Childhood Psychopathology Can Be *Really* Bad for Your Health

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It is not often that one is asked to provide a commentary on an article that one expects to become a cornerstone of justifications for new psychiatric research (and especially longitudinal and developmental research), but that is what “Childhood Problem Behaviors and Death by Midlife: The British National Child Development Study”1 is. It is also scary to claim that this is the first study of its kind (there is always the fear that someone better read will point out that X showed something similar in the 1920s), but I believe it is. This admirably concise presentation from the 1958 British birth cohort indicates that children rated by their teachers as being in the highest scoring quartile on a more than 40-year-old questionnaire about emotional and behavioral problems had about double the mortality by age 46 years of children scoring in the lowest quartile. Of course, mortality by age 46 years was low overall (approximately 1.5% in the lowest male and female quartiles and approximately 3% in the highest quartiles), but in epidemiological terms, that is a huge effect, affecting a large number of people. If this disparity were to continue throughout life, it would amount to an unprecedented number of life-years lost to an easily measured childhood risk factor affecting so large a proportion of the population.

The article also reports that “externalizing” problems had a greater effect on death rates than “internalizing” problems and that the “internalizing” effect was largely explained statistically by familial factors and “externalizing” problems. The problem is that the seemingly familiar terms externalizing and internalizing, as used here, do not mean what they usually mean. As described in the Measures section of the article, the 146 items of the Bristol Social Adjustment Guide measured 10 “syndromes” that were clustered into two overarching dimensions called overreaction and underreaction. Presumably, because this more than 40-year-old conceptualization has been entirely superseded, the authors simply renamed the syndromes “externalizing” and “internalizing.” However, the overreaction (“externalizing”) dimension included “anxiety about acceptance by children” and “anxiety about acceptance by adults,” although these items clearly belong under the internalizing umbrella as it is currently understood. Similarly, “writing off adult values” is now seen as part of the externalizing spectrum, but because it was part of the conceptualization of “underreaction,” it is placed in the “internalizing” scale. One further overreaction syndrome (“inconsequential behavior”) has no obvious parallel in the modern assessment of internalizing or externalizing disorders, but here it counts toward “externalizing.” The key point is that both the “externalizing” and “internalizing” scales here are mixtures of what we would now regard as internalizing and externalizing symptoms.

So we cannot conclude that behavioral problems had greater direct impact on mortality than did emotional problems. However, this question could have been addressed had the syndromes been grouped in a manner more in keeping with current definitions of internalizing and externalizing problems. Perhaps the original syndrome level scores were not recorded, or are no longer available, but if they are, then I would be interested to know what effects resoring along the lines I have suggested would have.

In discussing their results, the authors briefly consider two potential mechanisms by which these effects on mortality might have arisen—susceptibility to risky and self-harmful behavior (which they suggest might be principally responsible for their findings) and low socioeconomic achievement. Because no data on the actual causes of death were available (perhaps in future research, the authors could access such information from the death certificates flagged in the National Health Service Central), it is perhaps understandable that they decided not to present a detailed discussion of this issue. However, I think it is helpful to consider a rather broader and more differentiated set of possible mechanisms. In this regard, I find it useful to consider six groupings of explanations that

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cross-cut and expand upon those presented in the article: (1) Direct results of psychopathology, such as death by inanition in the case of anorexia nervosa; suicide in the context of depression, anorexia nervosa, or personality disorder; and death by drug overdose. (2) Secondary effects of psychopathology, such as deaths by accident or homicide resulting from behavioral problems and deaths from the health effects of chronic alcohol and drug (including tobacco) abuse. (3) Mortality from physical diseases known to be associated with psychiatric disorders; here, the two obvious examples are asthma as related to anxiety disorders, for example, and hostility and depression as related to cardiovascular morbidity and mortality. (4) Effects driven by the environmental correlates of psychopathology, including low social status and relatively dangerous jobs and lower quality housing and nutrition associated with low socioeconomic status. (5) Genetic and early life (including intrauterine) factors that predispose both to psychiatric and physical disorders and death, for example, low birth weight. (6) Iatrogenic fatalities related to treatments for psychiatric conditions or their physical disorder concomitants.

It will be immediately apparent that these groupings are not separated by “bright lines” and that other organizing schemata can easily be formulated. However, this schema does serve to show that risky and self-destructive behavior and low social status may be part of several meaningfully different paths to death associated with early psychopathology and that it is premature to focus on them as a primary causal explanations. This discussion also highlights the value of data on the causes of death and the need for further follow-ups of this cohort. We have to ask whether early psychopathology is associated only with early death or with continuing higher mortality rates throughout the life span. It is also possible that longer term data on the causes of death will indicate that different mechanisms are associated with mortality at different ages.

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REFERENCES