Temperament in young adulthood and later mortality: prospective observational study

P McCarron, D Gunnell, G L Harrison, M Okasha, G Davey Smith

Study objective: To determine the association between a clinician assessment of temperament in early adulthood and cause specific mortality.

Design: Prospective observational study.

Setting: Glasgow University.

Participants: 9239 male former students aged 16–30 (mean 20.5) years who participated in an ongoing health survey from 1948–68. A physician recorded free text assessment of temperament, which seemed to capture aspects of personality (trait) and mental health (state), was coded into: stable, anxious, schizoid, hypomanic, odd, depressed, immature, hypochondriacal, unstable, and obsessive. Associations between temperament and mortality were investigated using Cox proportional hazards models.

Main results: There were 878 deaths. Most students—8342 (90.3%)—were assessed as stable, the remaining 897 (9.7%) having at least one, and 103 (1.1%) having more than one, temperament type. The most common temperament was anxiety, recorded in 520 (5.6%) students. In multivariable analyses, having at least one temperament type was associated with increased all cause and stroke mortality, hazard ratios (95% confidence intervals): 1.23 (1.01 to 1.50) and 1.95 (1.06 to 3.59) respectively, compared with stable students. Students with more than one temperament type had higher risk of death from: all causes, 2.05 (1.36 to 3.09); stroke, 3.26 (1.01 to 10.56); and cancer, 2.90 (1.62 to 5.20). Anxiety was positively associated with all cause and cancer mortality, respective hazard ratios: 1.36 (1.07 to 1.72) and 1.51 (1.04 to 2.20). Men labelled hypomanic had increased cardiovascular mortality risk, 1.90 (1.05 to 3.44).

Conclusions: Markers of early adult psychological distress are associated with increased mortality. Mechanisms underlying these associations require investigation.

While it is well known that people with psychological disorders are at increased risk of suicide, evidence is accumulating that symptoms of both minor and major psychological abnormality are also associated with increased mortality from other causes. In particular, the risk of cardiovascular disease (CVD) is greater among people with: depression, anxiety and worry, anger and hostility. There has been less research into the association of psychological morbidity with cancer, and findings thus far are inconsistent.

Most studies to date have examined the association between mental health recorded in middle age and subsequent disease risk. In these studies the possibility of reverse causality—physical disease that is already present, although perhaps clinically silent, being responsible for psychological morbidity—cannot be discounted. Few studies have investigated whether psychological conditions in early life also predict mortality from causes other than suicide. In a study examining the association between childhood personality and longevity after 70 years of follow up, “cheerfulness” was inversely related to longevity. In an early report from the Harvard and Pennsylvania Alumni follow up, several traits including anxiety, sleeplessness, feeling self conscious, or being subject to moods were more common in people who subsequently died from coronary heart disease (CHD) or stroke. More recent follow up of the same cohort has confirmed that exhaustion, as reported in an earlier univariable analysis is also an independent risk factor for CHD.

In another college based study, students with higher hostility scores were more likely after 21–23 years follow up to consume more caffeine, to have a greater body mass index (BMI) and a higher cholesterol:HDL cholesterol ratio and to be current smokers compared with those with lower hostility scores. Finally, in a population based cohort study, high hostility level predisposed young adults to coronary artery calcification—a marker of atherosclerosis and coronary risk factors in older adults—after 10 years follow up.

Investigation of the relation between measures of personality or psychological morbidity in early adulthood and later cause specific mortality may improve understanding of the aetiology of chronic diseases and thus help inform future preventative initiatives. Such studies are important, particularly in the light of recent findings that indicate that the prevalence of mental disorder is high; in the National Psychiatric Morbidity Survey in Great Britain, 16.6% of 20–24 year olds were identified as having a psychiatric disorder. We report on the association between a crude assessment of young adult personality and mental health and cause specific mortality in a large cohort of male students attending Glasgow University between 1948 and 1968.

METHODS

Design

Full details of the Glasgow Student Cohort Study are presented elsewhere. Briefly, students attending Glasgow...
University between 1948 and 1968 were invited to participate in a medical examination carried out in the Student Health Department. A questionnaire was administered and a clinical examination was carried out. Data collected included socio-demographic information, details of health behaviours, and medical history, and, in addition, measurements of blood pressure, height, and weight.

