

The nature of behavioural correlates of healthy ageing: a twin study of lifestyle in mid to late life

Matt McGue,^{1,2*} Axel Skytthe^{2,3} and Kaare Christensen^{2,3,4}

¹ Department of Psychology, University of Minnesota, Minneapolis, MN USA, ²Department of Epidemiology, Institute of Public Health, University of Southern Denmark, Odense, Denmark, ³The Danish Twin Registry and Danish Ageing Research Centre, Institute of Public Health, University of Southern Denmark, Odense, Denmark ⁴Department of Clinical Genetics and Department of Clinical Biochemistry and Pharmacology, Odense University Hospital, Odense, Denmark

*Corresponding author. Department of Psychology/Elliott Hall, 75 East River Road, University of Minnesota, Minneapolis, MN 55455 USA. E-mail: mcgue001@umn.edu

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Background With the greying of the industrialized world has come increased interest in identifying the modifiable lifestyle factors that promote healthy and successful ageing. Whereas many of the behavioural correlates of late-life morbidity and mortality have been identified, relatively little is known about the origins of individual differences in these factors.

Methods A sample of 12 714 twins, including both members of 3806 pairs of known zygosity, ascertained through the Danish Twin Registry and aged 40 to 80 years, completed a self-report assessment of six lifestyle factors associated with ageing: smoking, drinking, diet and physical, social and intellectual activities. Standard biometric methods were used to analyse the twin data and determine the extent to which individual differences in each of the lifestyle factors are heritable.

Results For each of the six lifestyle factors, the estimate of heritability ranged from 32% (95% CI: 19–42%) for the diet scale to 69% (62–72%) for the smoking measure. Biometric estimates of the contribution of the twins' common rearing environment were uniformly small ($\leq 6\%$). There was little evidence that standardized biometric estimates varied by gender or age.

Conclusions Individuals likely construct lifestyles in part to complement and reinforce underlying genetically influenced dispositions and talents. The heritable nature of lifestyle factors implies that the behavioural and genetic contributors to ageing processes are not necessarily conceptually distinct but rather reflect the complexity of gene-environment interplay in ageing.

Keywords lifestyle and ageing, twin study, heritability, successful ageing

Introduction

With the continued greying of the industrialized world has come an increased interest in identifying and promoting the factors that help older individuals

live a life that is not only long but also engaged, relatively disease free and psychologically satisfying. Arguably, Rowe and Kahn¹ did more than anyone to redirect the attention of gerontological researchers

away from a near exclusive focus on age-related declines in functioning towards investigating as well the lifestyle factors that promote successful ageing. The hope is that identification of the modifiable factors that mitigate age-related physiological declines will ultimately lead to lifestyle changes that support healthy ageing.^{2,3} Consistently with this perspective, the World Health Organization's 2002 world health report estimated that the burden of chronic disease could be reduced by more than one-third by eliminating 10 known risk factors,⁴ and that unhealthy behaviours have a substantial influence on morbidity and mortality.⁵

Following Rowe and Kahn's landmark publication, a substantial research literature on the behavioural correlates of mortality, morbidity and late-life well-being has emerged.⁶ Not smoking or drinking heavily, remaining physically active and eating a healthy diet have all been consistently associated with longevity and various indicators of successful ageing.⁷⁻⁹ Alternatively, intellectual and social engagement also appear to be important components of the successful ageing construct, although the relevant research literature is more limited than with the other indicators.¹⁰⁻¹² Yet although we now know a considerable amount about which lifestyle factors are associated with a long, healthy and satisfying life, we know much less about how individual differences in these lifestyle factors arise. That is, why do some individuals eat a healthy diet and exercise regularly whereas others struggle to adopt healthy habits? Knowing the answer to questions such as this should help with understanding the nature of the relationship between lifestyle factors and ageing outcomes and with the design of effective lifestyle interventions.

