 2005; Lucas & Donnellan, 2007; Pavot & Diener, 1993). This suggests that wellbeing is relatively stable in most people in the investigated populations. However, individual differences in this degree of stability have been reported as well, with some individuals experiencing more fluctuations over time compared to others (de Vries & Bartels, 2023; Eid & Diener, 1999; Gadermann & Zumbo, 2007). Similarly, correlations of 0.3–0.6 imply that stability is far from perfect, indicating that fluctuations in wellbeing overtime occur. Recent research has shown that the stability of wellbeing can change during different stages of life, such as the steep decline in life satisfaction during adolescence and relative stability in adulthood (Goldbeck, Schmitz, Besier, Herschbach, & Henrich, 2007; Jebb, Morrison, Tay, & Diener, 2020; Orben, Lucas, Fuhrmann, & Kievit, 2022). Furthermore, there is an active discussion about a proposed U-shaped trajectory across the life span, i.e. a high wellbeing in childhood and young adulthood, a drop in wellbeing in middle adulthood, and increasing levels in late adulthood (e.g. Blanchflower and Graham, 2021; Galambos, Krahn, Johnson, and Lachman, 2020). These findings highlight the need for research into the causes of stability and change of wellbeing over time.

One explanation for the relatively stable level of wellbeing and the individual differences in these stable levels can be the influence of a stable set of genetic factors on wellbeing across the lifespan. In cross-sectional data, two meta-analyses found a meta-analytic heritability of 40% (CI 37–42%) (Nes & Røysamb, 2015) and 36% (95%CI 34–38%) for wellbeing (Bartels, 2015). Most samples in the meta-analyses only included adolescents or only adult participants, limiting the comparison of heritability across the lifespan, i.e. between childhood, adolescent, and adult wellbeing. Therefore, Baselmans et al. (2018) compared the heritability of wellbeing in seven different age groups from childhood, adolescence, and adulthood (ages 7, 10, 12, 14, 16, 18–27, and 27–99). In the childhood samples, wellbeing was rated by mothers and fathers,

from the age of 14 participants completed self-reports. The heritability estimates of wellbeing in the different age categories were similar to the meta-analytic estimates mentioned before, ranging between 31% and 47%. Shared environmental influences explained around 40% of the variation in childhood. These influences disappeared after the age of 14 (self-report), leaving only additive genetic and unique environmental influences on wellbeing. In childhood, the shared environmental influences are likely partly explained by rater bias of the parents. Rater bias can arise because of disagreement between mothers and fathers based on biases such as different information sources, response style or stereotyping and the actual behavior of the children (Bartels et al., 2004a; Hewitt, Silberg, Neale, Eaves, & Erickson, 1992). For example, in studies on rater bias in ratings of internalizing and externalizing problems, around 20% of the variance and covariance over time (stability) is due to rater bias (Bartels et al., 2004a, 2004b). If not taken into account, this rater bias results in an overestimation of shared environmental effects.

Although the heritability estimates in the different age categories appear relatively similar, longitudinal designs are needed to directly investigate the overlap and change in genetic and environmental factors influencing wellbeing across the life span. Two studies with genetically informative samples investigated this stability and change in genetic and environmental factors in a longitudinal design. Lykken and Tellegen (1996) investigated the heritability of wellbeing twice in a small sample of young adults ($n = 254$) over a period of 10 years (age 20–30). Genetic factors explained 44 and 52% of the variance at age 20 and 30, respectively. The phenotypic wellbeing scores correlated 0.50 over the 10-year period and genetic effects explained around 80% of this correlation over time. Similarly, Nes and colleagues measured wellbeing twice (~6 years apart) in a larger sample of young adults ($n = 4322$; mean age T1 = 21.7, T2 = 25.6) (Nes, Røysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006). The longitudinal phenotypic correlation for wellbeing was 0.50. Additive genetic factors accounted for 81% of this longitudinal correlation in males and 75% in females, with the unshared environment explained the remaining phenotypic association. The longitudinal genetic correlation was strong (0.85 and 0.78 for males and females), indicating largely shared genetic influences over time.

Based on the above results, the stability in long-term wellbeing seems to be mainly determined by similar genetic influences over time. However, both studies only included young adults around their 20s and 30s and measured wellbeing twice, either 10 or 6 years apart. To better understand the development of wellbeing and sources of individual differences in wellbeing across the lifespan, and to find directions for when wellbeing interventions are most effective, we need to investigate the stability and change in genetic and environmental factors across the life span and across development, i.e. including transitions from childhood to adolescence, adolescence to adulthood, and adulthood to older adulthood. To answer this question, longitudinal wellbeing data from people across the lifespan are needed. The Netherlands Twin Register (NTR) has collected such longitudinal data of wellbeing in participants of all ages (Ligthart et al., 2019).

In the current study, we combined these data in childhood, adolescence, and adulthood in a cohort-sequential longitudinal design. We used a genetic simplex model to investigate the phenotypic stability of wellbeing and the continuity and change of genetic and environmental influences on wellbeing across the lifespan. As explained in the methods in more detail, in the genetic simplex model, we can detect different mechanisms of transmission and innovation of genetic and environmental influences (Boomsma & Molenaar, 1987). First, we can detect stable genetic or environmental factors that have a continuous influence on wellbeing across the lifespan (transmission). Second, we can detect new genetic and environmental influences at different ages, that is transmitted to the next age, i.e. innovation variance. Finally, we can detect age-specific influences that are specific to a certain age and are not transmitted to the next age.

Furthermore, inconsistent results on gender differences in average wellbeing have been reported (Batz & Tay, 2018). For example, large studies or meta-analyses reported higher wellbeing in males compared to females (e.g. in adolescents [Yoon, Eisenstadt, Lereya, & Deighton, 2023], adults [Stevenson & Wolfers, 2009], older age [Pinquart & Sörensen, 2001]), or higher wellbeing in females compared to males (e.g. in adolescence [Esteban-Gonzalo, Esteban-Gonzalo, Cabanas-Sánchez, Miret, & Veiga, 2020]). However, based on a large meta-analysis on gender differences in life satisfaction across the lifespan, no significant differences were found (Batz-Barbarich, Tay, Kuykendall, & Cheung, 2018). Similar inconsistent, but mostly non-significant gender differences have been found in studies of gender differences in the heritability of wellbeing (Bartels, 2015; Pelt, de Vries, & Bartels, 2024; van de Weijer, de Vries, & Bartels, 2022). However, the effects of gender on average wellbeing levels and heritability could differ across the lifespan. Therefore, we compute the trends of wellbeing across the lifespan separately for females and males as well, and apply sensitivity analyses on the simplex models in subsets of twin and sibling data of respectively only females and males.

