Genetic Endowments, Same-Sex Sexual Behavior, and Parental Religiosity*

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Abstract: We study interactions between sexuality and parental religiosity in the development of mental health and human capital outcomes among adolescents and young adults. "Closeting" presents a challenge to quantitative studies of sexual orientation: non-heterosexual individuals may conceal or repress their behaviors or identity in the face of family or social stigma. We overcome this challenge by using observed genetic markers to measure one's propensity for same-sex sexual behavior (SSSB). Specifically, we use a recently developed polygenic index (PGI) which aggregates a large number of genetic markers. While not randomly assigned, this measure is fixed at conception and thus predetermined relative to endogenous family responses. Using a sample from Add Health, we present three major results. First, we find evidence of endogenous closeting. The relationship between the SSSB PGI and self-reported SSSB is significantly attenuated among those with highly religious parents. Second, we find a significant positive interaction between the SSSB and parental religiosity in predicting depressive mental health symptoms. While there is little relationship between the PGI and depressive symptoms among those with less religious parents, there is a significant positive relationship for those growing up in more religious environments. Importantly, we do not find this result when interacting direct self-reports of SSSB with parental religiosity. Third, we fail to find any consistent evidence of interactions between the SSSB PGI and parental religiosity in predicting human capital outcomes (educational performance and completed education).

Keywords: Child Development, Religiosity, Same Sex Sexual Behavior, Polygenic Scores.
JEL Codes: J01, J24, I24, I26, I28

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1 Introduction

A large literature in economics and related disciplines examines factors that influence child development. These studies generally focus on household inputs like time and financial resources, school quality, parental characteristics (e.g. education), and children's endowments (Todd and Wolpin, 2003; Cunha and Heckman, 2007; Todd and Wolpin, 2007). Far less attention has been paid to the role of interactions between the characteristics of children and parents in this process, including potential frictions arising from parent-child mismatch. By contrast, several studies document the importance of match or mismatch in relationships outside the family, such as race and gender differences between teachers and students or between doctors and patients (Dee, 2005; Dee, 2007; Fairlie et al., 2014; Antecol et al., 2015; Alsan et al., 2019; Gershenson et al., 2022; Ye and Yi, 2023; Cabral and Dillender, 2024). Of course, parents and children also can—and often do—differ on many dimensions. Some parent and child characteristics, when combined, may be particularly beneficial or detrimental for children's outcomes.

We study parent-child mismatch in the context of sexual orientation, where there is significant scope for friction between non-heterosexual kids and parents who disapprove of their child's sexuality. Specifically, using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), we examine interactions between a child's genetic propensity for same sex sexual behavior (SSSB) and the religiosity of their parents in shaping mental health and human capital outcomes.

Sexuality is a major part of adolescent development. The onset of puberty and sexual development is associated with changes in a variety of behaviors and outcomes including mental health, academic engagement, risk-taking, and social behavior (Blakemore, 2019;Forbes and Dahl, 2010; Koerselman and Pekkarinen, 2017). For non-heterosexual kids, this process can be far more complicated. Beyond dealing with the normal pressures associated with adolescent development, they often face discrimination, hostility, or rejection tied to their status as sexual minorities from peers and, often, from their own family. In the face of these pressures, non-heterosexual youth may repress their sexuality altogether or keep it hidden in the "closet." The consequences are often severe. LGBTQ youth face significantly higher rates of depression and suicide, with family support playing a critical role in moderating these risks (Ryan et al., 2009; Plöderl and Tremblay, 2015; Lucassen et al., 2017).

The ability to quantitatively study sexual orientation as part of the development process is greatly hindered by measurement challenges. Few studies ask directly about sexual orientation or behaviors. Even when data sets include explicit questions—as in Add Health — privacy concerns or repression may create endogenous measurement error. Individuals who

have repressed or hidden their sexuality may indicate that they are heterosexual on surveys, or may refuse to answer such questions. This creates a dilemma when studying the relationship between sexuality and individual outcomes. Individuals who face the most repression (and associated subsequent negative outcomes) may be the least likely to report their homosexuality - a process we refer to as "endogenous closeting." This may lead researchers to under-estimate the impact of repression or rejection on economic outcomes.

We overcome this particular measurement challenge by shifting the empirical focus from observed sexual behavior (an endogenous outcome) towards a set of genetic predictors of same-sex sexual behavior (SSSB). Specifically, we examine a polygenic index (PGI) developed by (Ganna et al., 2019). This index is constructed as a weighted sum of many genetic markers found to predict SSSB. We interpret this index as capturing [Robbee should fill this in]. While interpreting variation in this index requires care, it is predetermined at conception and does not reflect endogenous reactions of parents or choices of individuals to hide or repress their sexuality.

While many factors could moderate the relationship between a child's sexuality and later outcomes, parental religiosity is a natural object of study. Traditional forms of Christianity (the predominant religious tradition in our setting) feature prohibitions on homosexuality. Although views on sexuality vary considerably within and across religious groups, there is a strong average relationship between religiosity and negative views towards non-heterosexuals (Olson et al., 2006, Schulte and Battle, 2004). Non-heterosexual kids raised by religious parents with such views may feel unsupported or rejected. This could take a negative toll on mental health and other aspects of child development.

We present three main sets of results in the Add Health sample. First, we document that the relationship between the polygenic index for SSSB and self-reported SSSB depends on parental religiosity. We interpret this as evidence of endogenous closeting in more religious households. For adolescents raised in less religious households, a one standard deviation higher SSSB index is associated with a 2.7 percentage point increase in the likelihood of reporting any SSSB later in life. For those raised in more religious households, this coefficient falls significantly to 0.008. This interaction is consistent with parental reactions that push children to either repress or hide their sexuality. Importantly, this provides evidence that self-reported measures of SSSB are endogenous and at least partially reflect interactions between a child's endowments and their rearing environment.

Second, we find evidence of a negative interaction between the SSSB index and high parental religiosity when examining depressive symptoms. Among children raised by less religious parents, we find no evidence of a relationship between the SSSB index and depressive symptoms. However, among those raised by more religious parents, a one standard deviation

higher SSSB index is associated with a 2.7 percentage point increase in the likelihood of ever experiencing significant depressive symptoms based on the CES-D scale. Critically, we fail to find evidence of such an interaction when we repeat these analyses using self-reported SSSB instead of the SSSB index. In the presence of endogenous sexual identification, the study of interactions between SSSB and environmental factors could be significantly biased. Predetermined measures like the SSSB polygenic score thus offer a way to potentially to circumvent such concerns.

Third, we also test for interactions in education or human capital outcomes. We find no evidence of statistically significant interactions between the PGI and parental religiosity in models predicting high school GPA or completed years of education as an adult. One interpretation of these results is that the effects of parent-child mismatch in this context may be localized to mental health outcomes, and may not disrupt or otherwise affect academic and human capital outcomes.

Our work contributes to several literatures. First, we contribute to a literature on the economics of child development. Much of this literature has focused on estimating dynamic models of skill formation, with parental investments. Key questions in this literature relate to the timing of investments in children, e.g., whether later investments complement earlier investments (Cunha and Heckman (2007), Agostinelli and Wiswall (2025)), the role of policy (e.g., cash transfers) to spur investment in children (Parker and Todd (2017)), and heterogeneity in returns to investments Leibowitz (1974). This paper relates in that mismatch could be conceived of as a source of disinvestment in children if it leads to disadvantageous outcomes.

Second, we contribute to a literature on sociodemographic mismatch in key relationships. Most prominently, a large literature focuses on how race and gender differences between teachers and students can lead to different interactions and, often, worse outcomes, especially among minority children. For example, Delhommer (2022) and Gershenson et al. (2022) show this for Black students with white teachers; Lindsay et al. (2022) and Delhommer (2022) show this for Latin American students and teachers, and Lim and Meer (2020) find negative interactions due to gender differences. Another prominent set of papers shows that race and gender mis-match between doctors and patients leads to worse health outcomes (Alsan et al., 2019; Cabral and Dillender, 2024). Our paper pushes this literature towards a new context: mismatch parents and children. Because parents and children often share many attributes (e.g., race), this type of mismatch has generally been overlooked. However, children's sexuality and parents' religiosity present a context where mismatch can arise and have detrimental impacts.

Third, we contribute to a broad set of papers in economics examining homosexuality

and economic outcomes. This literature has considered a number of relationships, including: wage penalties for sexual minorities (Ahmed and Hammarstedt, 2010; Burn, 2020; Drydakis, 2022), HIV/AIDS and economic behavior and outcomes (Sood and Wu, 2013; Papageorge, 2016; Chan et al., 2016), Blah Blah. More closely related to this paper, Carpenter et al. (2021) and Badgett et al. (2024) examine how different environments can shape the way in which homosexuality relates to a host of economic outcomes [Chris Carpenter?]. Interestingly, literature outside of economics has also considered the impact of gay parents on children's outcomes and generally finds null effects, including on children's own sexuality, i.e., having a gay parent does not increase the likelihood that a child is gay.[1]

Fourth, our paper relates to a relatively small but growing literature across the social sciences that relates religion to economic outcomes. Papers have considered a number of important relationships, including: adolescent mental health outcomes and own religiosity (using the same data we use) (Fruehwirth et al. (2019)), parental religion and child development, religion and historical economic growth (Squicciarini (2020)), and religion and educational outcomes (Horwitz et al. (2020)), among others.[2] Many such papers are correlative, though Bartkowski et al. (2008) use within-sibling variation in their analyses and Fruehwirth et al. (2019) uses an instrumental variables approach based on peer religiosity, both to make causal arguments. In general, our findings that religion on its own predicts positive outcomes is align with many previous papers. However, our focus in on interacting with variables that predict mismatch between parents' religiosity and children's attributes, which predicts disadvantageous outcomes.

