A New, Near-Gaussian Measurement of Leftism Shows the Paternal Age Effect Replicates

Joseph Bronski

OpenPsych

Abstract

Previously, we showed that there is a paternal age effect on leftism (increasing leftism with increasing age of father when born), using a binary classification based on three items regarding Black Lives Matter, LGBT, and feminism [1]. A major limitation of that study was the use of the binary measurement. In this paper, we show that the same effect is detectable with a new, near-Gaussian measurement of leftism. The correlation between this measurement and paternal age was r = 0.12 (p < 0.001). This measurement has high reliability (Cronbach's α = 0.93) which far outperforms the commonly used Wilson-Patterson Conservatism Scale (α = 0.71) [2] as well as high criterion validity, as evidenced by the Cohen's d between Republicans and Democrat on the measurement (d = 2.31, p < 0.001). Likewise, we show that, as before, there is no significant correlation between general leftism and age when having a child in fathers, suggesting this result is not due to older fathers being more leftist.

1 Introduction

In a previous article, we showed that there is a paternal age effect on leftism (increasing leftism with increasing age of father when born), using a binary classification based on three items regarding Black Lives Matter, LGBT, and feminism [1]. As explained in the introduction of that article, this is potentially indicative of mutational pressure increasing the incidence rate of leftism in the population. Furthermore, we showed, with the same binary measurement of leftism, that older fathers and their wives were not more likely to be leftist than younger fathers and their wives.

A major limitation of that study was the use of the binary measurement. Binary variables can be problematic for a number of reasons [3]. Dichotomization at the mean can often lead to a reduction in effect sizes, occurrence of spurious significant main effects or interactions, risks of overlooking nonlinear effects, and problems in comparing and aggregating findings across studies.

In this article, we update our measurement to be continuous, and show that the paternal age effect is in fact present under the continuous metric. Our measurement is near-Gaussian, has high reliability (measured as Cronbach's α), high validity (measured as its ability to predict party alignment), and outperforms the commonly used Wilson-Patterson Conservatism Scale on these metrics.

1.1 The theory of mutation and leftism

To recap the introduction of the previous article [1], it has been hypothesized that leftism is caused by the accumulation of harmful mutations in the genome, which accumulate in the population under low post-industrial selection pressures. Leftism, as measured by

Wilson-Patterson conservatism, moral foundations, and openness, is highly heritable. Leftism has also been found to be correlated with diseases which show paternal age effects, like schizophrenia and ADHD, as well as face asymmetry. Men accumulate new mutations in their gametes as they age, so the effect of mutation on a trait can be estimated by examining the trait's correlation with paternal age.

1.2   Other theories of leftism

There are, broadly speaking, three groups of theories about the rise of leftism: the hereditarian theories, the idealist theories, and the environmentalist theories. Hereditarians hypothesize that leftism is increased by genetic change, whether it be natural selection or some other pressure; environmentalists stress economic pressures or chemical exposure; and idealists claim that leftism is an idea that became more common.

To this point, there is little if any evidence for any of these. The true cause of the increase of leftism over time remains unknown. This is mostly because hardly anyone attempts to approach leftism scientifically. This paper is an attempt to remedy that issue by investigating the extent to which mutational pressure can explain at least part of the rise of leftism.

The mutational load hypothesis has nice scientific properties. We can predict, from a simple model of the human genome and some facts from molecular genetics, what we expect the mutational pressure to be. This allows us to predict what percent of the rise of leftism it explains, and what our paternal age and leftism correlation should be.

1.3   Model of polygenic trait decay

It has been found that thousands of SNPs contribute to human educability [23]. Educability is highly correlated with IQ. When genes are mutated, they almost always decrease intelligence [13]. This makes sense if one views high IQ as an optimization problem. The human genome can be conceptualized like an optimization problem on an extremely high dimensional vector which is 800 megabytes in size. If gradient descent is run on this vector for millions of years, it will become close to an optimum. If afterwards random bit flips are introduced to the vector, fitness will tend to fall. It is not hard to see that there are a lot of ways to be dumb and one way to be smart; most human diseases decrease IQ scores because anything that saps energy will tend to decrease general brain function. Likewise, we can conceptualize conservatism as "moral IQ" and predict that random mutations will decrease conservatism.

A simple model based on this intuition can produce the following equation:

$$r = \frac{h}{3} \frac{n_m 2\sqrt{7}}{5\sqrt{n_{SNP}}}$$

Where r is the paternal age correlation with the trait, n_{SNP} is the number of SNPs, n_m is the average number of new SNP mutations a person inherits per generation.

The assumptions in this model are as follows:

1. Negligible linkage disequilibrium and dominance for relevant SNPs [23]

2. Mutational load effects are equivalent to known SNP effects [18]

3. Mutational load effects are deleterious [13]

4. SNP effects are Gaussian [20]

5. The average person has 40 de novo mutations from their father [21][22]

6. SNP frequencies are uniform between 0 and 1 [19]

Each of these assumptions are supported by the broader literature.

The model can be derived as follows: let G be an i by n_{SNP} random matrix with Bernoulli columns, where i is arbitrarily large and n_{SNP} represents the number of SNPs in the human genome. The column means are uniformly distributed: $p_{n_{SNP}} \sim U(0,1)$. Real SNP frequencies are similarly distributed [19].

Each of these assumptions are supported by the broader literature.
The model can be derived as follows: let G be an i by n_{SNP} random matrix with Bernoulli columns, where i

is arbitrarily large and n_{SNP} represents the number of SNPs in the human genome. The column means are uniformly distributed: $p_{n_{SNP}} \sim U(0,1)$. Real SNP frequencies are similarly distributed [19].

