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IS AFI ALL IN THE FAMILY? A MULTI-LEVEL FAMILY STUDY OF AGE OF FIRST INTERCOURSE

A DISSERTATION APPROVED FOR THE
DEPARTMENT OF PSYCHOLOGY

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Abstract

The importance of the timing of first intercourse in one's life history, and its significance in relation to a number of fertility and social outcomes, has been established in a number of studies. Studies have attempted to untangle the factors that contribute to its timing, and only some of these studies explore the possibility of selection influences on this outcome. This study uses National Longitudinal Survey of Youth (NLSY) samples and multilevel survival models to evaluate predictors of age at first intercourse (AFI) at both the family and individual level. The family structure among the NLSY samples enables the use of a children of siblings type design so that we may also investigate the possible influence of selection effects. Intelligence and educational goals are often implicated as factors motivating adolescents and young adults to delay AFI. Extended family, maternal, and child intelligence variables are the predictor variables of focus in this study. Other variables include maternal AFI, measures of the home environment, and family income, as these variables also relate to the evaluation of educational goals. Gender and race are also included as control variables. None of the intelligence variables were found to be significant predictors of AFI, though interesting trends emerged. Maternal AFI was consistently a significant predictor across models, but was later identified as non-significant relative to average AFI at the maternal family level. Possible explanations for these findings are offered.

Is AFI All in the Family? A Multi-Level Family Study of Age of First Intercourse

Initiation of sexual activity is a significant event in one's life history, and is related to a variety of other important life events through either correlation or causation. Those who initiate sexual activity early are at a high relative risk of not reaching their educational and career goals (Halpern, Joyner, Udry, & Suchindran, 2000), especially if a pregnancy results. Early first intercourse is also associated with higher levels of delinquency in adulthood (Harden et al., 2008), antisocial behavior and substance abuse (Boislard & Poulin, 2011). Further, early initiators are also more likely to have a greater number of sexual partners in adolescence (Udry et al, 1995), and another particular risk of early AFI involves sexually transmitted diseases. In a sample of adolescents from South Africa, Mathews et al. (2009) found that those with a better understanding of how HIV is transmitted and prevented are more likely to delay initiation of sexual activity. The United States has a relatively high rate of teen pregnancy when compared to other Western, industrialized nations (Kirby, 2002), despite decades of efforts to curb adolescent sexual activity and prevent its associated risks (Harden, 2013).

Those who delay the initiation of sexual intercourse, however, are at less risk of these negative outcomes. The motivation for delaying intercourse initiation may often be an explicit attempt to safeguard one's future from these associated risks (Halpern et al., 2000; Harden & Mendle, 2011). Some of the benefits to delayed intercourse seem to last into adulthood. Harden (2012) described delayed age of first intercourse (AFI) as a protective factor against relationship dissatisfaction in adulthood, perhaps due to interpersonal characteristic differences that mediate the way in which one approaches a relationship, protecting against the negative interactions in early relationships that may have a lasting effect on the individual. Further, those who delayed AFI were rated as

more attractive, had higher levels of educational attainment, and higher household incomes than those who initiated sexual activity earlier (Harden, 2012). Carvajal et al. (1999) found that adolescents with more positive attitudes and social norms, as well as greater self-efficacy about their ability to refrain from intercourse were more likely to delay its onset.

Paul et al. (2000) found that a number of predictive factors at the family level differed between males and females, but early maternal child bearing and measures of family structure and relations were predictive of early AFI for both. For males, low family SES and low educational attainment by both parents were associated with early AFI. For females whose mothers worked more than 35 hours per week, early AFI was more likely. Other studies have also found a link between low SES and earlier AFI (Mathew et al., 2009). Adolescents who make decisions without parental input are more likely to engage in sexual activity at an earlier age (Raffaelli & Crockett, 2003). A study by Boislard and Poulin (2011) suggested that adolescents who freely speak with their parents about their free time and those with low levels of parent-child conflict tend to delay sexual activity. This child attribute may either be a result of family-level influence, possibly from an environment of open-communication intentionally fostered by the parent(s), or it may be the result of individual differences in the child resulting from characteristics that are passed to children along with a predisposition for later initiation of intercourse. This transmission can either be genetic or environmental in origin.

In a comprehensive review of the literature on adolescent transitions into sexual activity, Rodgers (1996) discussed many theories regarding the decision to initiate

sexual activity. The social influence of the groups adolescents belong to, including family, religious, and cultural units are important, as are the dynamic relationships between the adolescents themselves. Rodgers, Harris, and Vickers (1992) found that June and July, when students are home from school, are particularly popular months in which adolescents lose their virginity, suggesting that supervision may make an important difference in defining when adolescents choose to initiate sexual activity. Research also indicates that males tend to have an earlier AFI than females, and black males in particular demonstrated early AFI, motivating our inclusion of race and gender in our models.

At the individual level, factors related to intelligence and education can affect AFI. Students who themselves are high achievers academically are less likely to engage in early sexual activity (Boislard & Poulin, 2011). Some have even found a bidirectional relationship between education and AFI. Those who have lower educational goals initiate sex earlier, and those who previously had higher goals indicate lower goals after initiating sexual activity (Schvaneveldt, Miller, Berry, & Lee, 2001). A study by Paul et al. (2000) made a similar point about the relationship between GPA and AFI, suggesting that low GPA is a consequence of early AFI, rather than a cause of it. A relationship between an individual's intelligence or educational goals and their timing decision regarding first intercourse has been found in a number of studies (Harden & Mendle, 2011; Kirby, 2002; Unger, Molina, & Teran, 2000). In fact, smarter teens appear to delay all forms of partnered activity, even activities such as kissing and hand-holding (Halpern, Joyner, Udry, & Suchindran, 2000) which do not carry the same risks as sexual activity, but may be seen by individuals choosing to delay intercourse as

precursors to it. The issue of causality is again in question here: Although it is possible that educationally related considerations directly influence an adolescent's decision to engage in intercourse, it is also possible that both the child's late AFI and higher educational goals and achievements are the product of family influence, either genetically or environmentally.

Because of the high cost of childbearing to females, it is likely that they will give particular consideration to the outcome of intercourse before deciding to initiate sexual activity. As such, it may be that females are even more affected than males by the influence of educational achievement goals. Previous research indicates that smarter teens tend to delay sex, and this relationship is even stronger for females (Halpern, Joyner, Udry & Suchindran, 2000). Interaction effects between gender and child intelligence measures are included in our model to account for such effects. In a sample of adolescent females, Raffaelli & Crockett (2003) found that those who perceive greater positive consequences from teenage child bearing are more likely to engage in risky sexual behaviors, and individuals with low self-regulation or high risk proneness are also more likely to engage in early sexual activity (Raffaelli & Crockett, 2003).

Harden (2013) noted the emphasis that has been placed on AFI in studies of adolescent sexual behavior, and provides an extensive review of literature regarding the hereditary mechanisms related to its timing. Numerous studies have explored what factors contribute to the timing of AFI. Ellis (2004) discussed AFI in a comprehensive review of literature on adolescent sexual development and behavior within the context of Life History theory, and linked its timing to that of physiological development. Many studies have evaluated the role of factors beyond the individual level that can affect the

timing of first intercourse. Kirby (2002) examined more than 250 such studies on the antecedents to early AFI, and found important community and family level influence on AFI. In those communities and families where adults emphasize the importance of education and career goals in conjunction with the avoidance of teen pregnancy, the risk of early AFI and early childbearing is relatively low. Daughters in families that attend church together also tend to have a later AFI (Udry et al., 1995). Previous research also shows that higher parental education is related to delays in AFI (Carvajal et al., 1999; Raffaelli & Crockett, 2003; Udry et al., 1995), which itself is likely to be related to an environment where the importance of education is underscored.