Finally, our paper relates to a growing literature that uses polygenic scores in economic analysis. This work is wide-ranging, but a lot of papers focus on so-called "gene-by-environment" or G×E interactions. For example, Barth et al. (2020) show that different pension schemes moderate the relationship between a polygenic score for education and financial wealth; and Papageorge and Thom (2020) find that the same polygenic score for education is more predictive of college-going when children grow up in wealthier households. This paper considers a novel G×E interaction: a genetic propensity for SSSB and parental religiosity.

A unique feature of polygenic scores is that they allow the researcher to observe tendencies, risks, and behaviors that might otherwise remain hidden. For example, Jeong et al. (2024) find that genetic risk for dementia predict disadvantageous economic outcomes among individuals who are never diagnosed for dementia. Indeed, they may not even be

[1] Some work suggesting otherwise has been soundly debunked on basic methodological grounds.

[2] A reasonable extension to our findings here would be to extend Fruehwirth et al. (2019) to examine how the polygenic risk of same sex sexual behavior interacts with own religion and how these interactions relate to economic outcomes.

aware of their cognitive decline. Use of polygenic risk scores allows the researcher to examine relationships that are otherwise unobservable due to mis-reporting or lack of diagnosis. Similarly, this paper examines relationships between homosexuality tendencies or behaviors and parental attributes that would otherwise remain hidden due to endogenous closeting.

2 Background on the Genetics of Same-sex Sexual Behavior

Research has suggested that same-sex sexual behavior runs in families (Pillard and Bailey, 1998), based on evidence demonstrating that this behavior is concordant more often in genetically identical twin pairs than in fraternal twin pairs (Bailey et al., 2000; Långström et al., 2010). However, while these studies show a clear heritable component to sexual preferences and/or orientation, identifying specific genetic variants link with these behaviors has proven more difficult. Previous studies linking genetic variants, including the controversial "gay gene" hypothesis, were based in small samples that do not meet the robust, replicable standards of genome-wide significance widely recognized in modern genetics research (Hamer et al., 1993; Rice et al., 1999; Sanders et al., 2017).

As access to enormous amounts of genetic data linked to health and behavioral outcomes has grown, so too has the ability to link specific genetic variants to outcomes of interest with high confidence. Genome-wide associations studies (GWAS) are the standard method through which this goal is accomplished (McCarthy et al., 2008). GWAS estimates the association between what is found at a particular location on the genome and an outcome of interest. Specifically, for a particular location in the human genome, a GWAS considers the effect a genotype (i.e., the number and type of alleles present) on the outcome. GWAS analyze millions of distinct locations in the genome, which are known as single nucleotide polymorphisms (SNPs). Further, GWAS applies a strict Bonferroni correction for 1,000,000 independent tests to arrive at a p-value of 5x10-8 for genome-side significance. GWAS results, called summary statistics, consist of a long vector of estimated effects, one per SNP, and are often made publicly available to researchers for use in downstream analyses. In 2019, the first well-powered GWAS of same-sex sexual behavior was conducted using the data from a sample of 477,522 individuals, identifying for the first time genetic variants linked to same-sex sexual behavior with high confidence (Ganna et al., 2019).

2.1 Polygenic Index Construction and GWAS Data

The measure we use to gauge genetic propensity for same-sex sexual behavior is a polygenic index (PGI). Polygenic indices aggregate or sum the effects on a phenotype of hundreds of thousands or even millions of single-nucleotide polymorphisms (SNPs), the type of genetic variant that is responsible for most genetic differences between humans. At each SNP, a person possesses two alleles, and a person's genotype at each SNP is the number of risk or reference alleles (0, 1, or 2) found there. A PGI for an individual is therefore a weighted sum of a person's genotypes at j loci,

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

where $\hat{g}_i$ denotes the polygenic index of individual i, $\hat{\beta}_j$ is the estimated additive effect size of the effect-coded allele at variant j, and g_{ij} is the genotype of individual i at variant j (coded as having 0, 1, or 2 instances of the effect-coded allele). To ease interpretation of results, we standardize each PGI to have a mean of 0 and standard deviation of 1 across the sample.

The indices in this analysis are constructed using a Bayesian index generation method called LDpred (Vilhjálmsson et al., 2015), a model of index generation that considers genetic architecture by accounting for linkage disequilibrium. Since we do not know the variance-covariance matrix of the effects in the training sample, we replace this matrix with a block diagonal matrix estimated using LD patterns from a reference sample of unrelated individuals with European ancestry. Our LD reference sample is specific to our prediction cohort; to estimate the block diagonal LD matrix, we use the genotyped data from each of the Add Health prediction cohort, after dropping cryptically-related individuals or ancestry outliers, to create our LDpred reference sample. All variants that passed our filters were used when constructing our indices.

After imputing the genetic data to the Haplotype Reference Consortium (HRC) using the Michigan Imputation Server (The Haplotype Reference Consortium, 2016; Das et al., 2016), we used only HapMap3 variants to construct our indices, which are well imputed and provide good coverage of common variation across the genome. We limited all analyses in the Add Health cohort to European-ancestry individuals. Finally, we used HapMap3 variants with a call rate above 98% and a minor allele frequency $> 1\%$. In our final index generating step, we used Plink to multiply the genotype probability of each variant by the corresponding LDpred posterior mean over all variants (Vilhjálmsson et al., 2015). We created our polygenic

indices for SSSB using the publicly available GWAS summary statistics from Ganna et al. (2019). The summary statistics are available at the GWAS Catalog[3] using Accession Number GCST008654[4]. These summary statistics are reported from a meta-analysis GWAS of SSSB in the UK Biobank[5] and the top 10,000 independent SNPs from 23andMe data. They include 329,546 European ancestry males and 338,218 European ancestry females. In total, we used 1,064,271 HapMap3 variants to construct the polygenic indices in Add Health.

A number of studies have discussed important caveats regarding the use of genetic data in economic analysis.[6] We briefly review the main concerns here. First, the polygenic index (PGI) for same-sex sexual behavior was discovered using individuals of European ancestry. Prior research has shown that PGIs discovered in one ethnic group are substantially less predictive when applied to other groups, making cross-ethnic comparisons largely misleading (Martin et al., 2017). Accordingly, our analysis is limited to individuals of European ancestry. Second, the phenotype used to construct the PGI is a binary, self-reported measure of whether respondents have ever had sex with someone of the same sex. Respondents may underreport this behavior, introducing measurement error. In addition, there may be estimation error in the GWAS weights used to construct the PGI (Ganna et al., 2019), which could further contribute to measurement error, though such error is likely to diminish as GWAS sample sizes increase. Third, PGIs are not exogenously assigned and may be correlated with aspects of the family environment shaped by parental genetic endowments. For example, parents with genetic predispositions toward same-sex sexual behavior may not only pass these traits to their children but may also create home environments that systematically differ from those of other families.

Despite these limitations, our analysis provides valuable insights into the joint influence of genetic predispositions toward same-sex sexual behavior and family environments on important life outcomes. While causal interpretations should be made with caution, the patterns we document reveal meaningful associations that deepen our understanding of the interplay between genetic and environmental factors in shaping sexual orientation and a range of socioeconomic outcomes.

3 Data

We use the restricted-access version of the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative sample of over 90,000 adolescents

[3]https://www.ebi.ac.uk/gwas/downloads/summary-statistics
[4]https://www.ebi.ac.uk/gwas/studies/GCST008654
[5]https://www.ukbiobank.ac.uk
[6]See Biroli et al. (2026) for a comprehensive discussion on this issue.

in grades 7–12 during the 1994–1995 academic year (Wave I). Following an initial in-school survey, a randomized subset of 20,745 individuals participated in comprehensive in-home interviews. Concurrently in 1995, parent interviews, completed largely by mothers, gathered supplemental data regarding both the household and the adolescent. The in-home cohort was subsequently followed across multiple waves: Wave II (1996), Wave III (2001–2002; ages 18–26), Wave IV (2008–2009; ages 24–32), and Wave V (2016–2018; ages 33–43). These surveys provide rich longitudinal data on sexual behavior, educational achievement, mental health, labor market outcomes, risky behaviors, and parental socioeconomic status (SES).