Let $P = Gb$ where $b \sim N(0, I)$. SNP effects are commonly modeled like this in the GWAS literature [20]. We must estimate $[V|P|n_{SNP}]$ and the effect of the mutation process in order to get the expected standardized mutational pressure.

$$\mathbb{E}[\mathbb{V}[P \mid n_{SNP}]] = \mathbb{E}\left[\mathbb{V}\left[G_1 b_1 + \ldots + G_{n_{SNP}} b_{n_{SNP}}\right]\right] = n_{SNP} \mathbb{E}[\mathbb{V}[G_1 b_1]]$$

This comes out to approximately 1/7 on simulation. This means $\mathbb{E}[\mathbb{V}[P \mid n_{SNP}]] \approx \frac{n_{SNP}}{7}$

Next, for mutation, we flip all the bits where the SNP effect is generated as negative. This gives the same genetic and trait variance as the distribution of p {n{SNP}} is unchanged. We compute the phenotype with $[b]$. This changes the trait mean, but not the variance.

Finally, we generate the mutation matrix $M = \text{diag}\left(X_1, \ldots, X_{n_{SNP}}\right)$ where $X_i \sim \text{Bernoulli}\left(\frac{n_m}{n_{SNP}}\right)$. This gives $P_m = \left(MG_+^T\right)^T |b| = G_+ M^T |b|$.

We want to know $G_+ M^T |b| - G_+ |b|$. This is clearly $\mathbb{E}[|b|] \cdot P(G = 0) \cdot \frac{n_m}{n_{SNP}} \cdot n_{SNP}$. This is because there are n_m expected mutations and each mutation is expected to have the effect $\mathbb{E}[|b|]$. However, $P(G = 0)$ of the mutation sites are already 0, so they have no effect.

It can easily be shown that $\mathbb{E}[|b|] \approx \frac{4}{5}, P(G = 0) = \frac{1}{2}$, so our effect simplifies to $\frac{2n_m}{5}$. Next we divide by the standard deviation $\sqrt{\frac{n_{SNP}}{7}}$ to get

$\Delta_m^g \approx \frac{n_m 2\sqrt{7}}{5\sqrt{n_{SNP}}} \Delta_m^g$

represents the effect on the gene score, so the effect on phenotype is: Δ_m^g represents the effect on the gene score, so the effect on phenotype is the mutational pressure $\Delta_m = h\Delta_m^g$.

The mutational pressure is about 3 times the causal paternal age correlation [15], so substituting $3r$ in for the mutational pressure recovers the original equation. n_m is about 40 per person and almost all of these come from the father [21][22]. The most recent educability PGS found 4000 SNPs significantly contributed to trait variance [23]. Plugging in 40 and 4000*2 for n_m (we multiply by two because our model uses binary SNPs while theirs uses Hardy-Weinberg distributed SNPs, which are the sum of two binary loci with identical effects) and n_{SNP} respectively (and using 0.50 for h^2), we get a predicted paternal age correlation of 0.11 for educability.

It should be said that we also assume each de novo mutation will most likely hit a non-SNP, but produce an effect similar to that of a "bad" SNP. In other words, there would not really be 40 mutations in the 4000 EA4 SNPs, but 40 mutations elsewhere each giving an expected effect similar to flipping 40 EA4 SNPs negative. This should be reasonable given a molecular study found similar effect sizes for the effect of a single de novo mutation on cognitive performance [18].

Although there is little work done on the molecular genetics of politics, we assume, because it is a Gaussian behavioral trait with relatively high heritability, it will likely have a similar genetic architecture to educability. We have then, a plausible quantitative model that yields a specific prediction for our observations, using parameters from other literature as its input. Our results will show that the model cannot be rejected given the present data.

3 Methods

In this paper, two studies are presented. The first surveys 1175 white American men, mean age 41.5 years (SD = 13.2) and gives them the general leftism test, and asks their father's age when they were born (mean = 61.3, SD = 7). We attempt to balance the number of liberals and conservatives by ordering an equal number of liberals and conservatives, using the data participants gave to Prolific when they signed up, since Prolific has an overrepresentation of liberals by default.

The second surveys 994 mothers and fathers over 35 years old (white Americans, mean age = 55.0, SD = 10.3) and gives them the same test, while asking what ages they were when their children were born. The first study establishes the correlation between paternal age (age of father when born) and leftism, while the second establishes the degree to which older parents are more or less leftist than younger parents.

The continuous metric mirrors the binary metric in that it centers around three topics: LGBT, feminism, and race ideology. These dimensions are hypothesized to be common to empire decline, and covary due to being the result of mutational pressure on the same genes [1]. Each question was on a Likert scale with the following answer choices: Strongly disagree, disagree, neutral, agree, and strongly agree.

The questions were as follows:

G1. Is LGBT good?

G2. Homosexual behavior is fine when it is private and chaste.

G3. There is nothing wrong with public depictions of homosexual relationships. G4. I support gay marriage.

G5. There is nothing wrong with attending a gay orgy.

G6. Children should be taught about gay sex in sex education classes.

F1. Is feminism good?

F2. The country would be better if women couldn't vote. (-1)

F3. Women should try to be married by the age of 25. (-1)

F4. The government should help ensure sexual equality by making sure women are not discriminated against in private hiring.

F5. Women should hold the majority of the positions of power in society.

F6. Marriage is oppressive for women, and monogamy should be moved away from.

R1. Is Black Lives Matter a good organization?

R2. Europe would be best if it remained all white. (-1)

R3. Immigration policy should be strict and heavily meritorious. (-1)

R4. The government should ensure racial equality by prohibiting racial discrimination in private business dealings such as hiring.

R5. Black people deserve reparations for the legacy of slavery. R6. I support open borders.

The questions were intended to get "harder" as they progressed in each category, meaning woker people tend to be the only ones to agree to the later questions, while a greater percent of respondents would agree with earlier questions. Also, items F2, F3, R2, and R3 were reversed.

Each of the 3 sub-scales was designed to be added up into a sum score. From the three sum scores, a general factor was derived by factor analysis with varimax rotation. The sum-scores were near-Gaussian, with Q-Q plot R-squareds of 0.956, 0.97, and 0.981 respectively. We achieved factor loadings of 0.88, 0.87, and 0.78 for race, feminism, and homosexuality support respectively. Cronbach's α for the three sums was 0.86, which is far over the typical acceptable reliability of 0.70. In contrast, Wilson-Patterson conservatism has had αs as low as 0.71 [2].