Clearly, a variety of variables at the individual and family levels have been implicated in the timing of AFI. However, the source of the relationship between these variables and an adolescent's AFI is not clear. Selection effects occur when individuals predisposed to an outcome are non-randomly selected into environments that have been associated with the risk for that outcome (Mendle et al., 2009). Here, selection would occur if individuals who are otherwise at risk for early AFI are non-randomly selected into families with low intelligence. Forces of selection and influence are both at play in the intergenerational transmission of traits (Harden, 2013). The relationship between AFI and its associated variables may be due to the confounded genetic and family-level environmental effects, rather than AFI being influenced by other factors themselves (Mendle et al, 2009). There are a number of ways in which these genetic and environmental forces might be correlated (Plomin, DeFries, & Loehlin, 1977). Passive gene environment correlations occur when a parent provides both the genes and an environment associated with a particular outcome. Reactive, sometimes called

evocative, gene environment correlations occur when people with different genotypes are treated differently by those around them, further encouraging the outcome to which they were already predisposed. Active gene environment correlations are the result of the individual seeking out environments conducive to their genotype.

Behavior genetic studies consistently indicate the influence of genetics on AFI, but also often show non-negligible shared environmental influence on the trait as well (Harden et al., 2008; Harden & Mendle, 2011; Mendle et al., 2009; Rodgers, Rowe, & Buster, 1999; also see Harden, 2013 for further review). These studies often show a stronger effect from genetic than environmental sources. This finding means that selection effects, whereby families share predispositions (either through genetics or environmental exposure) for both traits such as greater educational attainment and delayed AFI, may be responsible for their association. The existence of shared environmental influences on AFI, however, indicates that there are also important non-genetic family-level influences on the timing of AFI. Harden noted in her comprehensive review of the literature on adolescent sexuality that, despite the evidence for genetic effects, the emphasis of past studies has been on the family environmental, rather than the genetic, forces at play in studies of adolescent sexuality.

Children of siblings designs are quasi-experiments equipped to evaluate this type of effect (Lahey & D'Onofrio, 2010; Mendle et al., 2009; Rodgers, Bard, Johnson, D'Onofrio & Miller, 2008). By evaluating the children of siblings who are themselves related as cousins (and therefore share the same genetic and family environmental ancestral background), but who have had differing levels of exposure to potential causal environmental variables at the maternal level, one can examine the outcome variable for

selection effects as well as the direct influence of parental traits. Our methodology incorporates this type of design to untangle possible selection effects from more direct causal influences.

In the current study, multilevel modeling is used to evaluate the importance of both family level factors and individual level factors in determining AFI. The use of the National Longitudinal Survey of Youth (NLSY) datasets permits us adequate information on family-level variables from the NLSY79 sample. Because of the data structure, we are able to use three-level models, incorporating information from the level of the mother-aunt dyad (level 3), the child's nuclear family or maternal level (level 2), as well as information from the child him or herself (level 1). Individual-level factors come from the NLSY79 Children/Young Adult surveys, which sampled all of the biological children born to females in the NLSY79.

Many studies, as discussed above, indicate the importance of educational considerations as related to the decision to initiate sexual activity. As such, our models focus on factors that may affect how a child evaluates his/her educational goals in making the decision to initiate sex. As previous research suggests (Paul et al., 2000), we believe that higher maternal intelligence is likely associated with a family environment where educational goals are given priority, but also likely indicates the presence of a role model for attaining educational goals. Family level income, which may reflect how accessible a child sees long-term educational goals such as a college degree, will also be included. Intelligence at the individual level is also being considered. Above and beyond the effects of the family environment's encouragement of educational goals and the financial means to make such a pursuit feasible, the individual's capacity to

successfully pursue those educational ambitions is also an important factor when evaluating its position among life decisions. Gender and race will also be included in the model as covariates.

We believe that both individual and family-level factors will demonstrate significant influence on AFI, with measures of intelligence demonstrating the most prominent effects. It is likely that evidence of genetic influence on these factors will be apparent, given the previous research demonstrating its importance in factors relating to adolescent sexual behavior (Harden, 2013).

Methods

Sample

The National Longitudinal Survey of Youth 1979 (NLSY79) began in 1979 when a nationally representative sample of 14-22 year olds from 8,770 households was surveyed. These 12,686 individuals were surveyed annually until 1994 when a biennial schedule began, which continues to this day. Participants were surveyed on a variety of topics, covering labor force matters, intelligence, childcare, and more. The individuals from the NLSY79 are referred to within this paper as the Gen1 (First Generation) sample. Starting in 1986 the biological children of all the female respondents in the NLSY79 were surveyed as well, creating the NLSY79 Children and Young Adults (NLSY79-CYA) survey, which also continues on a biennial basis. The "children" subsample includes those age 15 or younger at the survey date, and are assessed through questionnaires of the mother and the child. When children surpass age 15, they become part of the "young adult" sub-sample and complete surveys modeled after those the

original NLSY79 sample received. These combined child and young adult samples are referred to as the Gen2 (Second Generation) sample within this paper.

Measures

Both samples were administered intelligence and/or cognitive performance tests. In 1980, when they were in late adolescence, the mothers were administered the full version of the Armed Services Vocational Aptitude Battery (ASVAB), a measure of knowledge and skills in areas including language and mathematics; a component of the ASVAB is the Armed Forces Qualifying Test (AFQT), designed to assess intelligence. The 5-13 year olds in the NLSY79-CYA sample were administered the Peabody Individual Achievement Test (PIAT), which provides separate subscale measures of reading recognition, reading comprehension, and mathematics ability. In addition, a Wechsler Memory for Digit Span test, which measures short-term memory for ordered number sets, was included for 7-11 year olds. The Peabody Picture Vocabulary Test (PPVT) was administered to 3-12 year olds. This test requires that children indicate which picture matches a sentence that has been read to them.

The NLSY79 respondents are asked at each interview about the total net family income. This variable will be used to evaluate the family level financial status, as it may affect the accessibility of educational and career outcomes for the children in the study. The NLSY79-CYA sample assessment includes a set of measures evaluating the home environment on variables affecting cognitive stimulation and social support, called the Home Observation for Measurement of Environment (HOME). These measures are age specific, ranging from birth to age 14, and include items to evaluate the frequency and manner of parent-child interactions, the number of age-appropriate, cognitively

stimulating books and toys the child has, the number of outings the child goes on, intentional cognitive stimulation such as being read to or being taught to count, self-care, and discipline. Individuals age 13 and older are also asked during the self-administered portion of the survey to report the age at which they first engaged in sexual intercourse.

Outcome Variable

For the modeling phase of this study, a single measure of age of first intercourse (AFI) is needed. AFI was asked of participants in the child sub-sample from 1988 through 2000. Young adults were asked this question from the 1994 survey on. Occasionally, very young AFI values were reported, which most likely reflected either a misunderstanding of the question, response duplicity, or involuntary sexual activity. Response values under age 10 were recoded as missing. A vast majority of respondents reported an AFI (*N* = 4843, 85.31%); most had only given one AFI response over the 12 surveys this question could have been asked. Once respondents provided a value for AFI future surveys skipped the question, with occasional exceptions (for example, one respondent had answered this question five times). Despite the sometimes large number of interview occasions, most respondents were only asked to report an age value for AFI during one assessment (N = 5112, 90.05%) and almost all respondents who had answered this question more than once gave values within one year of each other (*N* = 5374, 94.66%). The greatest difference between response values was nine years (*N* = 1). For the whole sample, the average AFI across responses was 15.78 years (*SD* = 2.13, *N* = 5677). Excluding those whose responses differed by more than three years made little difference (*M* = 15.82, *SD* = 2.14, *N* = 5526). The rounded average value of all AFI

values for each respondent was used as their AFI value. This treatment is consistent with previous AFI research using the NLSY data sets (Rodgers, Rowe, & Buster, 1998). Only two respondents in our final sample who provided an AFI response had reported a value over the age of 25. As such, and because differences in AFI beyond age 25 seem likely to be less influenced by family level effects, all AFI values over the age of 25 were recoded to 25 to reflect membership in an age 25 and up group.

