
Is there a genetic factor that produces a bias in favor of one sex for some families?

Does Having Boys or Girls Run in the Family?

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Several years ago, the first author's sister noted, without even a trace of doubt, that "Rodgers men produce boys." I never take those kinds of statements from my sister lightly. She is a journalist who writes popular articles about family and reproduction, and her husband is an M.D. who specializes in infertility. Further, she had the data on her side. At that point, the eight Rodgers men with children from the past four generations had collaborated with eight different women in the production of 24 biological children; 21 were boys and 3 were girls. Although she had not run the analysis, my sister's statistical intuition was excellent. In the United States, approximately 51% of the babies born are males. Under a binomial model of sex outcomes, having three or fewer boys out of 24 children would happen by chance around twice in 10,000 families.

Like my sister, many mothers and fathers believe that a tendency to have boys or girls runs in a family. Informally, we have noted that belief among many of our friends. Pregnant women appear particularly interested in and amenable to the notion that sex composition runs in the family. But research in the statistical and cognitive psychology literature suggests that humans are notoriously bad at distinguishing systematic patterns from random patterns. Even if the sex selection process is purely by chance, some parents will have all boys in families of size 1, 2, 3, or even 10 or 12. For example, mothers of four children who have all boys or all girls must naturally wonder if something system-



The first author's grandfather, father, and two uncles are part of a long line of predominantly male Rodgers.

atic contributed to their "unusual sex composition." Yet, around 1/8th of all four-child families are expected to be a same-sex family under a chance model, not an especially unusual occurrence. Yet, even parents with Ph.D.'s in statistics must be inclined to wonder whether such extreme outcomes are caused by an unusual chance event or by a bias in certain fathers or mothers (or combinations) to produce one or the other sex. Some dice really are loaded.

Many factors have been identified that can potentially affect the human sex ratio at birth. A 1972 paper by Michael Teitelbaum accounted for around 30 such influences, including drinking water, coital rates, parental age, parental socioeconomic status, birth order, and even some societal-level influences like

wars and environmental pathogens. A 1997 study in *Nature* by Komdeur, Daan, Tinbergen, and Mateman suggested that birds — Seychelles warblers — alter their sex ratios in response to the availability of food, and other examples of extreme sex ratios in insects exist in the literature. Further, some recent evidence suggests that the human sex ratio may be currently shifting in the United States toward more female babies. Of interest in the current article is whether family influences — especially genetic ones passed from parents to offspring — can legitimately be included among the factors affecting the sex ratio at birth.

In 1930, Sir Ronald Fisher explained why the sex ratio should tend toward a 50–50 equilibrium for species that

reproduce sexually. Then, in 1973, Trivers and Willard proposed a sex ratio theory based on parental sensitivity to the environment. Their evolutionary-based thinking was that male offspring would be better able to take reproductive advantage of good environments than females, while female offspring would do reproductively better than males in relatively poor environments. As a result, human parents who are well off should disproportionately produce sons, and those not so well off should produce relatively more daughters. A number of empirical tests have shown support for the Trivers/Willard hypothesis. But most of these arguments concern external environmental influences on the sex ratio. Are there influences from within the family?

In 1962, Edwards reviewed the literature on genetic influences on the human sex ratio and concluded that genetic influences were nonexistent (or at least immeasurably small). Teitelbaum summarized those findings: “the search for genetically-controlled variability and heritability of the sex ratio has not been fruitful.” However, he cautioned that this conclusion was based on studies with “data of questionable accuracy, and on occasion even numerical inaccuracies of analysis have affected interpretation of results.”

M. L. Samuels and J. A. Witmer’s 1999 statistics textbook describes a study published by Geisser in 1889 in which the sex composition of 19th century German families with 12 children were studied. Remarkably, 6,115 such families were identified, and Samuels and Witmer compared the patterns to those expected under the assumption of independent Bernoulli trials. They found a disproportionate number of families that were homogeneous with respect to sex composition, suggestive that having boys or girls does “run in the family.” They speculate that the probability of a male birth is not constant in the population. Variation in that probability would create the observed pattern. But what is the cause of this variation?

