Abstract

We exploit the findings from a recent large genome-wide association study of educational attainment to construct a genetic score designed to predict educational attainment. Using data pooled from two independent samples, we deploy this genetic risk score in models for educational attainment in order to test the hypotheses offered by prior researchers that social structure constrains genetic expression for low SES individuals. In contrast to this prior, twin-based research, we find that genetic effects are not moderated by socio-demographic variables such as parental education, age or gender. In fact, the effect of offspring genotype appears to be moderated by maternal genotype, suggesting that prior evidence of gene-by-environment interaction may have actually been gene-gene effects. These findings are consistent with the existence of two parallel systems of ascription: genetic inheritance and social inheritance. We caution, however, that at the presently attainable levels of explanatory power these results are preliminary and may change when better-powered genetic risk scores are developed.

Introduction

Despite the controversy surrounding such estimates, ascertaining the proportion of a quantitative trait—such as education or IQ—that is due to genetic variation has long been of interest to a wide range of scientists. (For a wide range of examples, see, e.g. Breen, Plomin, & Wardle, 2006; Plomin, Fulker, Corley, & DeFries, 1997; Plomin, Owen, & McGuffin, 1994; Plomin & Spinath, 2004; Plomin, 2009; Purcell, 2002; Rodgers, Buster, & Rowe, 2001; Rodgers, Rowe, & Buster, 1999.) Among human populations where experimentation is not possible, the workhorse of such analysis has been the twin or extended twin design, where the average relatedness of various kin pairs is correlated with their phenotypic similarity in order to ascertain the effect of shared genotype on a given outcome (Zaitlen et al., 2013). The reigning critique of this approach is that it is difficult to eliminate the possibility that increased similarity between, say, monozygotic twins as compared to, for example, dizygotic twins, is due to more similar exogenous environments and not just their greater degree of genetic similarity (Goldberger, 1978, 1979—for a defense, see, Conley, Rauscher, & Dawes, 2013; Scarr & Carter-Saltzman, 1979).

A recent meta-analysis that specifically examines the heritability of educational attainment across 36 different cohorts finds a heritability of ~40 percent, though there is significant variation among the individual studies (Branigan, McCallum, & Freese, 2013). For example, a study of Italian twins finds a heritability of ~50 percent (Lucchini, Bella, & Pisati, 2013). However, another recent paper uses U.S. data from the National Longitudinal Survey of Adolescent Health and accounts for assortative mating

and obtains a genetic component of educational attainment of just under a quarter (Nielsen & Roos, 2011).

In motivating these studies, some sociologists have persuasively argued that we should abandon raw or adjusted mobility rates (or, intergenerational earnings elasticities) as measures of openness and meritocracy. Rather, Guo and Stearns and Nielsen among others argue that perhaps we should compare the genetic component to the common environmental component of social status as determined by twin and other kin-based variance decomposition models (Guo & Stearns, 2002; Nielsen, 2006, 2008). In this paradigm, it is not the overall correlation between siblings, for instance, that measures the relative openness or closure of a stratification system (c.f. Björklund, Eriksson, & Jäntti, 2002; Corcoran, Gordon, Laren, & Solon, 1992; Hauser & Sewell, 1986; Hauser, Sheridan, & Warren, 1999; Kuo & Hauser, 1995; Olneck, 1976; Page & Solon, 2003; Warren & Hauser, 1997; Warren, Sheridan, & Hauser, 2002) but rather the proportion of that correlation that is due to genetics. A meritocracy has a high genetic component to achieved social position and a low common (read: familial) environmental component. According to this argument, policy should aim to enhance sorting on innate characteristics and not the social advantages or disadvantages that may be conferred on us by our conditions of birth and upbringing (Heath, Berg, Eaves, & Solaas, 1985).[1]

[1] However, this line of argument conflates genetics with merit. For example, if social sorting in the educational system (or labor market) took place based on eye color, it would be close to 100 percent heritable/genetic, yet few would argue that this form of assignment would be meritocratic, since meritocracy also assumes a legitimacy to the characteristics by which we sort individuals into roles—not merely a biological or natural basis to those characteristics. Admitting students to U.C. Berkeley based on basketball shots would be fair—in the sense that everyone might know the rules

In this vein, knowing whether or not genetic effects for a given outcome interact with social position helps direct policy-makers and researchers to a better understanding of the efficient levers available to them. If for certain groups genetic effects are muted, changing social structures to allow heritabilities to rise would allow for more "equal opportunity" without any costs to efficiency (Heath et al., 1985). By contrast, in a situation where heritabilities were high across the board, more equal outcomes could still be achieved, but such an effort would presumably trade off efficiency for equity.

Considering this line of reasoning, it is perhaps useful to revisit an argument made over 30 years ago by Christopher Jencks (Jencks, 1980). Among the points Jencks made in this classic article was that social scientists tend to think of something that is genetic has having to do with some basic biological process or function that is constant across time and place (i.e. invariant to social environment). But the total genetic component of the variance in an outcome may very well work through environmental channels. For example, children with faster neuronal transmission may seek out (or have bestowed upon them) more cognitively stimulating environments. Indeed, niche construction is a well-studied biological phenomenon whereby individual organisms seek to shape the environment and fitness landscape to best fit their phenotype (Day, Laland, & Odling-Smee, 2003; Laland & Brown, 2006; Laland, 1999; Odling-Smee, Laland, & Feldman, 1996, 2003a, 2003b). Jencks goes on to point out the fact that—as Goldberger had also argued (Goldberger, 1979)—just because something is

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beforehand and be subject to the same constraints in a task that is easily observable with minimal measurement error—but few would agree that this would be meritocratic since there would be a mismatch between the institutional raison d'etre of the university as an institution and the sorting mechanism.

associated with the distribution of genes does not mean that public policy cannot alter the distribution of phenotypes by say, changing the assignment mechanism to something that is orthogonal or even negatively correlated with the preexisting genotype-phenotype covariance. This is true irrespective of whether the proximal cause is biological or social.

