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Stronger Working Memory Reduces Sexual Risk taking in Adolescents, Even After Controlling for Parental Influences

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Abstract

This study examined the prospective influence of adolescent working memory (WM) on changes in impulsivity and sexual risk taking and assessed whether this relation could be explained by confounding effects of parental influences. Data from 360 community adolescents ($M_{age}=13.5\pm0.95$ years; 52% female; 56% non-Hispanic White; low-mid SES; recruited from Philadelphia area in 2004–2005) were analyzed using structural equation modeling to predict changes in impulsivity and sexual risk taking over a two-year follow-up, using baseline assessments of WM, parental monitoring, parental involvement, and socioeconomic status. Stronger WM predicted reduced involvement in sexual risk taking at follow-up, effects channeled through changes in impulsivity dimensions of 'acting without thinking' and 'inability to delay gratification'. Parental variables had a protective influence on adolescent impulsivity and risk involvement, but the effects of WM operated independently of parental influences.

Adolescence is the developmental period when sexual exploration and experimentation typically begins (Craver, Joyner, & Udry, 2003). As compared to adults, adolescents tend to have faster rates of turnover in sexual partners (Lansford et al., 2010) and a greater number of unplanned sexual encounters, making them more susceptible to sexually transmitted infections (STIs). In the United States, young people under the age of 24 account for more than half of the nearly 16 million new STI cases reported annually (Satterwhite et al., 2013), and have a disproportionately higher risk for unintended pregnancies compared to other sexually active age groups (Finer, 2010). Early sexual initiation during adolescence can further enhance the risk for these unhealthy sexual outcomes (O'Donnell, O'Donnell, & Stueve, 2001). Nevertheless, the high social and health costs associated with precocious and unplanned adolescent sexual activity are largely preventable. This serves as the guiding premise of the present research which focuses on identifying novel and modifiable risk

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factors associated with adolescent sexual risk behaviors, and examining their pathways of influence.

Past efforts to reduce early and risky sexual activity in adolescents have largely focused on imparting sexual knowledge and/or condom use skills (Picot et al., 2012), with a tacit assumption that all adolescents are equally capable of exercising caution and self-restraint in sexual encounters. In reality, contraception is a complex behavior, and adolescents often fail to engage in it effectively despite having the know-how (Brückner, Martin, & Bearman, 2004). To better understand the underlying reasons for this inconsistency, we focus on the heretofore unexamined role of working memory (WM), a core executive function, as a predisposing risk factor for sexual risk involvement, in particular condom non-use in adolescents.

Working Memory and Sexual Risk Behaviors

WM represents a limited-capacity neural system that allows for temporary storage and manipulation of goal-relevant information in the mental workspace, necessary for successfully executing higher-order tasks such as planning and decision-making (Miller & Cohen, 2001). WM relies on the key role of ‘executive attention’, which enables individuals to preferentially and flexibly attend to goal-relevant stimuli, while suppressing interference from dominant impulses (Kane, Conway, Hanbrick, & Engle, 2007). This top-down attentional control is necessary to maintain and process goal-relevant information during complex tasks. Indeed, some theorists have argued that individual differences in WM have less to do with differences in mental storage capacity and more to do with the ability to exert attentional control while maintaining or suppressing information in service of a goal (Engle, 2002). This ability to suppress otherwise powerful urges enables individuals with stronger WM to more readily evaluate the short and long-term consequences of their behavior. Not surprisingly, weaknesses in WM are found to be associated with impulsive behavioral tendencies (Hinson, Jameson, & Whitney, 2003; Winstanley, Olausson, Taylor, & Jentsch, 2010), including acting without thinking (AWT) (Romer et al., 2009) and inability to delay gratification (IDG) (Shamosh et al., 2008). We elaborate on these associations below.

AWT represents a classic form of behavioral impulsivity stemming from the failure to consider the consequences of one’s behavior (Eysenck, Easting, & Pearson, 1984). IDG is a related but largely independent indicator of impulsivity manifested as a tendency to discount future beneficial outcomes in favor of smaller, more immediate rewards (Madden & Bickel, 2010). These two distinct forms of impulsivity that are only modestly correlated (Reynolds, Penfold & Patak, 2008) are robust predictors of adolescent risk taking, including early sexual initiation (Khurana et al., 2012) and progressive trajectories of early drug use behaviors (Khurana et al., 2013, 2014). Thus, impulsive tendencies, such as AWT and IDG, are expected to mediate the influence of weak WM on adolescent risk taking behaviors. In particular, adolescents with weak WM are likely to have greater difficulty regulating dominant impulses (e.g., desire to have unprotected sex) in favor of considering longer-term outcomes (e.g., risk for STIs or unintended pregnancies).

Despite the plausibility of the link between WM and impulsivity, evidence in support of this relation has mostly been correlational (Khurana et al., 2012; Romer et al., 2009; Shamosh et al., 2008). Although a causal link between weak WM and IDG has been documented experimentally using WM training (Bickel, Yi, Landes, Hill, & Baxter, 2011), the effect was only evaluated over a brief one month follow-up and involved substance abusing adults. More research is therefore needed to better understand the role of WM in the development of self-control in adolescents.

Past research suggests that teens with lower cognitive ability engage in higher rates of risky sexual involvement, including early initiation (Halpern, Joyner, Udry, & Suchindran, 2000), early childbearing (Shearer et al., 2002), and multiple sexual partners (Fergusson, Horwood, & Ridder, 2005). However, the mechanisms of this association are poorly understood. Since WM is a critical component of cognitive ability (Miller & Cohen, 2001) that is necessary for top-down control of behavioral impulses during decision-making (Hinson et al., 2003), we argue that it may be a key factor driving the association between general cognitive ability and sexual risk taking.