The mothers of the CYA children were between 45 and 53 years of age during the 2010 interview, and had likely finished childbearing. That being said, there are still a number of very young participants in the 2010 NLSY79-CYA survey who have not yet aged into the typical range when first intercourse would occur. All individuals who were less than 10 years old at the 2010 interview are excluded from the study.

Individual Level Variables

As described above, the NLSY79-CYA group was measured on intelligence using the Wechsler Memory for Digit Span test (DS), the PPVT, and the PIAT subtests, which include subscales addressing mathematics, reading recognition, and reading comprehension. A single factor intelligence measure is desirable for both theoretical and modeling purposes. Studies on intelligence indicate that a variety of intelligence measures all consistently correlate with a general measure of intelligence, referred to as *g*. Further, such a general measure of intelligence is stable throughout adulthood, and using a single latent measure of intelligence allows researchers to capitalize on this consistency (Davis, Haworth, & Plomin, 2009; Haworth et al., 2010).

In order to obtain a single measure for intelligence for each child in the study, and thereby capture the effect of individual-level general intelligence in our models, the

age-standardized scores for each intelligence scale were averaged together for each child. These individual averages for PPVT, DS, and the PIAT subscales were then combined using factor analysis as a measurement model, so that a single measure of intelligence will be available for the multilevel model. A single factor solution best fit the data based on examination of skree plots and eigenvalues (Table 1), and each variable loaded positively on the factor. Table 2 provides the factor loadings and the proportion of each variable's variance explained by the factor. The resulting factor score for each individual will be used in the multilevel models. Race and gender will also be included as an individual-level variable, as previous research indicates there are significant differences in AFI between males and females and across races.

Gen1 Family Level Predictor Variables

Maternal intelligence was measured using the percentile scores of the AFQT scale from the ASVAB and will be used in the family level model. The HOME assessment offers a variety of measures in the NLSY79-CYA, including age-specific composite measures for the cognitive stimulation and emotional support subscales, as well as a total score for all items and a single age-standardized score for all participants. To obtain a single score for each individual, all their raw scores were averaged across all measurements occasions. Only scores before the age of ten were used to ensure that no measurements were included that might have occurred after engaging in first intercourse. The average of these individual composite HOME scores for the entire Gen2 sample was 186.42 (N = 10329, SD = 32.07). To obtain a family-level value for this variable all siblings' composite HOME scores were averaged together to provide an

aggregate score for all children in the household. Average HOME values varied little across most sibling pairs (average difference = 17.66, *SD* = 18.43).

Maternal reports for family net income over the years will be averaged to form a single, family-wide measure of income. Similar to the calculation of a family-wide HOME variable, for each child all reported values for net family income from age 10 or below will be averaged across all years, and siblings' means will then be averaged together to provide a family level value.

Genetic Relatedness

Included in some models are measures of genetic relatedness. Although levels of sibling relatedness were not originally distinguished in the NLSY datasets, the 2006 and later administrations ask direct questions regarding the type of sibling relationship between Gen1 household members and between siblings in the Gen2 sample. Previous projects (Rodgers, 1996) had inferred the relationships between siblings in both the Gen1 and Gen2 datasets using information contained in the datasets; these kinship links have been used successfully in a number of projects (Mendle et al., 2009; Rodgers et al., 2008; Rodgers, Rowe, & Li, 1994; Van Hulle et al., 2007). More recently, a project by Rodgers, Beasley, Bard, & Meredith (2012) made use of both the new explicit questions in the 2006 and later surveys, as well as the previously defined implicit links, to create an updated algorithm linking family members within and between the Gen1 and Gen2 datasets. As all Gen2 children are the biological children of Gen1 mothers, they therefore share an average of 50% of their genes with their mothers (giving an R value, or coefficient of genetic relatedness, of .50). Full siblings share an average of 50% (R = .50) of their genes and half siblings share an average of 25% (R = .25). The

algorithm was used to define the R value for Gen1 mother/aunt dyads (Gen1R). The R value describes the genetic relationship (i.e. average percent of genes shared) between Gen2 sibling pairs (SibR).

Sub-Sample

Because of the structure of our models, it was necessary to only include in the sample individuals who came from complete family units. This meant that each individual had to have at least one sibling and a pair of cousins born to a single aunt, all of whom had non-missing data for all predictors in the model, including the Gen1R and SibR measures. This strict inclusion criterion was necessary as any missing data (except for the AFI variable) would result in exclusion from model calculations. When multiple family units with non-missing data were available from a family only the first available family unit was used. Because some of our models use kinship data between the Gen1 mother/aunt pairs and between the sibling pair within a Gen2 family, we could only accommodate one family at each level. The grouping structure within the models necessitates that only one R coefficient could be associated with each Gen1 and each Gen2 family.

The final sample of 876 individuals used in the multilevel models included 647 individuals with non-missing AFI values (74%, M = 15.80, SD = 2.19). Missing AFI values will be handled as censored data within discrete time survival analysis incorporated into the multilevel model. This was necessary as excluding those with no AFI data would necessarily bias results, and the incorporation of survival analysis allows for those who have censored data on AFI to be included in the model. The 876 Gen2 subjects in the final sample came from 219 family units. The 219 Gen1 sisters in

these family units included nine cousin pairs (R = 0.125), twenty half-sibling pairs (R = 0.25), 189 full-sibling pairs (R = 0.50) and a single pair of monozygotic twins (R = 1.00) at the Gen1 level. The 438 Gen2 sibling pairs included 134 half-siblings, six ambiguous-sibling pairs (there was uncertainty as to whether they shared the same biological father, so their R value of 0.375 was derived by averaging that of full siblings and half siblings), 295 full-sibling pairs, and three sets of monozygotic twins. All predictor variables were grand mean centered across the final sample before inclusion in the models for ease of interpretation--when all predictors are grand mean centered the intercepts represent a participant with average values on each of the predictors (Singer & Willet, 2003).

Design

This study utilizes a children of siblings design (Lahey & D'Onofrio, 2010; Mendle et al., 2009; Rodgers, Bard, Johnson, D'Onofrio & Miller, 2008) to help untangle the direct influence of predictor variables in our model from the confounding effects of selection influences. By evaluating the children of Gen1 sisters, who vary in their degree of genetic relatedness but share family environmental histories and genetics, we are controlling for these confounding factors shared across the mom/aunt dyad (Dick, Johnson, Viken, and Rose, 2000). Because siblings at the Gen1 level share these selection influences, differences between their children's AFI related to maternal-level intelligence or maternal-level AFI values would indicate associations with variations on these maternal traits within the extended family. If, however, the selection effects that associate a child's AFI with maternal level intelligence or intercourse timing are responsible, we would expect to see similar values across the children of both Gen1

sisters due their shared genetic or environmental factors, regardless of their level of exposure to the maternal level trait. Because these Gen1 sister-sister pairs differ in genetic relatedness, we can leverage these differences by including interaction terms between their intra-familial deviations on the predictors and a measure of the amount of shared genes between them. Significant interactions with the R terms can help pinpoint whether genetic forces are the cause of significant Gen1 level effects by demonstrating the differences between children who have varying levels of exposure to familial intelligence varies as a function of the genetic relationship within families. If the association were all or mostly genetic in origin, we would expect to see an interaction that describes greater individual-level (maternal-level) predictive power among siblings (cousins) who share less genetic ancestry. In other words, if a strong gene-environment correlation was at play, individual-level (maternal-level) deviation scores should have little or no additional predictive impact, over that of the maternal-level (extended-family-level) predictors, on the outcome among siblings (cousins) who share greater amounts of genetic material.