For example, phenylketonuria (PKU) is a recessively inherited genetic disease where individuals cannot properly metabolize the amino acid phenylalanine. If phenylalanine is consumed by someone who suffers from PKU, they will suffer cognitive impairments. Jencks points out that knowing this condition is genetic in origin does not mean that we should treat it any differently than say, lead poisoning, which is much more purely environmental in its origins. However, the examples here—lead and phenylalanine exposure—both critically depend on environmental interactions. By preventing children from ingesting phenylalanine or lead, respectively, policy makers would equalize IQ and schooling. Down's syndrome, by contrast, is genetic in origin and fairly unresponsive to environmental conditions. Though there someday may be an environmental intervention—such as a medication—that mitigates the effects of Trisomy-21, at this point in history one does not exist. Thus, whether or not we believe that high heritability is an indicator of an efficient meritocracy, knowing whether the genetic component of a quantitative trait like educational attainment (or IQ) works through interaction with the environment—under present societal and historical conditions—is useful to aid policy makers as to whether there are interventions that would mitigate the effects of genetic predisposition without major efficiency costs.

By way of example, Turkheimer and Haley find that among low income children the heritability of IQ is lower than it is for higher income children (Turkheimer & Haley, 2003). Likewise, Guo and Stearns (Guo & Stearns, 2002) show that for blacks, the heritability of IQ is lower than for whites. In both cases, the researchers interpret this to mean that environmental disadvantages—such as a lack of parental resources or poor schooling conditions or simple racism—prevent the full realization of genetic variation in a given population. In other words, there is an implied GE conditionality such that potential intellectual ability is inherited but requires environmental conditions of human capital investment to be realized in the form of IQ (or educational attainment or income for that matter; c.f. Becker & Tomes, 1994; Behrman, Pollak, & Taubman, 1995; Behrman, Rosenzweig, & Taubman, 1996). If such genotype-by-environment interaction effects held true, this would augur policy interventions that target groups defined by social categories—SES or race—in order to equalize genetic effects (i.e. level the playing field) (Bearman, 2013; Fletcher & Boardman, 2013; Mitchell, 2013).

However, if we ascertain genetic influence (i.e. heritability) latently through twin comparisons (as they do), we can never know if a reduced or enhanced heritability for a given group is due to differences in 1. differential effects of prenatal conditions (c.f. Conley & Strully, 2012); or 2. differences in the level of variation in genotype (numerator) or the denominator (phenotype); or 3. whether, instead, it is truly a difference only in the covariance between genotype and phenotype by subgroup. However, if we measure genotype directly—as we do here—we can interrogate the distribution (i.e. variance) of genotypic propensity toward educational attainment by subgroup, the variance in phenotype by subgroup and estimate interaction effects

between genotype and subgroup (i.e. test whether the covariance between genotype and phenotype differs). Thus, here we aim to provide a much more direct test as to whether the "natural" genetic tendencies of one group—say women or those from low SES families—is repressed by social structure.

That said, an ideal test would be to see if underlying genotype interacts with exogenous environmental shocks—such as school or tax policy interventions (Fletcher & Conley, 2013). Such a research design is beyond the scope of the current paper, however, since we do not have a natural experiment that we know about to apply to the data at hand. Thus, it remains possible any significant interaction effects we discover are not true gene-by-environment effects but rather gene-gene interactions: between measured genotype and unmeasured genotype of parents or the offspring. To mitigate against this possibility, we also directly test for interaction effects between parental educational genotype and offspring educational genotype. While this does not guarantee that our measured environmental variables are truly environmental (and not proxying unmeasured genetic factors), such analysis should give us a sense of whether such confounding is likely to be driving our results.

To preview our findings: We test for interactions between educational genotype (described below) and maternal education, sex, age (birth cohort), study cohort and maternal genotype. The interaction with maternal education is meant as a test of the Turkheimer hypothesis that low-SES represses the true expression of phenotype. The interaction with sex is a test of whether the differential opportunity structure within the educational system for boys and girls is moderated by underlying genetic propensities. The interaction with birth cohort tells us whether an expansionary regime of higher

education (along with other temporal changes over the same period, such as skill-biased technological change [Bekman, Bound, & Machin, 1998; Card & DiNardo, 2002]) affects the expression of genotypic tendencies in the educational system. Finally, the interaction effect with maternal genotype is meant as a check to see if any significant interaction effects with measured maternal education may be indicative of unmeasured GxG interactions within the family (through social interaction) or the individual (by virtue of parental bequeathing of unmeasured genetic stock). We find none of the genotype-by-environment interaction effects tested to be significant in across- or within-family models. Rather, the only significant interaction effect we find is between child and maternal genotype. Thus, our approach casts doubt on the interpretations of prior twin-based researchers who claimed GxE interactions from their latent ascertainment of genetic effects by subgroups of twins.

The Age of Molecular Markers

Until recently, the study of human genetic variation has consisted mainly of behavior genetics studies, where twin and adoption designs were used to identify heritable, or genetic, variation in various traits (see, e.g., Björklund, Lindahl, & Plug, 2006; Plomin, Owen, & McGuffin, 1994; Plomin, 2009; Plug, 2004; Sacerdote, 2007). Whether or not one believes the estimates of genetic influence on phenotypes that emerge from such studies, the fact remains that they do not directly measure genotypes and thus are of limited utility. Today, however, the costs of comprehensively genotyping subjects have fallen to the point where major funding bodies, including those in the

social and behavioral sciences, can now begin to incorporate genetic and biological markers into major social surveys.

The recent addition of genetic markers to large datasets has opened up an opportunity for researchers to model directly how genetic propensities interact with other factors to differentially produce phenotypes. Indeed, a recent paper conducted a genome wide association study of 126,559 individuals from 54 distinct cohorts to search for alleles that may be associated with educational attainment (Rietveld, Medland, Derringer, & Yang, 2013). Rietveld et al. (2013) conducted what is called a genome-wide association study (GWAS), an atheoretical approach to gene discovery where hundreds of thousands of single nucleotide polymorphisms (SNPs) are tested for association with an outcome of interest. In what follows, we index SNPs by j and individuals by i. Each individual SNP is tested for association by running a regression of the sort:

$$y_i = \mu + \beta_j x_{ij} + Z_i \gamma_i + \epsilon_i,$$

where x_{ij} is the number of reference allele individual i is endowed with at SNP j and Z is a vector of controls, which include age, sex and the first four principal components (PC) of the variance-covariance matrix of the genotypic data. The PCs are included to guard against the well-known problem of population stratification; the tendency for allele frequencies to covary with unobserved environmental confounds. Because the number of hypotheses that were tested is very large, it is common to declare a SNP-association to be significant if it reaches a p-value of 5×10^{-8}. Rietveld et al. (2013) identified three SNPs that reached this level of significance, and all three replicated in an independent sample. However, the greater significance of this study is