Weakness in WM (Squeglia, Schweinsburg, Pulido, & Tapert, 2011) and the inability to control impulses can be further exacerbated by the use of drugs, in particular alcohol (Finn, Justus, Mazas, & Steinmetz, 1999), the use of which is associated with sexual risk taking (Poulin & Graham, 2001). Therefore, greater susceptibility towards early and heavy drinking may serve as another potential mechanism by which adolescents with weak WM are at heightened risk for engaging in unhealthy sexual behaviors.

Impulsivity versus Sensation Seeking

Long lines of research have linked sensation seeking (SS), the tendency to engage in novel and exciting experiences, with adolescent risk taking (Donohew et al., 2000). However, unlike AWT and IDG, SS tends to be *positively* related to WM (Khurana et al., 2012) and cognitive ability (Raine, Reynolds, Venables, & Mednick, 2002), suggesting that this tendency is not reflective of an underlying weakness in executive control. Although adolescents high in SS may be attracted to novel and exciting experiences because of their reward-seeking tendencies (Magid, MacLean, & Colder, 2007), they are not likely to exhibit loss of control over behavioral impulses and may therefore be able to consider alternative courses of action (Romer, Duckworth, Sznitman, & Park, 2010). Indeed, in early adolescent samples, SS no longer has a unique effect on adolescent risk taking once the influence of impulsivity dimensions characterized by weak executive control (i.e., AWT and IDG) is controlled (Khurana et al., 2012; 2013; Romer et al., 2011). SS is also not as related to long-term adverse effects of risk behaviors (e.g., addiction) as other personality dimensions reflective of weak executive control (Magid et al., 2007; Stautz & Cooper, 2013). In fact, while weakness in self-control (i.e., impulsivity) assessed as early as three years of age is found to be predictive of a variety of unhealthy developmental outcomes during adolescence and adulthood (Moffitt et al., 2011); SS in comparison tends to peak around mid-late adolescence (Romer & Hennessy, 2007) and is considered normative and enabling of healthy independence and identity development during adolescence (Spear, 2010). In light of these considerations as well as past evidence (Khurana et al., 2012, 2013), we expected WM's

effect on adolescent sexual risk behaviors to be mediated primarily by the impulsivity dimensions of AWT and IDG, and not by SS. However, because the current research examined sexual behaviors up to ages 15–16, we also explored any potential influence of SS on risk behaviors during these mid adolescent years when SS starts to peak.

Contrasting WM with Parental Influences

Executive functions, including WM are known to be susceptible to environmental influences, such as parental responsiveness (Farah et al., 2008) and family socioeconomic status (SES) (Evans & Shamberg, 2009). The influence of these contextual variables, that are also independently associated with adolescent risk taking (Huebner & Howell, 2003; Kerr, Stattin, & Burk, 2010), has not been accounted for in past studies focusing on the link between WM and adolescent risk behaviors. It is therefore possible that previously documented associations between weak WM and early risk taking may no longer be significant when third variables are taken into account. Therefore, to more clearly delineate the unique influence of WM on sexual risk involvement, we examine the potential confounding effects of parental SES, parental involvement, and parental monitoring in our model.

Several theoretical viewpoints on the development of self-control point to the importance of parental influences that may either predate the adolescent period or continue to moderate risky behaviors during adolescence. For instance, Gottfredson and Hirschi's (1990) self-control theory which was developed in the field of criminal justice, posits that parents may be able to directly impact children's self-control through active monitoring and by recognizing and rectifying risky behaviors through consistent discipline. Parents' monitoring and supervision of youth behaviors and whereabouts has also separately been linked to lower sexual risk taking (Huebner & Howell, 2003), although more recent examination suggests that these protective effects might be more a consequence of youth's self-disclosure than parents active monitoring per se (Kerr et al., 2010).

While Gottfredson and Hirschi (1990) theorized that parents can have a direct influence on their child's self-control, alternative pathways indirectly link parental effects to increased self-control and reduced engagement in risky sex. More specifically, parents may be able to impact their children's self-control by influencing their developmental trajectories of WM (Farah et al., 2008). Longitudinal studies have shown that children raised in family contexts characterized by nurturing and responsive parenting tend to show larger gains in executive functions, such as WM, during early years of development (Raver, McCoy, Lowenstein, & Pess, 2013; Rhoades, Greenberg, Lanza, & Blair, 2011). Adolescents with better WM are therefore more likely to have parents who are positively involved, and who can potentially continue to influence adolescents' developing regulatory abilities through other channels, such as behavioral modeling and opportunities for experiential learning (King, Lengua, & Monahan, 2013; Unnever, Cullen, & Pratt, 2003).

Greater parental involvement may also directly reduce adolescent sexual risk behaviors, apart from its potential influence mediated by WM and self-control. Parents who are more involved in their adolescents' lives may encourage greater self-disclosure of potentially risky

behavior (Kerr et al., 2010), or may share stronger emotional bonds that increase adolescents' motivation to adhere to safer behavior (Hirschi, 2001). Taken together, these findings suggest that parents who are positively involved in their children's lives may be able to influence their children's WM as well as the development of self-control during adolescence.

If the protective effects of WM on self-control and sexual risk behavior are largely attributable to more involved parenting, then the relation between these variables and sexual risk taking should be greatly attenuated once current parenting influences are controlled.