Method

Participants

Participants were voluntary members of the NTR. The NTR was established by the Department of Biological Psychology, Vrije Universiteit Amsterdam more than 30 years ago (Ligthart et al., 2019). The NTR collects data in children and adolescents, i.e. the Young NTR (YNTR) and in adults, i.e. the Adult NTR (ANTR). In the YNTR, parents complete questionnaires (including questions about the children's wellbeing), when their children are 5, 7, 10, and 12 years old. Furthermore, adolescents complete a self-report questionnaire including measures of wellbeing when 14, 16, and 18 years old. In adults from the ANTR and for those of the YNTR who are 18 years and older, every two/three years, longitudinal survey data about lifestyle, personality, psychopathology, and wellbeing in twins and their families are collected (Ligthart et al., 2019).

In the current study, we used parent-reported data on quality of life of the child when the child is 5, 7, 9, 10, and 12 years old, self-report data from adolescents when they are 14, 16, and 18 years old, and self-report data from adults from surveys 8 (2008), 10 (2013), 11 (2013), 12 (2015), and 14 (2019), which we reorganized in age cohorts. The sample included in this study consists of twins and their siblings for whom wellbeing data were available in one or more surveys ($n = 46885$, 56% females, $n_{twins} = 42\,505$, $n_{siblings} = 4380$). The average number of surveys completed per person was 1.66 (S.D. = 0.79, range = 1–5). In Table 1, the number of surveys, number of complete twin pairs, and siblings per age category can be found. In this cohort-sequential design, the availability of one or multiple surveys per person at different ages allows us to investigate wellbeing across

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Table 1. Number of surveys, complete twin pairs and descriptive statistics

Age	*N* surveys	MZ complete pairs	DZ complete pairs	Siblings	MZ cor	DZ cor	Mean WB	S.E. WB
5	7970	1572	2375	0	0.92	0.88	8.65	0.01
7	7514	1508	2213	0	0.84	0.69	8.43	0.01
10	11 160	2122	3385	0	0.82	0.66	8.36	0.01
12	11 178	2042	3476	0	0.82	0.64	8.26	0.01
14	7228	1102	1778	297	0.45	0.24	7.96	0.01
16	6407	912	1321	585	0.49	0.22	7.76	0.02
18	7103	957	1186	810	0.39	0.15	7.62	0.01
19–24	6048	673	713	1121	0.34	0.13	7.59	0.02
25–30	2938	380	247	393	0.34	0.25	7.68	0.02
31–40	4078	579	385	337	0.31	0.12	7.74	0.02
41–50	2921	390	233	237	0.25	−0.02	7.62	0.03
51–60	1768	326	160	125	0.26	0.16	7.65	0.03
61+	1496	300	133	103	0.20	0.24	7.79	0.03

MZ, monozygotic; DZ, dizygotic; *r*, correlation; WB, wellbeing.

the lifespan without the need to have data of all participants across the entire lifespan.

Measures

Wellbeing was assessed as quality of life with the Dutch version of Cantril's Self-Anchoring Striving Scale (Cantril, 1965). Parents (age 5, 7, 10, and 12) or participants themselves (from 14 years) were shown a ladder with 10 steps and to indicate the step of the ladder at which they place their lives in general. The top step (10) indicated the best possible life you can imagine, and the bottom step the worst possible life you can imagine. In one survey (survey 14), the item was scored on a scale from 0 to 10 instead of 1 to 10. Since almost no participants scored a 0 ($n = 3$) or 1 ($n = 2$; together <0.2%) in this survey, these two answer options were pooled together to consistently score from 1 to 10 across the different surveys.

Reorganization of the data

We reorganized the available wellbeing data in groups based on age, similar to earlier longitudinal NTR studies (Kan et al., 2013; Li-Gao et al., 2022; Nivard et al., 2015). Depending on the availability of wellbeing data across different ages of twins and their siblings, we created age bins spanning a minimum of 2 years. This resulted in 13 age categories: 5, 7, 10, 12 (parent ratings) and 13–15, 15–17, 17–19, 19–25, 25–30, 30–40, 40–50, 50–60, and 60+ years old (self-ratings, see Table 1 for the sample sizes). For the childhood data, we used only the mother report data in the model due to availability of largest sample sizes. (At age 5, only maternal ratings were available. At age 7, 10, and 12, for respectively 92, 96, and 93% of the children maternal reports were available compared to 53%, 55%, and 54% of paternal reports.)

Statistical analysis

Analyses were preregistered and can be found at https://osf.io/w6xzd. To test for gender differences in the average wellbeing across the lifespan, we computed the means separately for females and males, and used *t* tests to test the differences in means.

Next, to analyze the phenotypic stability of wellbeing, we analyzed the trend and computed the correlations, i.e. tracking coefficients, between the age groups, using generalized estimating equation (GEE) models (van der Zee, van der Mee, Bartels, & de Geus, 2019). In a GEE, we included family number as random factor to correct for family structure, wellbeing at time point n as independent variable, and wellbeing at time point $n + 1$ as dependent variable. We scaled the wellbeing scores to a mean of 0 and standard deviation of 1, therefore the β's can be interpreted as correlations.

Next, to investigate the innovation and stability of the effects of genetic and environmental factors over time, we applied a genetic simplex longitudinal model to the data (Boomsma & Molenaar, 1987) in OpenMx (Boker et al., 2011). To increase the power of the model, if available, we included data on the sibling closest in age to the twins as well and extend the model to a twin-sibling model. In the simplex model (see Fig. 1), the total variance in wellbeing within each age category is decomposed into genetic and environmental components according to the classical twin design (Boomsma, Busjahn, & Peltonen, 2002). In the classical twin design, the difference in genetic relatedness of monozygotic (MZ) twin pairs (share [nearly] all genes) and dizygotic (DZ) twin pairs (share on average half of their segregating genes) can be used to decompose the phenotypic variance of traits into additive genetic variance (A; variance explained by all alleles that influence the phenotype via a linear model), non-additive genetic variance (D; variance due to interactions between alleles), shared environmental variance component (C; variance shared by family members), and a non-shared environmental component (E; variance unique for an individual). In the classical twin design, the effects of C and D cannot be estimated simultaneously; therefore, a choice for an ADE or ACE model is made based on the pattern of twin correlations. An ADE model is appropriate if twice the DZ correlation (rDZ) is smaller than the MZ correlation (rMZ), $2 \times \text{rDZ} < \text{rMZ}$.