that it allows for the construction of a polygenic risk score for educational attainment. A common approach to constructing such a genetic risk score (GRS), which we label $\hat{g}$, is to take a weighted sum of SNP, where the weights are given by the estimated β_j coefficients from Equation 1:

$$\hat{g}_i = \sum_{j=1}^{J} x_{ij}\hat{\beta}_j$$

The results from the Rietveld et al. 2013 analysis are reproduced as Figure 1. (For other examples of GRS deployment, see, e.g., Belsky et al., 2012; Belsky, Moffitt, et al., 2013; Belsky, Sears, et al., 2013; Benjamin et al., 2012; SM Purcell, Wray, & Stone, 2009; Visscher, 2010; Yang, Benyamin, & McEvoy, 2010.) The steep slope of the lines shows the polygenicity of educational attainment. That is, while only three alleles reached what geneticists call genome-wide significance ($p < 5 \times 10^{-08}$) and replicated in the independent samples, these explained a trivial amount of the total variance in years of schooling or college attendance. Meanwhile, relaxing the threshold continually increases the predictive power of the genetic risk score all the way to the point where all autosomal SNPs are taken into account regardless of significance level. This suggests that to the extent that it is associated with genotype, educational attainment—as we might expect—is driven by many small effects across the entire genome.

FIGURE ONE ABOUT HERE

Two percent is a relatively small contribution to our understanding of educational outcomes, especially when compared to the published meta-analyses that find that genetic factors account for up to 40 percent of the variation (Branigan et al., 2013). There are several important explanations for the discrepancy. This “missing”

heritability (de Los Campos, Vazquez, Fernando, Klimentidis, & Sorensen, 2013) is due to a variety of factors. Most importantly, there is estimation error in the $\widehat{\beta_j}s$, which attenuates the score. The genotyping platforms capture common variants and ignore rare alleles that may matter; and even these common variants are not necessarily the causal loci but only correlated spatially with the "true" causal loci on the genome. Further, there may be complex, cross-allele interaction effects that explain part of the genetic component of the variability in the trait. These are not captured by the linear and additive functional form that is assumed in Equation 1.

With these caveats in mind, the approach of the present paper is to take the algorithm for this single polygenic educational "risk" score and apply it to data pooled from two samples that were not included in the original discovery or replication studies. Our study builds on Rietveld et al. (2013) because we examine *intergenerational* models of educational attainment that include genetic endowment in both the parental and offspring generations and examine interaction effects between the polygenic risk score and socio-demographic factors: namely, parental education, sex and age (i.e. cohort).

Of course, for the present analysis we only observe two percent of the putatively forty percent of educational-genotypic covariance, so the question becomes whether or not the two percent explained by our GRS is systematically biased with respect to the underlying, ~ 40 percent of educational variance that is associated with genotype on the one hand, and parental education, on the other hand. That is, if the missing 38 percent displays the same covariance with parental education, then the fact that we are measuring only two percent out of 40 merely implies that as we obtained a better measure of genotype, we would increase in a linear fashion the degree to which our

observed measure of genotype moderates the parent-offspring educational correlation. In this case of random measurement error being the problem behind the missing heritability in our sample, our inferences are still asymptotically unbiased, if attenuated, in the present specification.

Finally, it could be the case that alleles are non-randomly distributed across social grouping making the genetic effect spurious. Imagine white ethnic group A has higher education on average than white ethnic group B due for historical, cultural, or economic reasons. Meanwhile, white ethnic group A also scores higher on the polygenic risk score for education for random reasons of genetic drift. It could appear that the polygenic risk score causes educational attainment when it is really just acting as a proxy for socially observable differences (ethnic culture). This is what geneticists call population stratification and is illustrated by the chopsticks problem (Hamer & Sirota, 2000). The weights for the alleles that go into calculating the polygenic risk score that were calculated by Rietveld et al. (2013) attempted to eliminate population stratification by controlling for the first two principle components in the data structure. This is the typical approach in large scale, genome wide analysis. However, it is certainly possible that given the multi-national consortium of cohorts, this is inadequate when the score is then transferred to a specific cohort within a given country or region of a country (as our datasets are).

To address this concern, we run family fixed effects models. By comparing full siblings from the same family, concerns about genetic-environmental confounding are obviated since the differences in polygenic risk score between siblings stem wholly from the random segregation of grandparental alleles during the meiosis that produces the

parental gametes. Sibling differences in genotype, then, represent the result of random assignment at conception when a given spermatozoon fertilizes a specific oocyte. We can then multiply sibling differences in genotype with within-family factors gender and age to see if there are any significant interaction effects without any concern that they may be confounded by population structure. Finally, we also test for interactions between offspring educational genotype and parental educational genotype as a direct test of GxG interaction effects that may be spuriously generating any putatively observed GxE effects.

Data

The data for the present study come from the Framingham Heart Study (FHS) second and third generation respondents as well as from the Minnesota Twin Family Study (MTFS). Genotypes for the FHS were assayed using the Affymetrix GeneChip Human Mapping 500K Array and the 50K Human Gene Focused Panel. Genotypes were determined using the BRLMM algorithm (additional details can be found at [Jaquish, 2007]). Of the original 500,568 SNPs, 260,469 were left after cleaning (e.g., HWE screens and a MAF cut-off of 0.05). The screens were conducted using all available individuals with genetic data, not only those that were included in this analysis. The Minnesota Twin Family Study (MTFS) data were genotyped on the Illumina 660W Quad array. Detailed information on the sample (Iacono & McGue, 2002) as well as on genotyping and quality control (QC) can be found elsewhere (Miller MB, Basu, S., Cunningham, J. M., Eskin, E., Malone, S. M., Oetting, W. S., McGue, 2013). In brief, QC procedures were applied separately to each individual cohort. Individuals with a call

rate <0.95 (N=22), estimated inbreeding coefficient > 0.15 (N=2) (Yang et al 2011), and individuals showing evidence of non-European descent from multidimensional scaling (N=298, mainly individuals with Mexican ancestry) were removed. Individuals were considered outlying from European descent if one or more of the first four eigenvectors were more than three standard deviations removed from the mean. SNPs with minor allele frequency (MAF) < 0.01, call rate < 0.95 or Hardy-Weinberg Equilibrium test p-value <0.001 were removed. We show descriptive statistics against the non-Hispanic white sample of the 2012 General Social Survey for comparative purposes. The GSS is well-known to social scientists and has been described extensively elsewhere (Davis & Smith, 1992).