Measurement of exposures
A box in the health questionnaire labelled “personality” prompted a free text assessment by the examining physician. The assessments recorded in this box indicate that the physicians were assessing a mixture of concurrent psychiatric symptomatology, with free text comments such as “depressed”, “anxious” and, “hypomanic”, as well as aspects of students' personality at the time of examination, with comments such as “immature” and “schizoid”. As the examining doctors were not recording the more precise construct of personality in itself and as we recognise the limitations of the measure in this regard, we have chosen to refer to the original physician assessment as temperament throughout the remainder of the paper. Before the analyses, a psychiatrist (GH) and an epidemiologist with an interest in psychiatric disorders (DG) coded the temperament assessments into 11 broad categories. Students who did not have an atypical temperament assessment were categorised as “stable”—the term used by the examining physicians. Because it appears that these doctors were attempting to detect atypical temperaments we have chosen to include in the “stable” category students who were variously described as “cheerful”, “pleasant”, “quiet”, “sensible”, and “well balanced”. Further justification for this is that where these terms occurred in the original assessments it was almost always in combination with the term “stable”. Where the category term or a variation of that term (for example, depressive, depressed) appeared, the person was assigned to that category. Other summary descriptive statements (variations given in parentheses) were assigned to specific categories as follows: (i) anxious, (worried, sleeplessness); (ii) schizoid, (introverted, reticent, inhibited, unemotional); (iii) hypomanic, (volatile); (iv) odd, (abnormal); (v) depressed, (breakdown, mental breakdown, up and down); (vi) immature; (vii) hypochondriacal, (preoccupied—with health related matters); (viii) unstable (emotional, emotional instability); (ix) obsessive; (x) paranoid and; (xi) inadequate.

Temperament type Number (%)  
None (stable) 8342 (90.3)  
At least one 897 (9.7)  
More than one 103 (1.1)  
Anxious 520 (5.6)  
Schizoid 238 (2.6)  
Hypomanic 120 (1.3)  
Odd 5 (0.1)  
Depressed 14 (0.2)  
Immature 55 (0.6)  
Hypochondriacal 19 (0.2)  
Unstable 30 (0.3)  
Obsessive 1 (0.0)  
Paranoid 0 (0.0)  
Inadequate 0 (0.0)

*The same person may appear in more than one row if more than one temperament trait was identified.

Two further categories were created: one for people who had at least one atypical temperament recorded, regardless of its categorisation, and another for people who had more than one category of atypical temperament recorded.

Measurement of outcome
The National Health Service Central Register (NHSCR) in Edinburgh and its English equivalent in Southport carried out tracing of the students who took part in the original examinations. Dates of birth together with surname and forename were used to trace the cohort. For people who have been successfully identified using the NHSCR we are notified of date and cause of death, area of current residence, and emigration details.

Statistical analyses
Analyses are limited to men as there have been comparatively few female deaths. Men aged over 30 years at the time of examination, or who had emigrated from the UK at an undetermined date were also excluded from the analyses. Deaths were coded using the ninth revision of the International Classification of Diseases (ICD 9). Mortality rates for each cause of death per 1000 men in each temperament category were calculated. Cox proportional hazards models were then used to examine associations between temperament and mortality from the following causes: all cause; CVD (ICD 9: 390–459); CHD (ICD 9: 410–414); stroke (ICD 9: 430–438); cancer (ICD 9: 140–208); and mortality from accidents, suicides, and violence (ICD 9: 800–999). As there were fewer than 10 deaths among those classified as depressed, immature, hypochondriacal, or unstable we did not examine cause specific mortality patterns for these category types. Age adjusted and multivariable hazard ratios were computed. The following potential confounding factors were included in the multivariable models: smoking (none, 1–10, 11–20 and>20 cigarettes per day) father’s social class (I-V), height (m) and BMI (kg/m²), and both maternal and paternal vital status (alive, dead). For all cause, CVD, CHD, and stroke mortality additional adjustment was made for systolic blood pressure (mm Hg). All analyses were performed using Stata version 7.0 (StataCorp, College Station, TX, USA).