Computing α over all the sums yielded a value of 0.93.

We also performed PCA as an alternative factor analysis method. We found one component explains 80% of variance, strongly indicating the appropriateness of a one factor solution. The PCA factor correlated with the varimax-rotated factor at r = 0.96. For the analyses in this paper, we used the varimax factor because it was slightly more Gaussian, with a Q-Q plot r^2 of 0.992 vs. 0.986 for PCA.

These statistics suggest that the measurement has high reliability. We also have evidence of high validity in that it predicts party and wingness well.

Figure 2 shows general leftism is good at distinguishing between party loyalties. This d score is equivalent to an r of about 0.75, meaning general leftism correlates strongly with party alignment.

Figure 3 shows those under -1 SD General Leftism are more than 90% likely to be right-wing and Republican, and same for those above 1 SD General Leftism.

Finally, Figure 4 shows the distribution of General Leftism was nearly gaussian. We had trouble at the tails, but it was accurate up to the 98th percentile.
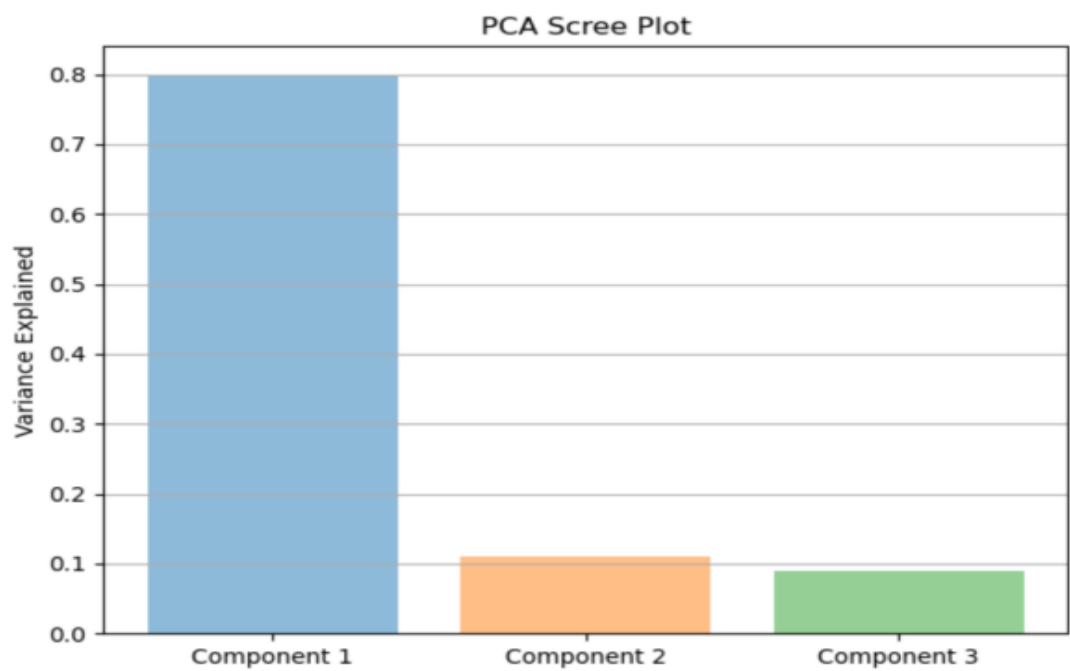
Scree plot for principal component analysis of Gay sum, Race sum, and Feminism sum.
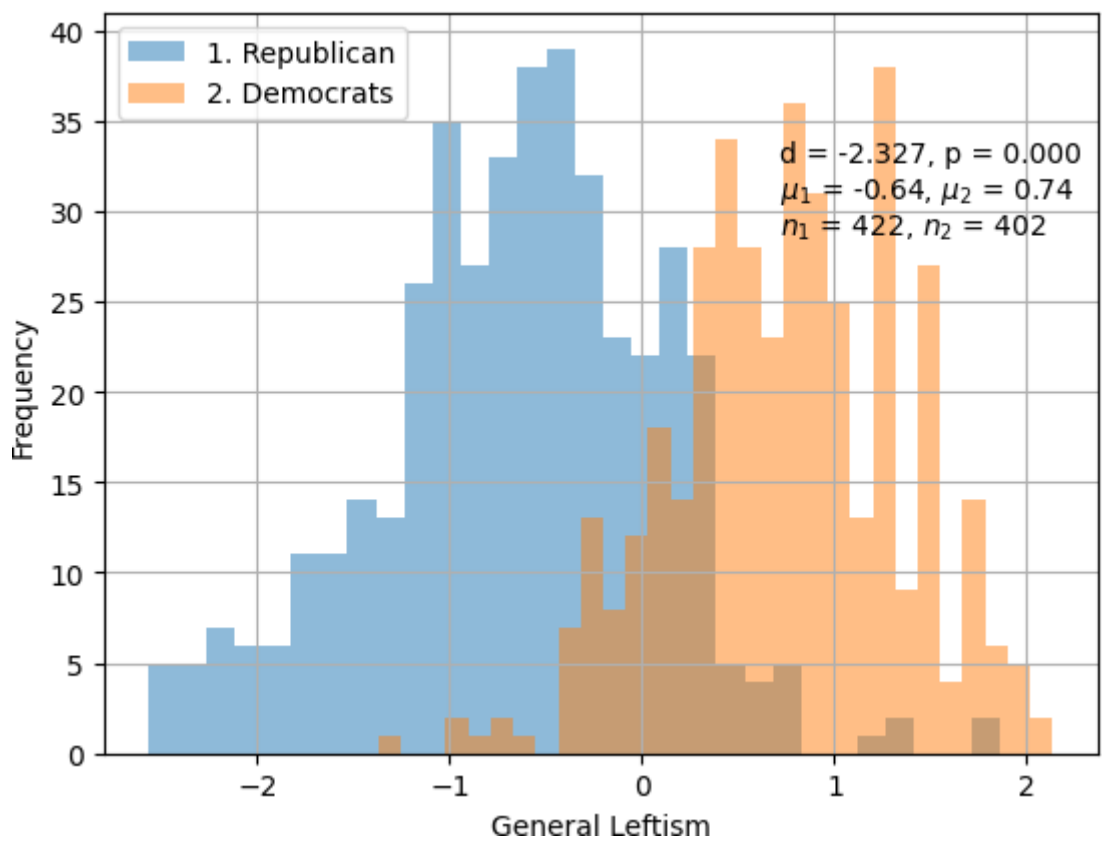
Figure 1. PCA Scree Plot