We did not focus only on non-Hispanic whites for reasons of political caution. Rather, the nature of our samples was that they were predominantly white to begin with. Added to this fact is that the polygenic risk score was obtained from a consortium that included only respondents of European heritage. Since there are vastly different allele frequencies, haplotype structure and much greater genetic diversity among those of African descent (Tishkoff et al., 2009), it was not feasible to apply the polygenic score to black (or Latino) U.S. respondents. It simply would likely fail to explain much variation in these populations. That said, given the association between allele frequencies and race, we wanted to make sure the claims we were making would not be attributed to race instead of genotype per se. This has been an issue in past research, especially when single, candidate gene approaches have been deployed (see, e.g., Freese & Shostak, 2009 for a critical examination). That said, within-family models of full-sibling differences obviates any confounding of race (or ethnicity) and genotypic effects. But we would not

have easily been able to draw unbiased inferences from the between-family component of the analysis.

[TABLE ONE ABOUT HERE]

Table 1, above, shows descriptive statistics for these three samples. The most stark difference between the populations is with respect to age. The GSS shows a mean age of almost 50 years (49.05) with considerable variability (standard deviation of 17.10 years). The 3rd generation respondents of the Framingham Heart Study who are included in our sample (i.e. have valid responses on both the social and genetic variables of interest as well as valid data on their parents) are just under a decade younger at 39.49 years of age on average, with concomitant lower variability as well (SD = 7.67 years). Meanwhile, the MTFS sample is much younger, with a mean age of 24.33 years and a standard deviation of less than a year (0.85 years) by virtue of its cohort design. While the age distribution does vary considerably, the sex ratio is almost the same, ranging from 50 to 54 % female across the samples. Finally, the mean education levels also vary between the different studies for a variety of reasons including attrition, region, age, and cohort effects. For example, both the FHS and MTFS display higher mean education levels for both the respondents and their parents. This may be due to the fact that Minnesota and Massachusetts are two states with high average education levels as compared to the nation writ large. For example, the mean rate of college graduation was highest in Massachusetts of all the states in the U.S. at 38.2 percent in 2009; Minnesota was ranked 10 with a 31.5 percent rate that same year; meanwhile the nation as a whole had a mean bachelor degree rate of 27.9 percent (U.S. Census Bureau, 2012). These state differences—along with cohort effects (namely that the GSS sample is

older)—probably account for the higher parental education levels in the two state samples. These differences are slightly mitigated in the respondent generation due to age effects—i.e. the fact that the offspring, who are younger than those in the GSS, probably have not all completed their educational careers. This would explain why the younger sample from Minnesota has a mean education level of one-third year lower than the Framingham sample.

Results

In column A of Table 2, we show a base model with no interaction effects. Here the two datasets are pooled to increase statistical power. We show parameter estimates for a basic model of educational inheritance that includes controls for sex, age, and for sample (i.e. MTFS v. FHS). (We have also tested a model that interacts the sample indicator variable with all the other predictors. This specification does not change our main results and thus for reasons of parsimony is not shown but is available upon request of the authors.) In this base model, the respondent's own genotype, as measured by the polygenic risk score, is standardized within each sample in order to make its magnitude interpretable. It is significant in predicting completed years of schooling such that each additional standard deviation in the score yields a 0.16 additional year of formal education. The effect of maternal phenotype, meanwhile, is not significant net of offspring genotype.

Next, in models B-D, we test whether measured genotype moderates (or is moderated by) the socio-demographic variables sex, age or parental education,

respectively. None of these interaction effects is significant. In Model E we even test whether the effect of genotype varies across the underlying populations. That is, we interact offspring genotype with the sample from which the respondent came. One could think of this as an omnibus test for any sort of unmeasured difference between the two populations, including, but not limited to: geography (Minnesota v. Massachusetts [and out-migrants from Massachusetts]), cohort (since the Minnesota sample is younger), or social class (since the education of the parents of the MTFS is higher on average than that of the FHS parents). Yet, this interaction is not significant either.

Of course, had we found significant interactions, it could have been the case that these putatively environmental measures we are interrogating were actually acting as proxies for unmeasured genotype. This is not a concern for age, which should be orthogonal to genotype within the evolutionary window we are examining. Likewise, though alleles on the X or Y chromosomes could have been driving an interaction with our measured autosomal alleles for the "sex" variable, this is unlikely, especially since we did not find a significant interaction effect. It is of most concern for parental education in this study and in prior studies, since parental education may be driven by the very same genotype as we were measuring in the offspring generation. In an ideal world, we would have an exogenous, environmental source of variation in parental education and interact offspring genotype with this instrumented measure of parental education. Of course, it is hard to envision what such an instrumental variable would be that would not violate the exclusion restriction (i.e. have no direct effect on offspring education other than through years of schooling of the parent). In lieu of this idealized study design, we control for maternal genotype in our models and do not find a

significant offspring genotype-parental phenotype interaction. If we had found a significant interaction effect, endogeneity concerns would warrant caution in interpreting this as a true GxE effect. Given our finding is null, such concerns are secondary.

That said, in Model F, we estimate a parental genotype-offspring genotype interaction effect directly. Parental genotype is the latent, lurking variable in prior studies that claim GxE interaction effects between genotype and social class (Turkheimer & Haley, 2003), so this is important test in conjunction with our null result in Column D. And indeed, here we find the only significant interaction effect in all the models: between maternal genotype and offspring genotype. This suggests that growing up with a genetically advantaged or disadvantaged mother enhances the effects of one's own standing in the genetic lottery. The coefficient is positive such that a child born to a genetically average mother who by the luck of recombination has a genotype that is one standard deviation above the mean for the offspring generation will complete, on average 0.16 more years of schooling than the kid at the mean of the genotype distribution. However, if that same child was born to a mother who herself was also one standard deviation above the mean in the genetic lottery, the child's advantage would be almost a quarter year of schooling. Note that this is net of how many years of schooling the mother actually completed—i.e. it is likely an interaction with her native cognitive or non-cognitive ability (that itself predicts education), not her achieved educational (social) status.