Furthermore, in the simplex model, across the age categories, the transmission of genetic and environmental factors from one

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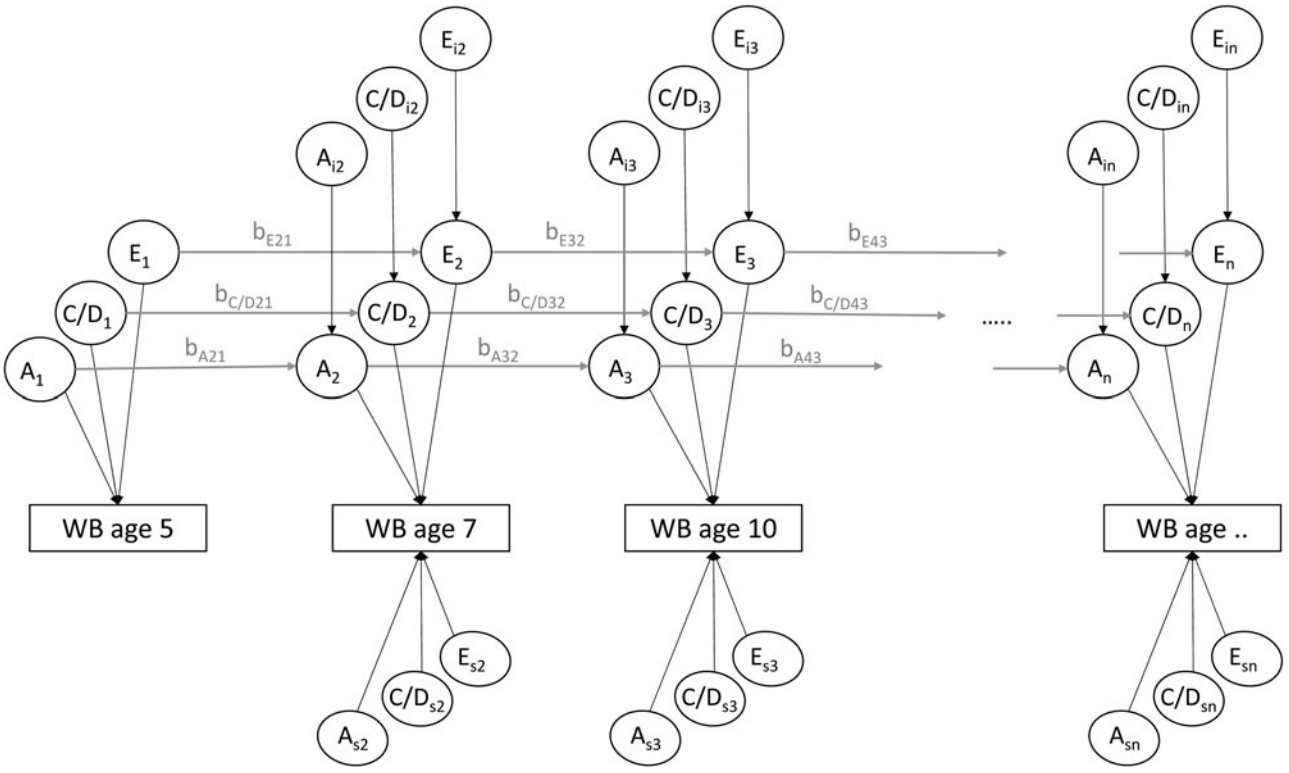

Figure 1. Genetic simplex model for wellbeing (WB). The variance within an age category is decomposed in variance transmitted from the last age category (stability: grey beta's) and new influences, divided into innovation components ($A_i/C/D_i/E_i$) and occasion-specific components ($A_s/C/D_s/E_s$) to the variance. A, additive genetic effects; C, shared environmental effects; E, unique environmental effects; WB, wellbeing.

age to the next and the extent to which new genetic and environmental factors (innovations) arise is estimated. The genetic simplex model results in estimates for (1) stable genetic and environmental effects across ages (transmission), (2) new genetic and environmental effects that are transmitted to the next age (innovation), and (3) age-specific variance. The stability is estimated with regressions between the genetic and environmental factors at two subsequent age categories (transmission, the grey betas in Fig. 1: To what extent is genetic or environmental variance explained by genetic or environmental variables in the previous age category?). Innovation variance is reflected in the top part of Fig. 1(Ai/C/Di/Ei). Innovation variance is variance new to the age category that is transmitted to the next age, i.e. this does explain part of the variance at the next age. Occasion-specific variance (lower part of Fig. 1, indicated as As/C/Ds/Es) is variance new to the age category that is not transmitted to the next age category (transient or age-specific variance).

Besides the estimates for transmission variance, innovation variance, and age-specific variance, we derived heritability estimates at each age group and calculated genetic and environmental correlations between the age groups.

Based on the twin correlations and earlier work in the same NTR data set (Baselmans et al., 2018), we estimated an ACE model for ages 5–12, and an AE model for ages 14 and above. Furthermore, for identification purposes, we constrained the residual variance to be equal over age.

We repeated both the computation of tracking coefficients and running the simplex model for subsets of the data with respectively only female and male participants to test if the trend in wellbeing and the influences on wellbeing across the lifespan is different depending on gender.

Results

Phenotypic stability

The average wellbeing score was the highest in childhood (WB_{age5} = 8.7, S.E. = 0.01), decreased significantly during mid- and late adolescence, with the lowest average wellbeing in young adulthood ($WB_{age19–24}$ = 7.6, S.E. = 0.02). From 25 years of age onwards, the average wellbeing score increased and then stabilized around 7.7 (see Fig. 2 and Table 1). Significant differences in average wellbeing between females and males can be seen at age 10, where girls are rated higher on their wellbeing by their mothers compared to boy (respectively 8.43 *v.* 8.33 out of 10), ages 14, 16, and 18 where adolescent boys rate their wellbeing higher (respectively M = 8.08, 7.87, and 7.69) compared to adolescent girls (M = 7.89, 7.65, and 7.55), and age 61+, where men report a higher wellbeing (M = 7.87) compared to woman (M = 7.67). However, the effects were negligible to small as indicated by the effect sizes (<0.20, see Table 2).