$$d = -2.327, p = 0.000$$
$$\mu_1 = -0.64, \mu_2 = 0.74$$
$$n_1 = 422, n_2 = 402$$

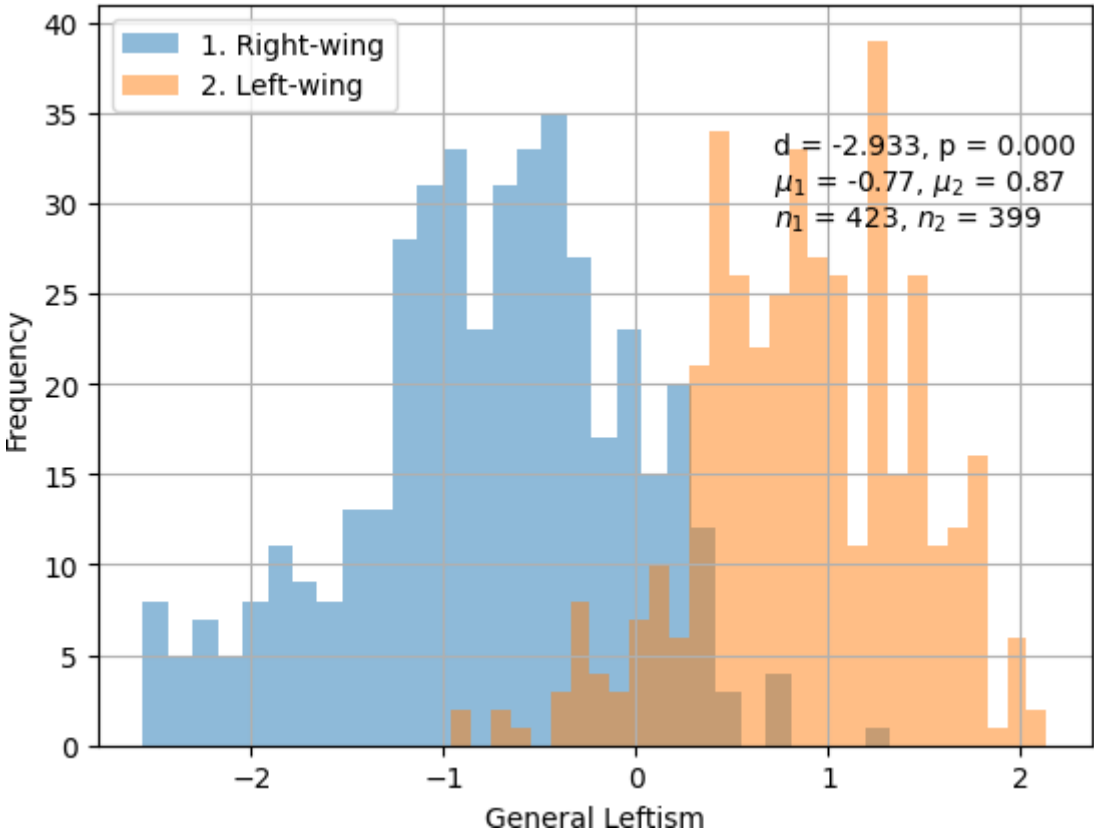**Figure 2.** General Leftism and Party

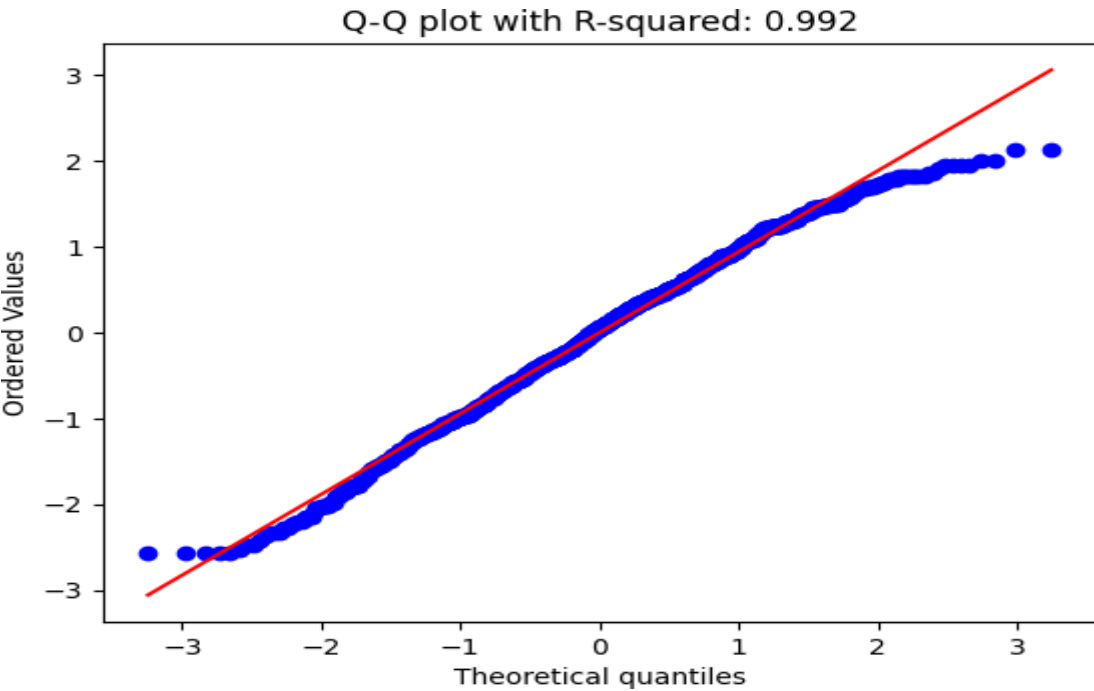**Figure 3.** General Leftism and Wingness