Methodologies to detect a reproductive bias for producing boys (or girls) fall broadly into three categories. First, medical studies can be used to search for a physiological mechanism (which might or might not be inherited) that would lead to a sex bias in

Table 1 — Kinship Correlations From Different Kinship Categories, for the NLSY Data

Kinship category	Response variable	
	Prop. of boys	First child
Cousins	.35 (33)	.21 (33)
Half siblings	.18 (98)	.12 (98)
Full siblings	.08 (1129)	.11 (1131)
DZ twins	-.03 (15)	-.11 (15)
MZ twins	-.20 (6)	-.03 (6)

Note: Sample sizes given in parentheses.

the production of children. For example, studies showing that boy-producing sperm (androsperm) have slightly less genetic material than female-producing sperm (gynospem) can help explain the male bias at conception and birth, to the extent that the smaller androsperm swim faster in the race for fertilization. Second, demographic studies can be used to examine population patterns to observe whether sex biases occur within families at rates higher than would be expected by chance. The Samuels and Witmer analysis of the German 12-child families is one example. For another example, a 1961 U.S. study by Westoff, Potter, Sagi, and Mishler found that parents waited longer to have their next child after a boy than after a girl. Finally, behavior genetic studies can use kinship pairs to identify whether shared sex bias is higher among more genetically related mothers. We are not aware of any previous behavior genetic studies of sex composition in the literature.

In this article, we will present empirical analyses that fall into the last two categories. In each case, mathematical models can be fit to empirical data to evaluate whether sex bias “runs in the family.” The data on which our results are based come from the National Longitudinal Survey of Youth (NLSY), a national survey with excellent family information. Our behavioral genetic study will compare respondents with different levels of relatedness to determine whether more closely related women are more similar in their children’s sex composition than those more distantly related. We used twins, full siblings, half siblings, and cousin pairs — all the pairs of which lived together in the same household — to compare kin-

ship correlations indexing kinship similarity. If kinship pairs with higher genetic relatedness (e.g., twins) are more similar to one another than those with lower genetic relatedness (e.g., cousins), then this pattern is suggestive of a genetic influence. Our demographic study will compare sex composition patterns from the NLSY respondents to those that would be expected by chance. The model that will be fit explicitly distinguishes between stopping behavior caused by sex composition and the probability of a particular sex. These analyses will suggest whether certain patterns occur more often than chance can explain (e.g., whether there are more “boy-biased” or “girl-biased” families than would be expected under a binomial model).

The Behavior Genetic Study

The Data

In 1979, over 12,000 respondents aged 14–21 were obtained from a household probability sample of 8,770 households in the United States with 2,862 of those households including more than one respondent in this age range. This original sample — the National Longitudinal Survey of Youth (NLSY) — has been followed yearly since then. In 1994, respondents were 29–36 years of age, and the majority of childbearing in this sample was completed. At this time, around 15,000 children had been born to NLSY parents. Among the many variables included in the NLSY dataset is information about sex of each biological child of each respondent and the information necessary to create genetic links

between biologically related respondents from the NLSY.

The Measures

Dependent variables for this study include indicators of sex-ratio of biological children of both mothers and fathers in the NLSY. Two such indicators were constructed. The first was the proportion of male biological children out of total children born to a respondent. The second was a dummy variable indicating the sex of the firstborn biological child.

The independent variables required for the behavior genetic study were indicators of genetic relatedness of the NLSY respondents who lived in the same household. For approximately 90% of the kinship pairs in the sample, we were able to define a particular kinship pair as monozygotic (MZ) twins, full siblings (or dizygotic (DZ) twins), half siblings, or cousins.

Results

Kinship correlations were computed across all of the different levels of relatedness. Correlations and sample sizes are presented in Table 1 (on the previous page). Kinship correlations for sex of first child were the following: $r = .21$ for cousins, $r = .12$ for half siblings, $r = .11$ for full siblings, $r = -.11$ for DZ twins, and $r = -.03$ for MZ twins. Two of the correlations were significantly different from 0 (for cousins and full siblings), but the pattern is not at all suggestive of genetic relatedness on sex of first child. If there were genetic influences, we would expect these kinship correlations to increase with increasing levels of genetic relatedness. In fact, the pattern is almost the exact opposite, with correlations decreasing systematically with increasing genetic relatedness. Behavior geneticists also model shared family environmental variance. This type of variance would occur if sex of child were somehow related to factors shared by the mothers in their common family environment. However, the correlations listed previously are not consistent with any shared environmental influences, either.