The tracking coefficients showed a median of β = 0.44, with a minimum of −0.19 and a maximum of 1 (see Table 3). Comparing the tracking coefficients in childhood, adolescence, and adulthood, we showed phenotypic stability in wellbeing within the childhood years (5–12, average β = 0.50), within adolescence (14–18, average β = 0.43), and within adulthood (18–61 +, average β = 0.56). The tracking coefficients between childhood and adolescence (average β = 0.18), and between adolescence and

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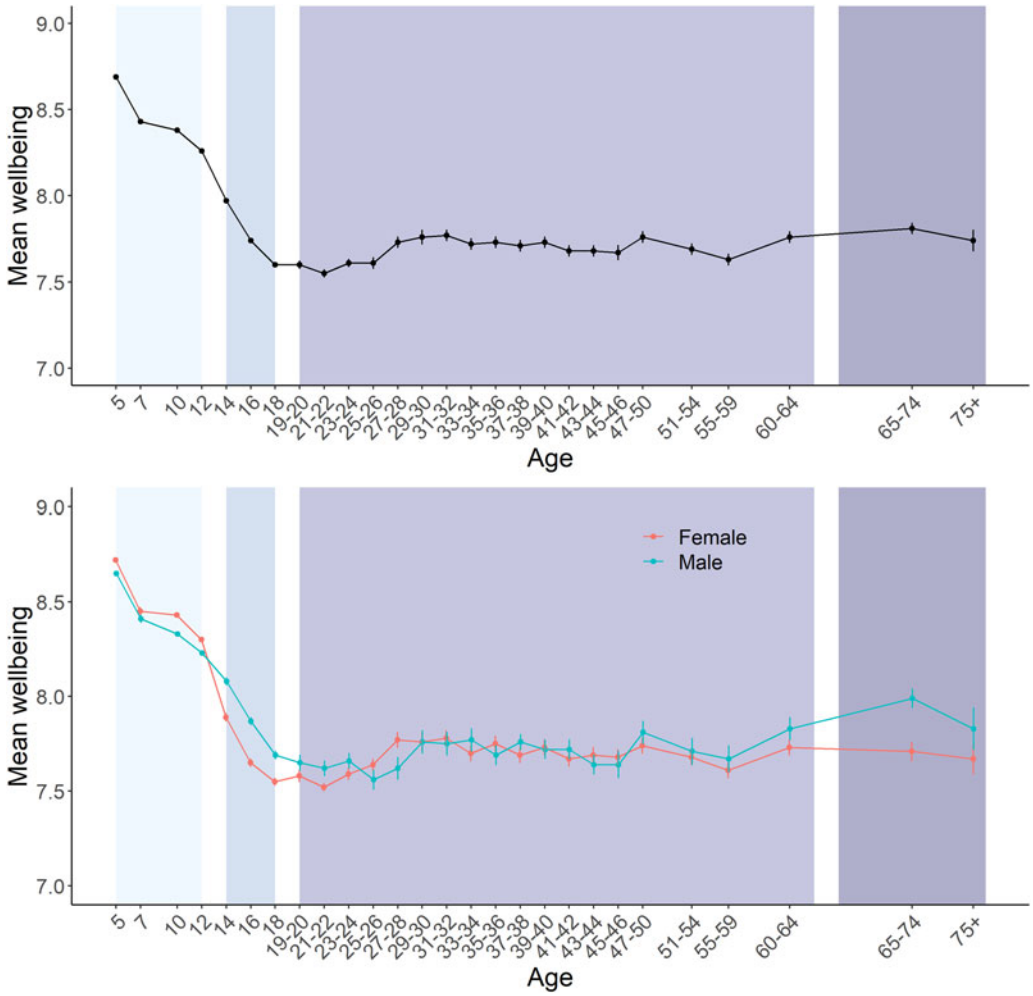

Figure 2. The phenotypic trend of wellbeing across the life span including 95% confidence intervals across all participants, and split for female and male participants. The different sections indicate childhood (mother reports), adolescence, adulthood, and late adulthood (all self-reports).

Table 2. Gender differences in average wellbeing across the lifespan

Age	*N* male	Mean male	s.d. male	*N* female	Mean female	s.d. female	*T* value	df	*p* value	Cohen's *d*
5	4005	8.65	0.94	3963	8.72	0.89	−3.34	7953.0	0.001	−0.07
7	3770	8.41	0.96	3742	8.45	0.94	−1.74	7506.7	0.082	−0.04
10	5663	**8.33**	1.01	5491	**8.43**	0.94	−5.43	11 138.1	$\mathbf{5.86 \times 10^{-8}}$	**−0.10**
12	5606	8.23	1.07	5564	8.30	1.07	−3.33	11 167.1	0.001	−0.06
14	3063	**8.08**	1.03	4158	**7.89**	1.14	7.68	6935.8	$\mathbf{1.74 \times 10^{-14}}$	**0.18**
16	2731	**7.87**	1.04	3671	**7.65**	1.16	7.83	6175.0	$\mathbf{5.88 \times 10^{-15}}$	**0.20**
18	2633	**7.69**	1.07	4467	**7.55**	1.14	5.21	5796.8	$\mathbf{1.94 \times 10^{-7}}$	**0.13**
19–24	2062	7.61	1.14	3982	7.55	1.09	2.11	4015.9	0.035	0.06
25–30	897	7.62	1.04	2040	7.71	1.05	−2.22	1735.3	0.026	−0.09
31–40	1332	7.73	1.05	2745	7.73	1.11	−0.13	2770.5	0.894	−0.01
41–50	901	7.67	1.07	2019	7.69	1.14	−0.44	1833.9	0.657	−0.02
51–60	528	7.68	1.19	1240	7.63	1.13	0.89	946.9	0.373	0.05
61+	535	**7.87**	1.06	961	**7.67**	1.08	3.52	1125.1	$\mathbf{4.48 \times 10^{-4}}$	**0.19**

Note: bold numbers indicate significant differences between the average male and female wellbeing at $p < 0.001$.