Present Study

To better understand the pathways by which parental variables and WM may impact adolescent self-control and sexual risk involvement, we examined both independent and mediated effects of these variables. The hypothesized pathways, depicted in Figure 1, are informed by past studies documenting an indirect relation between WM and early adolescent risk behaviors, the effects being channeled through impulsivity dimensions of AWT and IDG, but not through SS (Khurana et al., 2012, 2013). Pathways (A) and (B) represent baseline WM's unique effects on change in sexual risk involvement as mediated by change in self-control (measured using AWT and IDG), even after accounting for baseline parental influences on changes in self-control (C), alcohol use (D) and sexual risk involvement (E), as well as the correlation with WM due to earlier and continuing parenting influences (F). Pathways (G) and (H) represent indirect effects of weak WM and associated lack of self-control on change in sexual risk involvement, channeled through change in drinking behaviors. Finally, pathway (I) denotes the positive association between WM and SS. When controlling for the influence of AWT and IDG, SS was not expected to have a significant influence on either of the two risk behaviors. Nevertheless, we tested for any potential associations during these later years as depicted by pathways J and K in the model.

In sum, we posit that if WM is independently related to change in self-control and sexual risk behavior apart from concurrent parenting influences, then we would have stronger evidence that WM is an important and unique precursor for the development of these behaviors. To test our mediational hypotheses, we examined effects of WM (at baseline) on *changes* in self-control and sexual risk behaviors over a two-year follow-up. Given its association with WM (Khurana et al., 2013) and unprotected sexual involvement (Poulin & Graham, 2001), we expected alcohol use to operate as an additional mediator of WM's effect on sexual risk involvement.

Method

Participants

We used data from the last three annual assessments of a five year longitudinal study of a community sample of early adolescents (N=387), known as the Philadelphia Trajectory Study. Participants were enrolled at ages 9 – 12 (Mean age = 11.4 ± 0.87 years; 52% females) and tested annually for five consecutive years (2004– 2010). The sample was mainly recruited through flyers distributed in public and private schools in Philadelphia

neighborhoods, and posted in local venues such as libraries and community centers. Tasks were administered in a standardized manner using pencil and paper or on touch screen laptops with either e-Prime (Schneider, Eschman, & Zuccolotto, 2002) or Medialab software (Jarvis, 2004) using audio-computer-assisted self-interviewing (ACASI). The study was approved by the Institutional Review Board of the Children's Hospital of Philadelphia. Further details about recruitment and assessment procedures can be found in Romer et al. (2009).

The present analysis was conducted using data from 360 adolescents who participated in the last three waves of this 5 year longitudinal study. Loss to follow-up between initial recruitment (Wave 1) and Wave 3 of the study was approximately 5%. No statistically significant differences were noted in the demographics or baseline scores of key variables between those who were retained and those who dropped out. The decision to use only the last three waves of data (Waves 3–5) was made because of the low rates of sexual activity in the preceding waves. These three assessment time points will hereafter be referred to as T1 (baseline), T2 (first follow-up), and T3 (second follow-up). There was 8% attrition across these three assessment time points. Loss to follow-up was not related to baseline scores of key variables or demographic characteristics. Missing data were handled using full information maximum likelihood (FIML) in *Mplus* v7. The mean age of participants at T1 was 13.5 ± 0.95 years, with a near equal gender distribution (52% female). Non-Hispanic white adolescents represented 56% of the sample, followed by 27% Non-Hispanic black and 9% Hispanic youth, with the remaining 8% comprised of Native American and Asian American youth. The majority of the sample was from low-mid socio-economic backgrounds, assessed using the Hollingshead Two-factor Index of social status (Hollingshead, 1975). Table 1 presents the means and standard deviations of the key variables along with the correlation matrix.

Measures

Sexual Risk Involvement (T2 & T3)—Adolescents were asked at T2 and T3 – (a) “have you ever engaged in vaginal intercourse” (Y/N), and (b) “did you use a condom the last time you engaged in vaginal intercourse” (Y/N). Responses to these two questions were used to create a new composite variable for sexual risk involvement at the final follow-up (T3), coded such that 0 = virgin; 1 = non-virgin, used a condom at last intercourse; 2 = non-virgin, last sexual encounter was unprotected. At T2, only 4% of the sample ($n=15$) reported lack of condom use during last intercourse. Therefore, the sexual risk variable at T2 was coded as a binary variable representing sexual initiation (0 = virgin, 1= non-virgin). This variable was labeled as “virginity status (T2)” and was included as a predictor of sexual risk involvement at T3.

Working Memory (T1)—WM was assessed at baseline (T1) using four standardized tasks, namely *Corsi block tapping* (Milner, 1971), *Object two-back* (D'Esposito, Postle, Ballard, & Lease, 1999), *Digit span backwards* (Wechsler, 2003), and *Spatial working memory* (Owen, Downes, Sahakian, Polkey, & Robbins, 1990). All tasks were scored such that higher scores indicated better WM performance. A brief description of these tasks follows.

Corsi block tapping: This task is a non-verbal variant of the digit span task (Milner, 1971). Participants view a set of identical blocks that are spatially dispersed on the screen, and are individually lit up in a random sequence. Participants are asked to tap each box in the *reverse* order of the sequence of lit boxes. This task also assesses spatial working memory as the visual sequence must be maintained and reversed in WM in order to guide the response. We used the total correct score, ranging from 0–12, as an indicator of WM performance on this task.

