Transgenerational response and life history theory: a response to Peeter Hörak

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We are grateful to Peeter Hörak for his careful reading of our paper on the transgenerational response to childhood trauma.1 We reported2 that grandparental (generation 1, G1) death during generation 2 (G2) childhood predicted birthweight and prematurity in generation 3 (G3). The major point in Hörak’s response is that our results can be explained by ‘life history theory’, derived from evolutionary biology or evolutionary ecology. Our major conclusion, that the observed transgenerational response along the male line may well be epigenetic and triggered during the slow growth period, is hardly touched on by Hörak. Finally, Hörak states that human population data, like ours, can be used to test theories derived from evolutionary biology, such as life history theory. We disagree on Hörak’s first point but agree on the last. All three points are elaborated below.

Fast or slow life history

Genetics, according to Hörak, determine whether people have a ‘fast’ or a ‘slow’ life history strategy. The relevance of the concept of ‘fast life history strategy’ is that it involves both a strategy of taking risks and a ‘reduction of the amount of maternal somatic investment in fetal development, resulting in shortened gestation and low birthweight’. Men and women with a ‘fast life history’ tend to marry each other according to Hörak, so-called assortative mating. Genetic correlations in life history traits between spouses arise because of this assortative mating. Assortative mating is a relevant concept, well-known also in the social sciences, where it is usually applied to education. In our population, we were able to calculate the correlations in educational achievement between G2 spouses born before 1975 (n = 395 223 pairs). This is 0.31. The same correlation between G1 paternal grandparents is 0.28; between G1 maternal grandparents it is 0.29, in both cases based on 43 308 pairs. Clearly, assortative mating based on education exists in both G1 and G2 in our study.

However, we note that the particular outcomes that we focus on, birthweight and gestational length in G3, are influenced by assortative mating in G2. For a segment of generation 2, born 1973 to 1987, we have data on birthweight (n = 43,308 pairs) and gestational age (n = 42 971 pairs) for both G2 spouses. Birthweight correlations between spouses/parents in G2 are 0.02. Correlation in gestational length (measured in days) between spouses is even smaller: 0.008. It is obvious that there is no assortative mating whatsoever based on these traits.

Thus, if fetal growth and risk-taking behaviour are both indicators of a genetically driven ‘fast life history strategy’ and men and women chose partners based on such strategies, we would expect birthweights and gestational age to be correlated between spouses. Since they are not, we must conclude that birthweight and gestational age are not very important aspects of any genetically transmitted life history strategy, or alternatively, that life history strategies are not the basis for assortative mating. Hörak writes that ‘genetic correlations in life history traits are likely to cause the reported associations’, and that ‘genes predisposing fathers to premature death could cause reduced maternal investment in their granddaughters’ growth in utero’. This conclusion is not supported by our data.

Mechanisms of transgenerational response

From our study we concluded that exposure to childhood trauma, such as the death of a parent, triggers ‘a cascade of events, psychological, behavioural, metabolic and social’. Whereas this was true for both boys and girls suffering parental death, the transmission of this response to their offspring differed markedly. Along the female line, we assumed that it was most of all a socially mediated (partly through education) transgenerational response which gave rise to reduced birthweight and increased prematurity risk in offspring. Along the male line, we suggested that results were compatible with the hypothesis formulated by Pembrey and Bygren3: the period just before puberty may be extra sensitive to trauma, resulting in germ-line

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epigenetic change, transmittable to offspring; in our case, influencing offspring’s fetal growth.

Hörak does not address this very specific hypothesis directly. However, we take encouragement from his statement that ‘… as a predictive-adaptive response, a parent’s death, signaling harshness and unpredictability would trigger switching to a fast life history strategy …’ The idea of an adaptive switch appears to be similar to the idea of epigenetic change triggering a cascade of events; this is also how Eva Jablonka and colleagues4 understand such epigenetic changes which are transmitted to later generations. Clearly, this is a case of ‘cross-talk’ between genes and the environment, rather than a genetically driven response. Following Pembrey et al.,5 we prefer to be cautious about whether or not a specific epigenetic change, transmitted across generations, is in fact adaptive or not. This is another empirical question. Is it really obvious that low birthweight of children of early traumatized fathers is adaptive in modern society? In what way?

A new study by Svanes et al.6 highlights the complexity of transgenerational response to adverse environments. They noted that whether or not the paternal grandmother smoked when pregnant with her son, this would interact with epigenetic changes in puberty (due to the son’s own smoking), in such a way that the response in his offspring could either cause or protect against asthma. Surprisingly, boys smoking in puberty were less likely to transmit asthma to their offspring if the boys themselves had been exposed to smoking in utero. The hypothetical epigenetic change or ‘switch’ leading to asthma or not in later generations thus depends on a complex cross-talk between genes and the environment, spanning many years. Are concepts such as ‘fast life history’ really precise enough to describe such interactions?

Use of human population data

On the third point, we agree entirely with Hörak. Population data of the kind that we have analysed here also lend themselves to test hypotheses derived from life history theory, evolutionary biology or evolutionary ecology. Although these are strongly formulated theories, there is often a critical lack of data on human populations to underpin them. Hörak gives many examples of what could be done. It seems clear that demographers and epidemiologists have a crucial role here.

However, the sharp distinction in life history theory between fast and slow life history strategies, seems to be often based on studies comparing species, in particular primates. It appears to be a fruitful concept in studying very long evolutionary processes. But is it equally fruitful or appropriate for the study of individual or social variation in birthweight, prematurity and a number of other phenotypic traits among modern human populations?

Live fast and die young

‘Live fast and die young’ is the title of a biography7 of James Dean, a Hollywood icon who died in 1955, at age 24. His attitude influenced a generation of contemporaries, reminding us of the importance of culture for the life strategies that we choose to adopt, rather freely and under the influence of our peers.

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