Childhood Problem Behaviors and Death by Midlife: The British National Child Development Study

MARKUS JOKELA, PH.D., JANE FERRIE, PH.D., AND MIKA KIVIMÄKI, PH.D.

ABSTRACT

Objective: To examine childhood problem behaviors manifested as externalizing behaviors (e.g., aggression, impulsivity) and internalizing behaviors (e.g., anxiousness, avoidant behavior) as predictors of mortality by the age of 46 years and to assess whether these associations are dependent on childhood family background. Method: The participants were 5,426 girls and 5,716 boys born in 1958 and participants in the British National Child Development Study (total N = 11,142). Childhood problem behaviors were assessed by teachers at ages 7 and 11 years, and the participants were followed for mortality to the age of 46 years. Results: Both externalizing and internalizing behaviors were associated with mortality in adulthood. By the age of 46 years, the cumulative probabilities of death by increasing externalizing score quartiles were 1.4%, 2.2%, 2.3%, and 3.2%, respectively (odds ratio [OR] for mortality per 1 SD increase in standardized externalizing score 1.27; 95% confidence interval [CI] 1.13–1.44). The corresponding percentages for internalizing score quartiles were 1.8%, 1.9%, 2.3%, and 3.0% (OR 1.20; 95% CI 1.06–1.35). Adjusting for father’s social class, family difficulties, family size, and cognitive ability attenuated these associations for externalizing behaviors (OR 1.21; 95% CI 1.06–1.37) and for internalizing behaviors (OR 1.11; 95% CI 0.98–1.26). Childhood environment did not modify the association between problem behaviors and mortality. Conclusions: Childhood problem behaviors are associated with increased long-term mortality risk beyond childhood and adolescence. J. Am. Acad. Child Adolesc. Psychiatry, 2009;48(1):19–24. Key Words: externalizing, internalizing, life course, longitudinal, mortality.

Longitudinal studies extending from childhood and adolescence to adulthood suggest that early problem behaviors mark elevated risk for a range of negative adult outcomes. Childhood aggressiveness, impulsivity, and other externalizing behaviors have been found to predict antisocial behavior and substance abuse in later life. Childhood internalizing behaviors, such as withdrawn and avoidant disposition, in turn, have been related to increased risk of adult depression and anxiety disorders. Problem behaviors also predict lower socioeconomic achievement and delinquent behavior.

Because many of the outcomes associated with problem behaviors are related to excess mortality, it is plausible that problem behaviors expressed in childhood are a marker of increased adulthood mortality. To date, however, direct tests to confirm or refute this hypothesis are lacking. Moreover, childhood problem behaviors and mortality risk are both associated with adverse family environment, so it is important to consider the role of family background when examining the association between childhood problem behaviors and later mortality.

The ongoing British National Child Development Study has followed a large sample of children from birth to adulthood. At the ages of 7 and 11 years, the children’s teachers assessed the children’s problem behaviors. These data offer a unique opportunity to test the role of childhood problem behaviors and family background in early mortality. In addition to measures of family background, we adjusted the analyses for cognitive ability because low cognitive ability has been associated with increased problem behaviors and mortality risk.
METHOD

Participants

The British National Child Development Study (also known as the 1958 British birth cohort) was launched in 1958.18 The original participants were 17,634 individuals born in England, Wales, and Scotland during 1 week in March 1958. After the study baseline at the time of birth, data have been collected in follow-up phases at 7, 11, 16, 23, 33, 42, and 46 years of age. In the present study, we included all 11,142 participants (5,716 boys and 5,426 girls; representing 63% of the original cohort) who had data on teacher-assessed problem behaviors at the ages of 7 and 11 years and all other covariates used in the study. Ethical approval for the study was obtained from the South East Multi-Centre Research Ethics Committee.