Figure 4. Q-Q Plot of General Leftism

4   Results

4.1   *Study 1*

Figure 5 shows the correlation between leftism and paternal age (p < 0.001). There is a significant positive correlation. In this data, we cut off the tails of paternal age (>2.5 SD), but this did not change the results. The full data will be publicly available on the author's Github.
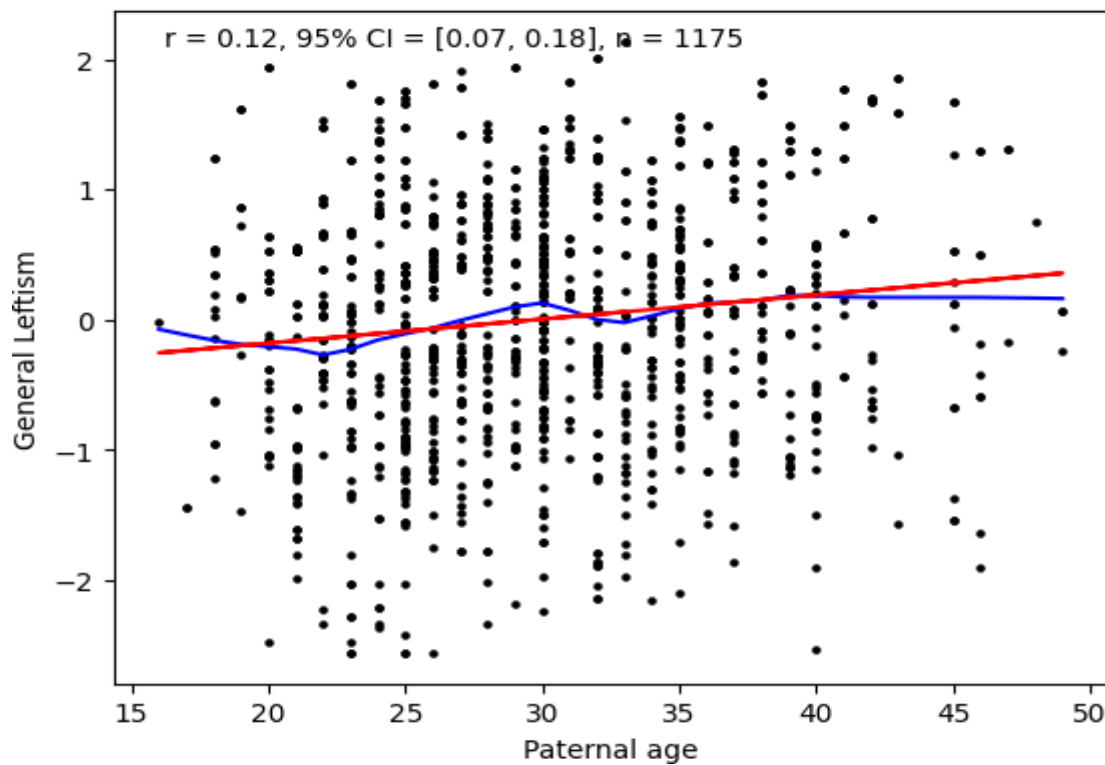

Figure 5. Leftism and Paternal Age

Figure 6 shows all of the correlation coefficients between all study variables. One star means p < 0.05, two means p < 0.01, and three means p < 0.001. Paternal age correlates with both factors, as well as the 3 sum scores, at .11 or .12 in all cases, p < 0.001 in all cases.

**Table 1.** OLS regression with Paternal age, participant age, on General Leftism. All standardized.

Covariates	β	p	S.E.
(Intercept)	0.000	1.000	0.270
Paternal Age	0.110	**0.000**	0.280
Individual Age	-0.090	**0.001**	0.270

Table 1 shows that the paternal age effect is present across father birth years. It is predicted by mutational load theory that age as well as paternal age will both independently predict general leftism. Age allows one to estimate the base mutational load of an individual's generation while paternal age allows one to estimate the expected deviation from that mean. We find that in the OLS model, the standardized beta of paternal age (.1104) with leftism was less than 0.01 under the correlation of leftism with paternal age (0.12), vindicating this prediction. Also, an interaction variable was tested, paternal age * age, to see if the paternal age effect varied with age, as Woodley et al. [11] found such an interaction when studying religiosity. We find no evidence of such an interaction in this study, the interaction being essentially 0.