Digit span backwards: This task tests the auditory-verbal working memory of participants by having them repeat back in reverse order, sequences of digits to the experimenter. The test was administered in standard format according to the procedures listed in the Wechsler Intelligence Scale for Children–Fourth Edition (WISC-IV) manual (Wechsler, 2003). The WISC digit span total raw score, ranging from 4 – 14, was used as an indicator of WM performance for this task.

Object two-back: This task involves monitoring a series of objects in the form of irregular polygons that are presented for 500 milliseconds each, separated by a one second interval. Participants have to continually update their working memory to compare the current object with the object presented two trials back (D’Esposito et al., 1999). The participants’ total correct score, ranging from 25–90, was used as an indicator of their WM performance on this task.

Spatial working memory: This self-directed computerized task requires the participant to search for hidden tokens one at a time within sets of four to eight randomly positioned boxes. Tokens are hidden only once in each box per trial. WM skills are tapped as the participant while searching must hold in WM the locations already checked and as tokens are found, must remember and update information about the locations of those tokens (Owen et al., 1990). *Between-search* errors on this task are made if the participant returns to a box where a token had already been found during a *previous* search sequence. In comparison, a *within-search* error is made when a participant returns to a box that has already been checked during the *same* search sequence. WM performance was indexed using the between-search errors score, since it places more demands on WM of participants as they have to remember where the token was found in the last search when beginning a new search for the next token. The between-search error score was reverse scored to indicate better WM performance, and ranged from 0 – 109.

These tasks were chosen because they tap into the executive control component of WM in addition to the storage component. The first three tasks (corsi block tapping, digit span backwards, and object two-back) require not just the ability to store task-relevant information but also manipulate this stored information in the mental workspace, for instance reversing the order of digits in case of digit span backwards. The spatial WM task also taxes the executive attention ability because it requires the participant to remember the sequence of tokens from the last search while remembering and updating the current search sequence.

All four tasks were found to significantly load on a single latent factor, with loadings ranging from 0.37–0.61. Model fit statistics [$\chi^2(2) = 1.06, p = 0.59$; RMSEA (Root Mean Square Error of Approximation) (90%CI) = 0.00 (0.00, 0.08); CFI = 1.00] and residual diagnostics (standardized residuals less than 3.0 and modification index below 4.0) suggested a well-fitting model. The latent factor of working memory was used as the predictor in the final model.

Self-control Dimensions (T2 & T3)—Data on two self-control dimensions, assessed at T2 and T3, were used in the present analysis, i.e., Acting Without Thinking (AWT), and Inability to Delay Gratification (IDG). AWT was assessed using a reliable and valid self-report measure (Eysenck et al., 1984), while IDG was assessed using a behavioral task with good psychometric properties (Reynolds & Schiffbauer, 2005). Further details about these measures are provided below.

Acting Without Thinking (AWT): Nine yes/no items were used from the Junior Eysenck Impulsivity Scale (Eysenck et al., 1984) to assess the tendency to act on impulse (e.g., “Do you usually do and say things without stopping to think?”). Given that these items significantly represented a single underlying latent factor, with high item-loadings (0.44–0.76), and a rho of 0.80, we averaged each participant’s response on these 9 items to create a single AWT score at T2 and T3 respectively.

Inability to Delay Gratification (IDG): This self-control dimension was assessed using a behavioral task (adapted from Green, Fry, & Myerson, 1994) in which the participants were asked in the context of payment for a job to choose between hypothetical monetary rewards ranging from \$10 – \$90, that, if received immediately, would be equivalent to receiving \$100 six months later. Respondents were initially asked if they would accept a payment of \$50 immediately in lieu of being paid \$100 in six months. Those who accepted the \$50 offer, were then asked if they would accept an amount lower than \$50 in \$10 decrements. The lowest amount they accepted was taken as their equivalent value. A comparable procedure with successively increasing values was used for those who did not accept \$50. Scores on this variable ranged from 10 – 100, which were reverse-scored such that higher scores were indicative of greater delay discounting (i.e. greater inability to delay gratification). Similar procedures have been shown to be valid with adolescents (Duckworth & Seligman, 2005) and to be valid indicators of the ability to delay gratification (Reynolds & Schiffbauer, 2005). The test-retest reliability of this measure at the last two annual assessments was high ($r = 0.52, p < .001$). IDG scores from T2 and T3 assessment were used in the present analyses.

Sensation Seeking (T2 & T3): In addition to the two impulsivity dimensions, we also included sensation seeking (at T2 and T3) as a potential mediator of WM’s effect. Four items representing each of the four dimensions (i.e., experience seeking, boredom susceptibility, thrill/adventure seeking, and disinhibition) of the Brief Sensation Seeking Scale (Hoyle, Stephenson, Palmgreen, Lorch, & Donohew, 2002) were used to assess SS. Responses were coded on a 4-point scale ranging from strongly agree to strongly disagree. These items loaded significantly on a single latent factor, with item loadings ranging from

0.57–0.74, and a high reliability coefficient (ρ) of 0.77 (Raykov, 2009). The scores on the four items were averaged to create a composite score for each participant at both time points.

Alcohol Use (T2 & T3)—Adolescents were asked about ever (yes/no) and recent (past 30 days) alcohol use ranging from 0 (none of the past 30 days), 1 (1–2 days), 2 (3–5 days)... 7 (all 30 days) at T2 and T3. Due to the low response rates in the higher categories resulting in highly skewed distributions, the responses were coded to create a new variable with the following categories - 0 (Never drank), 1 (Used, but not in past 30 days), and 2 (Used, and drank in past 30 days) to account for drinking behaviors at both T2 and T3.

