Room at the top: biological lock-in of social advantage

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0.1 Introduction

Charles Murray (1995) warned of "a merging of the cognitive elite with the affluent". On the opposite side of the political spectrum, Karl Marx (1844) wrote "I am ugly, but I can buy the most beautiful woman. Which means to say that I am not ugly, for the effect of ugliness, its repelling power, is destroyed by money." These quotations suggest that social advantages, such as wealth, caste or status, may be transformed into biological advantages in the next generation, via assortative mating between socially and genetically advantaged people. We call this process biological lock-in.

Figure 1 illustrates the idea using data for spouse pairs from UK Biobank. It plots one partner's mean polygenic score for educational attainment (EA3) against the other partner's university attendance. Members of the sample who attended university had spouses with higher EA3.¹

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However, Figure 1 is not enough to prove biological lock-in. It could be that one spouse's university attendance reflects their own genetic endowment, and that assortative mating takes place on the basis of genetics alone, without any role for social advantage. For example, this might happen if potential marriage partners are good at predicting each other's genetics (via their phenotype) and are only motivated to select partners on the basis of their genetic characteristics.

To demonstrate biological lock-in, we therefore need a source of social advantage which is exogenous to genetics. One possibility is <u>birth order</u>. It is well known that earlier-born children receive more parental care and have better life outcomes. (XXX is it? Go check.) On the other hand, early- and late-born full siblings have the same <u>ex_ante</u> expected genetic endowment. ² We can therefore use birth order as an instrument for social advantage.

We run regressions instrumenting one spouse's educational attainment with their birth order. We control for overall family (i.e. sibling group) size. Without this, then birth order would correlate with family size, which itself may correlate with genetics, since parents of different family sizes might have different genetic endowments. Indeed, respondents with more siblings overall have substantially lower EA3. We also control for subject's own EA3. Lastly, we allow the effect of birth order to vary depending on total family size.

Table 1 shows instrumental variables regressions using 2 stage least squares. We use only subjects with between 2 and 8 siblings, and run separate regressions by sex. The effect is positive for both male and female subjects, but twice as strong, and significant, for males.

Although all children of the same parents have identical expected values of polygenic scores, it is conceivable that parents might select family size on the basis of genetics. For example, if the first child had a phenotype

¹To minimize stratification concerns, EA3 is residualized by the first 100 principal components of UK Biobank array data.

²This might not be the case, if parents' choice of whether to have more children is endogenous to the genetic endowment of their earlier children. We will check for this below.

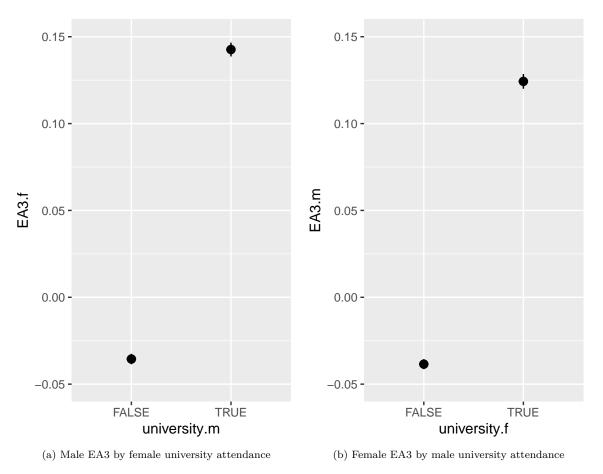


Figure 1: Social and genetic advantage among spouse pairs in UK Biobank

Table 1: Effects of university attendance on spouse's EA3

	Female spouse's EA3	Male spouse's EA3
University	0.130	0.238 **
	(0.086)	(0.080)
EA3	0.034 ***	0.024 **
	(0.009)	(0.008)
Family size dummies	Y	Y
R2	0.010	0.006
N	183777	182725
Sargan test (p)	0.954	0.906

^{***} p < 0.001; ** p < 0.01; * p < 0.05. University instrumented by birth order.

reflecting a high (or low) polygenic score, then that might affect the parents' decision to have a second child. To check this we regress birth order directly on EA3. As before, we control for family size and allow the effect of birth order to vary depending on family size. Birth order variables are not jointly significant (anova p 0.175) and their effect sizes are of the order of 0.01 of a standard deviation of EA3.