Analyses

Descriptive means analysis. Similar to the work by Mendle et al. (2009), descriptive means comparisons were first conducted to test the patterns of AFI values based on relative maternal intelligence. For this comparison, each Gen1 mother was marked as either being above or below average on the AFQT. The AFI of children will be compared based on whether they came from Gen1 families where both mothers were above average for intelligence (Group 1), their own mother was above average for intelligence but their aunt was below average (Group 2), their mother was below

average but their aunt was above average for intelligence (Group 3), and where both their mother and aunt were both below average for intelligence (Group 4) as measured by the AFQT. The averages of these groups can then be compared to patterns that would be expected if maternal intelligence directly influences the child's AFI, as well as patterns that would indicate the possibility of family-level influence from genetic or environmental factors that influence both intelligence and AFI across the family. If maternal intelligence influences a child's AFI, providing some sort of protective buffer within the family environment against early first intercourse, then we would expect to see the averages of Groups 1 and 2 to be approximately the same, and Groups 3 and 4 to be lower than the averages of Groups 1 and 2 but similar to each other. If, however, we find that the AFI averages for the groups appear to descend with each step from Group 1 to Group 4, reflecting the amount and proximity of above average intelligence in the family at the Gen1 level, then this may indicate that some shared genetic or family environmental protective effect on AFI emerging from the mother's family (Gen1), and shared across related individuals with above average intelligence in the family (Mendle et al., 2009). If selection effects are at play, then when there is above average intelligence in the extended family but not for the mother, some amount of protection could be provided to children by virtue of the background (genetic or environmental) they share with their above average aunt, but we would expect to see less protection than when this high intelligence is found in the child's own mother because of the greater proportion of background being shared with the individual demonstrating the higher intelligence.

As there is a great deal of missing data and many individuals who may not have aged into a first intercourse event at the time of the study, it would not be appropriate to perform statistical tests on this information. Further, by looking at only those with reported AFI we are necessarily biasing our results downward, as those who have reported an AFI would report lower ages than their same-age counterparts who have not yet experienced intercourse. As such, in the preliminary analysis, the means will be compared without drawing statistically backed conclusions.

Multi-level hazards models. Next, a series of multilevel, discrete-time hazards models will be fit to the data (Singer & Willett, 2003). Our models use logistic regression with intercepts randomly varying at levels two and three. First, a simple model including all predictor variables will be used to model the child's risk of first intercourse occurring in each time period from age 10 to age 25. The child's factor score across all five intelligence measures, their gender, and their race comprise the individual level predictors. At the family level Maternal AFI and AFQT score were used, as well as the averages for HOME scores and income reported for the children within the household.

The next model will include a third level of effects--those at the Gen1 family level. In addition to the predictors included in the first model (excluding the child's intelligence factor score and the mother's AFQT score, as both are incorporated in other covariates), the average intelligence measure across the mother/aunt dyad (MomAuntAvg), the mother's AFQT score's deviation from the MomAuntAvg measure (MomAuntDiff), and the deviation of the child's standardized intelligence factor from their mother's standardized AFQT score (MomChildDiff) are also included in order to

test the effects of family-level intelligence factors, both within and beyond the nuclear family, as well as the unique contribution of the child's own intelligence. As such, this model will be referred to as the maternal intelligence model. This model utilizes an extension of the children of siblings design by incorporating intelligence at the Gen1 family level as well as the effects of the mother's own intelligence as a force beyond that of the Gen1 family effects. Incorporating an interaction between mother's deviation from the Gen1 family average and the R value between her and her sister allows us to investigate whether the predictive power of the mother's intelligence changes as a function of how closely related the Gen1 pair is.

Another three level model, this one focusing on the effects of child-level intelligence, will also be fit. Instead of using mom/aunt intelligence averages for the extended family cluster effect, the average of the standardized intelligence factor scores of all four children in the family unit will serve as the level-three intelligence measure (Gen2IntellAvg). The average factor score of each child and their sibling (NucFamAvg) serves as a measure of nuclear family intelligence level, and the difference between this average and the family-wide intelligence measure (Gen2NucFamDiff) will serve to test the independent effects of household level intelligence on AFI. The deviation of the child's own factor score from the nuclear family average will serve as the level-one intelligence measure (NucFamChildDiff). We will call this model the child intelligence model. By incorporating interactions between these effects and the appropriate R coefficients it provides a framework for testing the effects of any shared forces related to intelligence coming from the Gen1 family unit, the unique effects of the sibling pair, and the effects of the child's own intelligence apart from family effects at either level.

In the basic model, the use of maternal AFQT as a predictor allows us to evaluate between-family variance based on this variable, just as the use of individual intelligence factor scores in the child intelligence model evaluates the effect of individual level variance in AFI related to child intelligence. With the use of within-family centered measures in the maternal intelligence and child intelligence models we now have within-family measures, enabling us to evaluate the effect of within-family variance on our outcome.

Both the maternal intelligence effects model and the child intelligence effects models will be fit including and excluding interactions between the intelligence deviation measures and the coefficient of genetic relatedness at the corresponding family level. In the maternal intelligence effects model this includes an interaction between Gen1R (the coefficient of genetic relatedness between the mom and aunt) and MomAuntDiff, as well as an interaction between SibR (the sibling pair's R value) and MomChildDiff. For the child intelligence model, the Gen1R will be tested for an interaction with Gen2NucFamDiff and SibR will be tested with NucFamChildDiff. As is standard practice, because the Gen1R and SibR effects will be included in the model in interactions with other variables, they will also be included in the model apart from any interactions effects. A number of variations of each model will be fit (though not all are reported) so that consistent patterns can be identified.

Results

Descriptive Means Comparisons

Table 3 presents the results of the descriptive means comparisons. This pattern of results, demonstrating an increase in AFI based on the number and proximity of

persons with above average intelligence in the child's family at the Gen1 level, suggests that some selection effects may be at play within our sample. This effect may be the result of a suite of shared genes or common environmental factors affecting both maternal/aunt intelligence and the child's decision on when to initiate sexual activity. We will further investigate these results statistically with our hazards modeling analysis.

Multilevel Hazards Models

The results of the three multilevel hazard models are presented in Table 4. Many variations of the models were run to evaluate the importance of the covariates in the models and overall trends in the results. The results presented in Table 4 focus on models including all of the listed covariates and, in all but the basic model, the cluster average effects discussed earlier. Table 5 gives the results of these same models with the addition of interaction terms between genetic relatedness variables and the deviation effects at the corresponding family level as discussed in the Methods section. Table 6 illustrates the time effects in the underlying hazards model. These parameter estimates assess the risk of event occurrence, here the initiation of sexual intercourse, within each time period of the model.

Figure 1 illustrates the hazard function defined by these estimates. Table 6 and Figure 1 are both based on the fitted hazards models from the basic model. As all predictors are standardized, the figure then represents the expected hazards for an individual with average values on all predictors. The hazard values assess the risk of event occurrence in each time period for those still at risk for an initial event (i.e., the "risk-set"). The figure illustrates that risk of AFI steadily increases across time, peaking in early adulthood. Within this dataset no AFI values were reported or predicted to

occur at age 23, so ages 23 and over were collapsed into one group. Trends indicate that risk peaks at around age 18 and then virtually levels off.

The covariates for mother's AFI, race, and gender, were significant in all of the models. In any time period, children whose mothers had an AFI later than the mean by one standard deviation were at only 73-78% the risk of first intercourse occurring (depending on the model referenced) as those whose mothers had an average AFI value. Likewise, females were at an approximately 30% lower risk of event occurrence within any time period than were males. Three categories were possible for race, with those in the non-Black, non-Hispanic category showing the lowest risk for event occurrence and the Black racial group as the highest risk. Non-Black, non-Hispanic individuals served as the baseline group. The families' averages on the HOME scales were also significant, such that those with higher HOME scores have a lower risk of event occurrence, but only in models that did not include interactions between deviation scores and the R values. Figures 2 thru 5 illustrate the effects of each significant variable on the hazard function of the basic model.

Surprisingly, no intelligence-related variables were found to be significant in any of the models. In the basic model the child's factor score among the intelligence measures failed to reach significance, despite the large amount of previous research implicating one's own intelligence in determining AFI. In the basic model, all predictors, other than the factor measure of intelligence, showed the predicted protective effects against early AFI. Only mother's AFI, the family HOME average, gender, and race reached statistical significance. Models were also run testing for interactions between intelligence and gender, to assess whether females' higher cost

associated with pregnancy risks caused a greater delay in AFI for smarter females relative to their male counterparts. This interaction was not significant, however, in the basic model or in models fit containing only gender, the child's intelligence factor score, and their interaction. As such, this effect was not pursued further in other models and detailed results are not presented.