Comparisons between included and excluded participants indicated that the probability of having complete data was positively related to father’s baseline social class (odds ratio [OR] 1.08; confidence interval [CI] 1.05–1.11; p < .001 per 1 category, see below), so that this probability was 0.57 in the lowest class and 0.66 in the highest class. Study participation was not associated with sex (OR 1.04; CI 0.98–1.11; p = .17), and there were no differences in mortality risk after the age of 11 years between included and excluded participants (OR 1.13; CI 0.81–1.64; p = .44 in survival analysis model, see below).

Measures

Behavior Problems. At the ages of 7 and 11 years, the participants’ problem behaviors were assessed by their teachers using the standardized Bristol Social Adjustment Guide.22,23 The 146 items of the instrument assess children’s problematic behaviors that are grouped into 10 “syndromes.” Based on the 10 syndromes, two general dimensions of problem behaviors can be assessed: overreaction (related to externalizing behavior) is assessed by 6 syndromes, that is, hostility toward children, hostility toward adults, inconsequential behavior, restlessness, anxiety about acceptance by children, and anxiety about acceptance by adults (Cronbach α of .72 and .74 at ages 7 and 11 years, respectively). Underreaction (related to internalizing behavior) is assessed by four syndromes, that is, depression, withdrawal, unreliability, and writing off adult values (Cronbach α of .70 and .68). In this article, we refer to overreaction as externalizing behavior and to underreaction as internalizing behavior, because these concepts have commonly been used to describe the two main dimensions of problem behaviors in earlier studies, including those based on the present cohort.4,19 These measures have previously been found to predict increased risk of adulthood psychiatric disorders4,5 and other adverse social outcomes.7

The correlation between the measurements at ages 7 and 11 was r = 0.43 for externalizing behavior (r = 0.59 when adjusted for attention to measurement error) and r = 0.35 for internalizing behavior (r = 0.51 adjusted for measurement error). To provide robust assessment of these behaviors, we calculated the mean values of the scales over the two measurement times and used these means in the analyses. Being measures of social maladjustment rather than normal personality variation, the distributions of the two scales were positively skewed. To reduce the leverage of extreme values, we transformed externalizing scale by inverse transformation and internalizing scale by logarithmic transformation and then standardized the scales (mean 0, SD 1) to facilitate the interpretation of the ORs.

Childhood Family Environment. Measures of childhood family environment included father’s social class, family size, and family difficulties when the participants were 7 years of age. Data on father’s social class and family size were obtained from mothers for 98% of the participants and from other parent figures for 2% of the participants. Father’s social class was assessed on the basis of Registrar General’s social class categorization on a 6-point scale (1 = unskilled, n = 697; 2 = semiskilled, n = 1,949; 3 = skilled manual, n = 4,957; 4 = skilled nonmanual, n = 1,104; 5 = managerial or technical, n = 1,581; 6 = professional, n = 545). Households with no father (n = 421) were included in the group of unskilled fathers because preliminary analyses indicated equal mortality risk for participants with no father in the household (OR 1.66; 95% CI 1.10–2.49; p = .015) and those from households of unskilled fathers (OR 1.65; 95% CI 0.93–2.94; p = .09) compared with participants whose father was from the skilled manual class. Family size is often used as an indicator of how material and social resources need to be shared within the family, so we included family size as a covariate (mean 5.1, SD 1.6).

Family difficulties were assessed by the health visitors performing the home interviews with the parents, that is, the family-difficulties instrument was not administered to the parents in this format in the structured interview. After the interview, the health visitor completed a 13-item scale that queried whether the family was having difficulties due to housing, finances, physical illnesses, mental illnesses, mental subnormality, father’s death, mother’s death, divorce or separation, domestic tension, “in-law” family conflicts, unemployment, alcoholism, or for other reasons. Each item was coded on a dichotomous scale (0 = no, 1 = yes), and an indicator of family difficulties was created by summing these scores together. Although a sum score of the items may represent a rather crude measure of family difficulties (e.g., one family may be severely affected by a single difficulty, whereas another family may function well even when affected by several items of the scale), previous research on social stressors indicates that the accumulation of stressors may be particularly important in predicting children’s maladjustment.24,25 Supporting the use of sum scores, the original scale ranged from 0 to 9, but because there were few families in the high end of the scale, we categorized the scale into 5-point scale ranging from 0 to 4 or more reported difficulties (mean 0.4, SD 0.9).