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Table 3. Tracking coefficients, with the 95% confidence intervals in brackets

Age	5	7	10	12	14	16	18	19–24	25–30	31–40	41–50	51–60	61 +
5	1												
7	**0.53** (0.49–0.58)	1											
10	**0.48** (0.44–0.52)	**0.57** (0.54–0.61)	1										
12	**0.40** (0.32–0.58)	**0.49** (0.43–0.55)	**0.54** (0.50–0.58)	1									
14	NA	NA	NA	**0.22** (0.17–0.27)	1								
16	NA	NA	NA	**0.14** (0.03–0.25)	**0.47** (0.41–0.53)	1							
18	NA	NA	NA	**0.30** (0.16–0.43)	**0.36** (0.30–0.42)	**0.47** (0.42–0.52)	1						
19–24	NA	NA	NA	−0.19 (−0.49 to 0.11)	**0.27** (0.20–0.34)	**0.39** (0.33–0.45)	**0.44** (0.39–0.49)	1					
25–30	NA	NA	NA	NA	0.24 (−0.24 to 0.72)	**0.17** (0.08–0.27)	**0.31** (0.23–0.38)	**0.41** (0.34–0.47)	1				
31–40	NA	NA	NA	NA	NA	NA	0.59 (−0.79 to 1.0)	**0.41** (0.21–0.62)	**0.51** (0.42–0.61)	1			
41–50	NA	NA	NA	NA	NA	NA	NA	NA	**0.69** (0.36–1.0)	**0.47** (0.41–0.54)	1		
51–60	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.44 (−0.35 to 1.0)	**0.62** (0.49–0.75)	1	
61+	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**0.48** (0.15–0.81)	**0.53** (0.45–0.61)	1

Note: bold numbers indicate significant tracking coefficients at $p < 0.001$.

adulthood (average $\beta = 0.36$) were lower, indicating less phenotypic stability in the transitions between these ages. Due to the sparseness of the sample data, tracking coefficients could only be computed for up to three neighboring age groups, limiting the ability to investigate the phenotypic stability between childhood and both young and late adulthood, and adolescence and late adulthood. In sensitivity analyses, we computed the tracking coefficients separately for female and male participants. As can be seen in online Supplementary Table S2, the tracking coefficients were similar to the main findings, and the differences between the results for females and males were small, with overlapping confidence intervals.

Heritability across age

The MZ and DZ twin correlations can be found in Table 1. Both the MZ and DZ correlations decrease across the life span, indicating an increase in influences of the unique environment across development. From the genetic simplex model, we obtained estimates of the total variance, the heritability, and contribution of the (shared) environment on the phenotypic variance in wellbeing across the age categories. The total variance increased slightly from 0.86 at age 5 to around 1.20–1.30 in adolescence and adulthood. As can be seen in Fig. 3 and Table 4, in the childhood years, there was a substantial influence of shared environmental effects on wellbeing ($C = 83$–45%), that decreased and became zero at 14 years. In childhood, the heritability was between 9% and 37%. The heritability was highest in adolescence, around 40–46%, and decreased toward old adulthood (h2 = 35–24%). In line with this decrease, the contribution of unique environmental effects increased during childhood, adolescence, and adulthood, from 7% to 76%.

In sensitivity analyses, we applied the simplex model on data of only female and male participants. As can be seen in online Supplementary Table S3 and Fig. S2, the heritability results were similar to the main findings, and the differences between the results for females and males were small with overlapping confidence intervals.

Innovation and stability

The estimates of innovation showed little new genetic influences in childhood, indicating that a similar set of genes is relevant for wellbeing across childhood (age 7–12). During adolescence, starting at age 12, there is an increase in new genetic influences, with innovation estimates ranging from 0.22 to 0.07. This suggests that there is a change in the genetic factors affecting wellbeing during this time. In adulthood, from age 18 onwards, there is little to no new genetic influences, indicating that the same set of genes continues to impact wellbeing across adulthood. To summarize, we report genetic stability in childhood, genetic changes in adolescence, and genetic stability in adulthood again.

With respect to the unshared environmental influences, the estimates for new environmental influences in childhood and at the start of adolescence were small, indicating mostly the same environmental influences having an effect on wellbeing. From age 14 onwards, the environmental innovation variance became substantial (innovation = 0.58–0.76), indicating changes in environmental influences on wellbeing across mid/late adolescence and across adulthood (see Fig. 3).

The transmission coefficients of stability, i.e. the genetic and environmental autoregressive coefficients, indicate the genetic and environmental stability. In childhood the stability in wellbeing was due to a mix of stable genetic and shared environmental influences. From the age of 14, the stability in wellbeing was mostly due to stable genetic effects (around 65–79%, see Table 4).

The age-specific residual variance, including measurement error, was small but significant for both genetic (0.05) and non-shared environmental variance (0.06), indicating little occasion-specific variance that is not submitted to the next age.

In sensitivity analyses, we applied the simplex model on data of only female and male participants. As can be seen in online Supplementary Table S3 and Fig. S2, the results were similar to the main findings, and the differences between the results for females and males were small.

Genetic correlations

In Table 5, the genetic and (shared) environmental correlations for wellbeing between the different age groups are reported. The genetic correlations were substantial between two adjacent age groups ($r_g = 0.51$–0.84), except for the genetic correlation between 12 and 14 years old ($r_g = 0.30$). In line with the findings on innovation, the genetic correlations between childhood and adulthood wellbeing were low ($r_g < 0.20$), whereas the genetic correlations in the adulthood age categories were high ($r_g = 0.69$–0.85).