The classical twin study, i.e. the comparison of the similarity of monozygotic (MZ) twins with that of dizygotic (DZ) twins, is 'one of the most widely used and powerful designs available to the (genetic) epidemiologist'.¹³ Specifically, a twin study seeks to characterize the nature of individual differences on an observed phenotype in terms of underlying genetic and environmental contributions. Here we report results from a study of six modifiable lifestyle factors (smoking, drinking, diet, physical activity, intellectual activity and social activity) in a sample 3806 pairs of Danish twins aged 40 to 80 years, with a goal of understanding the origin of individual differences in these factors. Although a twin study may seem an unusual approach for studying the origins of individual differences in lifestyle factors, in fact a twin study can help to characterize the complex interplay between genetic and environmental influences on ageing phenotypes and dispel the dichotomy that is sometimes made between lifestyle and genetic contributions to ageing and disease.^{14,15} In particular, a finding that lifestyle factors are heritable would suggest that, rather than being exogenous, lifestyles reflect at least in part qualities that are intrinsic to the individual.

Methods

Sample

The sample consisted of 12 714 individual twin participants from two studies undertaken under the auspices of the Danish Twin Registry (DTR). The first, the Middle Age Danish Twin (MADT) study, included 2387 twins born between 1931 and 1952 and first assessed in 1998, who completed the questionnaire used in the current study as part of a 10-year follow-up assessment. Overall, 62% of the surviving MADT intake participants (65% males and 59% females) participated at follow-up. At intake, those who participated at follow-up were on average 1.2 years younger and had higher cognitive test scores [standardized mean difference (CI), $d=0.39$ (0.33, 0.45)]; grip strength [$d=0.15$ (0.09, 0.2)]; self-rated health [$d=0.20$ (0.14, 0.26)] and lower levels of depression [$d=-0.16$ (-0.10, -0.22)] than non-participants. Although these differences indicate that participants were generally healthier than non-participants, the differences are small and participation rate was not associated with zygosity (rate varied from 61% to 63% among the three zygosity groups). The second study, also called the Middle Age Danish Twin study but given the distinct acronym MIDT, included 10 327 twins (40% of those invited to participate) born between 1931 and 1969, who completed the questionnaire as part of their initial assessment. Although the participation rate of MADT twins at the 10-year follow-up (62%) was higher than the rate of participation for the newly recruited MIDT twins (40%), the assessments they completed were identical, the birth cohorts overlapped and a comparison of comparably aged MADT and MIDT twins on the measures used here revealed minimal differences (all d less than 0.05 in absolute value). Consequently, results from the two samples are pooled here and for ease of presentation collectively referred to as MIDT. In addition to the questionnaire used in the present study, the MIDT assessment included an in-person examination and the provision of a biological specimen. The rate of participation in MIDT is lower than in previous DTR surveys, in all likelihood because participants needed to travel to an assessment centre to complete their assessment. The sample used in the present analyses included 3920 pairs of twins: 476 monozygotic males (MZM); 593 monozygotic females (MZF); 622 same-sex dizygotic males (DZM); 768 same-sex dizygotic females (DZF); 1347 opposite-sex dizygotic (OSDZ); and 114 of unknown zygosity. Additional details of the MIDT recruitment and assessment design are given by Skytthe *et al.*¹⁶

Measures

The lifestyle measures analysed in this study were derived from the MIDT self-report questionnaire, which was sent to participants' homes prior to their in-person assessment to which they were asked to

Table 1 Description of the lifestyle measures

| Scale | Number of items | Reliability | R ² | Description / sample items |
|-------------------------|-----------------|-------------|----------------|---|
| Pack-years | 2 | NA | 0.032 | Number of cigarettes, cigars, pipes smoked a day divided by 20 multiplied by the number of years smoked |
| Drinks per Week | 5 | NA | 0.136 | Average number of beers, wine, hard liquor drinks and dessert wine drunk each week in past half year |
| Diet | 5 | 0.56 | 0.054 | How often do you eat fruit? How often do you eat raw vegetables? |
| Physical Activities | 5 | 0.64 | 0.021 | How often do you run, work out, do aerobics? How often do you cycle at least 3 km? |
| Intellectual Activities | 8 | 0.67 | 0.013 | How often do you take a course or participate in study group? How often do you read a book, news magazine or technical report? |
| Social Activities | 5 | 0.71 | 0.039 | How often do you visit family or friends at their home? How often do you participate in a party or other social event? |