Cognitive Ability. Cognitive ability was assessed at the age of 7 years by three tests: arithmetic problems, draw-a-man test, and copying designs. The arithmetic test comprised 10 problems with graded levels of difficulty.26 Tests of perceptual and motor ability included the Goodenough27 draw-a-man test and the copying designs test, in which the children were presented with six designs (circle, square, triangle, diamond, cross, and star) that they copied twice. The three test scores were standardized and summed together, and the resulting scale was standardized (mean 0, SD 1) to obtain a single score for cognitive ability.

Mortality. Mortality status was assessed on the basis of study participation data in which the participants’ deaths have been recorded in each follow-up phase at 7, 11, 16, 23, 33, 42, and 46 years of age.Deaths were ascertained through receipt of death certification (the cohort is flagged in the National Health Service Central) or notification to the study team.19 The present sample included participants who were alive and participating in the study at the ages of 7 and 11 years. Because we did not have data on the exact year of death, age of death was determined to be the age the participant would have been at the follow-up phase in which the participant was first recorded as deceased; for example, for a participant who had
died between the ages of 33 and 42 years, age at death was assigned as 42. Of the participants who had data on behavior problems and other covariates included in the study, 243 were recorded as deceased by the age of 46 years: 23 participants at ages 12 to 16 years, 16 at ages 17 to 23 years, 73 at ages 24 to 33 years, 69 at ages 34 to 42 years, and 112 at ages 43 to 46 years.

Data Analysis

The association between problem behaviors and mortality risk was assessed with discrete-time survival analysis, which is the appropriate statistical method to study whether and when an event occurs within a given period. The follow-up period spanned from 11 to 46 years of age and was coded by dummy variables for age periods 17 to 23, 24 to 33, 34 to 42, and 43 to 46, with age period 11 to 16 as the reference category. The statistical estimates were expressed as ORs referring to change in hazard function, that is, the probability of dying at a given age period. The results were illustrated by calculating the cumulative probability of death by the age of 46 years by problem behavior levels (from −1 SD below the mean to +2 SD above the mean; the skewed scales of problem behaviors did not reach −2 SD).

Two tests were performed to examine the role of childhood family environment in the association between problem behaviors and mortality. First, both problem behaviors and early mortality may reflect the adverse impacts of early environmental adversity, so we examined whether the association between problem behaviors and mortality diminishes when adjusted for measures of family environment. Second, family environment may modify the association between problem behaviors and mortality so that the role of behavior problems is augmented by adverse childhood environments. Thus, we examined the significance of the interaction effect between behavior problems and family background on predicting mortality in a model also including the main effects.

RESULTS

Table 1 shows the correlations between childhood problem behaviors, childhood environments, and cognitive ability. Externalizing and internalizing behaviors were positively correlated with each other ($r = 0.38$), and they were both associated with adverse family environment and with low cognitive ability (Table 1). Table 2 shows the association between childhood covariates and mortality risk. As expected, in unadjusted models, high mortality risk was predicted by male sex, low social class, high family difficulties, large family size, and low cognitive ability (model 1). When the covariates were entered in a single multivariate model, an independent association with mortality risk was found for all of the covariates, with the exception of cognitive ability, which was no longer a significant predictor of mortality (model 2).

Behavior Problems, Mortality, and Childhood Environment

As a preliminary analysis, we categorized the participants into quartiles by externalizing score and calculated the cumulative mortality function, that is, the probability of dying by a given age period. We then plotted the cumulative mortality function in Figure 1A (the 95% CIs were calculated using Greenwood approximation). By the age of 46 years, the cumulative probabilities of death by increasing externalizing score quartiles were 1.4%, 2.2%, 2.3%, and 3.2%, respectively, suggesting approximately a twofold mortality risk in the highest quartile compared with the lowest quartile.