The shared environmental correlations within childhood were relatively high ($r_c > 0.60$), indicating similar shared environmental factors playing a role in wellbeing across childhood. The unique environmental correlations were lower, both across childhood,

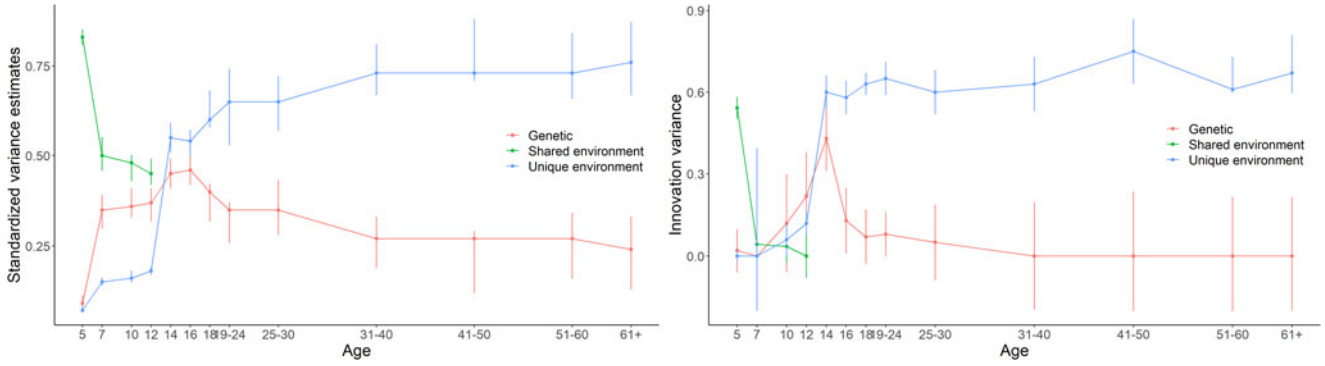

Figure 3. The standardized variance estimates at each age bin, including the heritability (proportion of variance that is explained by genetic effects), variance explained by the shared environment and the unique environment (left), including 95% confidence intervals and variance due to innovation at each age bin (right).

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Table 4. Estimates of variance components from the genetic simplex model, with the standard errors or 95% confidence intervals in brackets

	Transmission (β)			Innovation (Psi, ζ)			Residual				Variance decomposition		
Age	A	C	E	A	C	E	A	C	E	Total variance	a2	c2	e2
5	3.30 (6.07)	0.68 (0.03)	4.39 (14.2)	0.02 (0.04)	0.54 (0.02)	0.00 (0.01)	0.05 (0.04)	0.17 (0.02)	0.06 (0.01)	0.86 (0.81–0.88)	9% (7–11%)	83% (81–85%)	7% (7–8%)
7	0.82 (0.17)	0.94 (0.06)	0.68 (0.16)	0.00 (0.55)	0.04 (0.02)	0.00 (0.29)	0.05 (0.04)	0.17 (0.02)	0.06 (0.01)	0.93 (0.87–0.95)	35% (30–39%)	50% (46–55%)	15% (14–16%)
10	0.69 (0.14)	1.08 (0.08)	0.45 (0.10)	0.12 (0.09)	0.034 (0.03)	0.06 (0.03)	0.05 (0.04)	0.17 (0.02)	0.06 (0.01)	0.98 (0.94–1.00)	36% (33–41%)	48% (43–50%)	16% (15–18%)
12	0.38 (0.11)	0.16 (0.10)	0.24 (0.05)	0.22 (0.08)	0.00 (0.04)	0.12 (0.02)	0.05 (0.04)	0.17 (0.02)	0.06 (0.01)	1.14 (1.10–1.17)	37% (32–41%)	45% (42–49%)	18% (17–19%)
14	0.89 (0.08)		0.24 (0.04)	0.43 (0.06)		0.60 (0.03)	0.05 (0.04)		0.06 (0.01)	1.21 (1.16–1.24)	45% (41–49%)		55% (51–59%)
16	0.84 (0.06)		0.28 (0.04)	0.13 (0.06)		0.58 (0.03)	0.05 (0.04)		0.06 (0.01)	1.24 (1.19–1.28)	46% (42–50%)		54% (50–58%)
18	0.81 (0.05)		0.30 (0.04)	0.07 (0.05)		0.63 (0.02)	0.05 (0.04)		0.06 (0.01)	1.22 (1.16–1.25)	40% (32–42%)		60% (58–68%)
19–24	0.87 (0.09)		0.24 (0.05)	0.08 (0.04)		0.65 (0.03)	0.05 (0.04)		0.06 (0.01)	1.18 (1.13–1.22)	35% (26–37%)		65% (53–74%)
25–30	0.87 (0.17)		0.42 (0.08)	0.05 (0.07)		0.60 (0.04)	0.05 (0.04)		0.06 (0.01)	1.08 (1.01–1.14)	35% (28–43%)		65% (57–72%)
31–40	1.12 (0.26)		0.47 (0.05)	0.00 (0.10)		0.63 (0.05)	0.05 (0.04)		0.06 (0.01)	1.10 (1.03–1.16)	27% (19–33%)		73% (67–81%)
41–50	0.96 (0.20)		0.51 (0.06)	0.00 (0.12)		0.75 (0.06)	0.05 (0.04)		0.06 (0.01)	1.34 (1.22–1.39)	27% (12–29%)		73% (71–88%)
51–60	0.90 (0.21)		0.48 (0.06)	0.00 (0.11)		0.61 (0.06)	0.05 (0.04)		0.06 (0.01)	1.24 (1.16–1.36)	27% (16–34%)		73% (66–84%)
61+	NA		NA	0.00 (0.11)		0.67 (0.07)	0.05 (0.04)		0.06 (0.01)	1.21 (1.10–1.31)	24% (13–33%)		76% (67–87%)

A, additive genetic effects; C, shared environmental effects; E, unique environmental effects, NA, not applicable.

Table 5. Genetic (rG) and (shared) environmental correlations (rC/rE)

