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Author for correspondence:
Peeter Hõrak
e-mail: horak@ut.ee

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Animal behaviour

Father's death does not affect growth and maturation but hinders reproduction: evidence from adolescent girls in post-war Estonia

Peeter Hõrak[1] and Markus Valge[2]

[1]Department of Zoology, Institute of Ecology and Earth Sciences, Tartu University, Vanemuise 46, Tartu 51014, Estonia
[2]Institute of Psychology, Tartu University, Näituse 2, Tartu 50409, Estonia

The popular concept of predictive–adaptive responses poses that girls growing up without a father present in the family mature and start reproduction earlier because the father's absence is a cue for environmental harshness and uncertainty that favours switching to a precocious life-history strategy. Most studies supporting this concept have been performed in situations where the father's absence is caused by divorce or abandonment. Using a dataset of Estonian adolescent girls who had lost their fathers over the period of World War II, we show that father's death did not affect the rate of pubertal maturation (assessed on the basis of development of breasts and axillary hair) or growth. Father's death did not affect the age of first birth but, contrary to predictions, reduced lifetime reproductive success. Our findings thus do not support the concept of predictive–adaptive responses and suggest that alternative explanations for covariation between fatherlessness and early maturation are required.

1. Introduction

Constraints permitting, rates of maturation and reproduction of organisms should evolve to track the variation in mortality rates. Responding to cues of environmental harshness and uncertainty by speeding up reproduction—even at the expense of somatic and social costs—is adaptive [1,2], at least in situations where such cues turn out to be correct [3]. In humans, the focus of research on such predictive–adaptive responses has been on the timing of pubertal maturation, sexual debut and first pregnancy, in relation to childhood psychosocial adversities [4]. Of these, effects of the father's absence owing to divorce and abandonment have been studied particularly extensively. A recent meta-analysis of 33 studies confirmed that the father's absence was significantly related to earlier menarche [5].

Despite the valuable insight provided by such studies, they suffer a methodological problem in confounding the occurrence of adaptive plasticity with passive gene–environment correlations [6]. For instance, daughters may inherit a disposition to a life-history strategy from their mothers that links early puberty and reproduction with unstable partnerships [7], and genes predisposing the father to behaviours that include family abandonment may be passed on to their daughters, causing early puberty and precocious sexuality [8]. Such patterns may be further enforced by assortative mating in relation to cognitive abilities at all levels of intelligence [9]. Low cognitive abilities are often associated with steep discounting of future and early reproduction, for instance because reproduction is not compatible with schooling [10,11].

One possible way to disentangle putative predictive–adaptive responses from potentially confounding gene–environment correlations is to focus on the effects

of father's death on the development and reproduction of their children [12]. Such studies would be particularly informative in situations such as natural disasters and warfare, where causes of death appear random with respect to possible dispositions to any particular life-history strategy. Indeed, contrary to the findings of the majority of studies of the father's absence, stress owing to war conditions, including poverty as well as psychological and physical trauma, appeared to delay, rather than speed up the onset of menarche [13].

Here we use data from Estonian adolescent girls to assess the effects of father's death on rate of maturation and growth, onset of reproduction and eventual number of children. The dataset includes girls born between 1938 and 1953 and measured around the age of 17 [14]. Of 1678 girls, 12% (194) reported that their fathers were dead by the time of the study. Because the incidence of maternal mortality was only 2% (31/1678), the majority of paternal deaths were presumably caused by war and Stalinist repressions, creating the situation of a natural experiment where the paternal absence can be predominantly ascribed to external causes above and beyond genetic and socio-economic variables [15].

Relying on anthropometric measures of maturation (rate of development of breasts and axillary hair), growth and reproduction of adolescent girls, we were able to test the predictions of predictive–adaptive hypotheses [1,2,4,12,15,16] that father's death accelerates (i) sexual maturation and (ii) reproduction. Additionally, we tested (iii) whether father's death suppresses growth of daughters in order to elucidate the importance of paternal resource provisioning. Growing children may lack sufficient resources to boost maturation under the constraint of extreme nutritional limitation [16] and we expected that such a situation would be revealed by showing impaired growth of girls whose fathers were dead. Ultimately, the negative effect of father's death on somatic growth can also be predicted if the rate of pubertal maturation is traded off against the rate of growth [17]. Under such a scenario, we expected to see simultaneously boosting the effects of father's death on rate of maturation and negative effects on growth. Finally, we asked whether father's death affects the number of children eventually born to the girls under study. Fast reproductive strategies are associated with more offspring (reviewed in [4,16]), hence we predicted that (iv) originating from a family where the father was dead would result in a higher total number of children and lower probability of remaining childless. Our dataset is particularly suitable for testing the latter predictions because all women involved in the study had completed fertility by the time their reproductive parameters were recorded.