In the second analysis, kinship correlations were computed for the second dependent variable, overall sex ratio (measured as “proportion of boys”). Again, there was no indication that correlations increased with increasing genetic relatedness (i.e., no patterning

of these correlations in relation to genetic relatedness of the kinship pair), nor were shared family environmental influences indicated. These kinship correlations are also contained in Table 1.

If these correlations did show genetically or environmentally informative patterns, quantitative genetic and environmental models could be fit to these correlations to estimate behavior genetic parameters of interest. However, when the correlations are nonsystematic and most are not even significantly different from 0, there is little point in estimating parametric models. We simply conclude that neither genetic nor shared environmental factors are implicated as influences on sex composition in the NLSY data (which are themselves approximately representative — up to oversampling, attrition, missing data, and some slight right truncation in childbearing — of a broad cohort of developing adults in the United States).

The Demographic Study

A second type of statistical analysis directed toward the question of whether individuals are biased in the production of sex composition patterns of children is one in which overall sex composition patterns are inspected using conditional probabilities. If (for example) Rodgers men are more likely to produce boys than girls, then there should be more long runs of male babies than female babies by Rodgers men. In this case, the probability of having a boy, given one or more previous boys, should be different (and higher) than the probability of having a boy given one or more previous girls. Similarly, if others are more likely to produce girls, the frequency of certain sex composition patterns should respond to this tendency.

The Data

By 1994, when they were 29–36 years old, approximately half of the NLSY respondents reported having had one or more biological children ($N = 6,089$, 3,321 females and 2,768 males). In Table 2, we present the pattern of sex compositions for all NLSY respondents who were parents. There were so few respondents who had more than four children that we simply stopped counting after four children for those respondents.

We cannot simply inspect these frequencies to determine whether there are boy-biased or girl-biased families. The reason for this is that sex composition of a respondent’s family is potentially affected by another factor besides sex composition tendency — the “stopping behavior” of the parents. It is well-known by demographers that many societies have strong boy biases (alternatively, a few societies have girl biases). In such settings, two-child boy-boy families will be more common than two-child girl-girl families, and not because of any overall tendency to overproduce males. Rather, parents may stop having

Table 2 — Sex Composition Patterns in the NLSY Data (B = boy, G = girl)

Sex composition pattern	NLSY respondents (N = 6,089)	
	Frequency	Proportion
B	930	.1527
G	951	.1562
BB	582	.0956
BG	666	.1094
GB	666	.1094
GG	530	.0870
BBB	186	.0305
BBG	177	.0291
BGB	148	.0243
BGG	173	.0284
GBG	182	.0299
GBB	151	.0248
GGB	125	.0205
GGG	159	.0261
BBBB	43	.0071
BBGB	26	.0043
BGBB	30	.0049
BGGB	23	.0038
GBGB	23	.0038
GBBB	32	.0053
GGBB	29	.0048
GGGB	29	.0048
BBBG	34	.0056
BBGG	37	.0061
BGBG	22	.0036
BGGG	24	.0039
GBGG	28	.0046
GBBG	29	.0048
GGBG	28	.0046
GGGG	26	.0043
Total	6,089	1.000

children disproportionately after having two boys, while the majority of parents with two girls would go ahead to three or more children.

There are two different ways that we will account for parental stopping behavior in evaluating whether having boys or girls runs in the family. First, although the evaluation of only two-child families will confound parental stopping behavior and sex probability, the evaluation of the sex composition pattern among the first two children in *all* families eliminates the stopping behavior effect. In the data in Table 2, for example, the first two children were both boys in the BB, BBG, BBB, BBBB, BBBG, BBGB, and BBGG families. We can compile all patterns for the first two children (for which there are 4,208 NLSY parents) and for the first three children (for which there are 1,764 NLSY parents). In the second approach, we define a mathematical model that estimates parameters that separately reflect the parental stopping rule and the sex probabilities. In this modeling approach, we will compare predictions of various forms of a sex composition model with the data to determine which type of model — one that considers sex of the current child

to be independent of previous sex composition versus one that considers sex of the current child to depend on the sex of previous children — better fits the patterns in the NLSY data.