3.1 Sample Construction

During Wave IV, Add Health gathered genetic information via saliva samples from 96% of the in-home participants. Of this group, approximately 12,200 respondents (80%) authorized the long-term archiving of their genetic data. After quality control, genotyped data are available for 9,974 individuals (Braudt and Harris, 2020).[7]

Given that the discovery sample for the polygenic index for same-sex sexual behavior (SSSB) consisted of individuals of European descent, our analytical sample is restricted to respondents genetically categorized as European. From the 5,725 individuals in this demographic with complete genetic data, we further exclude those missing essential covariates, the polygenic index for educational attainment, parental socioeconomic status (SES), or parental religiosity. This yields a final sample of 4,927 individuals (2,574 females and 2,353 males), resulting in 21,630 person-wave observations across the five survey waves.[8] To mitigate concerns regarding nonrandom attrition into the genotyped subset, all estimations apply weights based on the inverse probability of genetic sample inclusion, conditional on observed characteristics.[9]

3.2 Parental Religiosity Measure

In the Wave I sample of 4,927 adolescents, 4,552 parents indicated a religious affiliation in 1995, leaving 375 unaffiliated. Affiliated parents answered four distinct items measuring their

[7]To adjust for population stratification (i.e., variations in genetics across ancestral backgrounds), Add Health restricts the genotyped sample to four ancestry categories: European, African, Hispanic, and East Asian. These classifications are derived via principal component analysis, utilizing the distance from the mean of the first two principal components of the genetic data (Braudt and Harris, 2020).

[8]Incorporating the polygenic index for educational attainment only slightly decreases the sample from 4,930 to 4,927.

[9]To construct the weights, we estimate a probit model predicting genotyped sample inclusion as a function of parental religiosity, religious affiliation, educational attainment, age and its squared, biological sex, and birth year dummies. We then utilize the inverse of the predicted probability, $\frac{1}{\hat{P}(\text{In Sample}|X_i)}$, as the weight.

religiosity: "How often have you gone to religious services in the past year?", "How important is religion to you?", "How often do you pray?", and "Do you agree or disagree that the sacred scriptures of your religion are the work of God and are completely without mistakes?" Each question featured four to five categorical response options. Our constructed measure of parental religiosity incorporates the first three items: service attendance, importance of religion, and prayer frequency. We omit the fourth question concerning scriptural infallibility due to its elevated nonresponse rate.[10] By summing the responses to the three retained questions, we generate a parental religiosity scale that spans from 1 to 11.[11] A maximum score of 11 corresponds to parents who gave the most religious response to each of the three questions.

?? plots the distribution of this constructed religiosity variable. A value of 0 on the horizontal axis corresponds to parents without any religious affiliation. For those with an affiliation, the distribution is strongly right-skewed, meaning very few parents chose the least religious responses across the survey items. In our primary specifications, we construct a binary indicator for whether a parent is classified as "more religious." As marked by the second vertical line in **??**, parents scoring 10 or 11 fall into the more religious category, whereas those scoring between 1 and 9, alongside the unaffiliated, are classified as less religious. Although this threshold is somewhat arbitrary, it effectively splits the weighted sample of affiliated parents in half.

3.3 Other Key Variables

3.3.1 Same-sex Sexual Behavior

The phenotype serving as the basis for the SSSB polygenic index is a binary variable indicating whether a respondent has ever engaged in sexual activity with a same-sex partner (Ganna et al., 2019). Add Health asks respondents to detail their sexual history regarding both male and female partners, which enables us to construct a variable that perfectly matches the one utilized in the original genome-wide association study. This specific survey question first appears in Wave IV, when the participants are generally in their mid-20s or older.

[10] The refusal rates for the first three items are between 0.2–0.4%, whereas roughly 10% of respondents either refused to answer the fourth question or responded with "don't know".

[11] Fruehwirth et al. (2019) also constructed a comparable religiosity measure by aggregating similar questions, although their metric utilized the adolescents' self-reported data rather than parental responses.

3.3.2 Mental Health

To evaluate mental health, the survey utilizes items drawn from the Center for Epidemiologic Studies Depression Scale (CES-D), one of the most widely used tool for measuring depressive symptoms (Radloff, 1977). While the standard CES-D contains 20 self-reported items, Add Health administered varying subsets of these items across different waves.[12] Specifically, Waves I and II include 19 items (CES-D 19), Wave III features 9 items (CES-D 9), Wave IV has 10, and Wave V retains 5 items (CES-D 5). To accommodate this variation, we construct consistent measures across specific waves: CES-D 19 covers Waves I and II, CES-D 9 spans Waves I through IV, and CES-D 5 is used for Waves I, II, and V.[13] Although the CES-D 19 and CES-D 9 capture a broader range of symptoms, the CES-D 5 remains a validated and reliable instrument for depressive symptoms within the Add Health cohort (Perreira et al., 2005).[14]

We construct several mental health variables using these scales. First, we compute a continuous score by aggregating the item responses. Second, for the CES-D 19, we apply the established binary threshold of 16 from the original 20-item scale to flag depressive symptoms. Alongside this standard threshold, we also evaluate a stricter cutoff of 20, as recommended by the broader literature (Radloff, 1977; Vilagut et al., 2016).[15] Third, for the 9- and 5-item scales, we define equivalent binary indicators by selecting thresholds that align with the prevalence rates of the longer scale. We apply cutoffs of 9 and 11 for the CES-D 9, and 5 and 6 for the CES-D 5. In both instances, the lower value captures mild to moderate symptoms, while the higher value indicates a greater likelihood of major depression.

Additionally, suicidality serves as another metric for severe mental health issues. A binary indicator tracks whether respondents seriously considered suicide within the prior 12 months, a measure collected consistently across all survey waves.

3.3.3 Educational Outcomes

During Waves I and II, when participants were in grades 7–12, they reported their frequency of excused full-day school absences. Responses fell into four brackets: "Never," "1 or 2 times," "3 to 10 times," and "More than 10 times." Participants also provided a continuous count of their unexcused full-day absences (skipped school). To further evaluate early educational outcomes, Wave I asks if the student had ever been held back or repeated a

[12] Relative to the standard CES-D, Add Health drops two items but introduces the prompt "You felt life was not worth living" (Perreira et al., 2005).

[13] Both Wave III and Wave IV exclude a single item from the CES-D 5 set.

[14] Perreira et al. (2005) further establish that the CES-D 5 in Wave V is suitable for cross-cultural and cross-ethnic analysis.

[15] Fruehwirth et al. (2019) similarly employs a threshold of 16 for the 19-item CES-D.

grade.

For high school achievement, Wave III supplies transcript-derived academic performance data, encompassing overall cumulative GPA as well as subject-specific GPAs in science, mathematics, and English for each year and cumulatively. We rely on the cumulative subject-specific and overall GPAs to gauge high school achievement. Data on final educational attainment are gathered in Waves III through V. As many Wave III respondents (aged 18–26) had not yet finished schooling, we primarily utilize Waves IV and V to measure completed educational attainment.

3.3.4 Parental Characteristics

In addition to the religiosity measure, the regression models incorporate controls for parental education level, biological sex, and age. Including these covariates is crucial, as they may correlate with parental religiosity and can independently impact the outcomes of interest. For instance, prior literature demonstrates that religious affiliation alters educational attainment and that higher religiosity mitigates depressive symptoms (Sander, 1992; Lehrer, 1999; Fruehwirth et al., 2019).[16]

3.4 Summary Statistics

Table 1 details the summary statistics for our analytical variables. Because the majority of outcomes were not tracked in every wave, observation counts fluctuate based on data availability. The pooled sample yields a maximum of 21,630 observations; specific analyses utilize subsets of this total depending on the outcome in question.

Female parents make up the majority of the sample, reflecting that mothers supplied most of the parental data. Roughly 39% of parents fall into the "more religious" category. After applying the inverse probability weights for genotyped sample inclusion, this proportion increases to approximately 46%, which roughly divides the sample of religiously affiliated parents in half. In terms of same-sex sexual behavior, nearly 12% of the respondents indicated they had ever had a same-sex partner.

Table 1 also outlines the mental health variables based on their wave availability. For the mild-to-moderate depression indicators, the prevalence rates for CES-D 19, CES-D 9, and CES-D 5 stand at 20%, 18%, and 15%, respectively. These proportions align closely because the thresholds were specifically calibrated to match prevalence rates in Wave I. Since

[16]While we could control for additional SES variables such as household income and participation in welfare programs, doing so severely restricts the sample size. Nevertheless, incorporating these variables does not alter our primary findings.

each measure utilizes a distinct combination of items, they collectively provide a nuanced, multi-faceted perspective on respondents' mental health.

We define severe depressive symptoms using higher binary cutoffs, which are similarly calibrated to match Wave I prevalence rates. Under these stricter thresholds, 11% of the sample is classified as severely depressed using CES-D 19, alongside 10% for both CES-D 9 and CES-D 5. By tracking these indicators across the 20-year span of the five waves, we find that roughly 37% of individuals experienced mild-to-moderate depression at some point, while 23% met the criteria for severe depression at least once. Suicidality is another metric utilized to gauge mental distress, and its average prevalence closely mirrors that of the severe depression indicators.

Mental health challenges during adolescence can carry over into long-term adult outcomes. To explore this, Table 1 details descriptive statistics for a range of education-related variables, covering early school engagement, high school GPA, and completed educational attainment sorted into five categories. Additionally, the table presents data on labor market variables, including hourly wages, annual earnings, average weekly hours, and employment status, as well as binary flags for risky behaviors like drug, alcohol, and tobacco use.

The primary variable of interest is the polygenic index for same-sex sexual behavior (SSSB index), which is standardized within the genotyped sample of Add Health. Because some respondents are excluded from the analytic sample due to missing covariates, the mean and standard deviation of the index within our final analytical sample deviate marginally from 0 and 1, respectively.[17]

To visualize summary statistics, Figure 1 shows a historgram of the parental religiosity measure. Figure 2 shows the density of the SSSB index for more and less religious households. Less religious households show a slightly higher SSSB index, i.e., a right-shifted density.