No family level intelligence means, deviation, or interaction effects at any level within the maternal intelligence model approached significance. Within the child intelligence model excluding interactions with R coefficients, however, both the Gen2NucFamDiff and the NucFamChildDiff values approached significance ($p = 0.07$, $p = 0.06$, respectively). Although having a higher average intelligence within the nuclear family provided the expected protection from early AFI, the almost significant effect of being smarter than one's sibling actually puts an adolescent at higher relative risk for AFI occurring in any time period.

Further, analyses were conducted to verify the direction of the relationship between higher within-family intelligence and earlier AFI, as the direction of this relationship is counter to the previous research suggesting that greater intelligence is related to later AFI (Harden & Mendle, 2011; Kirby, 2002; Unger, Molina, & Teran, 2000). First, the averages for the more and less intelligent siblings among pairs were computed. Their comparison supported this effect. Smarter siblings had an average AFI of 15.72 ($SD = 2.11$, $N = 321$) while the average for the less-smart siblings was 15.87 ($SD = 2.28$, $N = 326$), giving a difference of -0.14 years ($SD = 2.67$, $N = 263$), or about 51 days. When this comparison was conducted on the entire Gen2 sample (excluding those less than age ten), to ensure that the effect was not an artifact of selection into our

sub-sample, we essentially found no differences between the smarter and less smart siblings. Smarter siblings reported an average AFI of 15.65 years (*SD* = 2.14, *N* = 4822), whereas the less-smart sibling reported an average of 15.68 years (*SD* = 2.17, *N* = 4754). This difference was only -0.01 years (about two days), but still illustrates that intelligence differences within family do not demonstrate the robust smarter-means-later effect consistently reported in studies focusing on AFI for unrelated samples of subjects.

The NLSY-CYA still has a number of individuals who have not aged into our sample who are born to relatively older mothers. To ensure that effects found in our models were not the result of bias from sample restriction, a number of comparisons were made between mothers who were included in our sample and the entire Gen1 female sample. This analysis indicated that full-sample females (*N* = 5607, *M* = 17.44, *SD* = 2.12) and sub-sample mothers (*N* = 876, *M* = 17.51, *SD* = 2.19; *d* = 0.03) were similar in their reported AFI. The sub-sample mothers (*N* = 876, *M* = 36.54, *SD* = 29.01), however, showed slight differences from full-sample females (*N* = 5939, *M* = 42.17, *SD* = 28.13; *d* = 0.20) in terms of their AFQT measure. When the Gen2 subsample was compared to the total sample we found that they did not differ substantially on the intelligence factor measure (*N* = 876, *M* = -0.37, *SD* = 0.96; *N* = 7826, *M* = -0.01, *SD* = 0.97; respectively; *d* = -.37), the family HOME average (*N* = 876, *M* = 188.42, *SD* = 27.34; *N* = 8297, *M* = 189.05, *SD* = 27.67; respectively; *d* = 0.02), family income (*N* = 876, *M* = 28,752, *SD* = 31,291; *N* = 8306, *M* = 27,523, *SD* = 33,525; respectively; *d* = 0.04), or AFI (*N* = 647, *M* = 15.80, *SD* = 2.19; *N* = 5677, *M* = 15.78, *SD* = 2.13; respectively; *d* = -0.01).

We were particularly interested in the effects of intelligence at both the individual and maternal level, and both were significantly correlated with AFI (see Table 7). Further, separate, zero-order discrete-time survival models containing only one of these variables indicated each was a significant predictor of AFI when other variables were not included. As such, a series of models were fit with different sets of predictors to determine which additional predictors yielded the intelligence variables no longer significant.

Models containing the child's intelligence factor score were evaluated in this manner by adding each of the other predictors individually and determining in which models the child's intelligence factors score was no longer significant. Only in two predictor models containing the mother's AFQT score or the family HOME score along with the child's intelligence was their intelligence no longer a significant predictor of their AFI. The ability of the HOME score to supersede child intelligence in prediction strength was not surprising, given its significance in the final models. Maternal AFQT, however, was not significant in any of the models containing the entire set of predictor variables.

Our attention then turned to what variables were needed to reduce the prediction strength of the maternal AFQT score to a non-significant level. A similar set of model runs were done containing AFQT and each other individual predictor variable, but no individual variable's inclusion was enough to remove the significance of the AFQT predictor. Models containing every possible combination of another two variables again yielded no results where AFQT was non-significant. Models were then run containing combinations of additional sets of three variables, and only when maternal AFI, the

average HOME score, and race were included was AFQT no longer statistically significant.

Maternal AFI was consistently a significant predictor in our models. We then decided to evaluate a model including Gen1 cluster averages (Gen1AfiAvg) for maternal AFI and the deviation of the child's own mother from that cluster average (MomGen1AfiDiff). Similar to the analyses for maternal intelligence, an interaction between the maternal deviation from the cluster average and the Gen1 R value was included to determine if genetic influence is detectable in our model. The results from this analysis are presented in Table 8. As in previous models, the family HOME average, race, and gender remained significant predictors in this analysis. Interestingly, the Gen1AfiAvg predictor was the only significant predictor for maternal AFI measures, suggesting that the influence of maternal AFI found in previous models was due to an effect at the entire Gen1 level that passes through the measures of maternal AFI, rather than an effect specific to the mother's AFI itself. No evidence of significant genetic influence related to maternal AFI was found, but power to detect such an effect was low in our subsample.

Discussion

Although several predictors, both at the family and individual level, were found to have significant influence on AFI, by far the most surprising result was the failure to find significant influence of intelligence, at any level, on AFI. This may have been because of low power resulting from small sample size. Because of the unique structure of our models it was difficult to find entire family units eligible for inclusion in the model, as any missing data (other than AFI) anywhere in the family unit necessitated

omission. A repeat of this analysis, after more individuals within the sample have had the opportunity to age into the selected sample and initiated intercourse, even among late bloomers, may (or may not) show different results.

In the child intelligence model, the Gen2NuclFamDiff and the NucFamChildDiff values approached significance but did not reach that threshold. Had these effects been significant, especially without a significant influence from the Gen2IntellAvg measure, it would have indicated that household and individual intelligence affect AFI, but that extended family intelligence is not notably influential. Our results suggest that there is not a strong family-wide gene-environment correlation between intelligence and AFI, as such an influence would have shown up in the interaction terms with the measures of genetic similarity. Again, low statistical power might have affected our ability to find such effects. Most surprisingly, however, is that a Gen2NucFamDiff score indicated higher intelligence among the children protects against early AFI (odds ratio = 0.79), but a higher NucFamChildDiff score actually suggested an *earlier* AFI (odds ratio = 1.23). The surprising direction of this latter effect, even though it was not significant, prompted follow-up analyses to ensure the validity of this effect within our subsample. This effect did not was persist across all our full-sample checks; however, even in these full sample inspections, we did not see evidence for the usual smarter-means-later AFI effect. Instead, the full sample checks suggested (essentially) no differences in AFI among smarter and less-smart siblings.

This pattern suggests that extended family intelligence influence is not important, whereas nuclear family and individual intelligence may be. This result contrasts with the findings of the descriptive means comparison, where the pattern of

AFI among children with varying levels of aunt and maternal intelligence suggested that the high intelligence of extended family members may provide some protection against early AFI. However, the descriptive means analysis necessarily only included individuals who had provided an AFI value, and therefore had a downward bias in the AFI values. This difference in results may then have been because of the exclusion of young children in our sample, and older children who have not initiated intercourse and will therefore exhibit later AFI than children of the same age who were eligible for inclusion in the descriptive means analysis.