We take the combination of non-significant results for plausibly environmental interactions in conjunction with the significant genotype-genotype interaction as

suggestive that prior work that has claimed GxE interaction effects by parental class or education level may have actually been picking up GxG interaction effects, given the endogeneity of parental SES/education to parental genotype.

[TABLE TWO ABOUT HERE]

One concern might be that though the original parameters that went into the risk score calculation were based on a pooled analysis of 36 cohorts across a wide range of countries and controlled for principle components to address concerns of population stratification (Rietveld et al., 2013), the genetic score here could be acting as a proxy for unmeasured environment, in which case both its main effect and the interaction effects with other variables could be biased. To address this possibility, in Table 3, below, we show results from sibling fixed effects regressions. This design insures that genotype is a randomly assigned variable at conception since the only portion of it that varies is that which differs between siblings from the same parents. That is, conducting within-family analysis breaks all potential rGE and population structure. (Of course, there are environmental differences within families, but to the extent that these are correlated with genotype, they form part of the genetic effect as "endogenous environment" or niche formation [c.f. Jencks, 1980].) In the base model, A, we find that indeed, a respondent's genotype predicts her education even in this stricter test. However, under this approach, none of the interaction effects are significant—even the interaction between mother's genotype and offspring genotype that was significant in the OLS models of Table 2.

[TABLE THREE ABOUT HERE]

To further interrogate the maternal-offspring genotype interaction effect that was significant in OLS models, we bring fathers into the picture in Table 4, below. Any significant parent-offspring GxG interaction effect is, of course, likely working through the family environment, but in a way that is endogenous to family genotype and thus will play out differently with respect to educational or policy interventions than an interaction with parental exogenous phenotype. Namely, in the true G by exogenous E scenario, improving parental education would have a benefit of improving the returns of offspring genotype as well. However, in the GxG situation, improving parental education would not have such dynastic effects. Rather, an environmental intervention would need to be apply directly to the children to mimic higher innate ability of mothers.

In a random mating situation, maternal genotype would be orthogonal to paternal genotype. In our sample, the maternal and paternal scores are correlated at 0.09, implying some slight positive assortative mating on educational genotype. Thus, it could be that maternal genotype is acting as a proxy for paternal genotype. To address this concern, in Table 4, below we rerun the analysis controlling for paternal phenotype and paternal genotype (since more data are missing for fathers, this analysis suffers from a reduced N). The critical model is B, where even controlling for paternal variables, we find that the interaction effect of maternal and offspring genotype is still significant. Meanwhile, in Model C, a paternal-offspring genotype interaction is not significant. Given the prior that mothers are more critical to shaping the household learning environment for children than are fathers, this suggests that maternal genotype is not merely acting as a proxy for additional genetic influences in the offspring herself

(since father's genotype should have had the same proxy effect if this were true), but rather, is picking up a true interaction between maternal and offspring genetically-influenced ability.[2] Finally, Model D shows interactions between both paternal and maternal genotype, on the one hand, and offspring genotype, on the other. In this model, multicolinearity leads to null results for both coefficients. (Likewise, higher order interactions—not shown—are also insignificant.)

[TABLE FOUR ABOUT HERE]

Discussion

The move from twin based models to studying SNPs and other genetic polymorphisms (such as Copy Number Variants) has opened up a particularly promising research program on genetic-(social) environmental (GxE) interactions in human populations. The estimation of such interaction effects has long been a goal of social scientists fond of expressing the dependence of genetic expression on social structure. However, how do we get from the sociological adage that "a gene for aggression lands you in prison if you're from the ghetto, but in the boardroom if you're to the manor born" to a serious empirical research program on the study of GxE interactions? Since at least the publication in *Science* (Caspi et al. 2002, 2003) of empirical evidence of gene-environment interaction (GxE), there has been growing interesting in integrating biological and social science approaches, data, and models.

[2] This is really just a check since we deploy a linear, observed genotype constructed from alleles that are pruned for linkage disequilibrium, there is by design not likely to be any way that measured offspring genotype could be acting as a proxy for unmeasured genotype that varies systematically by parental genotype (and vice versa). This is yet another an advantage of using a genetic risk score constructed out-of-sample rather than latent genotype or single SNP approaches.

Caspi et al. (2002, 2003) suggested an important, genetic source of heterogeneity in responses to adverse early-life events, attempting to partially answer the question of why some individuals are resilient to stressors while others suffer deleterious psychological sequelae. While these studies created substantial interest in potential gene-by-environment interactions, they also required replication and extension by other researchers using alternative data. Indeed, there are now competing meta-analyses suggesting either that the original results linking differential response to stress by 5-HTT are reasonably robust (Karg et al. 2011) or lack consistent supporting replication (Risch et al. 2009).

The discussion generated by this line of research in the biological and social science communities has been productive because it has led to a greater appreciation of the shortcoming of Caspi et al.'s research design - namely that the alleles and the proposed environmental modifiers may not be randomly assigned in the population and may therefore correlated with unobserved causal factors. For example, it may be the case that an observed interaction between a genetic variant and environmental exposure actually reflects differential risk of exposure (e.g., "genes selecting environments") rather than the genetic modification of exogenous environmental exposures. This is known as gene-environment correlation (rGE). Further, measured environments—particularly when fashioned by parents who also pass on their genes to the respondents—may be correlated with unmeasured genetic variation and thus could be acting as proxy for a gene-by-gene interaction rather than a true gene-environment interaction.

While some researchers have attempted to deal with this problem by finding exogenous sources of environmental influences such as state cigarette tax policies

(Fletcher, 2012), in utero differences in nutrition (Conley & Rauscher, 2013; Cook & Fletcher, 2013), legal policies (Guo, Cai, Guo, Wang, & Harris, 2010) or regional economic conditions (Lee, 2013), they still interact single SNPs with the given environmental stressor. Since candidate gene studies like these cannot adequately rule out the possibility that alleles are not randomly distributed across unmeasured environments, it is entirely possible that such studies are detecting ExE interactions rather than GxE effects—between, for example, tobacco taxes and state educational differences.