### Table 1

| Correlations Between Independent Variables ($N = 11,142$) |
|------------|---|---|---|---|---|---|
|            | 1  | 2  | 3  | 4  | 5  | 6  |
| Sex        | —  | —  | —  | —  | —  | —  |
| Externalizing | −0.19 | —  | —  | —  | —  | —  |
| Internalizing | −0.16 | 0.38 | —  | —  | —  | —  |
| Father’s social class | 0.01 | −0.14 | −0.17 | —  | —  | —  |
| Family difficulties | −0.01 | 0.15 | 0.16 | −0.19 | —  | —  |
| Family size | −0.01 | 0.11 | 0.15 | −0.14 | 0.22 | —  |
| Cognitive ability | −0.01 | −0.28 | −0.34 | 0.20 | −0.18 | −0.14 |

*Note: All correlations with r ≥ 0.02 were statistically significant at the level of p < .001.

### Table 2

Predicting Mortality Risk by Study Covariates ($N = 11,142$)

<table>
<thead>
<tr>
<th></th>
<th>Model 1: Unadjusted</th>
<th>Model 2: Mutually Adjusted</th>
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<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
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<tr>
<td>11–16 y (reference)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>17–23 y</td>
<td>0.67 (0.34–1.31)</td>
<td>0.67 (0.34–1.31)</td>
</tr>
<tr>
<td>24–33 y</td>
<td>2.83* (1.72–4.66)</td>
<td>2.83* (1.72–4.66)</td>
</tr>
<tr>
<td>34–42 y</td>
<td>2.84* (1.73–4.68)</td>
<td>2.85* (1.73–4.69)</td>
</tr>
<tr>
<td>43–46 y</td>
<td>4.67* (2.91–7.49)</td>
<td>4.69* (2.92–7.52)</td>
</tr>
<tr>
<td>Sex (0 = male, 1 = female)</td>
<td>0.55* (0.42–0.72)</td>
<td>0.55* (0.43–0.72)</td>
</tr>
<tr>
<td>Father’s social class (per 1 category)</td>
<td>0.88* (0.82–0.95)</td>
<td>0.92 (0.85–1.00)</td>
</tr>
<tr>
<td>Family difficulties (per 1 difficulty)</td>
<td>1.24* (1.11–1.40)</td>
<td>1.14*** (1.01–1.30)</td>
</tr>
<tr>
<td>Family size (per 1 person)</td>
<td>1.12* (1.05–1.20)</td>
<td>1.08*** (1.01–1.15)</td>
</tr>
<tr>
<td>Cognitive ability (per 1 SD)</td>
<td>0.87*** (0.77–0.99)</td>
<td>0.93 (0.82–1.06)</td>
</tr>
</tbody>
</table>

*Note: Age was included in all models. The age coefficients in model 1 are for a model including only age. Odds ratios (and 95% confidence intervals) from discrete-time survival analyses.

*p < .001; **p < .01; ***p < .05; †p < .10.
The above procedure was then applied to internalizing behavior. This suggested a pattern similar to that observed for externalizing behavior. The cumulative probability of death was significantly lower in the lowest quartile than in the highest quartile of internalizing behavior (Fig. 1B) so that, by the age of 46 years, the percentages by increasing internalizing score quartile were 1.8%, 1.9%, 2.3%, and 3.0%, respectively.

We then applied survival analysis to model the associations between behavior problems and mortality. The upper part of Table 3 shows a stepwise survival analysis model examining whether externalizing behavior predicted mortality and whether this association was independent of internalizing behavior and family environment. Externalizing increased the odds of dying at a given age, and this association remained, albeit attenuated, when internalizing and family covariates were included in the model. The lower part of Table 3 shows the corresponding analyses for childhood internalizing behavior. Internalizing also increased the odds of dying at a given age period. Adjusting for externalizing or family covariates attenuated this association by half, and when adjusted for both externalizing and family measures, internalizing no longer predicted mortality.