The lack of significant intelligence effects must be considered in the context of the models used. Every predictor, including cluster averages and deviation scores, was significantly correlated with AFI in zero-order correlations (Table 7). In the context of the multilevel models, however, where other predictors and time elements are included in each model, intelligence related measures fail to reach statistical significance. This may mean that maternal AFI, a significant predictor in every model, along with the average HOME score, do more to predict AFI than intelligence at any level. It may be that factors affecting both maternal AFI choices and the kind of environment mothers provide to their children, in part measured by the HOME scale, are responsible for the effects typically seen between intelligence and AFI. The significant effects of race and gender have been found in previous research, and their significance within models including other predictors is, therefore, not surprising. Gender and intelligence, however, did not have a significant interaction effect on AFI.

Follow up analysis using Gen1 averages for AFI and maternal deviations from this cluster mean were used to further investigate the source of the influence maternal

AFI has on a child's AFI. Our findings suggest that the average Gen1 AFI is more predictive of a child's AFI than the mother's AFI itself, suggesting that a typically unmeasured ancestral variable of familial AFI is responsible for the patterns seen in our other models, and perhaps the relationships between maternal and child AFI cited in other research. A great deal of previous research implicates genetics in the timing of first intercourse (see Harden, 2013 for review), and would be a reasonable explanation for the prevalence of family-level effects on this outcome. However, the interaction between the specific timing of maternal AFI and the Gen1 R coefficient failed to reach significance within our model when extended family average for maternal AFI was accounted for. This may be due to low power from our small sample size, discussed above, and would warrant exploration in other contexts with better power.

The significance of the average HOME measure in most models and the importance of maternal AFI, later attributed to latent extended-family effects of AFI in our follow-up analysis, highlight the importance of family-level influence on the choice to initiate intercourse. The differential risks for early AFI based on gender and race found here have been established in previous research time and time again. Maternal AFI has previously been established as a predictor for a child's AFI (Newcomer & Udry, 1984), and may relate to her children's AFI simply as a proxy measure for maternal values that relate to sexual decision making. The last model discussed in this paper suggests that this effect may actually be due to family level effects on intercourse decisions from the Gen1 level, which may reflect cultural influences that then affect how a mother raises her children. A mother who was part of a family with later AFI may have been motivated to refrain from sexual activity for personal, moral, or cultural

reasons. The effect may have also passed through the elements measured in the HOME scale. This mother is likely to use that same moral framework in raising her children and foster a similar home environment, imparting the same effects on her offspring. Though we are not able to directly test this ad-hoc hypothesis in this set of models, in the absence of any effects of intelligence or evidence of genetically-linked influences on AFI, the idea of a transmission of moral motivations seems a reasonable explanation for these results. Such a moral framework or household environment may be a more important factor in sexual decision making than intelligence itself, as is suggested in the analysis presented here. Genetic effects should not be dismissed, however, given the prevalence of the support for its influence on sexual behavioral outcomes such as AFI (see Harden, 2013 for review) and the relatively small sample size used here.

Future studies with samples that have fully aged through the opportunity for first sex, especially with the inclusion of direct measures for attitudes on sexual permissiveness and the importance of education, would help to answer some of the questions left from this set of analyses. Larger samples would also help to identify underlying genetic effects that may have gone undetected in our small sample. Although the influence of family level effects was clear throughout our analyses, further study should include intelligence differences between siblings within families and the within-family effect of intelligence on AFI.

References

Boislard P, M.-A., Poulin, F. (2011). Individual, familial, friends-related and contextual predictors of early sexual intercourse. *Journal of Adolescence, 34,* 289-300.

Carvajal, S. C., Parcel, G. S., Basen-Engquist, K., Banspach, S. W., Coyle, K. K., Kirby, D., Chan, W. (1999). Psychosocial predictors of delay of first sexual intercourse by adolescents. *Health Psychology, 18*(5), 443-452.

D'Onofrio, B. M., Van Hulle, C. A., Waldman, I. D., Rodgers, J. L., Harden, K. P., Rathouz, P. J., & Lahey, B. B. (2008). Smoking during pregnancy and offspring externalizing problems: An exploration of genetic and environmental confounds. *Development and Psychopathology, 20,* 139-164.

Davis, O. S. P., Haworth, C. M. A., & Plomin, R. (2009). Dramatic increase in heritability of cognitive development from early to middle childhood: An 8-year longitudinal study of 8,700 pairs of twins. *Psychological Science, 20*(10), 1301-1308.

Dick, D. M., Johnson, J. K., Viken R. J., & Rose, R. J. (2000). Testing between-family associations in within-family comparisons. *Psychological Science, 11*(5), 409-413.

Ellis, B. J. (2004). Timing of pubertal maturation in girls: An integrated life history approach. *Psychological Bulletin, 130*(4), 920-958).

Halpern, C. T., Joyner, K., Udry, R., & Suchindran, C. (2000). Smart teens don't have sex (or kiss much either). *Journal of Adolescent Health, 26,* 213-225.

Harden, K. P. (2012). True love waits? A sibling-comparison study of age at first sexual intercourse and romantic relationships in young adulthood. *Psychological Science, 23*(11), 1324-1336.

Harden, K. P. (2013). Genetic influence on adolescent sexual behavior: Why genes matter for environmentally oriented researchers. *Psychological Bulletin.* Advance online publication.

Harden, K. P., & Mendle, J. (2011). Why don't smart teens have sex? A behavioral genetic approach. *Child Development, 82*(4), 1327-1344.

Harden, K. P., Mendle, J., Hill, H. E., Turkheimer, E., & Emery, R. E. (2008). Rethinking timing of first sex and delinquency. *Journal of Youth and Adolescence, 37,* 373-385.

Haworth, C. M. A., Wright, M. J., Luciano, M., Martin, N. G., de Geus, E. J. C., van Beijsterveldt, C. E. M., Bartels, M., Posthuma, D., Boomsma, D. I., Davis, O. S.

P., Kova, Y., Corley, R. P., DeFries, J. C., Hewitt, J. K., Olson, R. K., Rhea, S.-A., Wadsworth, S. J., Iacono, W. G., McGue, M., Thompson, L. A., Hart, S. A., Pertill, S. A., Lubinski, D., & Plomin, R. (2010). The heritability of general cognitive ability increases from childhood to young adulthood. *Molecular Psychiatry, 15*(11), 1112-1120.

Lahey, B. B., D'Onofrio, B. M. All in the family: Comparing siblings to test causal hypotheses regarding environmental influences on behavior. *Current Directions in Psychological Science, 19*(5), 319-323.

Mendle, J., Harden, K. P., Turkheimer, E., Van Hulle, C. A., D'Onofrio, B. M., Brooks-Gunn, J., Rodgers, J. L., Emery, R. E., Lahey, B. B. (2009). Associations between father absence and age of first sexual intercourse. *Child Development, 80,* 1463-1480.

Newcomer, S. F., & Udry, J. R. (1984). Mother's influence on the sexual behavior of their teenage children. *Journal of Marriage and Family, 46*(2), 477-485.

Kirby, D. (2002). Antecedents of adolescent initiation of sex, contraceptive use, and pregnancy. *American Journal of Health and Behavior, 26*(6), 473-485.

Mathews, C., Aarø, L. E., Flisher, A. J., Mukoma, W., Wubs, A. g., & Schaalma, H. (2009). Predictors of early first sexual intercourse among adolescents in Cape Town, South Africa. *Health Education Research, 24*(1), 1-10).

Paul, C., Fitzjohn, J., Herbison, P., & Dickson, N. (2000). The determinants of sexual intercourse before age 16. *Journal of Adolescent Health, 27,* 136-147.

Plomin, R., DeFries, J. C., Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Program, 84*(2), 309-322.

Raffaelli, M., Crockett, L. J. (2003). Sexual risk taking in adolescence: The role of self-regulation and attraction to risk. *developmental Psychology, 39*(6), 1036-1046.

Rodgers, J. L. (1996). Sexual transitions in adolescence. In Graber, J. A., Brooks-Gunn, J., & Petersen, A. C. (Eds.), Transitions Through Adolescence: Interpersonal Domains and Context (pp. 85-110). Location: Mahwah, New Jersey.