Such potential confounding is known as "population stratification"—a concept popularized by Hamer and Sirota (Hamer & Sirota, 2000) who used the example of a "chopstick gene" appearing because of data that mixes Asians and Caucasians. The above mentioned studies all limit their samples to whites; however, even within an ethnically homogenous population, genotypes may be acting as proxies for different places or social environments (Benjamin, Cesarini, Chabris, et al., 2012; Cardon & Palmer, 2003). By moving GxE research to analysis of genome-wide data, we can address this concern of population stratification using controls for principle components (Price et al., 2006).

Second, most complex, behavioral phenotypes are highly polygenic in nature as we have shown for educational attainment in Figure 1. Thus, knowing whether the "overall" measured genotype for a quantitative trait like educational attainment interacts with environmental conditions is arguably of more scientific importance than estimating GxE effects at particular locus—whose effects will no doubt be overwhelmed by the thousands of main effects (and interactions) at other loci.

Despite the advantages of our approach, in the present study we do not enjoy exogenous environmental shocks to parental education (i.e. a natural experiment) that we can apply within our data; thus, we cannot be 100 percent sure that we have broken the rGE and that our measured environmental variables are indeed not proxying for unmeasured genetic influences (GxG). That said, we take comfort in four facts: First, we deployed principle components in the construction of the index to address the potential for rGE. Second, at least two the environmental variables with which we interact genotype are putatively orthogonal to genotype: sex and age. (Sex is uncorrelated with our measured genotype since we construct the index only from autosomal genes, though any effect of sex could still be reflecting unmeasured X or Y linked genotype.) Third, sex and age, which vary within families, do not interact with genotype (which also varies within families) even in sibling fixed effects models.

Two much more important limitations to our study should provide grist for future researchers: First, as mentioned before, our measure of genotype, while highly robust to population stratification, only explains two percent of the variance in education. This is likely due to a number of factors, not the least of which is a lack of statistical power—notwithstanding the risk score's calculation on 126,559 individuals. Rietveld et al. 2013 show that with increased power such attenuation bias in the predictive power of the GRS would decline. A second concern with our operationalization of genotype rests in the fact that by design, it is a linear predictor of main effects based on meta-analysis across a wide range of environments. In one way, this represents an advantage to our approach in that we are working from well-established main effects to see if they vary by context. Such a directed search effectively

reduces the number of hypotheses tested from millions (of SNPs) times the number of environmental regimes to a handful (i.e. one index score times the number of environmental factors tested). However, a cost to this approach is that it may be the case that we are testing only that part of the genetic influence on education that is the most robust (i.e. inelastic) to environment.

Conclusion

If researchers studying educational attainment merely want to describe the extent to which children resemble parents on this dimension, they need not concern themselves with the mechanisms by which such an intergenerational correlation is obtained. However, if scholars seek to understand how this social fact comes into being and, further, wish to know whether policies that affect the distribution of education in one generation will have distributional consequences in the next generation, then whether or not the observed parent-child correlation in education varies by genotype should be of utmost importance.

We find absolutely no evidence that the effect of genotype varies by maternal education or any of the other variables in our analysis. Besides maternal education, we also tested interaction effects with sex, age and study cohort. All null findings. Meanwhile, we did detect a maternal genotype-offspring genotype interaction on offspring education. This provides at least limited circumstantial evidence that there are two parallel systems of educational inheritance at work—social and genetic—and the two do not articulate. However, it also suggests that if policymakers wish to counteract

the social sorting effects of genetics, they will have to look hard for levers since—at least in our observational data—we could not find any environmentally manipuable factor by which the effect of the GRS was muted (or enhanced).

This last point stands in contrast to the findings of Turkheimer and Haley who deploy traditional twin models and find that among low income children the heritability of IQ is lower than it is for higher income children (Turkheimer & Haley, 2003) as well as research by Guo and Stearns (Guo & Stearns, 2002) who show that for blacks, the heritability of IQ is lower than for whites. In both cases, the researchers interpret this to mean that environmental conditions—such as a lack of parental resources or poor schooling conditions or simple racism—prevent the full realization of genetic variation in this population. In other words, there is an implied GE covariance such that potential intellectual ability is inherited but requires environmental conditions of human capital investment to be realized in the form of IQ (or educational attainment or income for that matter; c.f. Becker & Tomes, 1994; Behrman, Pollak, & Taubman, 1995; Behrman, Rosenzweig, & Taubman, 1996). They obtain this result from traditional ACE twin models and not from measured molecular data. Thus it could be that differences in the numerator (i.e. genetic heterogeneity) are driving their results and not differences in the denominators (i.e. phenotypic variability). By contrast, here we know that the underlying variance of GRS is not different by population subgroups (see Supplemental Table 1 for these figures), and we do not find any evidence that its effect varies by social class position (i.e. parental education), or for that matter, age, sex or cohort.[3]

[3] Of course, by definition, when we group respondents by phenotype, the variance within those categories difference. The more critical data point is that the standard

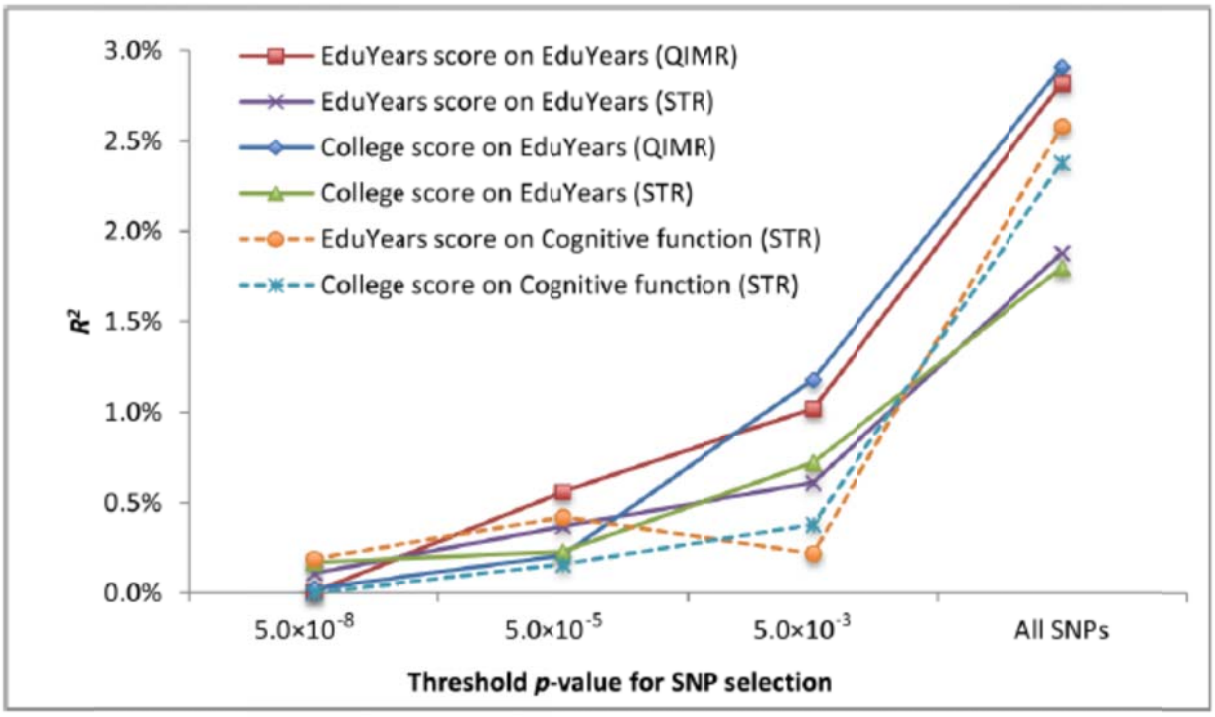
Meanwhile the fact that maternal genotype *does* moderate the effect of offspring genotype suggests that perhaps prior researchers claiming to have found parental SES-genotype GxE interactions are actually picking up GxG interactions.