2. Methods

Anthropometric data from 1678 schoolgirls born between March 1938 and November 1953 were collected between January 1956 and September 1969 by a single person, Juhan Aul, as described previously [14]. Their average age at measurement was 16.85 ± 0.89 (s.d.) years, ranging from 14.6 to 20.0 years. Along with anthropometric measures, number of siblings, the presence of father and mother (alive versus dead) and parental occupations were recorded. Rate of sexual maturation was described on the basis of development of breasts and axillary hair (determined by visual inspection) and divided into five stages according to the Tanner scale [18]. Breast and axillary hair development are not necessarily synchronous, as they are driven by different though closely related hormonal systems (gonadal and adrenal steroids, respectively [18]). Unfortunately, we lacked data on menarcheal age. We used three measures of growth that appeared most sensitive to different biosocial factors at different ages (legs grow faster than trunk before puberty; see [14]).

Data for age of primiparity and number of children were obtained from the Estonian Population Register and processed while maintaining anonymity of subjects (see Ethics footnote). From the original study, 1054 participants could be identified.

Effect of the father's absence on anthropometric and reproductive variables was tested with ANCOVAs, using Statistica v. 10 (StatSoft Inc). All models were adjusted for relevant developmental and biosocial covariates [14,18]; only significant predictors were maintained. Sample sizes differed between the models because we had an incomplete dataset for some variables. Residuals of the models did not diverge from normality. Number of children and probability of remaining childless were not affected by any biosocial variables, hence *T*-tests and χ^2-statistics were used for comparing girls with dead versus alive fathers. All tests were two-tailed with a *p*-level below 0.05 as a criterion for significance.

3. Results

We did not find any evidence that father's death had affected measures of maturation, growth or age of primiparity (tables 1 and 2). Breast and axillary hair development scores correlated negatively with height but these correlations were not affected by father's death (interactions for father's death × height for breast: $F_{1,1671} = 0.18$, $p = 0.672$; hair: $F_{1,907} = 0.27$, $p = 0.600$). Women from families where the father was dead had significantly fewer children than those from families where the father was still alive in their adolescence (1.48 ± 1.10 (s.d.), $n = 123$ versus 1.73 ± 1.19, $n = 931$; $t = 2.17$, $p = 0.030$); the effect size ($d = 0.13$) was, however, small. Of women originating from families where the father was dead 23.6% (29/123) remained childless, in contrast with 16.5% (154/931) of women whose father was still alive by the time they reached adolescence. This difference was marginally non-significant ($\chi^2_1 = 3.75$, $p = 0.053$).

4. Discussion

The hypothesis that father's death accelerates maturation and/or reproduction was not supported by our data. The absence of paternal effects cannot be ascribed to low sample size because we had sufficient test power for detecting even small effects. We can also exclude the possibility that the absence of paternal effects could be caused by insufficient plasticity in maturation rates because both indices of sexual development correlated negatively with body height. The correlation was particularly notable in the case of the breast development score ($\beta = -0.21$), indicating that early maturation had significant somatic costs. However, the trade-off between the rate of maturation and growth was not mediated by the father's absence. The father's absence did not suppress any measures of vertical growth, indicating that father's death did not cause somatic costs comparable to those exerted by high number of siblings or low parental SEP. We can thus reject the possibility that girls with dead fathers were short of bodily resources for speeding up their maturation.

The absence of somatic effects of father's death is particularly intriguing in the context that girls with dead fathers had

Table 1. Test of originating from the family with dead versus alive fathers on parameters of maturation and age of primiparity and height in ANCOVAs. In all models, only significant covariates are retained. *N* (dead/alive) denotes sample sizes for girls whose fathers were either dead or alive by the time of measurement. SEP is socio-economic position, β is standardized regression coefficient, VIF is variance inflation factor. Average trait values are presented in table 2. Minimal effect size is minimal detectable effect size in s.d. units, calculated on the basis of least squares means and s.d.s for one-tailed tests with 80% power, adjusting for covariates.

trait	breast development score				axillary hair score				age of primiparity[a]			
effect	*F*	*p*-value	β (s.e.)	VIF	*F*	*p*-value	β (s.e.)	VIF	*F*	*p*-value	β (s.e.)	VIF
birth date	5.6	0.018	−0.06 (0.02)	1.10	9.6	0.002	−0.10 (0.03)	1.07	26.6	<0.0001	−0.19 (0.04)	1.15
age	6.1	0.013	0.06 (0.02)	1.12	51.6	<0.0001	0.22 (0.03)	1.13				
height	57.5	<0.0001	−0.21 (0.03)	1.47	6.6	0.011	−0.09 (0.04)	1.48				
weight	218.8	<0.0001	0.41 (0.03)	1.50	79.7	<0.0001	0.33 (0.04)	1.55				
parental SEP									10.4	0.001	0.12 (0.04)	1.16
father dead	1.1	0.290	−0.02 (0.03)	1.01	1.9	0.169	0.04 (0.03)	1.02	0.2	0.639	−0.02 (0.03)	1.03
N (dead/alive)		194/1484					115/799				82/740	
minimal effect size	0.19				0.25				0.29			