Rodgers, J. L., Bard, D. E., Johnson, A., D'Onofrio, B. M., & Miller, W. B. (2008). The cross-generational Mother-Daughter-Aunt-Niece design: Establishing validity of the MDAN design with NLSY fertility variables. *Behavior Genetics, 38,* 567-578.

Rodgers, J. L., Beasley, W., Bard, D. E., Meredith, K. M. (2012, June). *Validity studies of the 2012 NLSY kinship links using height and weight.* Paper session presented at the meeting of the Behavior Genetics Association, Edinburgh, UK.

Rodgers, J. L., Harris, D., Vickers, K. B. (1992). Seasonality of first coitus in the United States. *Social Biology, 39,* 1-14.

Rodgers, J. L., Rowe, D. C., & Buster, M. (1998). Social contagion, adolescent sexual behavior and pregnancy: A nonlinear dynamic EMOSA model. *Developmental Psychology, 34*(5), 1096-1113.

Rodgers, J. L., Rowe, D. C., & Li, C. (1994). Beyond nature vs. nurture: DF analysis of nonshared influences on problem behaviors. *Developmental Psychology, 30,* 374-384.

Raffaelli, M., & Crockett, L. J. (2003). Sexual risk taking in adolescence: The role of self-regulation and attraction to risk. *Developmental Psychology, 39*(6), 1036-1046.

Schvaneveldt, P. L., Miller, B. C., Berry, E. H., Lee, T. R. (2001). Academic goals, achievement, and age of first intercourse: Longitudinal, bidirectional influences. *Adolescence, 36*(144), 767-787.

Singer, J. D., & Willett, J. B. (2003). *Applied Longitudinal Analysis: Modeling Change and Event Occurrence.* New York: Oxford University Press.

Stigum, H., Samuelsen, S.-O., Traeen, B. (2010). Analysis of first coitus. *Archives of Sexual Behavior, 39,* 907-914.

Udry, J. R., Kovenocik, J., Morris, N. M., van den Berg, B. J. (1995). Childhood precursors of age at first intercourse for females. *Archives of Sexual Behavior, 24*(3), 329-337.

Unger, J. B., Molina, G. B., Teran, L. (2000). Perceived consequences of teenage childbearing among adolescent girls in an urban sample. *Journal of Adolescent Health, 26,* 205-212.

Van Hulle, C. A., Rodgers, J. L., D'Onofrio, B. M., Waldman, I. D., & Lahey, B. B. (2007). Sex differences in the causes of self-reported adolescent delinquency. *Journal of Abnormal Psychology, 116,* 236-248.

Appendix A: Tables

Table 1

Eigenvalues and Variance Explained by Each Factor in the Intelligence Factor Analysis

Factor	Eigenvalue	Proportion Variance Explained
1	3.16	1.05
2	0.06	0.02
3	0.02	0.01
4	-0.09	-0.03
5	-0.13	-0.04

Table 2

Factor Loading Patterns for Intelligence Variables

Variable	Factor Loading	Variance Explained
Digit Span	0.59	0.34
PIAT Math	0.84	0.71
PIAT Reading Recognition	0.89	0.80
PIAT Reading Comprehension	0.90	0.81
PPVT	0.73	0.53

Note: N = 8341.

Table 3

Mean of Children's AFI by Maternal/Aunt Relative Intelligence

	Mean AFI	*SD*	*N*
Group 1. Mom and aunt above average on AFQT	16.91	1.71	187
Group 2. Only mom above average on AFQT	16.38	1.83	110
Group 3. Only aunt above average on AFQT	15.83	1.69	130
Group 4. Mom and aunt below average on AFQT	15.37	1.39	345

Table 4

Parameter Estimates and Odds Ratios from Multilevel Survival Models Excluding Interactions with Coefficients of Genetic Relatedness (N = 876).

Parameter	Basic	Maternal Intell.	Child Intell.
	Est. / *OR*	Est. / *OR*	Est. / *OR*
Mother's AFI	-0.29** / 0.75	-0.28** / 0.76	-0.31** / 0.73
Mother's AFQT	-0.13 / 0.88	---	-0.14 / 0.87
Family Income Avg.	-0.07 / 0.93	-0.06 / 0.94	-0.06 / 0.94
Family HOME Avg.	-0.19* / 0.82	-0.19* / 0.83	-0.17* / 0.84
Child's Race	-0.22** / 0.80	-0.21** / 0.81	-0.25** / 0.80
Child's Gender	-0.22** / 0.80	-0.22** / 0.80	-0.22** / 0.80
Child's Intelligence Factor Score	0.03 / 1.04	---	---
Gen1IntellAvg/Gen2IntellAvg		-0.12 / 0.89	0.04 / 1.04
MomAuntDiff/Gen2NucFamDiff		0.01 / 1.01	-0.24 / 0.79
MomChildDiff/NucFamChildDiff		0.04 / 1.04	0.21 / 1.23
-2 Log Likelihood	2728	2727	2721

Note: Parameter estimates are followed by odds ratios.
*Denotes effects significant at the $p < .05$ level.
**Denotes effects significant at the $p < .01$ level.

Table 5

Parameter Estimates and Odds Ratios from Multilevel Survival Models Including Interactions with Coefficients of Genetic Relatedness (N = 876).

Parameter	Maternal Intelligence		Child Intelligence	
	Est.	*OR*	Est.	*OR*
Mother's AFI	-0.25**	0.78	-0.27**	0.76
Mother's AFQT Score	---	---	-0.13	0.88
Family Income Average	-0.06	0.94	-0.07	0.93
Family HOME Average	-0.14	0.87	-0.13	0.88
Child's Race	-0.19**	0.83	-0.21**	0.81
Child's Gender	-0.23**	0.79	-0.22**	0.80
Gen1R	0.51	1.67	0.59	1.80
SibR	-1.12	0.33	-1.19*	0.30
MomAuntAvg/AllChildFactor	-0.15	0.86	0.02	1.02
MomDev/NucFamDev	-1.46	0.23	-0.38	0.68
MomDev/NucFamDev * Gen1R	3.05	21.33	0.28	1.32
MomChildDev/ChildFactorDev	0.15	1.16	-0.14	0.87
MomChildDev/ChildFactorDev * SibR	-0.32	0.73	0.83	2.29
-2 Log Likelihood		2720		2715

Note: Parameter estimates are followed by odds ratios.
*Denotes effects significant at the $p < .05$ level.
**Denotes effects significant at the $p < .01$ level.

Table 6

Parameter Estimates for Time Elements in Basic Model

Age	Parameter Est.	Fitted Odds	Fitted Hazard
10	-6.29	0.00	0.00
11	-5.83	0.00	0.00
12	-4.36	0.02	0.02
13	-3.14	0.07	0.07
14	-2.41	0.15	0.13
15	-1.96	0.23	0.19
16	-0.89	0.68	0.40
17	-0.34	1.19	0.54
18	0.25	2.14	0.68
19	0.49	2.72	0.73
20	-0.03	1.61	0.62
21	0.26	2.16	0.68
22	1.86	10.70	0.91

Note. These values are from a model containing centered predictors and therefore reflect a baseline group with average values on all predictor variables.

Table 7

Zero-Order Correlations between AFI and Predictor Variables (Among Those Reporting and AFI Event)

	1.	2.	3.	4.	5.	6.	7.	8.
1. AFI	1.00	0.28**	0.26**	0.23**	0.30**	0.15**	0.22**	0.20**
2. Maternal AFI		1.00	0.33**	0.28**	0.39**	-0.07*	0.07*	0.25**
3. Maternal AFQT			1.00	0.51**	0.57**	-0.06	0.49**	0.54**
4. Family Income				1.00	0.45**	-0.02	0.25**	0.34**
5. Family HOME					1.00	0.02	0.28**	0.54**
6. Gender						1.00	-0.02	0.02
7. Race							1.00	0.28**
8. Intelligence Factor								1.00

Note: Sample sizes for correlations involving AFI were $N = 647$. All other variables came from a complete subsample of $N = 876$.
*Denotes significance at $p < .05$.
**Denotes significance at $p < .01$.