An important caveat to mention to all this analysis is that heritability—i.e. genetic influence—is, of course, highly contingent on social structure whether measured latently through kin correlations or directly by a GRS. Indeed, heritability is not a fixed parameter across time and place but is always a "local perturbation analysis" estimate as cogently argued by Feldman and Lewontin 35 years ago (Feldman & Lewontin, 1975: 1163). These authors go on to claim that "a complete analysis of the causes of variation would involve predicting the changes in the IQ distribution of genotypes and environments Φ(G,E). However, such analysis would require that we know the first partial derivatives of the unknown function f(G,E)." Instead, behavioral geneticists (and we here) typically pursue on a strategy that relies a small fluctuation around the observed mean values. So while we cannot reject the null hypothesis that genetic effects on education do not depend on social structure, we cannot reject it in perpetuity. Nor can we aver with any certainty that our polygenic risk score will be at all predictive in future generations as the social institution of education changes form. That said, we still believe it is useful to understand the relationship between genotype and phenotype even in partial equilibrium when it comes to important social outcomes like educational attainment. Our estimates then become the fodder for future analysis of waning or waxing articulation between social and genetic reproduction.

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deviations for our genotypic measure do not systematically vary by grouping. (See Table S1.)

Figure 1. Solid lines show results from regressions of EduYears on linear polygenic scores in a set of unrelated individuals from the Queensland Institute for Medical Research Sample (QIMR, N = 3526) and STR (N = 6770) cohorts. Dashed lines show results from regressions of Cognitive function on linear polygenic scores in a sample from STR (N = 1419). The scores are constructed from the meta-analysis for either EduYears or College, excluding the QIMR and STR cohorts. Reproduced from Rietveld et al., 2013.



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Table 1

Descriptives by Sample

	GSS		FHS		MTFS	
.	Mean	SD	Mean	SD	Mean	SD
Education (HGC)	14.25	2.79	15.08	2.06	14.75	2.35
Maternal Education (HGC)	12.29	3.12	13.66	2.26	13.87	1.9
Paternal Education (HGC)	12.23	3.74	14.41	3.04	14.24	2.35
Female Sex	0.54	0.5	0.5	0.5	0.54	0.5
Age	49.05	17.1	39.49	7.67	24.33	0.85
Polygenic Risk Score	--	--	-0.00000971	7.21E-06	9.65E-07	9.65E-07
Maternal Polygenic Risk Score*	--	--	-9.89E-06	7.51E-06	1.14E-06	8.44E-06
Paternal Polygenic Risk Score*	--	--	-9.65E-06	7.12E-06	6.28E-07	8.49E-06
N	1052		1256		1291	
Families	1052		361		727	

*N for FHS for this variable is 741 individuals from 241 familie

Table 2

OLS Regresion Models with Controls for Sample and Standard Errors Robust to Clustering on Family ID*

	A		B		C		D		E		F	
	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE
Female Sex	0.4232836	0.0822	0.4247446	0.08223	0.4254049	0.0821	0.425081	0.08209	0.4257097	0.0821	0.4255738	0.08199
Age	0.016606	0.0093	0.0165869	0.00926	0.0163079	0.0092	0.0165013	0.00923	0.0163064	0.0092	0.017037	0.0092
Mother's Education	0.3953004	0.0292	0.3953289	0.02919	0.3987243	0.0288	0.3983944	0.02918	0.3980654	0.029	0.3954789	0.02923
Respondent's Genetic Score	0.1600584	0.0483	0.2514799	0.13361	0.3282805	0.1645	0.2119399	0.08067	0.1079933	0.0683	0.156169	0.04846
Mother's Genetic Score	*-0.0353382*	*0.0517*	*-0.0345381*	*0.0517*	*-0.034284*	*0.0518*	*-0.0337779*	*0.05187*	*-0.0336817*	*0.0519*	*-0.041464*	*0.0513*
Sample Indicator (MTFS=1)	3.700511	0.3455	3.698393	0.34601	3.731763	0.342	3.725737	0.34486	3.721953	0.3435	3.717785	0.3455
Respondent Score * X:												
Female Sex			*-0.0609164*	*0.0812*								
Age					*-0.005678*	*0.0052*						
Mother's Education							-0.0069269	0.00792				
Sample Indicator (MTFS=1)									*0.0843433*	*0.0838*		
Mother's Genetic Score											**0.0879634**	**0.03838**
Constant	8.417656	0.5781	8.41689	0.57868	8.376916	0.5724	8.380826	0.5758	8.388687	0.5743	8.341605	0.57893
R^2	0.1387		0.139		0.1393		0.1391		0.1392		0.1412	
N	2478		2478		2478		2478		2478		2478	
Families	1088		1088		1088		1088		1088		1088	

*Coefficients in italics are*not* significant at p<.05 (one-tailed); bold indicates interaction effect significant

a fully saturated model with interactions between sample indicator and all other variables does not change main results.