4 Results

4.1 Self-Reported Same-Sex Sexual Behavior (SSSB)

We begin by examining the relationship between the SSSB PGI and self-reported SSSB in the Add Health Sample. Figure 3 presents a non-parametric local polynomial plot of this relationship. There is a smooth, monotonically increasing relationship between the PGI and self-reported SSSB, with the fraction reporting any SSSB rising from less than 0.10 at PGI values around -2 standard deviations below the mean to nearly 0.20 at values around 2 standard deviations above the mean. Figure 4 plots this relationship separately for those

[17] Within the analytical sample, the mean of the SSSB index is –0.008 and the standard deviation is 1.001.

growing up in households that are more and less religious, respectively. For those from less religious households, there is again a smooth, monotonically increasing relationship. By contrast, the plot for those from more religious households is much flatter, with statistically significant divergence appearing for values of the PGI above -1.

Table 2 presents regression results that quantify these relationships. In Column (1), we regress self-reported SSSB on the PGI and our baseline controls. The estimates suggest that a one-standard deviation higher PGI is associated with a statistically significant 2 percentage point increase in the probability of reporting any SSSB. Compared to the outcome mean of 0.12, this represents a 17 percent increase. In Column (2), we add the binary indicator for high parental religiosity. We find that children raised in more religious households are significantly less likely to report SSSB (by 3.6 percentage points), while adding this regressor does little to attenuate the main association of the SSSB PGI.

In Column (3) of Table 2, we add an interaction between the SSSB PGI and the indicator for higher parental religiosity. We find a statistically significant (p-val = 0.054) negative interaction between these variables. Our results suggest that, among those growing up in a less religious households, a one standard deviation higher SSSB PGI is associated with a 2.8 percentage point increase in the likelihood of self-reported SSSB. By contrast, our estimates suggest this relationship is only 1.1 percentage points for those growing up in a more religious household. In Columns (4)-(6) we find that the main associations and interactions remain robust when adding additional controls for parental SES, the EA PGI and it's interaction with the SSSB PGI, and an interaction between parental religiosity and the EA PGI. This collection of results is consistent with the process of endogenous closeting suggested earlier. In the presence of a rearing environment that may be less supportive of non-heterosexual behavior, individuals with higher SSSB PGIs might be less likely to engage in same-sex sexual behavior, or could be less likely to report them.

4.2 Depressive Symptoms

We next turn to results for depressive symptoms. Panel (a) of Table 3 presents results for a specification in which the dependent variable is a binary indicator for elevated depressive symptoms based on the 19 item CES-D battery. Column (1) reports results for a specification that includes the PGI in addition to our baseline control set. We find that, ceteris paribus, a one standard deviation increase in the PGI is associated with a 1 percentage point increase in the probability of depressive symptoms. Columns (2)-(3) add an indicator for growing up in a more religious household, and the interaction between this indicator and the PGI. Focusing on the results in Column (3), we find that growing up in a more religious household appears

to be protective; our estimates suggest that growing up in such households is associated with a statistically significant 3.6 percentage point reduction in the likelihood of elevated depressive symptoms. However, we find a significant negative interaction between parental religiosity and the PGI. Whereas our estimates suggest basically no relationship between the PGI and depressive symptoms among less religious households, we find that a one standard deviation higher PGI is associated with a 2.2 percentage point increase in the probability of depressive symptoms for those from more religious households. Given that about 20 percent of the sample meets the threshold for elevated depressive symptoms, this relationship within more religious households is substantial. Columns (4)-(6) add additional controls for Parental SES, the EA PGI, and interactions between the EA PGI and other regressors. The main results are robust to these added controls.

Importantly, our estimates *do not* imply that higher-PGI children in more religious households have systematically higher rates of depressive symptoms than similar children in less religious households. Recall that there is a significant negative main association between parental religiosity and the risk of depressive symptoms – growing up in a more religious household appears to be protective of mental health. In this context, a the negative interaction we find suggests that higher-PGI children growing up more religious households do not benefit from these protective effects, or perhaps suffer negative consequences that (but do not overwhelm) these effects. A visual treatment of the data highlights this point. Figure 5 plots the raw relationships between the PGI and the binary depressive symptoms measure (CESD-19) separately for those growing up in more and less religious households. For those from less religious households, this relationship is essentially flat. For those from more religious households, the relationship is monotonically increasing, but nearly always *below* the plot for less religious households.

Our main results for depressive symptoms center on the 19 item CESD scale, which was administered during Waves 1-2 when respondents were still mostly in grades 7-12. A less comprehensive 9 question screener was administered in Waves 3-4, while a 5 question screener was administered in Wave 5. As described in the Data Section, we also construct binary depressive symptom measures based on the CESD-9 an CESD-5 batteries. Panel (b) of Table 3 presents results for a binary depressive symptom measure based on the CES-D 9 scale (available in Waves 1-4). With this less comprehensive screener, we fail to find statistically significant interactions between the PGI and parental religiosity, although we do again find a negative main association between parental religiosity and depressive symptoms. Panel (c) presents results using a binary based on the the CES-D 5 question set (available in Waves 1, 2, and 5). Here we again find a statistically significant positive interaction between the PGI and parental religiosity, with most parameter estimates being quite similar to those

found in Panel (a). Finally, in Panel (d), we pool together all person-year observations and construct a binary indicating if an individual has ever met a threshold for depressive symptoms based on the most comprehensive question set available in a given year. We against find evidence of statistically significant positive interactions between the PGI and parental religiosity.

As a robustness exercise, we replicate all of the specifications in Table 3, but now use a more stringent threshold for determining depressive symptoms, as described in the Data Section. We present these results in Table 4 We generally find results that are qualitatively similar to those found in Table 3, though the estimates for the interaction coefficients tend to be smaller, and p-values tend to be modestly larger. Whereas we found no statistically significant interaction using the CES-D 9 scale, we now do find a statistically significant interaction for this scale when using a more stringent threshold. On the other hand, while we did find a statistically significant interaction with the standard threshold for the CES-D 5 scale, this is no longer significant using the more stringent threshold. Importantly, in Panel (d), when pooling all person-year observations, we again find a statistically significant positive interaction between the PGI and parental religiosity.

4.3 Human Capital Outcomes

Table 5 presents results for three human capital related outcomes: high school GPA, an indicator for completing high school, and an indicator for completing college. Here, we do find some weak evidence of negative interactions between the PGI and parental religiosity, but these are not robust to the inclusion of additional parental controls. For example, in Column (3) of Panel (a), we find that a one standard deviation higher PGI is associated with a 0.045 lower GPA, and that this association *doubles* for those growing up in more religious households. However, this borderline significant interaction coefficient becomes substantially attenuated and loses statistical significance when Parental SES controls are added in Column (4). A similar pattern appears in Panel (b) for the college completion outcome, where we find a significant negative interaction between the PGI and parental religiosity in Column (3) which is substantially attenuated when controlling for Parental SES.

Taken together, the results in Table 5 fail to provide any evidence of significant interactions between the SSSB PGI and parental religiosity in predicting academic and human capital outcomes. In the case of GPA, this could be partly due to sample size – we have significantly fewer non-missing observations for GPA than we do for depressive symptoms, reducing the precision of our estimates. It may also be the case that the interaction effects for mental health outcomes may not be large enough to pass through to downstream outcomes

(or to be detected with the sample sizes offered by the Add Health).

4.4 Self-Reported SSSB v.s. the SSSB PGI

Our empirical strategy involves a focus on genetic predictors of SSSB summarized in the SSSB PGI. The genetic markers that determine the value of the PGI are fixed at conception, and thus before any endogenous choices made by children and parents. An alternate approach to studying this question might instead rely directly on self-reported SSSB or sexual orientation as a way of identifying individuals who are non-heterosexual. Although this may be problematic, it has also been – until now – the only option for studying questions about how environments or policies affect non-heterosexual individuals.

As a final exercise, we therefore ask whether we would get similar results if we had used self-reported SSSB instead of the SSSB PGI as a way to study interactions between parental religion and non-heterosexuality. Tables 6-8 replicate the analyses of Tables 3-5, but now instead of the SSSB PGI, we use a binary indicator for self-reported SSSB. In Tables 6-7, we find a consistent pattern. Although self-reported SSSB is positively associated with depressive symptoms, we consistently fail to find evidence of positive interactions between SSSB and parental religiosity. In fact, we consistently estimate *negative* interactions, though none of these are significantly different from zero at conventional levels of significance. In Table 8, we also fail to find any statistically significant interactions between self-reported SSSB and parental religiosity in predicting our human capital outcomes.

The contrast between the depressive symptom results using the SSSB PGI and self-reported SSSB highlight the potential value in using genetic predictors or proxies for a trait when it is difficult to directly measure or endogenously determined. One possible explanation for the discrepancy involves endogenous closeting or repression. Some individuals with a high SSSB PGI might choose not to engage in SSSB because of stigma or disapproval that may be felt more strongly in more religious households. These individuals will not be observed as engaging in SSSB, but this process of suppression or internal conflict could increase the risk of depressive symptoms. This effect on mental health would be detected in a research strategy using the PGI, but would be missed by an approach using self-reported SSSB.