R² gives the square multiple correlation between that measure and Age, Squared Age and Sex. Twin and biometric analyses were based on the residuals from this regression analysis. Reliability estimate is based on internal consistency, alpha, method. NA, reliability estimation not applicable.

bring the completed questionnaire. The measures used in the present study are described in Table 1 and included: (i) Smoking Pack-years, which was derived from responses to the number of cigarettes, cigars or pipes smoked in a typical day and the number of years smoked (lifetime non-smokers were coded 0 on this measure); (ii) Drinks Per Week—the number of alcoholic drinks (beer, wine and hard spirits) typically consumed each week (non-drinkers were coded 0 on this measure); (iii) Diet—self-reported frequency of consuming fruit or vegetables (on an 8-point scale ranging from 1 = Never, to 8 = Four or more times a day); (iv) Physical Activities—self-reported frequency of engagement in exercise and other non-work related physical activities; (v) Intellectual Activities—self-reported frequency of engagement in reading and other forms of intellectual activity; and (vi) Social Activities—self-reported frequency of engagement in social activities with friends and families. Items from the latter three activities scales were all answered on a 5-point scale (ranging from 1 = Never to 5 = Daily), and adapted from a longer activities questionnaire¹⁷ using an exploratory factor analytic approach. Due to a small number of missing responses to individual items on the lifestyle scales, available sample sizes vary slightly from measure to measure. Except for the Intellectual Activities scale, which correlated 0.45 with educational attainment, all of the lifestyle scales were only minimally associated with socioeconomic background (all other correlations with educational attainment ≤ 0.22). Zygosity in same-sex twin pairs was determined from responses to four questions on physical similarity that the twins completed in an earlier DTR survey. This method of zygosity determination has been found to have an accuracy of greater than 95% in

predicting zygosity as determined from DNA markers.¹⁸

Statistical analysis:

Twin data were analysed using standard biometric methods.¹⁹ We first estimated the twin correlations and then determined whether the magnitude of each correlation varied by age using the Defries-Fulker method²⁰ as described by McGue and Christensen.²¹ Next we used Mx²² to fit a standard ACE model to the twin data, where A corresponds to the additive genetic contribution to phenotypic variance, C to the shared environmental contribution (i.e. the effect of environmental factors twins share by virtue of their common rearing and assumed to be independent of zygosity) and E to the non-shared environmental contribution (i.e. the effect of environmental factors the twins do not share). In the standard ACE model, the total phenotypic variance is assumed to be an additive function of A + C + E; the MZ twin covariance is assumed to be equal to A + C; and the DZ twin covariance is assumed to be 1/2 A + C. The parameters of the ACE model were estimated from the observed twin variance-covariance matrices using maximum likelihood as implemented in the Mx software. The ACE components were initially allowed to vary by gender and then constrained to be equal to determine whether the variance components varied by gender. In an analogous fashion, we determined whether the variance components varied as a function of age using a quantitative moderator analysis in Mx.²³ Because the Packyears and Drinks measures were positively skewed, both were log-transformed prior to analysis and all results reported here other than descriptive statistics are based on the log-transformed scores.