Parental Monitoring (T1)—Adolescent perceptions of parental monitoring were assessed at T1 using three self-report items. Specifically, participants were asked to report on how often their parents or adults who take care of them (i) “expect you to call them if you are returning home late”, (ii) “ask you where you are going when you go out”, and (iii) “want to know the names of the kids with whom you are going before you go out”. Response options included 1 (never), 2 (some of the time), 3 (most of the time) and 4 (all the time). Together these items represent both the key dimensions of parental monitoring as outlined by Kerr et al. (2010). The first item represents “control/rule-setting”, while the remaining two items represent “parental solicitation”. Scores on these items loaded on a single latent factor, with loadings ranging from 0.56 to 0.85, and a ρ of 0.77. The latent factor of parental monitoring was included in the final model.

Parental Involvement (T1)—Parental involvement was assessed at T1 using adolescent responses to the following statements – (a) “my family does something fun together”; and “my parents or the adults who take care of me...”: (b) “spend time just talking with me”, (c) “push me to do my best”, and (d) “help me with school work when I don’t understand”. Response options included – 1 (Almost never), 2 (A few times a month), 3 (A few times a week), and 4 (Almost every day) for the first two items, and 1 (Never), 2 (Some of the time), 3 (Most of the time), and 4 (All of the time) for the last two items. These four items loaded on a single latent factor with high loadings (0.57 – 0.71), and a ρ of 0.72. Confirmatory factor analysis conducted with parental monitoring and parental involvement as separate latent factors revealed strong loadings and good global and local model fit [$\chi^2(12) = 11.41$, $p = 0.49$; RMSEA (95% CI) = 0.00 (0.00, 0.05); CFI = 1.00].

Parental socio-economic status—Parental SES was assessed using the Hollingshead Two-Factor Index of Social Status (Hollingshead, 1975). Primary caregivers reported on a 7-point scale their “current occupation”, ranging from 1 (unskilled) to 7 (higher executive) and “years of education”, ranging from 1 (fewer than 7 years) to 7 (greater than 16 years). Scores on these two measures were averaged to create a single index of parental SES.

Control Variables—Because age, gender, pubertal development, and race-ethnicity are associated with adolescent sexual risk involvement (Abma, Martinez, & Copen, 2010; Udry, 1979), we included these variables as potential controls in our hypothesized model. Pubertal development was measured by adolescents’ self-reports using sketches of the Tanner stages of pubertal development (Tanner, Whitehouse, Marubini, & Resele, 1976). For boys, the development of genitals and body hair growth was assessed; while for girls, the development

of breasts and pubic hair growth was assessed. Sex-specific scores of pubertal maturation were used in the analyses, with higher scores signifying greater pubertal development.

Analytic Plan

Structural equation modeling (SEM) was used to test the study hypotheses outlined in Figure 1. More specifically, we examined the direct and indirect effect of WM (at T1), mediated by changes in SS, AWT, and IDG (from T2-T3), on sexual risk involvement (at T3), controlling for virginity status (at T2). In addition, we tested direct and mediated effects of parental monitoring, involvement, and socio-economic status at T1 on the sexual risk outcome. Change in drinking behaviors from T2-T3 was included as an additional mediator of the effect of the WM and parental influences on sexual risk behaviors. All analyses were conducted in *Mplus* v7 using the robust maximum likelihood estimator to account for any violations of normality. All exogenous predictors were assessed at T1, while the mediators and risk behavior outcomes were assessed at T2 and T3. Confidence intervals for mediated effects were obtained using the bias-corrected bootstrap re-sampling method (MacKinnon, Lockwood, & Williams, 2004). Model fit was evaluated using multiple indices of global fit and a close examination of residual diagnostics. The criteria for good model fit included a low χ^2 test-statistic, RMSEA value less than 0.05, and values of the comparative fit index (CFI) and the Tucker-Lewis Index (TLI) greater than 0.90.

Results

At the final follow-up (T3; Mean age = 15.7 years), slightly over a quarter of the sample (28%) had initiated vaginal intercourse, reflecting a near 100% increase from T2 initiation rate of 15%. Of those who had initiated sexual activity by T3 ($n=91$), nearly 1 in every 4 adolescents (27.5%) reported not using a condom during their last sexual intercourse. Significant age differences were observed in the rates of sexual initiation with older adolescents more likely to have initiated intercourse ($t=5.14$, $p<0.001$). No age differences were observed in condom use among those who had initiated sexual intercourse. Similarly, we noted no gender differences in the rates of sexual initiation or condom use in our sample. In terms of racial-ethnic variations, black and Hispanic youth were more likely to have initiated sexual intercourse at T2 and T3, as compared to non-Hispanic white, Asian, and Native American youth. Black (34.5%) and Hispanic (46.2%) youth also had relatively higher rates of condom non-use as compared to white youth (18.7%) in the non-virgin subsample at T3.

As expected, the latent factor of WM was found to be positively related to SS at T2 ($r=0.11$) and negatively related to both AWT ($r=-0.14$) and IDG ($r=-0.15$), such that adolescents with stronger WM reported higher levels of SS and lower levels of AWT and IDG. All three personality dimensions were positively correlated (AWT $\leftrightarrow$ SS = 0.44; AWT $\leftrightarrow$ IDG = 0.16), with the exception of SS and IDG, which were not significantly associated. In bivariate regression models, all three dimensions assessed at T2 were positively related to sexual risk involvement at T3: SS ($t=2.87$, $p<0.01$), AWT ($t=5.93$, $p<0.001$), and IDG ($t=3.91$, $p<0.001$). However, when the outcome variable was simultaneously regressed on all three dimensions, only AWT ($t=4.54$, $p<0.001$) and IDG ($t=3.15$, $p=0.002$) had a

significant effect. Controlling for the effect of AWT and IDG, SS was no longer a significant predictor of sexual risk involvement ($t = 0.91$, $p = 0.36$). Consistent with our hypothesis, SS also did not have a significant effect on any of the key outcomes in the full model, and hence the decision was made to drop it from the final model.