RESULTS
A total of 11 755 male students, of whom 11 043 were aged 16–30 years at the time of examination and had data on temperament, participated in the original survey. Of these, 9239 (83.7%), have been traced, and are representative of the original participants in terms of social class, height, weight,
Table 2  Baseline characteristics* of male students according to temperament type

<table>
<thead>
<tr>
<th>Temperament type</th>
<th>Age (y)</th>
<th>SBP (mm Hg)</th>
<th>Height (m)</th>
<th>BMI (kg/m2)</th>
<th>Social class I and II (%)</th>
<th>Non-smoker (%)</th>
<th>Non-drinker (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable</td>
<td>20.5</td>
<td>130.2</td>
<td>174.8</td>
<td>21.6</td>
<td>56.0</td>
<td>66.8</td>
<td>45.0</td>
</tr>
<tr>
<td>At least one</td>
<td>21.0</td>
<td>135.4</td>
<td>174.5</td>
<td>21.0</td>
<td>54.8</td>
<td>67.4</td>
<td>53.9</td>
</tr>
<tr>
<td>More than one</td>
<td>21.3</td>
<td>136.1</td>
<td>174.3</td>
<td>20.8</td>
<td>59.0</td>
<td>67.3</td>
<td>49.4</td>
</tr>
<tr>
<td>Anxious</td>
<td>21.3</td>
<td>135.6</td>
<td>174.3</td>
<td>20.9</td>
<td>53.2</td>
<td>66.6</td>
<td>49.4</td>
</tr>
<tr>
<td>Schizoid</td>
<td>21.0</td>
<td>133.4</td>
<td>174.8</td>
<td>21.3</td>
<td>57.4</td>
<td>67.6</td>
<td>51.6</td>
</tr>
<tr>
<td>Hypomanic</td>
<td>20.9</td>
<td>143.0</td>
<td>175.3</td>
<td>20.8</td>
<td>62.7</td>
<td>69.2</td>
<td>72.4</td>
</tr>
<tr>
<td>Odd</td>
<td>19.4</td>
<td>124.6</td>
<td>176.7</td>
<td>20.7</td>
<td>40.0</td>
<td>75.0</td>
<td>50.0</td>
</tr>
<tr>
<td>Depressed</td>
<td>20.6</td>
<td>124.8</td>
<td>175.1</td>
<td>21.1</td>
<td>58.3</td>
<td>35.7</td>
<td>45.5</td>
</tr>
<tr>
<td>Immature</td>
<td>18.9</td>
<td>132.7</td>
<td>172.5</td>
<td>20.0</td>
<td>53.7</td>
<td>77.4</td>
<td>71.2</td>
</tr>
<tr>
<td>Hypochondriacal</td>
<td>22.9</td>
<td>131.0</td>
<td>173.6</td>
<td>21.5</td>
<td>55.6</td>
<td>57.9</td>
<td>14.3</td>
</tr>
<tr>
<td>Unstable</td>
<td>21.4</td>
<td>133.6</td>
<td>175.3</td>
<td>20.6</td>
<td>50.0</td>
<td>71.4</td>
<td>38.0</td>
</tr>
</tbody>
</table>

*Values are means (SD) unless stated otherwise.

Table 3  Mortality rate per 1000 men for each cause of death by temperament type*

<table>
<thead>
<tr>
<th>Temperament type</th>
<th>All causes</th>
<th>Cardiovascular disease</th>
<th>Coronary heart disease</th>
<th>Stroke</th>
<th>Cancer</th>
<th>Accidents suicide and violence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable</td>
<td>2.20</td>
<td>0.86</td>
<td>292</td>
<td>0.64</td>
<td>218</td>
<td>0.16</td>
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<tr>
<td>At least one</td>
<td>3.43</td>
<td>1.47</td>
<td>57</td>
<td>0.83</td>
<td>32</td>
<td>0.39</td>
</tr>
<tr>
<td>More than one</td>
<td>5.62</td>
<td>1.80</td>
<td>8</td>
<td>0.67</td>
<td>3</td>
<td>0.67</td>
</tr>
<tr>
<td>Anxious</td>
<td>3.80</td>
<td>1.19</td>
<td>31</td>
<td>0.67</td>
<td>15</td>
<td>0.36</td>
</tr>
<tr>
<td>Schizoid</td>
<td>3.02</td>
<td>1.39</td>
<td>31</td>
<td>0.68</td>
<td>7</td>
<td>0.39</td>
</tr>
<tr>
<td>Hypomanic</td>
<td>3.87</td>
<td>2.21</td>
<td>12</td>
<td>1.29</td>
<td>7</td>
<td>0.55</td>
</tr>
<tr>
<td>Depressed</td>
<td>7.64</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Immature</td>
<td>2.52</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Hypochondriacal</td>
<td>4.81</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Unstable</td>
<td>4.59</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Total number deaths = 745+133. Single temperament categories may include deaths that occur in more than one category. Number duplicated deaths = 24 accounted for in the “more than one” category.