rG	5	7	10	12	14	16	18	19–24	25–30	31–40	41–50	51–60	61+
5	1												
7	0.51	1											
10	0.40	0.65	1										
12	0.25	0.41	0.54	1									
14	0.09	0.14	0.18	0.30	1								
16	0.07	0.12	0.16	0.26	0.78	1							
18	0.07	0.11	0.14	0.23	0.71	0.82	1						
19–24	0.06	0.10	0.13	0.20	0.62	0.72	0.78	1					
25–30	0.05	0.09	0.12	0.19	0.56	0.65	0.71	0.79	1				
31–40	0.05	0.09	0.11	0.18	0.55	0.64	0.69	0.78	0.84	1			
41–50	0.05	0.09	0.11	0.19	0.56	0.65	0.71	0.79	0.85	0.83	1		
51–60	0.05	0.09	0.11	0.18	0.56	0.65	0.70	0.79	0.85	0.83	0.84	1	
61+	0.05	0.09	0.11	0.18	0.55	0.64	0.69	0.77	0.83	0.81	0.83	0.82	1
rC/rE	5	7	10	12	14	16	18	19–24	25–30	31–40	41–50	51–60	61+
5	1												
7	0.64/0.19	1											
10	0.60/0.12	0.59/0.37	1										
12	0.61/0.05	0.61/0.15	0.65/0.25	1									
14	0.75/0.01	0.74/0.02	0.79/0.03	0.81/0.09	1								
16	0.00	0.00	0.01	0.02	0.22	1							
18	0.00	0.00	0.00	0.01	0.06	0.24	1						
19–24	0.00	0.00	0.00	0.00	0.02	0.07	0.27	1					
25–30	0.00	0.00	0.00	0.00	0.00	0.02	0.07	0.23	1				
31–40	0.00	0.00	0.00	0.00	0.00	0.01	0.03	0.09	0.36	1			
41–50	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.04	0.15	0.40	1		
51–60	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.02	0.08	0.21	0.49	1	
61+	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.04	0.10	0.23	0.44	1

adolescence, and adulthood, as well as within adulthood (average $r_e = 0.07$, range = 0.00–0.49). Similar to the findings of innovative environmental influences, the low environmental correlations indicate different sets of environmental influences playing a role in wellbeing for the different age categories.

Discussion

We investigated the influence of genetic and environmental influences on the stability and change of wellbeing throughout the lifespan, using longitudinal data of the NTR. Phenotypically, we showed the highest wellbeing in childhood, and a decrease in wellbeing during mid- and late adolescence which stabilized during adulthood. In childhood and adolescence, there were significant, but negligible to small ($d < 0.20$) differences in average wellbeing between females and males. In childhood and adolescence, around 40% of the individual differences in wellbeing was explained by genetic effects. The heritability decreased toward old adulthood (h2 = 35–24%), whereas the contribution of unique environmental effects increased during childhood, adolescence, and adulthood, from 7% to 76%. Genetic innovation was only observed during adolescence, between the ages 10 and 18. In childhood and adulthood, the absence of innovation indicates the same set of genes influencing wellbeing. In contrast, environmental innovation was found at every age and is especially prevalent after the age of 14 up to old adulthood. This suggests that environmental factors contribute less to the stability and can be a source of changes in individual differences wellbeing over time.

The decline in wellbeing during adolescence and stable wellbeing during adulthood in this study is in line with previous research (Goldbeck et al., 2007; Jebb et al., 2020; Orben et al., 2022). In a recent meta-analysis, it was reported that the trajectory of wellbeing depends on the measure of wellbeing, i.e. positive affect decreased across the lifespan, whereas life satisfaction was found to decrease during adolescence, increase in young and middle adulthood, and decreased again in late adulthood (Buecker et al., 2023). We used quality of life as measure, which is strongly related to life satisfaction (Bartels & Boomsma, 2009; Pavot, 2008), and showed a similar trajectory. We did not report the discussed *U* shape of wellbeing across the lifespan. The replicated drop in wellbeing during adolescence is in line with adolescence as a time of increased stress, substantial changes, identity development, and the time during which emotion regulation develops (Zeman, Cassano, Perry-Parrish, & Stegall, 2006). Furthermore, adolescence is characterized by large developmental changes in the body, brain, behavior, and interpersonal relationships (Blakemore, 2008; Goldbeck et al., 2007). For example, large physical changes occur during puberty, adolescents have more conflicts with parents, and can experience more uncertainty in friendships and peer situations. These changes and increased stress are proposed to underlie the decrease in wellbeing in adolescence.

Similarly to the phenotypic changes and stability, the simplex modelling showed that the genetic effects on wellbeing are mostly stable in adulthood, whereas new genetic variance arises during adolescence. The eventful period of adolescence and the (environmental) changes associated with this period could trigger the involvement of different genes having an influence on wellbeing, or the other way around, the expression of new genes could trigger changes in the environment, reflecting gene–environment interactions. The absence of genetic innovation from 18 years onwards indicates the same set of genes influencing wellbeing across the adult years. This finding is reassuring for large genome-wide association studies (GWAS) of wellbeing, in which samples of adults with diverse ages are pooled together (Baselmans et al., 2019; Okbay et al., 2016).

In contrast, the significant environmental innovation across the lifespan indicates new environmental influences on wellbeing at every age. Whereas the genetic stability can be seen as responsible for the phenotypic stability in wellbeing, the environmental innovation could be a source of fluctuations around the average wellbeing for a person. The specific environmental influences should be further investigated, but the results suggest that the environmental influences that are important for wellbeing differ across the lifespan, such as school and friends in childhood and adolescence to influences from employment, relationships, and possible parenthood in adulthood, to environmental influences specific to older age, for example, health-related factors. However, due to the interaction and dynamics of different environmental influences, the specific environmental variables playing a role in individual differences in the changes in wellbeing across the life span are difficult to determine. Examples of environmental influences on wellbeing are positive and negative life events. Life events, including the death or illness of a close one, marriage, having children, and getting fired affect wellbeing, but the effects depend on specific event and individuals (innate) differ in their reactivity to them (Kettlewell et al., 2020; Luhmann, Hofmann, Eid, & Lucas, 2012). Bivariate twin models have also shown that wellbeing and life events partly share genetic influences (Wootton, Davis, Mottershaw, Wang, & Haworth, 2017). Possible gene–environment correlation can result in the genetic predisposition for wellbeing seeking out environments where positive life events are more likely and negative life events are less likely, or vice versa.

The heritability estimates were mostly in line with previous research on wellbeing, i.e. around 40% in adulthood and only influence of shared environmental effects in childhood (Bartels, 2015; Baselmans et al., 2018; Nes & Røysamb, 2015). However, we did show a decreasing heritability into older adulthood, which was mostly due to an increase in environmental variance in old adulthood. The overall environmental innovation could explain this finding. For example, the effect of life events can accumulate over time, resulting in environmental effects becoming more important for individual differences in wellbeing as the differences in experienced life events increase when people are older. The increasing influence of environmental effects on individual differences across the lifespan also suggests interventions to increase wellbeing should focus on environmental influences.