Table 3

Fixed-Effects Regresion Models with Controls for Sample and Standard Errors Robust to Clustering on Family ID*

	A		B		C		D		E		F	
	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE
Female Sex	0.4173009	0.0822	0.4095299	0.1165	0.4190047	0.1163	0.4190689	0.11634	0.4185908	0.11631	0.4232678	0.11653
Age	*-0.0034561*	*0.0093*	*-0.0032992*	*0.0109*	*-0.002817*	*0.0109*	*-0.0041424*	*0.01093*	*-0.0034115*	*0.0109*	*-0.003946*	*0.0109*
Mother's Education												
Respondent's Genetic Score	0.2139994	0.0483	0.3999248	0.1704	-0.050436	0.2654	0.0370288	0.1763	0.2577071	0.07295	0.2129351	0.06493
Mother's Genetic Score												
Sample Indicator (MTFS=1)												
Respondent Score * X:												
Female Sex			*-0.124644*	*0.1056*								
Age					*0.007416*	*0.0072*						
Mother's Education							*0.0152166*	*0.01409*				
Sample Indicator (MTFS=1)									*-0.2093718*	*0.1597*		
Mother's Genetic Score											*0.0545919*	*0.06*
Constant	14.28969	0.5781	14.29666	0.3791	14.27067	0.3796	14.30508	0.37938	14.2885	0.37904	14.26542	0.3801
R^2	0.684		0.6844		0.6843		0.6843		0.6845		0.6842	
N	2478		2478		2478		2478		2478		2478	
Families	1088		1088		1088		1088		1088		1088	

*Coefficients in italics are*not* significant at p<.05 (one-tailed); bold indicates interaction effect significant

a fully saturated model with interactions between sample indicator and all other variables does not change main results.

Table 4

OLS Regresion Models with Controls for Sample and Standard Errors Robust to Clustering on Family ID; Father's Characteristics Included*

	A		B		C		D	
	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE
Female Sex	0.4479851	0.0887	0.4511392	0.08223	0.4529339	0.0887	0.4543166	0.08851
Age	0.0197813	0.0104	0.0203089	0.00926	0.0200848	0.0103	0.0204484	0.0103
Mother's Education	0.286311	0.04	0.2864074	0.02919	0.286009	0.0402	0.2861657	0.04028
Respondent's Genetic Score	0.18227	0.0625	0.1754178	0.13361	0.18421	0.0625	0.1780027	0.06278
Mother's Genetic Score	*-0.0720091*	*0.0576*	*-0.0748196*	*0.05172*	*-0.074117*	*0.0576*	*-0.07593*	*0.0575*
Sample Indicator (MTFS=1)	4.438245	0.4081	4.463687	0.34601	4.446688	0.4079	4.465786	0.40831
Father's Education	0.1570781	0.0382	0.1579459	0.03827	0.1573671	0.0384	0.1580183	0.03836
Father's Genetic Score	*-0.0035049*	*0.0565*	*-0.00225*	*0.05642*	*-0.010108*	*0.0563*	*-0.007395*	*0.0565*
Respondent Score * X:								
Mother's Genetic Score			**0.0789458**	**0.04184**			*0.0658736*	*0.0419*
Father's Genetic Score					*0.062559*	*0.0401*	*0.0467692*	*0.0405*
Constant	7.473592	0.6543	8.41689	0.65549	7.415155	0.653	7.358418	0.65546
R^2	0.1509		0.1528		0.1523		0.1535	
N	2032		2032		2032		2032	
Families	980		980		980		980	

*Coefficients in italics are *not* significant at p<.05 (one-tailed); bold indicates interaction effect significant

a fully saturated model with interactions between sample indicator and all other variables does not change main results. higher order interactions (such as between mother's and father's genetic score and between that interaction and child's score are not significant and not shown.

Table S1

Distribution of Key Independent and Dependent Variables by Subgroup

Framingham Heart Study Sample												
	Score		Mom Score		Dad Score		Education		Mom Education		Dad Education	
.	SD	N	SD	N	SD	N	SD	N	SD	N	SD	N
Maternal Education <13	7.11E-06	548	7.05E-06	410	6.99E-06	374	2.04839	548	0.96181	548	2.41386	548
Maternal Education 13+	7.18E-06	708	7.59E-06	558	6.95E-06	519	1.855789	708	1.75959	708	2.780829	708
Female Sex	7.02E-06	671	7.28E-06	522	6.97E-06	466	2.000245	671	2.1298	671	2.984441	671
Male Sex	7.43E-06	585	7.78E-06	446	7.04E-06	427	2.121904	585	2.39459	585	3.099313	585
Age < 40	7.45E-06	647	7.45E-06	549	7.14E-06	515	1.932445	647	2.08129	647	2.866482	647
Age 40 +	6.94E-06	609	7.54E-06	419	6.78E-06	378	2.182196	609	2.38851	609	3.09353	609
All respondents	7.21E-06	1256	7.51E-06	968	7.00E-06	893	2.063438	1256	2.25656	1256	3.039379	1256
Minnesota Twin Family Study Sample												
	Score		Mom Score		Dad Score		Education		Mom Education		Dad Education	
.	SD	N	SD	N	SD	N	SD	N	SD	N	SD	N
Maternal Education <13	8.01E-06	714	7.33E-06	796	8.02E-06	611	1.916257	508	0.56615	814	1.734476	654
Maternal Education 13+	8.03E-06	1653	8.62E-06	1534	8.49E-06	1357	1.800485	1143	1.55932	1566	2.316541	1436
Female Sex	7.84E-06	1286	8.46E-06	1176	8.25E-06	1058	1.806518	868	1.96626	1176	2.437781	1054
Male Sex	8.27E-06	1099	8.12E-06	965	8.54E-06	841	1.933303	783	1.81187	965	2.201467	837
Age < 25	8.15E-06	980	8.12E-06	903	8.37E-06	809	1.839472	935	1.89157	903	2.43601	803
Age 25 +	7.98E-06	1387	8.33E-06	1427	8.35E-06	1159	1.955743	716	1.90677	1477	2.26996	1287
All respondents	8.05E-06	2367	8.25E-06	2330	8.36E-06	1968	1.89106	1651	1.90066	2380	2.334907	2090