Table 2 Descriptive statistics [mean (SD)] and sample sizes

| | Monozygotic | | Same-sex dizygotic | | Opposite-sex dizygotic ^a | | Unknown zygosity | | Total |
|-------------------------|----------------|----------------|--------------------|---------------|-------------------------------------|-------------------|------------------|----------------|----------------|
| | Male | Female | Male | Female | Male | Female | Male | Female | |
| Total | 1358 | 1479 | 2335 | 2388 | 2221 | 2581 | 157 | 195 | 12 714 |
| No. of pairs | 476 | 593 | 622 | 768 | 1347 ^a | 1347 ^a | 42 | 72 | 3920 |
| Age (years) | 59.0 (9.6) | 56.2 (10.0) | 60.2 (9.3) | 58.5 (9.5) | 59.9 (9.6) | 59.0 (9.4) | 60.7 (9.8) | 56.6 (10.1) | 58.9 (9.6) |
| Pack-years | 13.1 (19.0) | 8.3 (13.4) | 14.2 (20.4) | 9.1 (14.1) | 13.9 (19.3) | 9.2 (13.8) | 11.1 (17.5) | 6.9 (11.6) | 11.2 (17.0) |
| Drinks per Week | 10.3 (9.4) | 5.4 (5.8) | 11.1 (9.9) | 5.6 (5.8) | 10.7 (9.5) | 5.6 (5.5) | 10.9 (17.5) | 5.0 (4.9) | 8.1 (8.4) |
| Diet | 20.5 (4.5) | 22.4 (4.2) | 20.7 (4.3) | 22.4 (4.0) | 20.5 (4.4) | 22.6 (4.1) | 21.2 (4.3) | 21.9 (4.2) | 21.6 (4.3) |
| Physical Activities | 12.9 (4.2) | 14.0 (4.0) | 12.9 (4.2) | 14.0 (4.0) | 12.9 (4.3) | 14.0 (4.0) | 12.6 (4.1) | 13.3 (4.1) | 13.5 (4.2) |
| Intellectual Activities | 21.8 (4.3) | 22.1 (4.0) | 21.9 (4.2) | 22.1 (4.1) | 21.8 (4.3) | 22.1 (4.1) | 22.3 (3.9) | 21.8 (4.2) | 22.0 (4.2) |
| Social Activities | 14.1 (2.2) | 14.9 (2.2) | 13.9 (2.2) | 14.6 (2.2) | 13.8 (2.3) | 14.7 (2.2) | 14.0 (2.3) | 14.7 (2.2) | 14.3 (2.2) |

^aPairs in the male and female opposite-sex dizygotic columns refer to the same pairs.

Because the main effects of age and sex can confound biometric analysis,²⁴ each of the six lifestyle factors was regressed on sex, age and squared age and the residuals from these regressions used in all analysis of twin similarity.

Results

Descriptive statistics

Table 2 gives descriptive statistics for all study measures in the overall sample, as well as broken down by zygosity group. The sample is on average 59 years old with a range of 40 to 80 years. A total of 3920 complete twin pairs are included in the sample, although zygosity could not be determined for 114 of these pairs, leaving 3806 pairs available for the biometric analysis. The overall mean of 21.6 on the Diet scale corresponds to an average frequency of approximately a few times a week for consuming each of the five healthy items; the average scores for the Physical, Intellectual and Social Activities scales correspond to engaging in each of the items on those scales an average of a few times a month. Sex was moderately associated with all six of the lifestyle measures (standardized mean difference ranging from 0.28 to 0.65) except for the Intellectual Activities scale for which the effect size of 0.08 is small. On average, women reported healthier lifestyles than men, i.e. less smoking and drinking and more frequent eating of fruits and vegetables and engagement in physical, intellectual and social activities. Age was also moderately

associated with all of the measures [correlation ranging from -0.06 (-0.05 , -0.07) for Physical Activities to 0.19 (0.18 , 0.20) for Drinks], with younger individuals reporting on average healthier lifestyles on all six factors. The squared multiple correlation (R^2) between each scale and age, squared age and sex is reported in Table 1. These demographic factors accounted for between 1.3% (for Intellectual Activities) to 13.6% (for Drinks) of the variance in the scales. All subsequent twin analysis is based on the residuals from these regressions.