Table 8

Parameter Estimates and Odds Ratios from Multilevel Survival Models with Cluster Analysis on Maternal AFI (N = 876).

Parameter	Maternal AFI	
	Est.	*OR*
Mother's AFQT Score	-0.07	0.93
Family Income Average	-0.08	0.92
Family HOME Average	-0.16*	0.85
Child's Race	-0.23**	0.79
Child's Gender	-0.22**	0.80
Gen1R	0.51	1.66
Gen1AFIAvg	-0.41**	0.66
MomGen1AFIDiff	0.30	1.35
MomGen1AFIDiff * Gen1R	-0.77	0.46
-2 Log Likelihood	2721	

Note: Parameter estimates are followed by odds ratios.
*Denotes effects significant at the $p < .05$ level.
**Denotes effects significant at the $p < .01$ level.

Appendix B: Figures

Figure 1

Hazard of AFI occurring at each age in Basic Model.

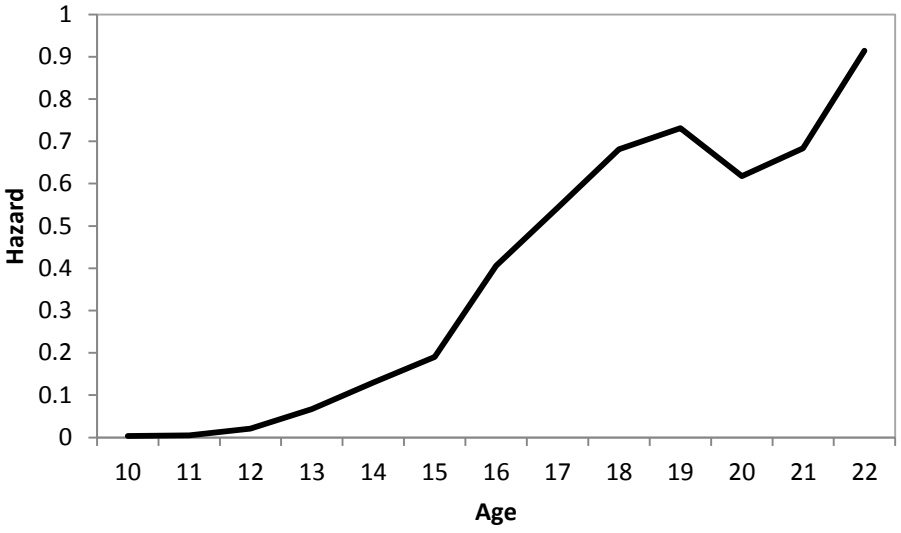

Figure 2

Change in hazard function of basic model resulting from variation in maternal AFI.

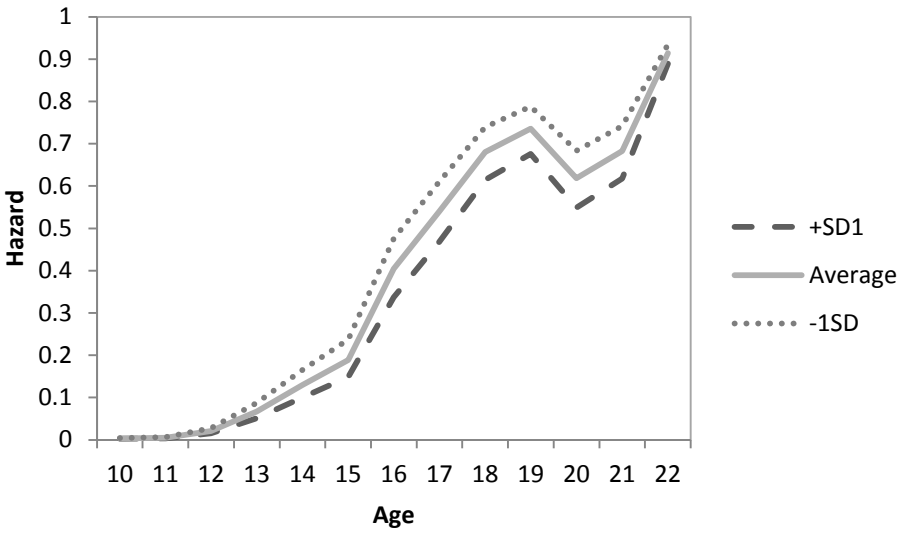

Figure 3

Comparison of hazard functions for males and females in the basic model.

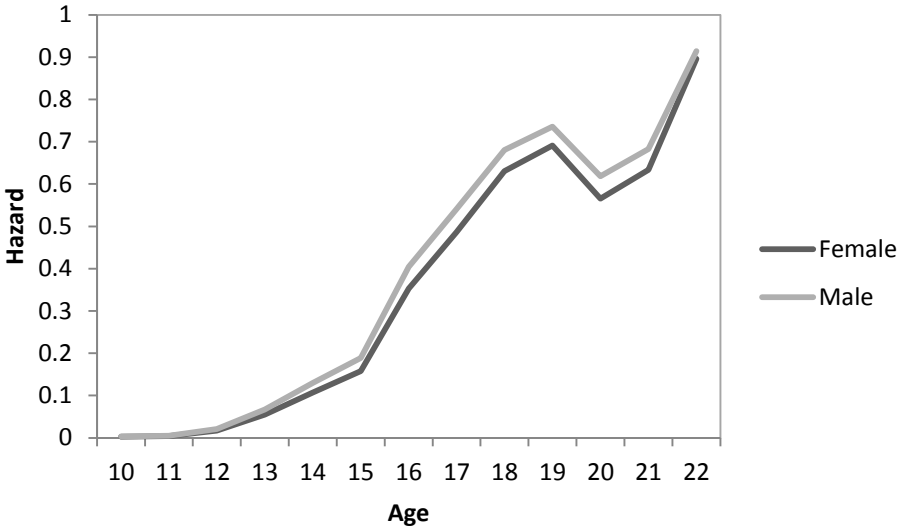

Figure 4

Comparison of hazard functions by race in basic model.

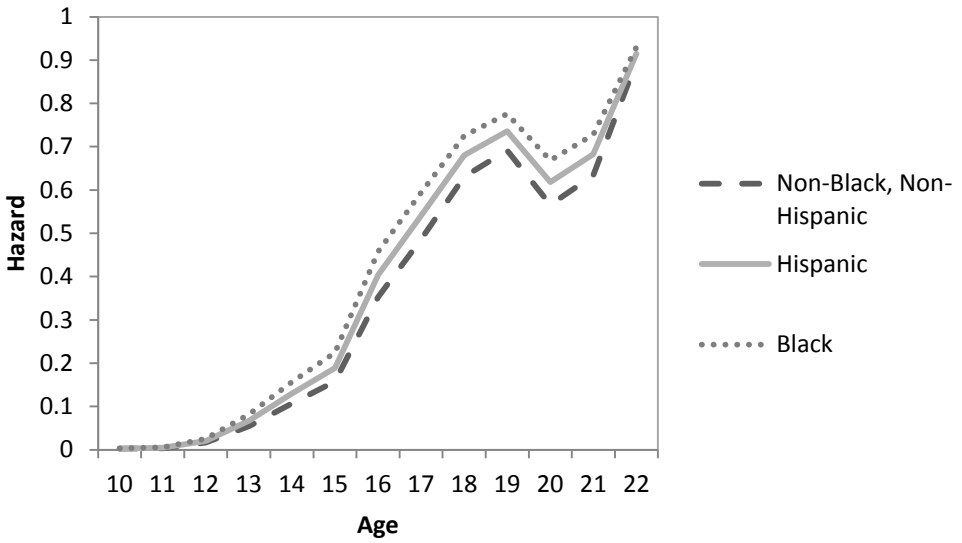

Figure 5

Change in hazard function of basic model resulting from varying HOME scores.