Figure 6. Correlation matrix of study variables.
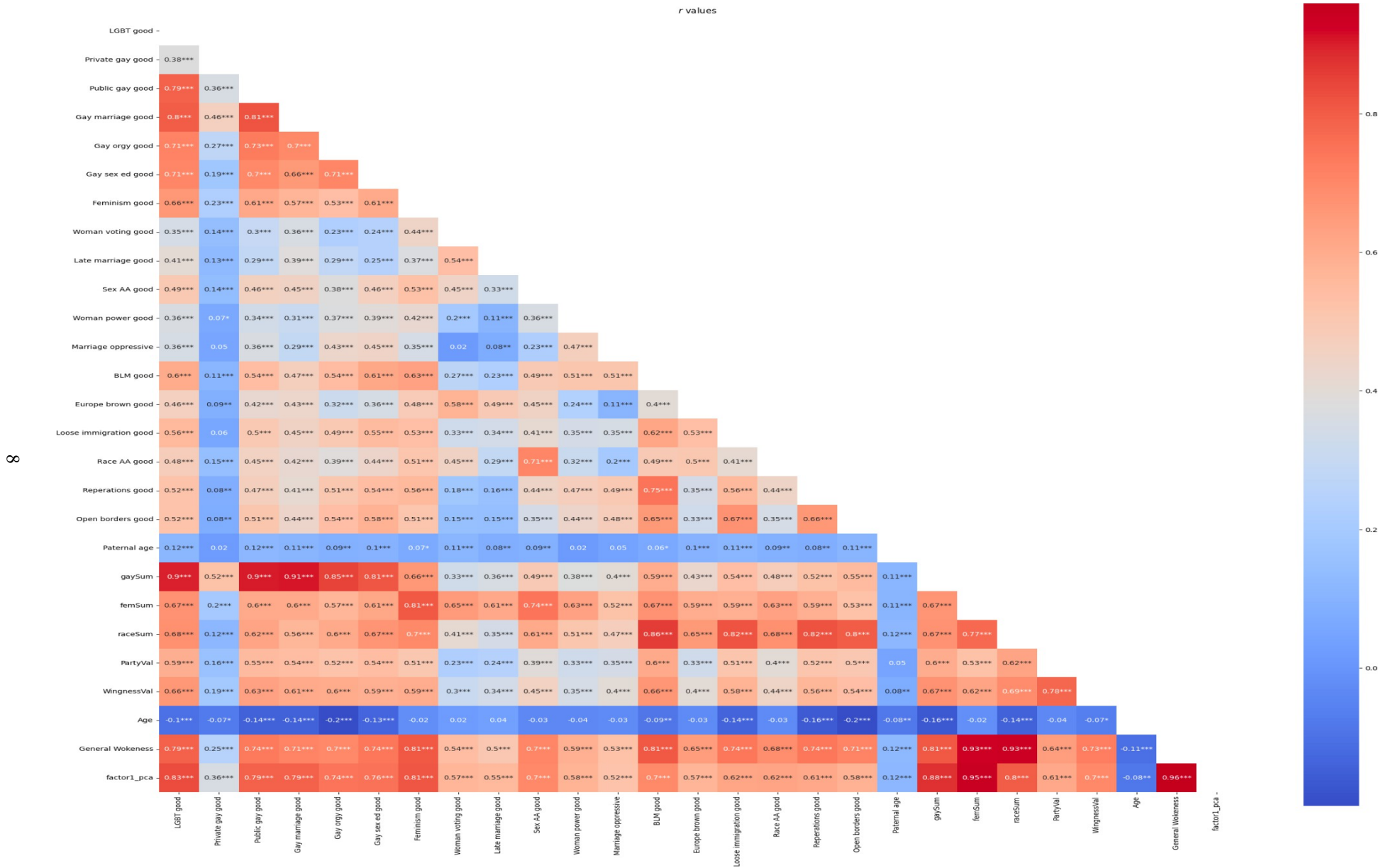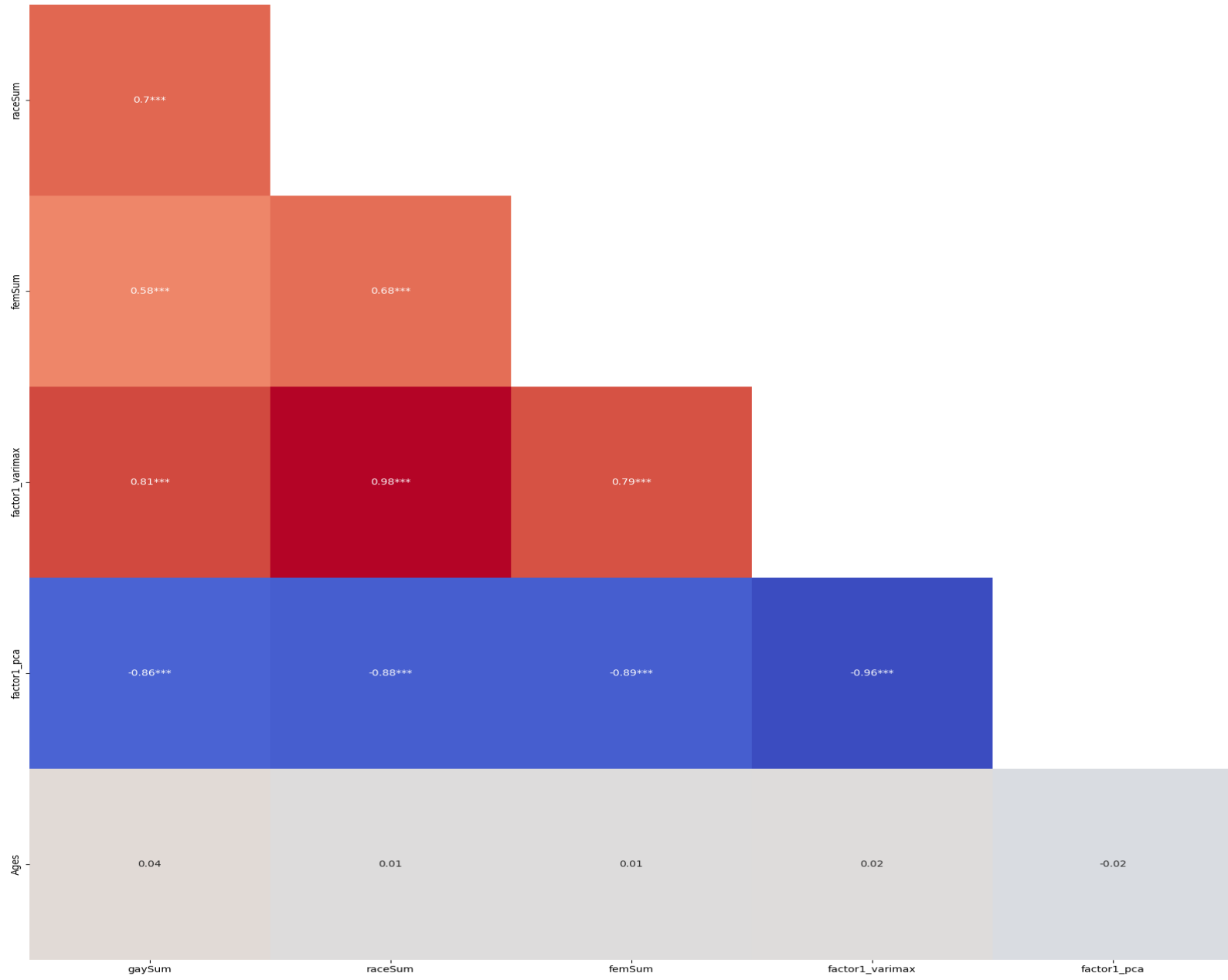
Figure 7. Age of Parents and Parent's Politic