Table 3 shows the predicted cumulative probability of death by problem behavior levels. Given the low values of the percentage points, the ORs give an approximation of the relative risk of death by a given age associated with 1 SD increase in problem behavior score. In other words, an externalizing score of 1 SD above the mean was associated with 27% increased relative risk of death (from 2.1% to 2.7%), which was attenuated to 19% when controlling for family covariates and internalizing. An increase of 1 SD in the internalizing score was associated with an increased relative mortality risk of 19% when adjusting for sex (from 2.1% to 2.5%), which was attenuated by half when adjusted for family background and decreased to 6% risk when further adjusting for externalizing (Table 3).

Additional analyses were then applied to examine the association between problem behaviors and mortality in more detail. First, we examined whether the association between problem behaviors and mortality changed over age by including problem behavior–age interaction effects in the models. These interactions were not significant for externalizing or internalizing (p > .11), suggesting that problem behaviors predicted mortality with a similar magnitude during different age periods. Second, we assessed whether the association between problem behaviors and mortality was nonlinear. However, the quadratic terms of internalizing or externalizing were not significant (p > .41). Third, we tested for sex differences, but there were no interaction effects between sex and externalizing (p = .49) or between sex and internalizing (p = .64), suggesting no differences between men and women.

Finally, we examined whether the association between problem behaviors and mortality was moderated by the three family background variables or by cognitive ability. None of the interaction effects between externalizing or internalizing behaviors and the covariates were significant (all p > .34), indicating that childhood problem behaviors predicted mortality
indicate that approximately one third of and p, (mean). Odds ratios (and 95% confidence intervals) of two nested (+1 SD)/ and that increase the risk j. It is therefore plausible that the 23 < .001; ** < .01. = 11,142). CI = confidence All models adjusted for sex. The odds ratios refer to change j adjusted for sex 1.7 2.1 2.6 3.0 1.21** (1.06–1.37). Adjusted for covariates* adjusted for sex 1.7 2.0 2.9 1.19** (1.04–1.36). Adjusted for covariates* and internalizing

<table>
<thead>
<tr>
<th>Externalizing</th>
<th>−1 SD</th>
<th>Mean</th>
<th>+1 SD</th>
<th>+2 SD</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjusted for sex</td>
<td>1.7</td>
<td>2.1</td>
<td>2.7</td>
<td>3.4</td>
<td>1.27* (1.13–1.44)</td>
</tr>
<tr>
<td>Adjusted for internalizing</td>
<td>1.7</td>
<td>2.1</td>
<td>2.6</td>
<td>3.1</td>
<td>1.23** (1.08–1.39)</td>
</tr>
<tr>
<td>Adjusted for covariates*</td>
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<td>2.0</td>
<td>2.5</td>
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<td>1.21** (1.06–1.37)</td>
</tr>
<tr>
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<td>2.0</td>
<td>2.4</td>
<td>2.9</td>
<td>1.19** (1.04–1.36)</td>
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<table>
<thead>
<tr>
<th>Internalizing</th>
<th>−1 SD</th>
<th>Mean</th>
<th>+1 SD</th>
<th>+2 SD</th>
<th>OR (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjusted for sex</td>
<td>1.8</td>
<td>2.1</td>
<td>2.5</td>
<td>3.0</td>
<td>1.20** (1.06–1.35)</td>
</tr>
<tr>
<td>Adjusted for externalizing</td>
<td>1.9</td>
<td>2.1</td>
<td>2.3</td>
<td>2.6</td>
<td>1.11 (0.98–1.27)</td>
</tr>
<tr>
<td>Adjusted for covariates*</td>
<td>1.9</td>
<td>2.1</td>
<td>2.3</td>
<td>2.5</td>
<td>1.11 (0.98–1.27)</td>
</tr>
<tr>
<td>Adjusted for covariates* and externalizing</td>
<td>1.9</td>
<td>2.0</td>
<td>2.2</td>
<td>2.3</td>
<td>1.06 (0.93–1.21)</td>
</tr>
</tbody>
</table>