Sex Composition Among the First Two and First Three Children

In Table 3, we compile the frequencies of each sex from Table 2 for the first two and first three children for all NLSY parents. For the first two children, families heterogeneous by sex were more likely than expected in an independence constant probability model. This is exactly opposite from the results of Samuels and Witmer, in which homogeneous families were more prevalent. For the first three children, the observed frequencies are in the same direction as the finding in Samuels and Witmer's analysis of the Geisser data. These conflicting results leave the status of our basic question in doubt, however. A more sophisticated way to address our basic question is to define a model that accounts for the probability of having a boy (or a girl) conditional on the previous sex composition of the family. We turn now to a formal modeling exercise to attempt to resolve the question of whether having boys or girls runs in the family.

The Models

We have formalized the probabilities of different sex composition patterns into a set of mathematical models, each of which accounts for two different components. The first component is the topic of this article, the probability of having a boy (or a girl). Some of the models define unconditional versions of this probability and assume that the likelihood of having a girl (or a boy) does not depend on the sex composition of previous children. Other models define this probability as being conditional on having had previous boys (or girls). The second component of these models is the one identified previously that is not the direct focus of this article, but which can act to contaminate the probabilities that we are trying to estimate — parental stopping probabilities.

We accounted for sex probability in two different ways. The first ignored the sex composition of previous children and assumed that the probability of a boy or a girl is independent of the previous family composition. In this model,

the “sex-unconditional model,” there is a single parameter for the probability of a boy, PB, with the probability of a girl, PG = 1 – PB. The second type of model accounted for the sex of previous children by defining separate probabilities for a boy or a girl, depending on whether previous children define a boy-biased family, a girl-biased family, or a sex-neutral family. In this type of model, there is a different parameter for the probability of a boy in previously boy-biased families, in previously girl-biased families, and in sex-neutral families, PBB, PBG, and PBN, respectively. The probability of a girl in the families, PGG, PGB, and PGN, are the complements of the boy probabilities. We will call the second model the “sex-conditional model.” If having boys or girls really does run in the family, then the sex-conditional model should fit the data better than the sex-unconditional model.

We accounted for stopping behavior in a number of different ways. In the first, a parity-specific stopping strategy, we defined three different stopping parameters. These reflected the probability of terminating or stopping after the first child (t_1), the probability of stopping after the second (t_2), and the probability of stopping after the third (t_3). (There were so few NLSY respondents who had more than four children by 1994 that our model assumed automatic stopping after four children.) But stopping might be conditional on the sex of the most recent child. To account for this, we defined a gender-specific/parity-specific strategy that separated each of t_1 , t_2 , and t_3 into two parameters — b_1 and g_1 to account for stopping after a first boy or girl, b_2 and g_2 for the second child, and b_3 or g_3 . The third type of stopping parameter accounted for another type of parental stopping strategy. It is well-known by demographers that there is another important dynamic besides parity and sex that potentially influences stopping behavior. Particularly in the United States, many parents have a “balance preference,” which translates into a desire to have at least one child of each sex. Our final set of stopping-rule parameters accounts for this goal by defining additional parameters for stopping after two children when sex balance was reached or after three children when the third child resulted in at least one child of each sex. We

Table 3 — Sex Composition Patterns for the First Two Children and the First Three Children for all NLSY Parents (B=boy, G=girl)

Sex composition Pattern	Frequency
First two children	
BB	1,085
BG	1,086
GB	1,111
GG	926
First three children	
BBB	263
BBG	240
BGB	200
GBB	212
BGG	220
GBG	233
GGB	182
GGG	214

denoted these balance parameters with the symbol m (for “mixed-sex” families) and define m_2 or m_3 for balance being achieved after the second or third child, respectively.