Twin correlations

Twin correlations for the six lifestyle measures are reported in Table 3. Several general trends are apparent. First, correlations among MZ twins are consistently higher than those for same-sex DZ twins, implicating genetic factors. Second, correlations among same-sex twins are generally similar across the male and female subsamples, suggesting that the genetic and environmental contributions to phenotypic variance are of similar magnitude in males and females. Third, the opposite-sex DZ twin correlations are slightly less than the corresponding same-sex DZ correlations, suggesting that the genetic factors underlying variance on the six measures may differ somewhat for men and women. The purpose of the biometric analysis is to investigate these impressions within a formal statistical framework. Prior to undertaking the biometric analyses, however, we used DeFries-Fulker analysis to model the MZ, same-sex

Table 3 Twin correlations and standardized variance component estimates

| | Twin correlations | | | | | Standardized biometric estimates | | | |
|-------------------------|-------------------|---------------|---------------|---------------|----------------|----------------------------------|----------------------|----------------------|----------------------|
| | (number of pairs) | | | | | (95% CI) | | | |
| | MZM | DZM | MZF | DZF | DZOS | a ² | c ² | e ² | r _a |
| Packyears | 0.66 (460) | 0.32 (606) | 0.71 (566) | 0.35 (737) | 0.20 (1292) | 0.69 (0.62, 0.72) | 0.00 (0.00, 0.07) | 0.31 (0.28, 0.34) | 0.57 (0.37, 0.72) |
| Drinks per Week | 0.38 (446) | 0.23 (579) | 0.47 (533) | 0.25 (661) | 0.18 (1220) | 0.45 (0.28, 0.49) | 0.01 (0.00, 0.13) | 0.54 (0.51, 0.61) | 0.83 (0.23, 1.0) |
| Diet | 0.33 (475) | 0.18 (620) | 0.43 (592) | 0.23 (766) | 0.19 (1339) | 0.32 (0.19, 0.42) | 0.06 (0.00, 0.17) | 0.62 (0.58, 0.67) | 0.84 (0.13, 1.0) |
| Physical Activities | 0.45 (474) | 0.20 (620) | 0.45 (593) | 0.25 (762) | 0.13 (1342) | 0.45 (0.32, 0.49) | 0.01 (0.00, 0.11) | 0.55 (0.51, 0.59) | 0.54 (0.02, 1.0) |
| Intellectual Activities | 0.55 (473) | 0.29 (617) | 0.50 (588) | 0.33 (765) | 0.25 (1338) | 0.47 (0.30, 0.55) | 0.06 (0.00, 0.20) | 0.47 (0.44, 0.52) | 0.79 (0.20, 1.0) |
| Social Activities | 0.35 (464) | 0.09 (616) | 0.40 (590) | 0.28 (766) | 0.17 (1336) | 0.38 (0.24, 0.43) | 0.01 (0.00, 0.12) | 0.61 (0.57, 0.66) | 0.85 (0.57, 1.0) |

MZM, monozygotic males; DZM, same-sex dizygotic males; MZF, monozygotic females; DZF, same-sex dizygotic females; DZOS, opposite-sex dizygotic; a², additive genetic variance; c², shared environmental variance; e², non-shared environmental variance; r_a, correlation in additive genetic effects in males and females.

Estimates were based on the biometric model in which standardized estimates were constrained to be equal in males and females.

DZ and opposite-sex DZ correlations as a function of age and squared age. Of the 18 possible tests for age moderation (i.e. three correlation types by six scales), no *P*-values were <0.01 and only two were <0.05; the opposite-sex DZ correlation increased slightly with age for Packyears and the same-sex DZ correlation decreased slightly with age for Physical Activities. None of the 18 tests for the incremental moderating effect of squared age had *P* < 0.05. The modest magnitude of the age-moderated effects and the failure to find consistent effects across the three types of twin correlations in the DeFries-Fulker analysis suggest that twin similarity for lifestyle factors is stable over the age range spanned in MIDT.

Biometric Analysis

We first fitted a model in which the A, C and E components were allowed to vary by sex. Included in this model was a parameter, r_a, which is the correlation in the additive genetic factors for men and women. An r_a < 1.0 would indicate that the genetic factors underlying phenotypic variance in men and women were not the same, accounting for lower opposite-sex than same-sex DZ correlations. We subsequently fitted models in which the C components were fixed to 0 (the AE model) and the A components were fixed to 0 (the CE model). In every case, the CE model fitted very poorly (χ^2 on 3 df ranged from 39.1 for Social Activities to 299.6 for Packyears, with all *P*-values < 0.0001) but the AE model fitted well (χ^2 on 2 df ranged from 3.9 for Social Activities to 0.1 for Packyears, with all *P*-values > 0.14). Tests for gender moderation of the biometric components in the ACE