DISCUSSION

Our findings show that a brief clinician assessment of a crude measure of temperament and mental health in early adulthood can predict later mortality risk. This observation points to the importance of psychological adjustment in early adulthood in predicting future mortality risk and extends...
previous findings reporting associations between mental health and mortality. This is a large prospective study in which temperament was measured many years before death occurred, ruling out the problem of reverse causality. Data on several potential confounding variables, including parental vital status, were available but controlling for these had little influence on the overall effect estimates. Apart from anxiety, few students had any other atypical temperament codings, thus decreasing the power of the analyses, as reflected in the occasionally wide confidence intervals. Also, as students attending university during the period of the study were overwhelmingly from affluent backgrounds and participants in the health survey comprised 50% of the male student population of Glasgow University, there could be selection bias. It is also possible that the associations with temperament may be different among people from poorer backgrounds than those represented in this study, because of associations between socioeconomic position and the occurrence of stressful life events, low social support, and other psychosocial factors that have economic position and the occurrence of stressful life events, presented in this study, because of associations between socio-economic position and the occurrence of stressful life events.

**Measurement and meaning of temperament**

The major limitation of the study is that the assessment of temperament was not standardised. Essentially, the assessments were impressionistic free text codings of a global assessment that seemed to capture elements of state (mental health at the time of the assessment) and trait (personality). Our stratification of these free text statements into broad categories of temperament is likely to represent a conflation of trait and state psychological markers, with uncontrolled variability in thresholds for recognition and severity. Nevertheless, the prevalence of atypical temperament types reported here is broadly comparable with rates of psychological distress reported in other unselected population studies, lending some credence to their usefulness as markers for different domains of mental health.

**Explanations of findings**

Several mechanisms may account for the current findings. An important consideration is whether the findings may be explained by residual confounding. Although controlling for several important confounding variables had little effect on the results, one of these variables, father’s social class, is a comparatively crude indicator of the socially patterned exposures in childhood. However, while we have no information on the students’ socioeconomic circumstances after graduation, it is probable that the majority (over 75%) had affluent social positions in later adulthood.32 Temperament in young adulthood could, of course, influence later mortality risk through its influences on later life social circumstances.

The role of health behaviours, such as smoking and alcohol consumption, may be important in explaining the findings. Men who were positive for at least one atypical temperament assessment were more likely to smoke than stable people. Controlling for tobacco and alcohol intake had little effect on the findings, but as with social class, these measures, crudely assessed at one point in time, may give a poor indication of participants’ subsequent adherence to these health behaviours. It is known, for example, that depressed people are less likely to quit smoking and therefore may be at increased risk of CVD and certain cancers in later life, compared with non-depressed people. The strong association between depression and all cause mortality although based on small numbers of deaths (two from CVD and two from cancer), provides some support for this hypothesis.

A range of biological mechanisms for the positive associations between psychiatric morbidity and CVD have been advanced. These include the greater prevalence of adverse

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**Table 4**  
**Age adjusted and fully adjusted hazard ratios for association between temperament type and cause specific mortality**