SEM analyses testing the final model revealed that WM had a significant mediated effect on sexual risk involvement at the final follow-up (T3), $B(SE) = -0.05 (0.01)$, $p < 0.001$, controlling for virginity status (at T2) and the effects of parental SES, involvement, and monitoring at baseline (T1). WM's effect was primarily channeled through change in the self-control dimension of AWT (see Figure 2). Stronger WM at T1 was negatively associated with change in AWT, $B(SE) = -0.04 (0.02)$, $p < 0.01$, and IDG, $B(SE) = -4.00 (1.78)$, $p < 0.05$, from T2 – T3. On average, adolescents with stronger WM at baseline had lower AWT and IDG scores at T3, controlling for T2 levels.

Change in AWT (from T2-T3) was significantly associated with change in alcohol use (from T2-T3) and sexual risk involvement at T3, controlling for T2 virginity status. Some of AWT's effect on sexual risk involvement was direct, $B(SE) = 0.25 (0.10)$, $p < 0.01$, while some of it was channeled through change in alcohol use, $B(SE) = 0.09 (0.03)$, $p < 0.01$. IDG's effect on sexual risk involvement at T3 was only indirect, and channeled through its influence on virginity status at T2, $B(SE) = 0.002 (0.001)$, $p < 0.01$. In other words, adolescents who displayed greater difficulty in delaying gratification (at T2) were more likely to have initiated sexual activity at that time point, which in turn increased their risk for involvement in condom non-use at T3.

A key question in our analysis was whether any observed relation between WM and sexual risk involvement could be explained by parental influences during adolescence. The parental variables included in our model did have independent protective effects. Adolescents who reported greater parental involvement at T1 were less likely to engage in sexual risk taking at T3, $B(SE) = -0.04 (0.01)$, $p < 0.001$, controlling for virginity status at T2. This effect was indirect and mainly channeled through AWT and alcohol use. Nevertheless, parental involvement did not predict change in either form of self-control. In addition, parental involvement was not significantly related to adolescent WM abilities ($p = 0.06$), and the net mediated effect of parental involvement on risk behaviors, channeled through WM, was non-significant. Thus, although parental involvement was independently related to self-control and risk behavior, it could not explain the mediated effect of WM on sexual risk involvement.

In comparison to parental involvement, monitoring had a marginally significant direct effect on change in sexual risk behavior. Adolescents who reported higher levels of parental monitoring at T1 were less likely to engage in sexual risk behaviors at T3, controlling for virginity status at T2, $B(SE) = -0.07 (0.04)$, $p < 0.06$. Parental monitoring did not influence any other outcome in our model, and thus could not explain the relation between WM and risk taking.

In the case of parental SES, there was a direct effect on sexual risk involvement at T3, $B(SE) = -0.004 (0.002)$, $p < 0.05$, controlling for virginity status at T2, as well as an indirect

effect that was channeled through WM, $B(SE) = 0.013(0.005)$, $p < 0.01$. More specifically, this indirect effect was channeled through the protective effect of parental SES on AWT change, which was mediated by WM, $B(SE) = -0.001(0.001)$, $p < 0.05$. In comparison, the effect of parental SES on IDG change was largely direct, $B(SE) = -0.18(0.09)$, $p < 0.05$. Path coefficients of significant effects, including influence of covariates, can be found in Table 2. Consistent with our past research (Khurana et al., 2012), pubertal development was positively associated with WM, but this effect was conditional on gender, and significant only in the case of females. Notably, WM and associated self-control dimensions were found to be significant mediators of the risk-enhancing influence of low parental SES and black racial identity. However, controlling for these effects, the relation between WM and risk behavior was left largely intact.

Interactions were explored between WM and parental variables, as well as between WM and alcohol use; none were significant. The model fit was excellent, with a non-significant chi-square value, RMSEA value less than 0.05, and CFI and the TLI values greater than 0.90 [Chi sq. (248) = 330.44, $p < 0.001$; RMSEA (90% CI) = 0.03 (0.02, 0.04); CFI = 0.95; TLI = 0.93]. Overall, our model accounted for a significant amount of variance in alcohol use and virginity status at T2 (19% and 18% respectively), and alcohol use and sexual risk involvement at T3 (34% and 50% respectively), underscoring the importance of WM and self-control as protective factors in preventing adolescent risk taking. To rule out the possibility that changes in risk behaviors were predicting changes in impulsivity, we also tested an alternative model reversing the direction of effects between risk behaviors (alcohol use, sexual risk taking) and impulsivity dimensions (AWT and IDG). Changes in risk behaviors (from T2-T3) failed to predict changes in AWT and IDG. The alternative model also had a relatively poor fit compared to our current model.

Finally, since our outcome variable at T3 was a composite of early sexual initiation and condom non-use – outcomes that can have unique predictors, we re-analyzed our models separately for sexual initiation (using the virgin-only sub-sample at T2, $N = 305$) and condom non-use (using the non-virgin sub-sample at T3; $N=91$). We found the same pattern of findings in both models - weakness in WM and associated lack of self-control was found to increase adolescents' risk of early sexual initiation and involvement in unprotected sexual intercourse. Parental involvement was a significant predictor of AWT, drinking behaviors, and sexual initiation (virgin sub-sample). The effect of parental involvement on condom non-use failed to reach significance in the sexually active sub-sample ($N=91$), possibly due to low power.