models indicated that whereas overall phenotypic variance varied by sex (males had greater variance than females), there was little evidence that the standardized estimates varied. For the test constraining standardized estimates to be equal in males and females, only for the Social Activities scale was a *P*-value < 0.05 observed (χ^2 on 2 df = 8.5, < 0.01, *P* < 0.05; all other χ^2 on 2 df were less than 5.7 with *P* > 0.05). Finally, we tested for whether there was age moderation of the biometric variance components in the AE model. Although there was evidence for an increase in phenotypic variance with age, there was little evidence that standardized estimates varied with age (for the latter all χ^2 on 1 df < 3.5, *P* > 0.05).

Table 3 reports the standardized biometric variance component estimates (a², c² and e²) along with estimates of r_a from the ACE model in which the standardized components had been estimated under the constraint that they were equal in the male and female subsamples. We include standardized estimates of C, even though they are consistently small, to ensure that the standardized estimate of heritability (a²) is not upwardly biased by assuming a small value of C is zero by dropping it from the model. The estimated heritabilities are generally moderate in magnitude except for Packyears in which case the estimate is substantial (a² = 0.69).

Discussion

Biometric analysis of six modifiable lifestyle factors (smoking, drinking, diet and physical, intellectual

and social engagement) in a sample of 3806 Danish twin pairs aged 40 to 80 years support the following conclusions. First, there is a moderate to strong genetic contribution to individual differences in each of the lifestyle factors. Second, the magnitude of this genetic contribution does not appear to be moderated by either sex or age. Third, whereas environmental factors contribute substantially to individual differences in all of the factors, the relevant environmental factors appear to be those that contribute to differences rather than similarities among twins.

Before discussing the relevance of each of these findings to research on the relationship of lifestyle with ageing, it is important to acknowledge the limitations of our research design. First, our lifestyle assessment was brief and based exclusively on self-report. The scope of our lifestyle assessment was constrained by the limited amount of time that could be devoted to any single assessment given the multiple research objectives in MIDT. Despite their brevity, however, the self-report scales have reasonable psychometric properties and a coherent pattern of empirical relations in a large sample. Nonetheless, we recognize that, for example, a 5-item self-report cannot provide a comprehensive assessment of an individual's dietary habits. Second, the breadth of our lifestyle assessment is also limited. Nonetheless, the six factors we considered feature prominently in most discussions of behavioural contributors to successful ageing. Third, even though a classical twin study is the most widely used design within behavioural genetics and there is considerable empirical support for its essential validity,²⁵ our estimates of heritability and shared environmental influence require the assumption that MZ and DZ twins share environmental similarity to the same degree and that all genetic effects are additive. Replication of findings from twin studies using alternative designs that do not require these assumptions should always be sought. Fourth, the rate of participation in the survey was lower than in our previous Danish surveys. Because a subset of the sample had completed a previous survey, we were able to identify factors associated with participation, showing that in general participants were better functioning than non-participants. Nonetheless, the differences between participants and non-participants were generally small, suggesting that any selection biases are likely to be minimal. Finally, our estimates of heritability are cross-sectional and so do not address the important question of whether lifestyle change is also heritable. To our knowledge, there is no relevant longitudinal research on lifestyle change that would allow us to evaluate this issue.

On the one hand, our finding that lifestyle factors are moderately to substantially heritable is not altogether unexpected in the context of a vast behavioural genetic literature demonstrating that most behavioural characteristics are at least somewhat heritable.²⁶ Indeed, for at least two of the factors we

investigated, smoking and drinking, there is a large behavioural genetic literature documenting heritable contributions.^{27,28} Nonetheless, the relevant behavioural genetic literature, including that for smoking and drinking, is overwhelmingly based on adolescent or young adult samples. It may be that environmental influences cumulate over time, so that the relative contribution of heritable factors decreases with age. In this case, we might expect little heritability in a sample such as ours, which is predominantly elderly. Our finding of moderate to strong heritability for lifestyle factors, that is stable over age, is however consistent with earlier research showing that heritable contributions to cognitive ability, physical ability and emotional health are stable throughout most of the latter half of the lifespan.²¹ It is our hypothesis that the stability of heritable influences on age-related outcomes like cognitive and physical ability are a consequence in part of the individual's ability to create lifestyles that complement and reinforce their underlying genetically influenced talents.