5 Discussion

We relate a polygenic index for same sex sexual behavior with parental religiosity to predict economic outcomes. We find evidence of less same sex sexual behavior in more religious households (endogenous closeting) and of higher rates of depressive among adolescents with

high SSSB indices in more religious households. We do not find similar effects on other education and labor outcomes.

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Tables and Figures

Table 1: Summary Statistics

Variables	Mean	Std	N
Age	23.5	8.12	21630
Female (=1)	0.52	0.50	4927
Parental Characteristics			
Age	41.7	6.01	4927
Female (=1)	0.93	0.25	4927
LT High School (=1)	0.10	0.30	4927
High School (=1)	0.44	0.50	4927
Some College (=1)	0.21	0.41	4927
College (=1)	0.15	0.36	4927
HT College (=1)	0.096	0.29	4927
Religiosity (0-11)	7.86	3.22	4927
More Religious (=1)	0.39	0.49	4927
Sexual Behavior			
Number of Sexual Partners	13.3	24.3	8015
Ever Had Sex (=1)	0.70	0.46	21547
Age at First Sex	16.9	2.83	11836
Ever Had Anal Sex (=1)	0.48	0.50	8566
Ever Had Same-sex Sex (=1)	0.12	0.32	8530
Mental Health			
CES-D19	10.4	7.30	8788
CES-D19 (=1)	0.20	0.40	8788
CES-D19 Severe (=1)	0.11	0.31	8788
CES-D9	5.06	4.11	17899
CES-D9 (=1)	0.18	0.38	17899
CES-D9 Severe (=1)	0.10	0.30	17899
CES-D5	2.31	2.44	12449
CES-D5 (=1)	0.15	0.36	12449
CES-D5 Severe (=1)	0.097	0.30	12449
Ever Depressed (=1)	0.37	0.48	19370
Ever Depressed Severe (=1)	0.23	0.42	18857
Suicidality (=1)	0.097	0.30	21416

Variables	Mean	Std	N
Education			
Never Absent from School	0.098	0.30	8388
1-2 Absent from School	0.29	0.45	8388
3-10 Absent from School	0.47	0.50	8388
10+ Absent from School	0.14	0.35	8388
Number of Times Skipped School	1.57	5.78	8384
Ever Repeated / Been Held Back a Grade	0.18	0.39	4925
Overall GPA	2.72	0.81	3556
Math GPA	2.36	0.94	3543
English GPA	2.52	0.93	3546
Science GPA	2.44	0.96	3520
LT High School (=1)	0.063	0.24	12813
High School (=1)	0.41	0.49	12813
Some College (=1)	0.24	0.43	12813
College (=1)	0.18	0.38	12813
HT College (=1)	0.11	0.31	12813
Labor Market Outcomes			
Earnings ($1000)	35.4	38.9	11804
Hourly Wage	17.6	22.8	10460
Work for Pay (=1)	0.80	0.40	11875
Hours Worked per Week	41.2	15.5	11097
Risky Behavior			
Any Risky Behavior (=1)	0.86	0.35	21577
Ever Smoked Cigarettes (=1)	0.69	0.46	12966
Ever Injected Illegal Drug (=1)	0.17	0.37	13094
Ever Consumed Alcohol (=1)	0.79	0.41	21573
Ever Used Marijuana (=1)	0.49	0.50	21553
Ever Used Cocaine (=1)	0.12	0.33	17849
Ever Regularly Smoked Cigarettes (=1)	0.48	0.50	18733
llegal Drug Frequency	0.50	10.7	12942
Binge Drinking (=1)	0.45	0.50	21588
Marijuana Frequency	2.59	16.7	12893
Cocaine Frequency	0.24	10.2	8072

Note: Earnings and hourly wage are adjusted to 2010 dollars using the Consumer Price Index. Summary statistics are calculated without applying sample weights.

Table 2: Genes, Parental Religiosity, and SSSB

	(1)	(2)	(3)	(4)	(5)	(6)
Dep: Ever Had Same-sex Sex			Wave 4-5			
SSSB Index	0.020***	0.019***	0.028***	0.027***	0.028***	0.028***
	(0.004)	(0.004)	(0.006)	(0.006)	(0.006)	(0.006)
More Religious (=1)		-0.036***	-0.037***	-0.036***	-0.036***	-0.036***
		(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
SSSB Index × More Religious (=1)			-0.017*	-0.017*	-0.018**	-0.017*
			(0.009)	(0.009)	(0.009)	(0.009)
Observations	8,530	8,530	8,530	8,530	8,530	8,530
R^2	0.030	0.033	0.034	0.035	0.036	0.036
Parental SES	N	N	N	Y	Y	Y
SSSB Index × Other Polygenic Index	N	N	N	N	Y	N
Parental Religiosity × Other Polygenic Index	N	N	N	N	N	Y

Note: All specifications include the following controls: 20 principal components of the genetic data, age and its squared term, biological sex, birth year dummies, and wave dummies. The "Parental SES" controls consist of biological sex, age, and full dummies for education level. "SSSB Index × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the SSSB index. "Parental Religiosity × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the parental religiosity. Robust standard errors clustered at the family level are in parentheses.

Table 3: Genes, Parental Religiosity, and Depressive Symptoms

	(1)	(2)	(3)	(4)	(5)	(6)
	Binary CES-D Scale					
Panel (a): CES-D 19 Wave 1-2						
SSSB Index	0.010*	0.009*	-0.002	-0.001	-0.002	-0.002
	(0.005)	(0.005)	(0.007)	(0.007)	(0.007)	(0.007)
More Religious (=1)		-0.037***	-0.036***	-0.026**	-0.023**	-0.023**
		(0.011)	(0.011)	(0.011)	(0.011)	(0.011)
SSSB Index × More Religious (=1)			0.024**	0.022**	0.022**	0.023**
			(0.011)	(0.011)	(0.011)	(0.011)
Observations	8,788	8,788	8,788	8,788	8,788	8,788
R^2	0.031	0.033	0.034	0.041	0.043	0.043
Panel (b): CES-D 9 Wave 1-4						
SSSB Index	0.008**	0.007*	0.004	0.004	0.004	0.003
	(0.004)	(0.004)	(0.005)	(0.005)	(0.005)	(0.005)
More Religious (=1)		-0.033***	-0.033***	-0.023***	-0.021***	-0.021***
		(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
SSSB Index × More Religious (=1)			0.008	0.006	0.005	0.007
			(0.008)	(0.008)	(0.008)	(0.008)
Observations	17,899	17,899	17,899	17,899	17,899	17,899
R^2	0.026	0.028	0.028	0.035	0.037	0.037
Panel (c): CES-D 5 Wave 1,2,5						
SSSB Index	0.010***	0.010**	-0.000	0.001	-0.000	-0.000
	(0.004)	(0.004)	(0.005)	(0.005)	(0.005)	(0.005)
More Religious (=1)		-0.031***	-0.030***	-0.022***	-0.021**	-0.021**
		(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
SSSB Index × More Religious (=1)			0.021***	0.019**	0.020***	0.020**
			(0.008)	(0.008)	(0.008)	(0.008)
Observations	12,449	12,449	12,449	12,449	12,449	12,449
R^2	0.020	0.022	0.022	0.028	0.028	0.028
Panel (d): Ever Depressed (=1)						
SSSB Index	0.017***	0.016**	0.002	0.003	0.002	0.001
	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
More Religious (=1)		-0.065***	-0.064***	-0.051***	-0.048***	-0.049***
		(0.014)	(0.014)	(0.014)	(0.014)	(0.014)
SSSB Index × More Religious (=1)			0.030**	0.027**	0.027**	0.028**
			(0.013)	(0.013)	(0.013)	(0.013)
Observations	19,370	19,370	19,370	19,370	19,370	19,370
R^2	0.103	0.108	0.109	0.119	0.120	0.121
Parental SES	N	N	N	Y	Y	Y
SSSB Index × Other Polygenic Index	N	N	N	N	Y	N
Parental Religiosity × Other Polygenic Index	N	N	N	N	N	Y

Note: Each panel presents results based on different CES-D measures available across survey waves. Panel (a) uses the 19-item CES-D scale from waves I-II. Panel (b) uses the 9-item CES-D scale from waves I-IV. Panel (c) uses the 5-item CES-D scale from waves I, II, and V. Panel (d) reports results based on a binary indicator for whether respondents have ever been classified as depressed, using CES-D19 in waves I and II, CES-D9 in waves III and IV, and CES-D5 in wave V. All specifications include the following controls: 20 principal components of the genetic data, age and its squared term, biological sex, birth year dummies, and wave dummies. The "Parental SES" controls consist of biological sex, age, and full dummies for education level. "SSSB Index × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the SSSB index. "Parental Religiosity × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the parental religiosity. Robust standard errors clustered at the family level are in parentheses.