Discussion

Adolescence is a period of rapid development associated with the onset of puberty. It is a time when novel experiences in social contexts interact with emerging regulatory abilities to influence the developing adolescent brain (Crone & Dahl, 2012). Although these experiences are important for learning and development, lack of self-control in such contexts can increase risk for adverse outcomes. With a novel focus on WM and executive control, our primary goals in this study were: First, to examine whether individual differences in WM and self-control were predictive of sexual risk involvement over a two-year follow-up

in a community sample of adolescents ages 12 to 15 at baseline, and second, whether these effects held independent of the continued or earlier impact of parental influences. We distinguished between parental monitoring, which parents can use to restrict and maintain oversight of risk taking, versus maintaining involvement with their child, which could reduce risk taking either by enhancing self-control or by directly reducing risky behavior. We also controlled for a host of other confounds that might be related to WM and sexual risk, such as parental SES. These potential influences could have operated as “third variables” resulting in a spurious relation between WM and adolescent risk taking.

Our results are the first to show that individual differences in WM can predict both early sexual initiation and unprotected sexual involvement during adolescence and that these effects hold despite controls for important parental characteristics that have been associated with both WM and sexual behavior in adolescents. In addition, in support of the underlying role of WM in promoting increases in self-control as adolescents age, we show for the first time that WM predicted change in both AWT and IDG over a one-year time span (from T2 to T3). Adolescents with relatively weak WM (at T1) were significantly more likely to have initiated sexual intercourse (by T2) and to report that their last sexual encounter was unprotected (at T3). Consistent with past research (Khurana et al., 2012), we found that the influence of WM on sexual risk taking was not direct. Instead, it was mediated by changes in the impulsivity dimensions of AWT and IDG that reflect an underlying lack of self-control. Although WM predicted change in IDG, this impulsive tendency was only predictive of sexual risk involvement at T3 by virtue of its relation to sexual initiation at T2. Thus, the effects of WM on AWT change were more robust than IDG in predicting increases in sexual risk behavior, and included mediated effects channeled through changes in alcohol use. Notably, adolescents with weak WM were more likely to engage in sexual risk behavior apart from the effects of alcohol use and early sexual initiation.

As compared to AWT, IDG bore no relation to alcohol use. In supplemental analyses we found that IDG was moderately correlated with binge drinking at follow-up ($r = 0.10$, $p < 0.07$), suggesting that adolescents who have difficulty delaying gratification might be more prone to progressive drinking and loss of control over alcohol use. Due to its low rate, binge drinking was not included in the present model. Nevertheless, it is possible that during later years, the effect of IDG on sexual risk taking may also be channeled through binge drinking, resulting in a stronger mediated influence for this facet of self-control.

The results also support the causal hypothesis between WM and sexual risk behavior by showing that this relation is robust to controls for parental influences, including parental monitoring, involvement, SES, as well as non-white ethnic-racial identity. Previous research examining the relation between WM and various risk behaviors has not controlled for all these influences, leaving open the possibility that WM and risk behaviors may have been related due to the confounding effect of parental or other influences. The strongest potential confound, family SES, was positively related to WM and negatively related to sexual risk involvement. However, after controlling for these relations, we still found that WM was directly related to two forms of self-control (AWT and IDG) and that these associations mediated the influence of weak WM on sexual risk involvement.

Although our model suggests that WM influences sexual risk taking indirectly through its relation with impulsivity, it can nevertheless be argued that instead of being a mediated relation, the association between WM and impulsivity is indicative of a higher-order, latent factor of executive function that is common to both WM and impulsivity. If indeed this were the case, then the exogenous predictor of WM in our model (i.e., parental SES) should affect both indicators of this underlying latent factor, i.e. WM and impulsivity. To test this alternative hypothesis, we examined if the effects of parental SES, instead of being channeled through WM, were mediated by AWT and IDG. However, unlike the current model, the indirect effects of parental SES on change in WM, as mediated by AWT and IDG, were non-significant. These findings are consistent with our observation that the effects of parental SES on WM were present at baseline, with little variability in WM scores thereafter. In contrast, there was significant variability in AWT and IDG post-baseline that was predicted by individual differences in baseline levels of WM. As such, we believe that the association between WM and impulsivity is unlikely to reflect the effects of a common underlying factor.

We found some support for the involvement hypothesis in that greater parental involvement was associated with both greater self-control and lower alcohol use, which in turn was associated with lower sexual risk-involvement at the final follow-up. In comparison, parental monitoring had a direct influence on the sexual risk outcome with trend-level significance. Nevertheless, its effect was not mediated by greater ability to exert self-control or to avoid alcohol use, suggesting that it had little effect on these important precursors to adolescent sexual risk taking. Instead, its effect appears to have been primarily produced by parental restrictions on adolescent behavior. Other prospective research has supported the efficacy of this parental strategy in reducing adolescent sexual risk behaviors (Dittus, Michael, Becasen, Gloppen, & Guilamo-Ramos, 2013). However, our findings suggest that parental monitoring is ultimately less protective than maintaining ongoing parental involvement because involvement is associated with greater adolescent self-control, in particular less AWT, which can have a more pervasive influence on risk taking outside of the direct purview of parents.