*Note: All models adjusted for sex. The odds ratios refer to change in the odds of death at a given age period. They also give an approximate relative risk of death by the age of 46 years associated with 1 SD change in problem behavior score, that is, RR = p (+1 SD)/ p (mean). Odds ratios (and 95% confidence intervals) of two nested discrete-time survival analysis models (N = 11,142). CI = confidence interval; OR = odds ratio.

*Father’s social class, family difficulties, family size, and cognitive ability in childhood.

*p < .001; **p < .01.

in a similar way irrespective of family background or cognitive ability.

DISCUSSION

In a large British birth cohort, childhood externalizing and internalizing behaviors predicted increased mortality between 11 and 46 years of age so that individuals with high problem behaviors in childhood had a heightened mortality risk even in adulthood. There were no interaction effects between problem behaviors and measures of childhood environment, implying that adverse childhood environment did not modify the role of problem behaviors in predicting mortality risk. The findings provide evidence of the long-term mortality risk associated with childhood social maladjustment and psychiatric vulnerability.

The study has some notable strengths, including a large national sample, assessment of childhood problems behaviors at two time points with a standardized instrument, assessment of childhood family environment characteristics, mortality data ascertained from a national registry, and a long follow-up period. One weakness is that 37% of the original cohort was lost because of missing data. There were no differences in mortality rates between the included and excluded participants, suggesting that health-related selection is an unlikely explanation for our results, despite individuals with low parental social class were slightly underrepresented among the participants.

Deaths between 11 and 46 years of age account for only a small proportion of total mortality in the population, so the present study cannot tell how important childhood problem behaviors are in creating overall mortality differentials over the life span. This can be assessed with data from future follow-ups of the National Child Development Study. However, deaths before the age of 46 years represent a substantial loss of life, considering that, in 1958, the life expectancy at birth in Britain was 68 years for men and 73 years for women.

Cause-specific mortality data may have provided clues to mechanisms mediating the influence of externalizing and internalizing behaviors, but unfortunately, such data were not available. The British national statistics indicate that approximately one third of deaths before the age of 45 years are due to external causes, for example, traffic accidents, suicides, and unintentional injuries. Moreover, externalizing and internalizing behaviors have been found to increase the risk of unintentional injuries in childhood and adolescence. It is therefore plausible that the association between problem behaviors and mortality is driven mainly by the consequences of risky and self-harmful behavior and exposure to dangerous environments. The association may also reflect other adverse effects of childhood problem behaviors, for example, low socioeconomic achievement, that increase the risk of early death.

Externalizing and internalizing behaviors both predicted mortality risk, but externalizing behavior had a stronger independent effect when controlling for childhood family circumstances and cognitive ability. Externalizing also had a stronger independent effect than internalizing when both problem behavior dimensions
were mutually adjusted, and the association between internalizing and mortality risk was attenuated to a nonsignificant level when further adjusted for family covariates. These findings suggest that the influence of problem behaviors on mortality risk is driven primarily by externalizing behaviors, whereas the heightened mortality risk associated with internalizing behavior may reflect comorbidity with externalizing behavior in addition to adverse childhood family environment. However, in the present study, we could not determine the causal relations between family environment and problem behaviors that may flow in both directions and also reflect common genetic influences. 

Previous life-course research of childhood problem behaviors and psychiatric vulnerability has established connections between childhood psychosocial maladjustment and a wide range of negative outcomes later in life. The present findings add to this literature by showing that problem behaviors expressed in childhood may carry a heightened risk of mortality over the life course.

Disclosure: The authors report no conflicts of interest.

REFERENCES