Table 4: Genes, Parental Religiosity, and Major Depressive Symptoms

	(1)	(2)	(3)	(4)	(5)	(6)
	Binary CES-D Scale (Major Depression)					
Panel (a): CES-D 19 Wave 1-2						
SSSB Index	0.011***	0.011***	0.005	0.005	0.005	0.005
	(0.004)	(0.004)	(0.005)	(0.005)	(0.005)	(0.005)
More Religious (=1)		-0.021**	-0.021**	-0.012	-0.011	-0.011
		(0.008)	(0.008)	(0.009)	(0.009)	(0.009)
SSSB Index × More Religious (=1)			0.013	0.012	0.011	0.012
			(0.008)	(0.008)	(0.008)	(0.008)
Observations	8,788	8,788	8,788	8,788	8,788	8,788
R^2	0.029	0.030	0.030	0.036	0.037	0.037
Panel (b): CES-D 9 Wave 1-4						
SSSB Index	0.007**	0.007**	0.001	0.001	0.001	0.001
	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)
More Religious (=1)		-0.018***	-0.017***	-0.010	-0.009	-0.009
		(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
SSSB Index × More Religious (=1)			0.013**	0.011*	0.011*	0.012**
			(0.006)	(0.006)	(0.006)	(0.006)
Observations	17,899	17,899	17,899	17,899	17,899	17,899
R^2	0.021	0.021	0.022	0.028	0.029	0.029
Panel (c): CES-D 5 Wave 1,2,5						
SSSB Index	0.008***	0.008**	0.004	0.004	0.004	0.004
	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)
More Religious (=1)		-0.023***	-0.022***	-0.016**	-0.015**	-0.015**
		(0.006)	(0.006)	(0.007)	(0.007)	(0.007)
SSSB Index × More Religious (=1)			0.008	0.007	0.008	0.007
			(0.006)	(0.006)	(0.006)	(0.006)
Observations	12,449	12,449	12,449	12,449	12,449	12,449
R^2	0.017	0.019	0.019	0.024	0.025	0.025
Panel (d): Ever Depressed (=1)						
SSSB Index	0.021***	0.020***	0.009	0.010	0.009	0.009
	(0.006)	(0.006)	(0.008)	(0.008)	(0.008)	(0.008)
More Religious (=1)		-0.046***	-0.046***	-0.033***	-0.031**	-0.031**
		(0.012)	(0.012)	(0.012)	(0.012)	(0.012)
SSSB Index × More Religious (=1)			0.023**	0.021*	0.020*	0.021*
			(0.012)	(0.012)	(0.012)	(0.012)
Observations	18,857	18,857	18,857	18,857	18,857	18,857
R^2	0.094	0.097	0.098	0.109	0.110	0.110
Parental SES	N	N	N	Y	Y	Y
SSSB Index × Other Polygenic Index	N	N	N	N	Y	N
Parental Religiosity × Other Polygenic Index	N	N	N	N	N	Y

Note: Each panel presents results based on different CES-D measures available across survey waves. Panel (a) uses the 19-item CES-D scale from waves I-II. Panel (b) uses the 9-item CES-D scale from waves I-IV. Panel (c) uses the 5-item CES-D scale from waves I, II, and V. Panel (d) reports results based on a binary indicator for whether respondents have ever been classified as depressed, using CES-D19 in waves I and II, CES-D9 in waves III and IV, and CES-D5 in wave V. All specifications include the following controls: 20 principal components of the genetic data, age and its squared term, biological sex, birth year dummies, and wave dummies. The "Parental SES" controls consist of biological sex, age, and full dummies for education level. "SSSB Index × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the SSSB index. "Parental Religiosity × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the parental religiosity. Robust standard errors clustered at the family level are in parentheses.

Table 5: Genes, Parental Religiosity, and Human Capital Outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
				Wave 3		
Panel (a): High School GPA						
SSSB Index	-0.073***	-0.067***	-0.045**	-0.057***	-0.052***	-0.051***
	(0.014)	(0.014)	(0.019)	(0.018)	(0.018)	(0.018)
More Religious (=1)		0.275***	0.274***	0.209***	0.192***	0.193***
		(0.028)	(0.028)	(0.027)	(0.027)	(0.027)
SSSB Index × More Religious (=1)			-0.045*	-0.016	-0.016	-0.020
			(0.027)	(0.026)	(0.026)	(0.025)
Observations	3,556	3,556	3,556	3,556	3,556	3,556
R^2	0.077	0.105	0.106	0.202	0.236	0.236
				Wave 4-5		
Panel (b): College (=1)						
SSSB Index	-0.023***	-0.020***	-0.007	-0.015*	-0.012	-0.011
	(0.007)	(0.007)	(0.009)	(0.008)	(0.008)	(0.008)
More Religious (=1)		0.119***	0.118***	0.066***	0.054***	0.054***
		(0.014)	(0.014)	(0.014)	(0.013)	(0.013)
SSSB Index × More Religious (=1)			-0.027*	-0.011	-0.011	-0.014
			(0.014)	(0.013)	(0.013)	(0.013)
Observations	8,617	8,617	8,617	8,617	8,617	8,617
R^2	0.055	0.070	0.070	0.196	0.226	0.225
Panel (c): High School (=1)						
SSSB Index	-0.008***	-0.007**	-0.007	-0.009*	-0.008*	-0.008
	(0.003)	(0.003)	(0.005)	(0.005)	(0.005)	(0.005)
More Religious (=1)		0.058***	0.058***	0.045***	0.043***	0.044***
		(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
SSSB Index × More Religious (=1)			0.001	0.004	0.004	0.003
			(0.006)	(0.006)	(0.006)	(0.006)
Observations	8,617	8,617	8,617	8,617	8,617	8,617
R^2	0.022	0.038	0.038	0.088	0.091	0.093
Observations	8,617	8,617	8,617	8,617	8,617	8,617
Parental SES	N	N	N	Y	Y	Y
SSSB Index × Other Polygenic Index	N	N	N	N	Y	N
Parental Religiosity × Other Polygenic Index	N	N	N	N	N	Y

Note: Panels (a), (b), (c), and (d) present the results for overall, English, mathematics, and science grade point averages (GPA), respectively. All specifications include the following controls: 20 principal components of the genetic data, age and its squared term, biological sex, and birth year dummies. The "Parental SES" controls consist of biological sex, age, and full dummies for education level. "SSSB Index × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the SSSB index. "Parental Religiosity × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the parental religiosity. Robust standard errors clustered at the family level are in parentheses.

Table 6: Self-Reported SSSB, Parental Religiosity, and Depressive Symptoms

	(1)	(2)	(3)	(4)	(5)	(6)
	Binary CES-D Scale					
Panel (a): CES-D 19 Wave 1-2						
SSSB (=1)	0.092***	0.088***	0.104***	0.101***	0.100***	0.100***
	(0.016)	(0.016)	(0.020)	(0.020)	(0.020)	(0.020)
More Religious (=1)		-0.036***	-0.030***	-0.018*	-0.016	-0.017
		(0.010)	(0.011)	(0.011)	(0.011)	(0.011)
SSSB × More Religious (=1)			-0.046	-0.048	-0.047	-0.047
			(0.033)	(0.033)	(0.033)	(0.033)
Observations	8,729	8,729	8,729	8,729	8,729	8,729
R^2	0.035	0.037	0.037	0.045	0.046	0.046
Panel (b): CES-D 9 Wave 1-4						
SSSB (=1)	0.094***	0.091***	0.101***	0.099***	0.096***	0.098***
	(0.012)	(0.012)	(0.015)	(0.015)	(0.015)	(0.015)
More Religious (=1)		-0.032***	-0.028***	-0.018**	-0.017**	-0.017**
		(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
SSSB × More Religious (=1)			-0.029	-0.030	-0.028	-0.029
			(0.026)	(0.025)	(0.025)	(0.025)
Observations	17,773	17,773	17,773	17,773	17,773	17,773
R^2	0.032	0.034	0.034	0.040	0.042	0.042
Panel (c): CES-D 5 Wave 1,2,5						
SSSB (=1)	0.076***	0.074***	0.082***	0.081***	0.080***	0.081***
	(0.012)	(0.012)	(0.015)	(0.015)	(0.015)	(0.015)
More Religious (=1)		-0.030***	-0.026***	-0.017**	-0.017**	-0.016**
		(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
SSSB × More Religious (=1)			-0.026	-0.027	-0.026	-0.027
			(0.025)	(0.025)	(0.026)	(0.025)
Observations	12,377	12,377	12,377	12,377	12,377	12,377
R^2	0.022	0.024	0.024	0.030	0.030	0.030
Panel (d): Ever Depressed (=1)						
SSSB (=1)	0.126***	0.120***	0.138***	0.136***	0.133***	0.133***
	(0.019)	(0.019)	(0.022)	(0.022)	(0.023)	(0.022)
More Religious (=1)		-0.063***	-0.055***	-0.040***	-0.038***	-0.039***
		(0.013)	(0.014)	(0.014)	(0.014)	(0.014)
SSSB × More Religious (=1)			-0.057	-0.059	-0.058	-0.057
			(0.039)	(0.039)	(0.039)	(0.039)
Observations	19,244	19,244	19,244	19,244	19,244	19,244
R^2	0.112	0.115	0.116	0.126	0.128	0.128
Parental SES	N	N	N	Y	Y	Y
SSSB Index × Other Polygenic Index	N	N	N	N	Y	N
Parental Religiosity × Other Polygenic Index	N	N	N	N	N	Y

Note: Each panel presents results based on different CES-D measures available across survey waves. Panel (a) uses the 19-item CES-D scale from waves I-II. Panel (b) uses the 9-item CES-D scale from waves I-IV. Panel (c) uses the 5-item CES-D scale from waves I, II, and V. Panel (d) reports results based on a binary indicator for whether respondents have ever been classified as depressed, using CES-D19 in waves I and II, CES-D9 in waves III and IV, and CES-D5 in wave V. All specifications include the following controls: 20 principal components of the genetic data, age and its squared term, biological sex, birth year dummies, and wave dummies. The "Parental SES" controls consist of biological sex, age, and full dummies for education level. "SSSB Index × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the SSSB index. "Parental Religiosity × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the parental religiosity. Robust standard errors clustered at the family level are in parentheses.