<table>
<thead>
<tr>
<th>Temperament type</th>
<th>All cause</th>
<th>Cardiovascular disease</th>
<th>Coronary heart disease</th>
<th>Stroke</th>
<th>All cancer</th>
<th>Accidents, suicide and violence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>At least one</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.22</td>
<td>1.00 to 1.48*</td>
<td>1.32 (0.98 to 1.78)</td>
<td></td>
<td>0.94</td>
<td>1.42 (1.01 to 1.83)</td>
</tr>
<tr>
<td>Fully adjusted</td>
<td>1.23</td>
<td>1.01 to 1.50*</td>
<td>1.28 (0.94 to 1.74)</td>
<td></td>
<td>0.90</td>
<td>1.35 (1.06 to 1.69)</td>
</tr>
<tr>
<td>More than one</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>2.04</td>
<td>1.36 to 3.06*</td>
<td>1.67 (0.83 to 3.36)</td>
<td></td>
<td>0.88</td>
<td>2.74 (0.96 to 9.81)</td>
</tr>
<tr>
<td>Fully adjusted</td>
<td>2.03</td>
<td>1.36 to 3.09*</td>
<td>1.60 (0.79 to 2.35)</td>
<td></td>
<td>0.83</td>
<td>2.60 (1.01 to 10.56)*</td>
</tr>
<tr>
<td>Anxious</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.37</td>
<td>1.07 to 1.73*</td>
<td>1.24 (0.85 to 1.82)</td>
<td></td>
<td>0.83</td>
<td>1.42 (0.64 to 3.11)</td>
</tr>
<tr>
<td>Fully adjusted</td>
<td>1.36</td>
<td>1.07 to 1.72*</td>
<td>1.18 (0.80 to 1.73)</td>
<td></td>
<td>0.77</td>
<td>1.43 (0.65 to 2.19)</td>
</tr>
<tr>
<td>Schizoid</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.00</td>
<td>0.67 to 1.48</td>
<td>0.96 (0.51 to 1.80)</td>
<td></td>
<td>0.67</td>
<td>1.64 (0.69 to 5.21)</td>
</tr>
<tr>
<td>Fully adjusted</td>
<td>1.00</td>
<td>0.67 to 1.48</td>
<td>0.93 (0.49 to 1.74)</td>
<td></td>
<td>0.64</td>
<td>1.56 (0.71 to 3.76)</td>
</tr>
<tr>
<td>Hypomanic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.39</td>
<td>0.90 to 2.15</td>
<td>2.00 (1.12 to 3.55)*</td>
<td></td>
<td>1.63</td>
<td>3.35 (2.82 to 8.58)</td>
</tr>
<tr>
<td>Fully adjusted</td>
<td>1.41</td>
<td>0.91 to 2.20</td>
<td>1.90 (1.05 to 3.44)*</td>
<td></td>
<td>1.49</td>
<td>2.23 (0.81 to 5.87)</td>
</tr>
<tr>
<td>Depressed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>4.11</td>
<td>1.54 to 11.00</td>
<td>4.26 (1.59 to 11.40)**</td>
<td></td>
<td>1.28</td>
<td>2.86 (1.14 to 7.14)</td>
</tr>
<tr>
<td>Fully adjusted</td>
<td>4.67</td>
<td>1.66 to 3.30</td>
<td>1.47 (0.66 to 3.00)</td>
<td></td>
<td>1.39</td>
<td>3.71 (1.48 to 9.34)</td>
</tr>
<tr>
<td>Immature</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.28</td>
<td>0.57 to 2.86</td>
<td>–</td>
<td></td>
<td>1.47</td>
<td>3.30 (1.14 to 9.11)</td>
</tr>
<tr>
<td>Fully adjusted</td>
<td>1.29</td>
<td>0.48 to 3.46</td>
<td>–</td>
<td></td>
<td>1.39</td>
<td>3.71 (1.48 to 9.34)</td>
</tr>
<tr>
<td>Hyperchondriacal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.39</td>
<td>0.52 to 3.71</td>
<td>–</td>
<td></td>
<td>1.29</td>
<td>3.46 (1.20 to 9.64)</td>
</tr>
<tr>
<td>Fully adjusted</td>
<td>1.40</td>
<td>0.30 to 2.89</td>
<td>–</td>
<td></td>
<td>1.07</td>
<td>3.34 (0.34 to 3.34)</td>
</tr>
</tbody>
</table>

*Fully adjusted analyses: adjusted for smoking, father’s social class, BMI, maternal and paternal vital status. In models examining all cause, cardiovascular, coronary heart disease, and stroke mortality systolic blood pressure was also controlled for. **p<0.05, ***p<0.01.

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health behaviours in those with psychiatric illness and disordered autonomic function leading to changes in heart rate variability and subsequent hypertension. In keeping with this latter explanation, systolic blood pressure was somewhat higher in those with one or more atypical temperament types. While controlling for systolic blood pressure did not affect the findings, the fact that blood pressure tracks into late adulthood points to the likelihood that people who raised blood pressure in early adulthood are also at increased risk of hypertension and therefore CVD in later life. Other potential mechanisms that have been proposed to explain associations between psychological morbidity and later mortality include deleterious changes in lipid metabolism, promotion of cardiovascular risk through increased basal levels of cortisol in adulthood as a result of early life stressors, and increased platelet activity secondary to depression leading to accelerated the thrombin formation and atherosclerosis. Evidence on these pathways is currently thin, however.

Conclusions
Psychological wellbeing has been recognised as an important factor for students in determining both educational and later occupational performance. We have shown that health in later life may also be dependent on personality/psychological health—although measured in a comparatively crude way—in early adulthood. The findings are provocative but the limitations highlighted point to the need for replication of the analyses in studies with standardised early life measures of mental health and with detailed measures of later life mediating factors.

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Conflicts of interest: none declared.

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