4.2 Study 2

In study 2, we sampled n=994 white men and women who had had at least one child. We asked them at what ages they had had their children, and administered the general leftism test. We then correlated parent's politics with the parental age. If the association between paternal age and child leftism is explained by additive heritability or shared environment, then the parents of children with higher paternal age will be more leftist. Thus, parental politics will correlate with paternal age. However, as with the binary measurement from Bronski [1], we found no evidence of such a correlation.

The bottom row of figure 7 shows that parent's age when his children are born had no correlation with any sum scores, the varimax rotation factor, or the PCA factor

Table 2. OLS regression of Parental Age and Parent's Politics. Controlled for Sex and current age, all variables standardized. Cluster robust standard errors were used, because many individuals had multiple children, therefore reporting multiple ages at breeding.

Covariates	β	p	S.E.
(Intercept)	0.000	1.000	0.037
Age at Breeding	0.025	0.413	0.031
Age at Survey	−0.086	**0.017**	0.036
Sex	0.094	**0.012**	0.038

Table 2 shows parental breeding age and its association with the varimax factor. In this sample, it is non-significant, but a correlation of up to 0.086 cannot be ruled out under conventional significance standards. Such a correlation would deduct about 0.043 from the paternal age effect, assuming that the additive heritability of leftism is 0.50, per the breeder's equation [12] and the omitted variable bias formula [7]. Since a relation between paternal age and general leftism of just 0.055 cannot be ruled out given the confidence interval from Table 1, there is a small chance that this confounder, along with other uncontrolled confounders, could explain all of the measured relationship here. There was no significant interaction between age at breeding and sex, meaning the relationship between breeding age and leftism did not significantly differ between fathers and mothers.

Table 3. Descriptive statistics for both studies.

Name	Mean	SD	n
Paternal Age (1)	29.823	6.336	1175
Age (1)	41.490	13.145	1175
Age at Survey (2)	55.002	10.315	1872
Age at Breeding (2)	30.586	5.840	1872
Sex (2)	0.408	0.491	1872

Finally, table 3 shows the mean and SDs for the variables used in OLS. This is provided so that the OLS coefficients may be unstandardized.

6 Limitations

The key limitation in this analysis is the lack of controls for maternal age and birth order effects, as well as direct controls for the leftism of parents at different ages.

There is a chance that this omission is confounding to the results presented here. Certain other literature on the same topic is similarly weak. IQ has a similar polygenic structure to conservatism, and a paternal correlation of a similar magnitude to the one found here has been found by Wang with controls for parental IQ [8]. This correlation did not weaken when controlled for maternal age, but the analysis lacked the power to properly control for birth order, so it is unclear what the independent effect of birth order on IQ is and how this confounds the paternal age correlation. Mental illness correlates with leftism on other scales [10], and paternal age effects do not weaken when birth order is controlled for in mental illness [9]. It is possible that birth order explains some of the paternal age correlation, and it would be advantageous to verify this directly for leftism as well as IQ.

However, robust correlations between paternal age and mental illnesses have been found in high-powered family designs which find the effect of paternal age on a trait within a family controlled for birth order and maternal age [16][9]. These effects are of the magnitude observed here and predicted by the model. Additionally, there is direct molecular evidence for the impact of de novo mutations operating through paternal aging on schizophrenia, autism, congenital heart disease, epilepsy, and intellectual disability [17]. Assuming the effect on intellectual disability is through shifting the IQ mean downwards, this implies a robust paternal age correlation with IQ of at least 0.05, in line with the predictions of the model for a highly polygenic trait [18].

However, the only direct evidence for a paternal age effect on leftism is this study and the previous one using a binary measurement. With the resources, it is possible to do a large study which removes uncertainty in this area. This study should collect family level data, and should genotype everyone. It should have a sample size of at least 10,000, and IQ as well as leftism should be measured for parents as well as children. Leftism should be measured in multiple ways, using this scale, as well as Wilson-Patterson conservatism, self-classification, party alignment, and support for a representative sample of specific policies. Due to having family level information, the independent effects of birth order, paternal age, maternal age, and natural selection would be able to be approximated for different measures of leftism with low uncertainty.

7 Conclusion

Based on the results, we conclude that there is some evidence for a paternal age effect for leftism. The paternal age correlation with leftism is robust to controls for age. Older parents are not strongly more leftist, but some confounding from this variable cannot yet be ruled out.

A further step, in addition to a much larger study with vast family data, is molecular confirmation. Studies which confirm the role of de novo mutation in being more leftist than parents, as well as studies which show increasing polygenic scores for leftism associated traits like openness and individualizing through time can molecularly confirm the role of mutational load and genetics more generally in the rise of leftism.

Some researchers in the standard social science model tradition will also worry about sociological variables confounding this result. Because the contribution from the shared environment on conservatism has been estimated to be near 0, this is unlikely [1]. However, an ideal study could also collect family SES, religion, education, and other sociological variables, and examine their statistical connection to the paternal age effect.