It is reasonable to ask why a gerontology researcher should be interested in the heritability of ageing-related lifestyle. We believe there are several reasons why our findings are broadly relevant but, before discussing this issue, we would like to emphasize what our findings do not imply. Our finding that lifestyle is heritable does not mean that lifestyle is somehow genetically fixed. Indeed, it seems nearly self-evident that factors such as smoking, diet and physical activity are amenable to intervention even if effecting behavioural change may be challenging in some cases.⁵ It is an unfortunate feature of the behavioural genetic literature that some researchers have erroneously interpreted heritability as evidence for the genetic determination of behaviour.

Our finding a heritable basis to ageing-related lifestyle factors has several implications for understanding the nature of these factors. First, our results imply that lifestyle, rather than being extrinsic and distinct from genetic factors,⁶ is at least in part a manifestation of qualities that are intrinsic to the individual. Lifestyles are shaped by individual decisions that are likely guided by heritable personality and ability traits. Individuals who are socially skilled and enjoy the company of others are, for example, more likely to seek out the company of others than are individuals who are introverted and socially awkward.²⁹ Similar mechanisms likely influence how physically and intellectually active we are or even whether we eat fruits and vegetables or drink a lot of alcohol. We might also expect that lifestyle choices reflect in part early family experiences, yet become increasingly differentiated as we get older. We do not, however, find support for either proposition in our biometric analyses, although the inference on these matters here is admittedly indirect. The shared environment corresponds to the effects of exposures that the two members of a twin pair share regardless of their zygosity.

This would include the effects of factors the two twins shared by virtue of being reared in the same home (e.g. the socioeconomic level of the home, their parents' diet and drinking behaviours). The standardized estimates of the contribution of shared environmental effects to the six lifestyle factors were uniformly small.

These findings are consistent with a larger behavioural genetic literature showing that shared environmental contributions to various behavioural outcomes are minimal in adulthood³⁰, but are nonetheless somewhat unexpected for a phenotype such as lifestyle on which parents are thought to have an important influence,³¹ at least in early life.³² Our failure to find shared environmental effects may largely be owing to the age of our sample; most of the twins stopped living together decades before their participation in MIDT. It is also of interest that the lifestyle factors we investigated may affect gene expression through epigenetic processes.^{33,34} How inherited factors and epigenetic processes combine to affect ageing processes is an active area of research and our findings suggest that lifestyle factors may be a useful target of that research.

Our results also have implications for interpreting twin studies of ageing-related outcomes. A fundamental assumption of twin studies is that MZ twins are no more environmentally similar than DZ twins. Yet our findings indicate that MZ twins are more

similar in their diets, level of activity and drinking and smoking behaviours than are DZ twins. This raises the possibility that twin similarity for ageing outcomes such as cognitive and physical functioning may be due in part to the similarity in lifestyles twins adopt. Ironically, the study of twin lifestyle differences may provide a powerful design for investigating the nature of the associations between lifestyle and ageing outcomes, given the existence of a heritable basis to both. For example, if increased physical or social activity contributes to healthy ageing then we should find that within MZ twins pairs discordant for level of activity, the more active twin is healthier.³⁵ It is for this reason that the discordant-twin design is seeing increased application in ageing research.³⁶

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KEY MESSAGES

- Lifestyle factors such as smoking, diet and physical activity contribute to late-life functioning.
- Based on findings from a large twin study, these lifestyle factors all appear to be at least partially heritable.
- Our lifestyles are not entirely exogenously determined but rather also reflect our intrinsic interests and talents.
- Heritability of late-life functioning may in part be due to heritable contributions to lifestyle factors.

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