Authors’ response regarding ‘Does low intelligence really cause pain? The importance of measurement, methodology and implications when drawing conclusions’

We were taken aback by the comments of Ms Bailey and Ms Schinkel on our article.[1] Their intimation that our findings should not be put in the public domain in case some individuals misinterpret them betrays a lack of understanding of the process of scientific research. Their assertion that that the conclusions we drew are “irresponsible” and the research “flawed” suggests that they misconstrue the purpose of epidemiological studies. Moreover, their comments about the apparent flaws in our study’s methodology highlight a fundamental lack of comprehension of some of the most basic elements of observational epidemiology.

The main criticism put forward by Bailey and Schinkel is that we suggest “that low intelligence is causally related to the development of chronic pain” based on our observation in a longitudinal study that higher scores on a test of intelligence at age 11 years were associated with a lower likelihood of reporting chronic widespread pain in adulthood. They comment that “causation should not be inferred from a correlation study, even if it is a prospective correlation design”. Leaving aside the fact that no study can correctly be said to have a “prospective correlation design”, at no point in our paper do we state or infer that the association between childhood intelligence and chronic widespread pain in middle age is causal. We report the association and discuss potential mechanisms that might explain it. This is common practice when positive results are reported: associations do have causes and scientists should seek them. The fact that our findings are based on a prospective study—as opposed to one with a cross-sectional or case-control design—which uses data on intelligence measured many years before the assessment of chronic widespread pain is a
major strength because it makes it more likely that intelligence, or some factor or factors associated with it, is involved in the aetiology of chronic widespread pain.

Bailey and Schinkel go on to suggest that by reporting an association between childhood intelligence and chronic widespread pain in adults our study may result in “individuals with low intelligence having their pain symptoms inadequately examined or inaccurately diagnosed” or in adults with chronic pain being viewed as “having low intelligence”. They appear to take the view that results of epidemiological studies should be suppressed and not written up for publication if there is any risk of the findings being misunderstood or misused by some individuals. Such an approach would be seriously detrimental to scientific progress.

The other major criticism put forward by Bailey and Schinkel concerns the measure of intelligence used in our study. They state that “the IQ scores were arbitrarily constructed and were obtained from a test that was not standardized with the same rigorous methodology” as modern intelligence tests. There is no cogent criticism here. The scores on the single test of verbal and non-verbal ability that our study participants completed at age 11 years were shown to correlate highly (r=0.93) with scores on an IQ test used to select children for secondary school suggesting a high degree of validity as a measure of general cognitive ability.[2] General cognitive ability as assessed by such tests is among the best-validated constructs in psychological science,[3] and it is general cognitive ability and not especially ability at specific cognitive tasks that correlates with many health outcomes.[4] However, if their suggestion is that specific cognitive tests—all of which have some general cognitive ability variance—would have provided stronger associations, then they are merely
stating that our effect sizes might be under-estimates. Ultimately, though, we did find a significant association with the test that was used and they should not look past that.

Bailey and Schinkel are concerned about our interpretation of group differences on this test. In describing our findings we clearly made the point that the difference in intelligence between those study participants with and without chronic widespread pain was small (0.21 of a standard deviation). Bailey and Schinkel choose to reiterate this. From a clinical perspective where the interest is in the individual patient, such a difference is of little consequence, but from the epidemiological perspective even a small difference between individuals can be scientifically informative regardless of whether it is clinically significant for one individual. In this cohort study, as in others, individual differences on that single measure of intelligence in childhood have been shown to be a significant predictor of several important life outcomes, such as earnings, socioeconomic position, obesity, psychological distress, and mortality. [5-9]

Bailey and Schinkel conclude their letter by accusing us of irresponsibility. We judge that that is an indefensible statement. Chronic widespread pain is a common and disabling musculoskeletal condition. Understanding its aetiology is crucial if we are to find ways to prevent it. Our study shows that intelligence in childhood may be a risk factor for the condition and points the way to further research to understand the underlying mechanisms.

Conflict of interest
The authors have no financial arrangements or conflict of interest to disclose as regards this manuscript. Sponsor’s role: none.

References


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