trait	height				sitting height				leg length			
effect	*F*	*p*-value	β (s.e.)	VIF	*F*	*p*-value	β (s.e.)	VIF	*F*	*p*-value	β (s.e.)	VIF
birth date	34.4	<0.0001	0.15 (0.03)	1.17	81.0	<0.0001	0.23 (0.03)	1.17				
age	22.8	<0.0001	0.12 (0.03)	1.10	49.0	<0.0001	0.17 (0.02)	1.10				
number of siblings	10.5	0.001	−0.08 (0.02)	1.08	8.6	0.003	−0.07 (0.02)	1.08	6.9	0.004	−0.07 (0.03)	1.06
parental SEP	12.1	0.0005	0.09 (0.03)	1.20	7.4	0.004	0.07 (0.03)	1.20	10.4	0.006	0.08 (0.03)	1.08
father dead	0.1	0.826	−0.005 (0.02)	1.03	1.6	0.200	−0.03 (0.02)	1.03	0.6	0.439	0.02 (0.02)	1.02
N (dead/alive)		166/1480					166/1480				167/1481	
minimal effect size	0.22				0.22				0.22			

[a]Removing parental SEP from the model increases the sample size to 94/777 and reduces the minimal detectable effect size to 0.27.

Table 2. Raw and least-square means of variables analysed in table 1. Breast and axillary hair development are measured in 5-grade Tanner scale. LS, least squares.

trait	father alive mean ± s.d. (*n*)	father dead mean ± s.d. (*n*)	father alive LS mean ± s.d.	father dead LS mean ± s.d.
breast stage	2.96 ± 0.50 (1484)	3.01 ± 0.55 (194)	2.96 ± 0.47	3.00 ± 0.47
axillary hair stage	2.10 ± 0.58 (799)	1.98 ± 0.61 (115)	2.09 ± 0.53	2.02 ± 0.53
age of primiparity (years)	25.32 ± 3.97 (740)	25.58 ± 4.58 (82)	25.31 ± 3.97	25.6 ± 4.02
height (cm)	161.56 ± 5.48 (1480)	161.19 ± 5.78 (166)	161.51 ± 5.38	161.20 ± 5.44
sitting height (cm)	86.17 ± 2.99 (1480)	86.15 ± 2.87 (166)	86.13 ± 2.86	86.44 ± 2.89
leg length (cm)	75.36 ± 3.77 (1481)	75.07 ± 4.00 (167)	75.38 ± 3.77	75.14 ± 3.80

fewer children and marginally higher probability of remaining childless. At present, we can only speculate that reproduction was suppressed by perceiving the absence of paternal social and/or economic support. In line with this reasoning, Hungarian girls with dead fathers married at later ages than girls with fathers present (although the eventual number of children was similar [19]). The generality of these findings is not known because studies focusing explicitly on the effects of father's death on daughters' maturation and/or reproduction are extremely scarce [12,20] and only two of them [13,19] were performed in developed countries. However, outcomes of such studies, including the current one, contrast with the findings of studies where the father's absence occurs owing to divorce/abandonment, which often enhances maturation and/or reproduction [1,4,5,12].

We can see two mutually non-exclusive explanations for the discrepancy between the findings of studies of father's death versus divorce/abandonment. First, the nature of psychosocial adversities can be qualitatively different. For instance, children who experience their parents' divorce or paternal abandonment are likely to have had prolonged exposure to conflict between parents, which is less likely in the case of children of widowed mothers [21]. The second explanation is based on genetic correlations between maturation rate and propensity to divorce/abandonment as described in §1. Such an explanation seems to be supported by recent developments of genome-wide association studies. Such studies have already demonstrated that the same genes are largely responsible for the covariation between a family's socio-economic position and children's educational attainment [9,22]. Given the universally negative covariation between women's education and their reproductive rates [23] (which can also be seen in the current dataset), we predict that genetic correlations between age of primiparity and educational attainment will be discovered. To summarize, we found no support for the idea that the mere absence of a father triggers predictive–adaptive responses that result in faster maturation and/or reproduction of their daughters. In the context of previous research, our findings are consistent with the views [12,15,20,21] that different causes of the father's absence will lead to different reproductive outcomes for daughters, depending on the ecological and psychosocial context leading to fatherlessness.

Ethics. The research was carried on under the licence of the Research Ethics Committee of the University of Tartu (protocol no. 235/T-16, issued on 17 March 2014).

Data accessibility. An Excel file is included as the electronic supplementary material.

Authors' contributions. The study was conceived by M.V., who located the data collected by Prof. Juhan Aul (1897–1994), built up and complemented the database. P.H. and M.V. analysed and discussed the data jointly, both authors wrote and critically reviewed the manuscript, approve the final version and agree to be held accountable for the work performed.

Competing interests. We have no competing interests.

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