To illustrate how a model of this type works we describe one example in detail. We refer to completed families using B to denote a boy and G to denote a girl and order those letters in the order in which they are born. Thus, a B family refers to a completed one-child boy family, a GB family refers to a completed family in which the first child was a girl and the second was a boy, and GGGBG refers to a family with four children, the first, second, and fourth of which were girls.

The model we use as an example is the sex-unconditional model with the most complex set of stopping parameters. There is one sex-probability parameter in this model, PB (with of course $PG = 1 - PB$). PB and PG are assumed fixed, unconditional on the previous sex composition. There are eight stopping parameters. b_1 and g_1 account for the separate probability of stopping after the first child, conditional on whether the child is a boy or girl. $b_2, g_2,$ and m_2 account for stopping after a BB family, a GG family, or a mixed-sex family, respectively. b_3 and g_3 account for stopping after a third boy or a third girl, unless that boy or girl achieves sex balance in the family, in which case the parameter is an m_3 parameter. Thus, for example, m_3 is the stopping parameter for a BBG or a GGB family. (Note that for other balanced three-child families — BGB, GBG, BGG, GBB — the balance was achieved with the birth of the second child and not the third; for these parents that chose to go on despite achieving balance, we simply model the third stopping probability as b_3 or g_3 .) Within this model, the probability of stopping after the fourth child is unity (because our data only supported analysis through the fourth child). Note that this is a 9-parameter model.

The probabilities for a subset of sex composition outcomes under this particular model are shown in Table 4. The probability of a family with one male child, the first line in Table 4, is the product of the probability of having a boy (denoted PB in the sex-unconditional model) and the

Table 4 — Selected Probabilities of Several Sex Composition Outcomes Under the Unconditional Model. (B = boy, G = girl)

$P(B) = PB*b_1$	
$P(G) = PG*g_1$	
$P(BB) = PB*(1-b_1)*PB*b_2$	
$P(BG) = PB*(1-b_1)*PG*m_2$	
$P(GB) = PB*(1-g_1)*PG*m_2$	
$P(GG) = PG*(1-g_1)*PG*g_2$	
$P(BBB) = PB*(1-b_1)*PB*(1-b_2)*PB*b_3$	
$P(BBG) = PB*(1-b_1)*PB*(1-b_2)*PG*m_3$	
$P(BGB) = PB*(1-b_1)*PG*(1-m_2)*PB*b_3$	
$P(BBBB) = PB*(1-b_1)*PB*(1-b_2)*PB*(1-b_3)*PB*1$	
$P(BBBG) = PB*(1-b_1)*PB*(1-b_2)*PB*(1-b_3)*PG*1$	
$P(BBGB) = PB*(1-b_1)*PB*(1-b_2)*PB*(1-m_3)*PB*1$	
Parameters: stopping probability parameters — $b_1, g_1, b_2, g_2, m_2, b_3, g_3, m_3$; sex probability parameters — PB (with $PG = 1 - PB$).	

probability of stopping after having one boy (denoted b_1 , and read “the probability of stopping given that the first child was a boy”). Further, $b_1, g_1, b_2, g_2, \dots$ are used as shorthand for the conditional stopping probabilities. The probability of having a two-boy family, $P(BB)$, is the probability of having a first boy (PB) times the probability of not stopping ($1-b_1$) multiplied by the probability of having a boy on the second child (PB) times the probability of stopping after the second child (b_2): $P(BB) = PB*(1-b_1)*PB*b_2$. And so forth for each possible outcome.

The values of the parameters are estimated by fitting the probabilities in Table 4 to the observed frequencies in Table 2. The specific criterion used to estimate the parameters was the minimum chi-squared criterion.

Model Fitting

We evaluated the fit of a number of different forms of these models, varying the stopping rules and the sex probabilities. In this article, we report the result of only the best-fitting model out of many different possible models that competed to explain the NLSY data. The estimated model parameters from this best-fitting model are shown in Table 5.