Table 7: Self-Reported SSSB, Parental Religiosity, and Major Depressive Symptoms

	(1)	(2)	(3)	(4)	(5)	(6)
	Binary CES-D Scale (Major Depression)					
Panel (a): CES-D 19 Wave 1-2						
SSSB (=1)	0.080***	0.079***	0.085***	0.084***	0.082***	0.083***
	(0.013)	(0.013)	(0.016)	(0.016)	(0.016)	(0.016)
More Religious (=1)		-0.022***	-0.019**	-0.010	-0.010	-0.009
		(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
SSSB × More Religious (=1)			-0.018	-0.020	-0.019	-0.021
			(0.027)	(0.027)	(0.027)	(0.027)
Observations	8,729	8,729	8,729	8,729	8,729	8,729
R^2	0.034	0.035	0.035	0.041	0.041	0.041
Panel (b): CES-D 9 Wave 1-4						
SSSB (=1)	0.073***	0.072***	0.082***	0.081***	0.079***	0.081***
	(0.010)	(0.010)	(0.013)	(0.013)	(0.013)	(0.013)
More Religious (=1)		-0.018***	-0.014**	-0.007	-0.006	-0.006
		(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
SSSB × More Religious (=1)			-0.032	-0.033	-0.030	-0.032
			(0.021)	(0.020)	(0.021)	(0.020)
Observations	17,773	17,773	17,773	17,773	17,773	17,773
R^2	0.026	0.027	0.027	0.032	0.033	0.033
Panel (c): CES-D 5 Wave 1,2,5						
SSSB (=1)	0.076***	0.074***	0.083***	0.082***	0.081***	0.082***
	(0.010)	(0.010)	(0.013)	(0.013)	(0.013)	(0.013)
More Religious (=1)		-0.021***	-0.017***	-0.010	-0.010	-0.009
		(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
SSSB × More Religious (=1)			-0.029	-0.030	-0.028	-0.030
			(0.022)	(0.022)	(0.022)	(0.021)
Observations	12,377	12,377	12,377	12,377	12,377	12,377
R^2	0.022	0.024	0.024	0.028	0.029	0.029
Panel (d): Ever Depressed (=1)						
SSSB (=1)	0.138***	0.134***	0.146***	0.144***	0.140***	0.142***
	(0.018)	(0.018)	(0.022)	(0.022)	(0.022)	(0.022)
More Religious (=1)		-0.045***	-0.040***	-0.026**	-0.025**	-0.024**
		(0.011)	(0.012)	(0.012)	(0.012)	(0.012)
SSSB × More Religious (=1)			-0.036	-0.039	-0.035	-0.039
			(0.037)	(0.037)	(0.037)	(0.037)
Observations	18,734	18,734	18,734	18,734	18,734	18,734
R^2	0.104	0.107	0.107	0.117	0.118	0.118
Parental SES	N	N	N	Y	Y	Y
SSSB Index × Other Polygenic Index	N	N	N	N	Y	N
Parental Religiosity × Other Polygenic Index	N	N	N	N	N	Y

Note: Each panel presents results based on different CES-D measures available across survey waves. Panel (a) uses the 19-item CES-D scale from waves I-II. Panel (b) uses the 9-item CES-D scale from waves I-IV. Panel (c) uses the 5-item CES-D scale from waves I, II, and V. Panel (d) reports results based on a binary indicator for whether respondents have ever been classified as depressed, using CES-D19 in waves I and II, CES-D9 in waves III and IV, and CES-D5 in wave V. All specifications include the following controls: 20 principal components of the genetic data, age and its squared term, biological sex, birth year dummies, and wave dummies. The "Parental SES" controls consist of biological sex, age, and full dummies for education level. "SSSB Index × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the SSSB index. "Parental Religiosity × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the parental religiosity. Robust standard errors clustered at the family level are in parentheses.

Table 8: Self-Reported SSSB, Parental Religiosity, and Human Capital Outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
				Wave 3		
Panel (a): Overall GPA						
SSSB (=1)	-0.152***	-0.129***	-0.091*	-0.088*	-0.070	-0.073*
	(0.039)	(0.039)	(0.049)	(0.046)	(0.044)	(0.044)
More Religious (=1)		0.272***	0.286***	0.215***	0.198***	0.200***
		(0.027)	(0.029)	(0.028)	(0.027)	(0.028)
SSSB × More Religious (=1)			-0.108	-0.104	-0.107	-0.105
			(0.080)	(0.075)	(0.073)	(0.073)
Observations	3,537	3,537	3,537	3,537	3,537	3,537
R^2	0.070	0.097	0.097	0.195	0.231	0.231
				Wave 4-5		
Panel (b): College (=1)						
SSSB (=1)	-0.091***	-0.080***	-0.083***	-0.078***	-0.077***	-0.074***
	(0.019)	(0.019)	(0.022)	(0.020)	(0.020)	(0.020)
More Religious (=1)		0.125***	0.123***	0.067***	0.054***	0.055***
		(0.014)	(0.016)	(0.015)	(0.014)	(0.014)
SSSB × More Religious (=1)			0.010	0.007	0.014	0.009
			(0.040)	(0.037)	(0.036)	(0.036)
Observations	8,571	8,571	8,571	8,571	8,571	8,571
R^2	0.053	0.069	0.069	0.203	0.231	0.230
Panel (c): High School (=1)						
SSSB (=1)	-0.024**	-0.019**	-0.024*	-0.022*	-0.020	-0.021
	(0.009)	(0.009)	(0.013)	(0.013)	(0.013)	(0.013)
More Religious (=1)		0.054***	0.052***	0.038***	0.037***	0.038***
		(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
SSSB × More Religious (=1)			0.016	0.018	0.016	0.017
			(0.017)	(0.016)	(0.016)	(0.016)
Observations	8,571	8,571	8,571	8,571	8,571	8,571
R^2	0.018	0.031	0.031	0.079	0.083	0.084
Parental SES	N	N	N	Y	Y	Y
SSSB Index × Other Polygenic Index	N	N	N	N	Y	N
Parental Religiosity × Other Polygenic Index	N	N	N	N	N	Y

Note: Panels (a), (b), (c), and (d) present the results for overall, English, mathematics, and science grade point averages (GPA), respectively. All specifications include the following controls: 20 principal components of the genetic data, age and its squared term, biological sex, and birth year dummies. The "Parental SES" controls consist of biological sex, age, and full dummies for education level. "SSSB Index × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the SSSB index. "Parental Religiosity × Other Polygenic Index" refers to the polygenic index for educational attainment and its interaction with the parental religiosity. Robust standard errors clustered at the family level are in parentheses.

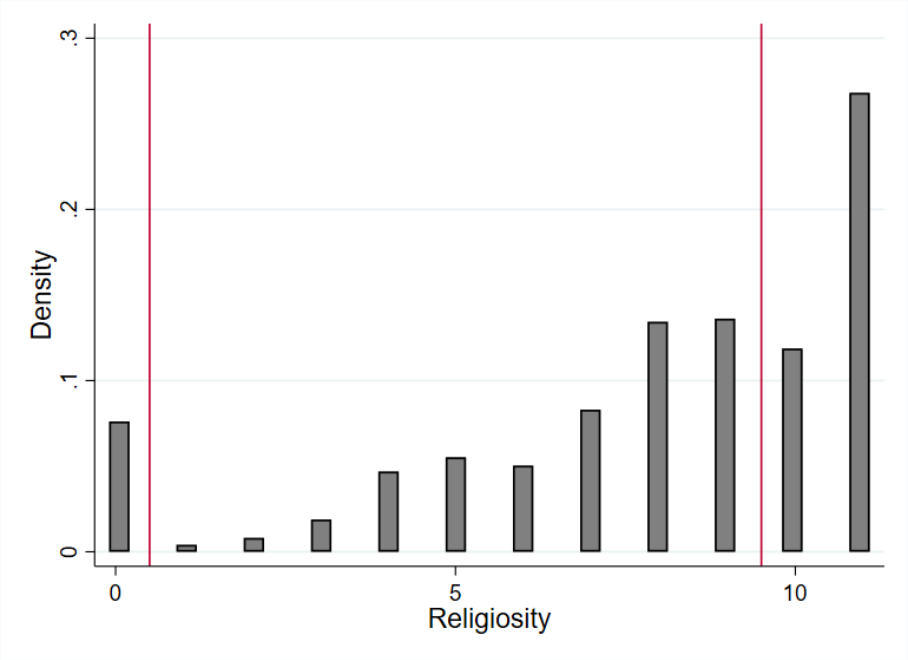


Figure 1: Distribution of Parental Religiosity

Notes: A value of 0 indicates individuals with no religious affiliation; values from 1 to 9 correspond to those whose parents have a religious affiliation and are less religious; and values of 10 or 11 represent individuals whose parents are affiliated and highly religious. The weighted proportions of these categories are 6%, 47%, and 46%, respectively. In the analysis, values from 0 to 9 are classified as "less religious," while values of 10 and 11 are classified as "more religious."