The decline of asabiyyah (defined as a population's ability for collective action and expansion)

[5] seems to be a general feature of empire decline. We propose that the mechanism of asabiyyah decline is in fact mutational load increasing leftism in a population, potentially alongside immigrant gene flow. Further quantitative studies investigating the universality of the rise of features of leftism like feminism (decreased fertility, increased female driven sexual selection), homosexuality, and mass immigration of foreigners can further confirm this view. It may even happen in animals, especially social mammals with similar patriarchal societies to humans like lions, chimpanzees, gorillas, and wolves. An interesting, though expensive and time consuming experiment, could be to take one of these species and give them great wealth in an area over many generations. We might expect them to begin by defending their wealthy territory from outsiders. Over the generations, free from selective pressures, we would expect to see the decline of fertility and increases in female driven sexual selection, with decreases in the ability and drive for males to dominate the females. We might

expect to see the ability to defend the territory weaken; gene flow from outsiders increases. And perhaps homosexual behavior would increase as well. This could be done most easily with wolves, because they can reproduce the fastest among the animals listed (2 year generations) and they are found outside of Africa, in Western nations. Just 20 years would be enough to simulate 10 generations, which is 250 years for humans, approximately the time since the American and French Revolutions. An experiment of similar reach, the aim of which is to domesticate foxes, has been run for the last 60 years in Siberia, with good results [4], so this is not unprecedented.

References

Bronski, J. (2023). Evidence for a Paternal Age Effect on Leftism. *Openpsych*.

Bouchard, T. J., Segal, N. L., Tellegen, A., McGue, M., Keyes, M., & Krueger, R. (2003). Evidence for the construct validity and heritability of the Wilson–Patterson conservatism scale: a reared-apart twins study of social attitudes. *Personality and Individual Differences*, 34(6), 959–969. doi: `10.1016/s0191-8869(02)00080-6`

MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods*, 7(1), 19–40. doi: `10.1037/1082-989x.7.1.19`

Dugatkin, L. A. (2018). The silver fox domestication experiment. *Evolution: Education and Outreach*, 11(1), 1–5.

Turchin, P. (2018). Historical dynamics: Why states rise and fall (Vol. 26). *Princeton University Press*.

Kondrashov, A. S. (2017). Crumbling genome: The impact of deleterious mutations on humans. Pg. 114.

Angrist, J. D., & Pischke, J. S. (2009). Mostly harmless econometrics: An empiricist's companion. *Princeton university press*.

Wang, M. (2023). Estimating the parental age effect on intelligence with controlling for confounding effects from genotypic differences. *Personality and Individual Differences*, 207, 112137.

D'Onofrio, B. M., Rickert, M. E., Frans, E., Kuja-Halkola, R., Almqvist, C., Sjölander, A., . . . & Lichtenstein, P. (2014). Paternal age at childbearing and offspring psychiatric and academic morbidity. *JAMA psychiatry*, 71(4), 432–438.

Goldberg, Z. (2020). Liberals more likely to have a mental health condition. `https://threadreaderapp.com/thread/1248823584111439872.html`

Woodley of Menie, M. A., Kanazawa, S., Pallesen, J., & Sarraf, M. A. (2020). Paternal age is negatively associated with religious behavior in a post-60s but not a pre-60s US birth cohort: testing a prediction from the social epistasis amplification model. *Journal of religion and health*, 59, 2733–2752.

Fisher, R.A. (1930). *The Genetical Theory of Natural Selection*.

Huguet, G., Schramm, C., Douard, E., Tamer, P., Main, A., Monin, P., . . . Jacquemont, S. (2021). Genome-wide analysis of gene dosage in 24,092 individuals estimates that 10,000 genes modulate cognitive ability. *Molecular Psychiatry*, 26(6), 2663–2676. `https://doi.org/10.1038/s41380-020-00985-z`

Salzberg, S. L. (2018). Open questions: How many genes do we have? *BMC biology*, 16(1), 1–3.

Bronski, J. (2023). How to Compute Mutational Pressure. `https://www.josephbronski.com/p/how-to-compute-mutational-pressure`

Wang, S. H., Wu, C. S., Hsu, L. Y., Lin, M. C., Chen, P. C., Thompson, W. K., & Fan, C. C. (2022). Paternal age and 13 psychiatric disorders in the offspring: a population-based cohort study of 7 million children in Taiwan. *Molecular Psychiatry*, 1–

Taylor, J. L., Debost, J. C. P., Morton, S. U., Wigdor, E. M., Heyne, H. O., Lal, D., . . . Robinson, E. B. (2019). Paternal-age-related de novo mutations and risk for five disorders. *Nature Communications*, 10(1), 3043.

Bronski, J. (2024). Estimating Mutational Pressure on IQ through the Effect of De Novo Mutations in the Exome on Intellectual Disability. `https://www.josephbronski.com/p/estimating-mutational-pressure-on`

Park, J.-H., Gail, M. H., Weinberg, C. R., Carroll, R. J., Chung, C. C., Wang, Z., . . . Chatterjee, N. (2011). Distribution of allele frequencies and effect sizes and their interrelationships for common genetic susceptibility variants. *Proceedings of the National Academy of Sciences*, 108(44), 18026–18031. doi: `10.1073/pnas.1114759108`

Yang, J., Lee, S. H., Goddard, M. E., & Visscher, P. M. (2011). GCTA: a tool for genome-wide complex trait analysis. *The American Journal of Human Genetics*, 88(1), 76–82.

Nachman, M. W., & Crowell, S. L. (2000). Estimate of the mutation rate per nucleotide in humans. *Genetics*, 156(1), 297–304.

Kong, A., Frigge, M. L., Masson, G., Besenbacher, S., Sulem, P., Magnusson, G., . . . Stefansson, K. (2012). Rate of de novo mutations and the importance of father's age to disease risk. *Nature*, 488(7412), 471–475.

Okbay, A., Wu, Y., Wang, N., Jayashankar, H., Bennett, M., Nehzati, S. M., . . . Young, A. I. (2022). Polygenic prediction of educational attainment within and between families from genome-wide association analyses in 3 million individuals. *Nature genetics*, 54(4), 437–449.