Wellbeing and mental health problems, like depressive symptoms, are strongly (genetically) negatively related (Baselmans et al., 2018). This interplay and overlap of mental health problems and wellbeing across the lifespan is important to consider when interpreting the findings of this study. In line with this overlap, our results on the genetic and environmental stability and change of wellbeing are similar to the longitudinal findings reported on symptoms of anxiety and depression (Nivard et al., 2015). Anxiety and depression increased during adolescence, and similar to what was found for wellbeing, the longitudinal stability was mostly attributable to stable genetic factors. However, in contrast, only genetic innovation in adolescence was found for wellbeing, whereas for anxiety and depression during both childhood and adolescence there was significant genetic innovation. Furthermore, recently, we showed unique genetic aspects of

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wellbeing, independently from depressive symptoms in our recent GWAS-by-subtraction study (de Vries et al., 2024). The slightly different longitudinal results on wellbeing and depressive symptoms indicate as well that the patterns of genetic and unique environmental factors throughout life are not completely mirror images for wellbeing and depressive symptoms. Similarly, the results of stability and change in genetic and environmental factors across the lifespan in ADHD (Kan et al., 2013) are partly different, indicating different developmental processes for different mental health-related traits. Therefore, longitudinal studies including different stages of development across the lifespan for diverse mental health traits are needed to increase our understanding of the development and genetic architecture of these phenotypes. Moreover, to effectively disentangle the trajectories of wellbeing and, for instance, depression, studies wherein one variable is regressed against the other or with a comparable method similar to GWAS-by-subtraction are needed to investigate the distinct components of these phenotypes.

The results of this study should be interpreted in light of some limitations. We grouped longitudinal data across 25 years in bins depending on age. This allowed us to investigate genetic and environmental effects across the development and life span. However, this design does not take the generational differences into account. For example, being 20 in the 1990s is environmentally quite different from being 20 in 2023, which could influence the ratio between genetic and environmental effects on wellbeing.

Furthermore, the cohort-sequential design allows us to investigate wellbeing across the lifespan without the need to have data of all participants across the entire lifespan. However, this limited availability of surveys per person did lead to limitations in computing the tracking coefficients for the phenotypic wellbeing data. Tracking coefficients could only be computed for up to three neighboring age groups, limiting the ability to investigate the phenotypic stability between childhood and both young and late adulthood, and adolescence and late adulthood.

In addition, the longitudinal data include a switch from parent ratings in childhood (up to 12) to self-report during adolescence (age 14). At the age of 12, the same rater, i.e. the mother, reports on both twins. At the age of 14, the twins provide self-reports, each serving as distinct raters with unique perspectives and potential biases. This switch results in a large increase in absolute unique environmental variance and more innovation variance at age 14 (Lubke, McArtor, Boomsma, & Bartels, 2018). To test if rater bias of the mother during childhood affects the estimates of the simplex model, we ran multiple rater models for age 7, 10, and 12, including both the mother and father ratings of wellbeing (see online Supplementary material for details). This enables us to investigate the degree to which rater bias is present (Bartels et al., 2004a; Hewitt et al., 1992). Whereas in the main model the shared environmental factor reflects influences shared between twins, in the multiple rater model, the rater-specific shared environmental factor reflects the rater bias. The results of the multiple rater model show that rater bias by the mother accounts for at most 19–27% of the shared environmental variance for wellbeing at age 7, 10, and 12 (see online Supplementary Table S1). Therefore, the inclusion of only the mother ratings in the simplex model does seem to have affected the results to some extent, with rater bias inflating the shared environmental effects estimates. In the interpretation of the simplex model results, this rater bias should be considered. However, the inflation in the shared environmental effect is only moderate, and is not expected to meaningfully change the results of the simplex model.

Furthermore, in the longitudinal study on symptoms of anxiety and depression (Nivard et al., 2015), both maternal rating and self-reports were available at age 12. Nivard et al. (2015) reported a moderate correlation between the mother and child rating ($r = 0.35$), and the largest part of this correlation could be attributed to genetic effects (56%). This indicates that both the mother and child seem to agree to a large extent on the genetically influenced phenotype of symptoms of anxiety and depression. Unfortunately, we could not test this for wellbeing in the current study, because parent-reports and self-report data at the same age were not available. However, because of the strong overlap between wellbeing and depressive symptoms, and similar results of the simplex model of Nivard et al. (2015) and our results on wellbeing, we believe an equally strong overlap between maternal and self-reports will be present.

Finally, in the current study, we included a large Dutch sample with a mostly high socio-economic status. Most of the research on the stability in wellbeing is performed in (white) samples from such high-income Western countries, i.e. Europe and the USA (Buecker et al., 2023). However, there are national differences in the average level of wellbeing experienced across the world (Helliwell et al., 2023) as well as cultural and societal differences in the experience of wellbeing (Joshanloo, 2014; Lomas et al., 2022; Tov & Diener, 2009). Western societies, like West-Europe and the USA, interpret wellbeing as more individualistic notions, whereas Eastern cultures emphasize the communal form of wellbeing, where wellbeing of the group is more important than the individual alone (Hitokoto & Uchida, 2018). Future research should therefore investigate the trajectory and stability of wellbeing in different cultures and different income levels to be able to generalize the findings across populations.

Conclusion

To conclude, using longitudinal data of a large twin-sibling sample, we showed that, on average, wellbeing decreases in adolescence and reaches a relatively stable level in adulthood. Similarly, the individual differences in wellbeing stabilize in adulthood, with little to no new genetic effects emerging after 18 years. However, there is continuing environmental innovation influencing individual differences in wellbeing throughout life. These results led to a better understanding of the stability and change in the sources of individual differences in wellbeing across the lifespan. The results can help to develop interventions to increase wellbeing at the most effective time in life and indicate the need for future research into the specific environmental influences at different ages.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0033291724000692

Data availability statement. The preregistered analytical plan and the online Supplemental materials are shared on OSF (https://osf.io/w6xzd). The data that support the findings of this study can be requested via the Netherlands Twin Register (https://ntr-data-request.psy.vu.nl)

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Author contributions. Lianne P. de Vries: conceptualization, methodology, formal analysis and investigation, writing – original draft preparation. Dirk H. M. Pelt: conceptualization, writing – review and editing. Meike Bartels: conceptualization, writing – review & editing.

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Ethical standards. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration. The data collection was approved by the ethical committee of the VU medical center. Informed consent was obtained from all individual participants included in the study.

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