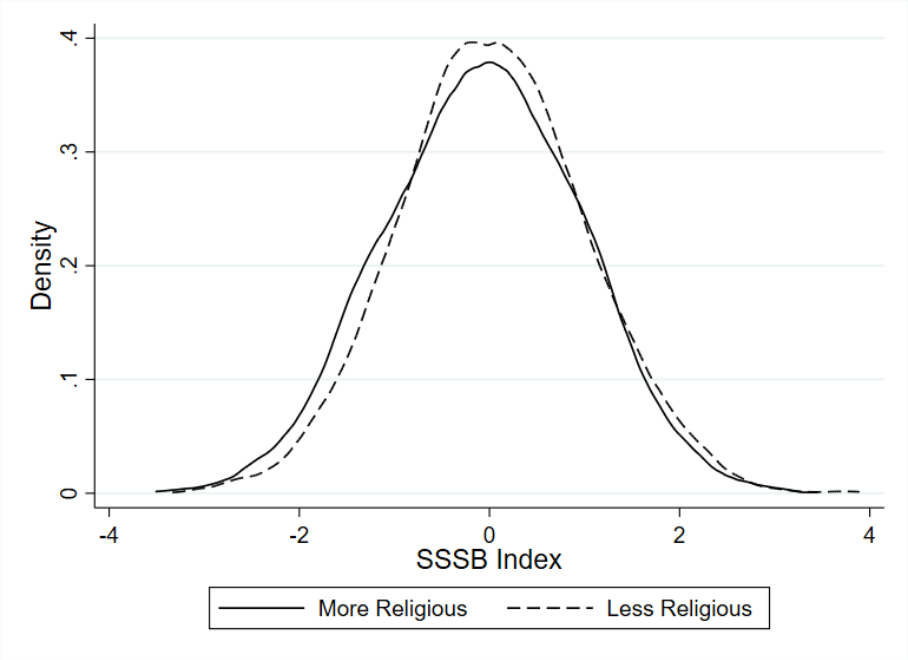


Figure 2: Distribution of SSSB Index by Parental Religiosity

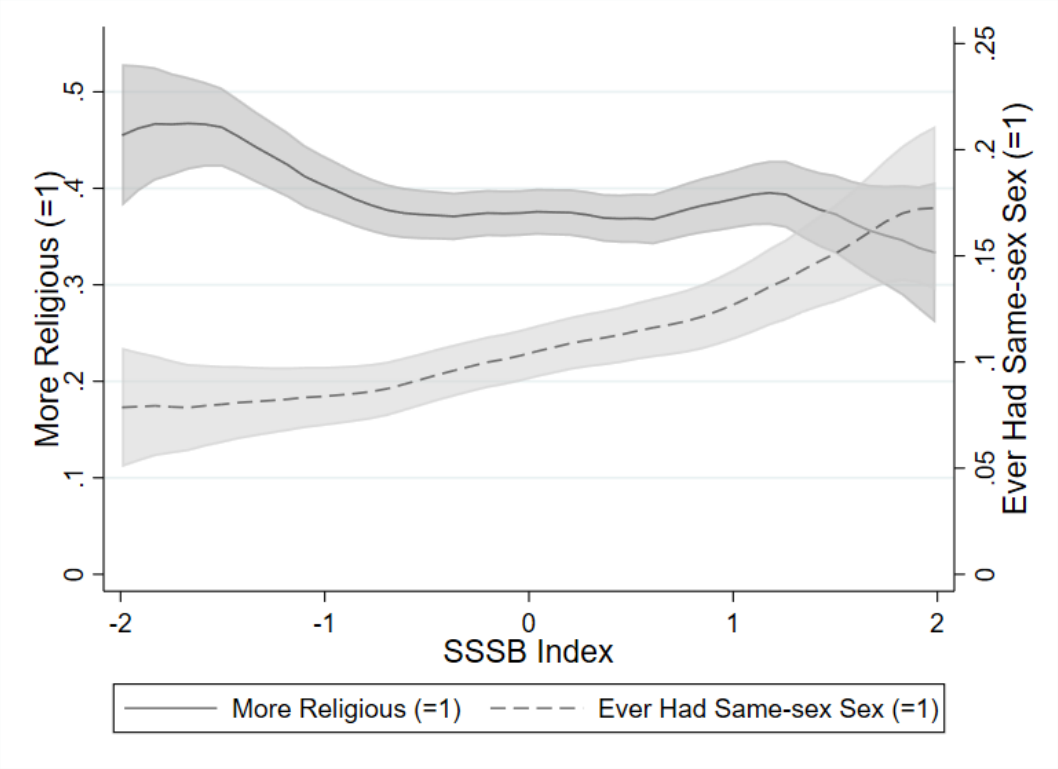


Figure 3: SSSB Index, Same-sex Sexual Behavior and Parental Religiosity

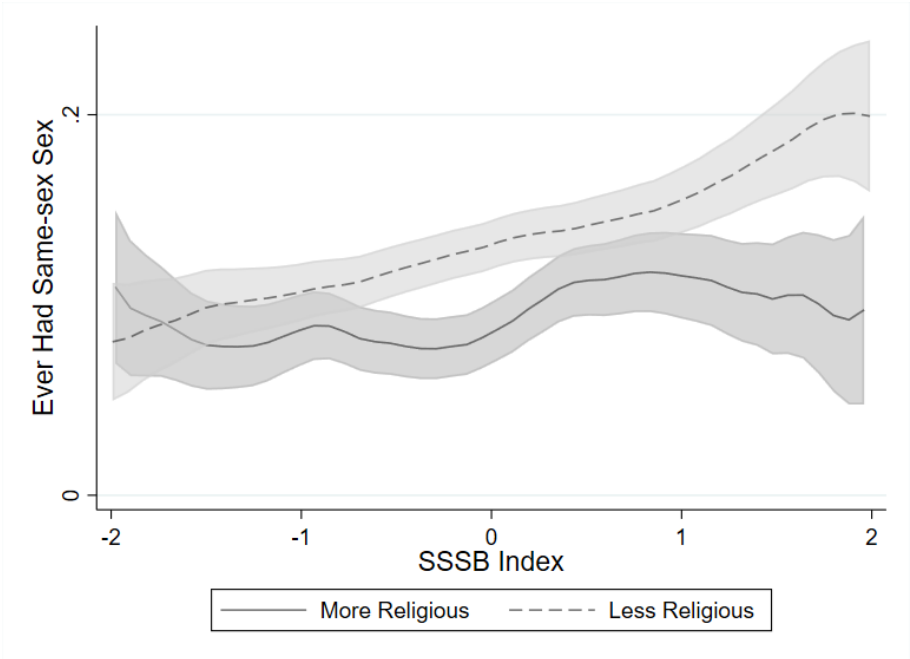


Figure 4: SSSB Index and Same-sex Sexual Behavior by Parental Religiosity

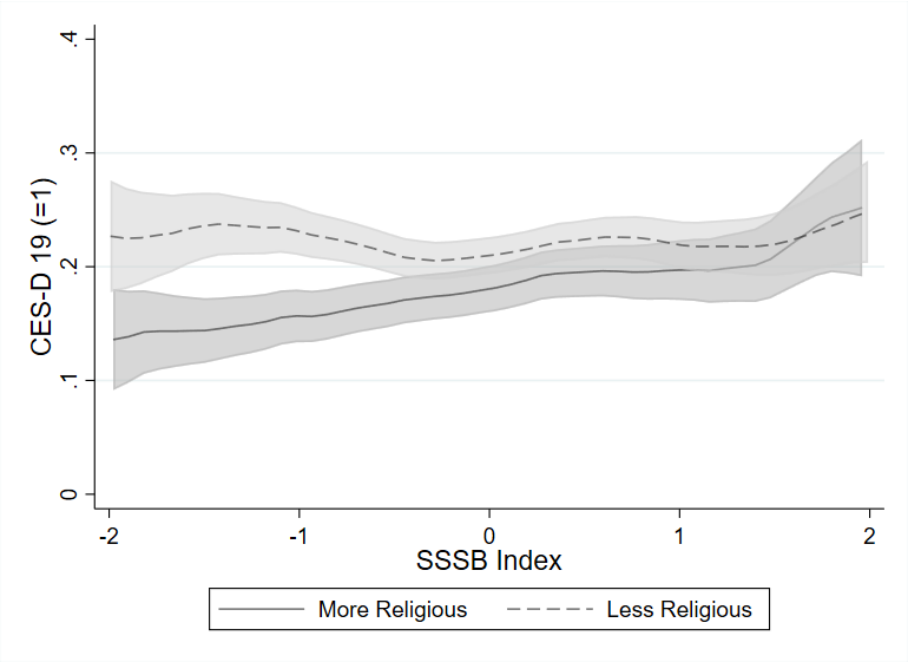


Figure 5: Relationship between Mental Health (Binary CES-D19) and SSSB Index by Parental Religiosity