This best-fitting model contained four parental stopping-rule parameters and the sex-unconditional probability parameters. The stopping-rule parameters included three reflecting a parity strategy for stopping, with $t_1=.31, t_2=.53,$ and $t_3=.74$ (and note that t_4 was fixed to be 1.0 because the model

assumed that parents would always stop after four children). These were plausible and interesting indicators of the probabilities with which NLSY respondents stopped after one, two, and three children, respectively. But the best-fitting model also included a mixed-sex stopping rule, suggesting that parents were more likely to stop at two children after achieving a mixed-sex family than if they achieved a BB or a GG family ($m_2=.63$ versus $t_2=.53$). These stopping-rule parameters are interesting in and of themselves but are not the focus of this study.

The sex probabilities, PB and PG, are the ones that address the theme of the current research. Though we fit many sex-conditional models, the best-fitting model was one in which the sex of a given child did not depend on the sex composition of previous children in the family. The chi-square measure of fit suggests that this best-fitting model was an effective fit to the NLSY data. This

Table 5 — Model-Fitting Results for the Best-Fitting Model

Sex probability parameters	PB	.51
	PG	.49
Stopping rule parameters	t_1	.31
	t_2	.53
	m_2	.62
	t_3	.74

model estimated $PB=.51$ and $PG=.49$, matching the generally accepted values for these probabilities for U.S. child-bearing. In this model-fitting exercise, there was a straightforward and unambiguous answer to the sex composition question when parameters for parental stopping behavior separated that process from the sex probabilities. The answer to the question, “Does having boys or girls run in the family,” was clearly “No.”

Discussion

Consistent with earlier findings using much more limited datasets and methods, there was no evidence from our behavior genetic study of the NLSY that having boys (or girls) has either genetic or shared environmental sources of influence. Our kinship correlations showed those to be generally nonsignificant, with no obvious pattern across levels of kinship relatedness that matched either genetic or shared environmental patterns.

Our demographic study provided further support for this finding. In one of the two family sizes investigated, there did appear to be a disproportionate number of same-sex families (and in the other the pattern reversed). But our model-fitting exercise showed that there was not a tendency for male-biased families to produce males with greater frequency than female-biased families.

It is of interest to compare these results with those from Samuels and Witmer’s analysis of the Geisser 19th century parents in Germany with 12 children. Samuels and Witmer found a

disproportionate number of homogeneous families compared to an independence constant probability model. In particular they speculated about the possibility that the probability of a male child varies from family to family. On the other hand, our sex-unconditional model was consistent with an independence model and our behavior genetic study rules out genetic factors. These two different findings can, in fact, be reconciled. Mealey and Mackey (1990) discussed two different possible mechanisms by which sex ratios might be influenced, a genetic process versus an “environmentally influenced facultative” process. The latter refers to a reproductive process in which parents respond to environmental contingencies (as the Seychelle warblers were shown to do in the Komdeur et al. study). Our study shows no genetic influence on sex ratios, a result consistent with previous research using much smaller and less representative samples. If the human sex ratio is indeed the result of a facultative response to environmental conditions, it would be no surprise at all to discover different sex composition patterns among large families in 19th century Germany compared to small families in late 20th century America. The specific environmental mechanisms to which parents were responding in each case are as yet unspecified, and these provide a fascinating question for future researchers to address.

Conclusion

When my sister announced several years ago that “Rodgers men produce male children,” the stage was set for the question addressed in this article. Do sex patterns run “in the family?” The question has been answered using a larger data source with more internal and external validity than most previous ones used in this assessment. We found no compelling evidence that sex bias runs in the family.

And what of the Rodgers men? Have we maintained our talent at turning out male children? Since my sister’s pronouncement, my brother and cousin have had a total of three children, two daughters and one son. And by now, I’ve also contributed children to the family reproductive pool. When they’re older, 7-year-old Rachel and 5-year-old Naomi may be

quite interested in their aunt’s pronouncement and in this particular study.

So the stakes have changed. Instead of 21 boys out of 24 children, those four generations of Rodgers men (for which fathering children is, quite likely, complete) have now produced a total of 22 boys out of 29 children — still a remarkably strong boy bias but one that appears to be weakening as the evidence comes in. Has the overproduction of Rodgers sons been an accident of chance or some grand design? The evidence emerging from the current study would support the former.

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The first author’s daughters weaken the case for boy bias in the Rodgers family.