Given its more pervasive influence on self-control, positive parental involvement may be an important variable to focus on in family-based preventive interventions (Deptula, Henry, & Schoeny, 2010). Although parental involvement did not predict change in AWT or IDG, its relation with AWT was separate from its potential association with WM. Greater parental involvement during adolescence likely reflects similar involvement during younger years, thereby fostering the development of self-control prior to their child's entry into adolescence. Children who enter adolescence with greater capacity for self-control are less likely to engage in impulsive and risky behaviors (Moffitt et al., 2011). Thus, even after controlling for any association between parental involvement and WM, the relation we observed between parental involvement and AWT may reflect the enduring effects of early parenting behaviors on child self-regulation.

Supplemental analyses using the virgin-only sample showed that parents who were more involved in the lives of their adolescents were also more likely to have adolescents who did not initiate sexual activity during the one-year follow-up. These protective effects of parental involvement could potentially operate through warm and responsive parenting that

encourages greater attachment to parents (Hirschi, 2001), along with other favorable mechanisms, such as greater disclosure of potential risky behavior that parents could prevent (Kerr et al., 2010).

We interpret the robust relation between WM and risky sexual behavior as a reflection of the relative inability of some adolescents to control impulses that encourage early sexual initiation and failure to use protection when engaging in sex. An alternative explanation, that adolescents engaging in unprotected sex are unaware of the hazards of doing so, seems unlikely given that these risks are well known and knowledge of these risks does not seem to be especially protective on its own (Pedlow & Carey, 2004). Nevertheless, we examined a related measure of how often parents communicated with the adolescent about relationships with members of the opposite sex and other sexual health topics. The frequency of these discussions was unrelated to risk taking in our sample. Thus, our findings provide one possible explanation for the lack of success of interventions that focus primarily on knowledge (Pedlow & Carey, 2004), by showing that unless adolescents have the executive ability to control impulses and consider healthy alternatives, they will be at greater risk of engaging in unsafe practices regardless of their knowledge of the consequences.

Theories of Adolescent Risk Taking

Our results have implications for recent theories of adolescent risk taking, e.g., the dual systems theory (Steinberg, 2010) and the imbalance theory (Somerville & Casey, 2010), which posit that the rise in risk taking during adolescence is attributable to the asynchronous development of neural reward and control systems. This creates a regulatory imbalance characterized by an overactive reward system that is inadequately controlled by an underdeveloped executive control system. Although Casey and Caudle (2013) emphasize the role of individual differences, the present results suggest that these theoretical models may be an oversimplification of developmental trends observed during adolescence. Indeed, these models have been challenged by inconsistent evidence from neuroimaging studies (Pfeifer & Allen, 2012), a mismatch with the observed peak in risk taking behaviors (Willoughby, Good, Adachi, Hamza, & Tavernier, 2013), as well as a failure to appreciate individual differences (Romer, 2010) and contextual influences (Gerrard, Gibbons, Houlihan, Stock, & Pomery, 2008). Our findings suggest that it is primarily adolescents with weak self-control who are susceptible to adverse forms of risk taking. Furthermore, SS, which increases during adolescence, does not coincide with weak executive control and therefore does not necessarily place adolescents at risk of adverse outcomes.

Consistent with Reyna and Farley's (2006) review of the literature, the present as well as other research (e.g., Romer et al., 2010) suggests that it is critical to distinguish between adaptive and maladaptive forms of adolescent risk taking. Although sensation seekers may be attracted to risky behavior, they do not appear to do so out of lack of control over impulses. Instead, in line with Reyna and Farley's (2006) fuzzy trace theory, adolescents' risk evaluation may be quite rational, taking into account both the risks and the rewards of their behavior. For instance, a recent study by Reyna and colleagues (2011) showed that adolescents' risky sexual behavior was related more to fuzzy trace decision processes than to sensation seeking. This interpretation is consistent with the recognition that the rise in SS

during adolescence has been conserved across mammalian species and is likely an adaptive behavior for this period of development (Spear, 2010).

Limitations

There are a few limitations to our findings. First, data on adolescent sexual risk and alcohol use behaviors were collected using self-reports, which may not always be accurate. To reduce reporting bias, we made use of the computer assisted self-interviewing technique when gathering information on sensitive topics. Second, our sample was recruited from urban neighborhoods in Philadelphia, which limits the generalizability of our findings. Third, our alcohol use measure only assessed the consumption of alcohol in the past 30 days; thus we could not examine the influence of alcohol use on condom use before or during sex, or the effects of alcohol consumption prior to the past month. Other indicators of risk involvement, such as binge drinking and number of sexual partners, could not be considered in the present model because of the low response rates. Fourth, we may have underestimated the sexual risk involvement in our sample by not explicitly asking adolescents about prior sexual experiences beyond the last occasion or their sexual experiences with same-sex partners. It is possible that some of the adolescents who reported being virgins may have engaged in same-sex sexual activity. Fifth, we cannot rule out the effect of other parenting behaviors that were not assessed in our study but could influence WM and self-control, such as parental disciplining practices (King et al., 2013). In a related vein, the effects of exogenous variables included in the present model (i.e., parental influences, SES, racial-ethnic identity) could be channeled through other risk factors not assessed in the present study, such as availability of risk opportunities and favorability towards risk prototypes in certain social contexts (Gerrard et al., 2008). Future research should also explore the confounding influence of deviant peers, especially as a potential mediator of negative parental influences. Finally, since we did not have information about early parenting behaviors in our sample, we are unable to confirm whether the effect of parental involvement on AWT represents carry-over effects from younger years or more temporally proximal influences.

Our main finding that WM has a protective influence on adolescent sexual risk behaviors, independent of parental influences, suggests that interventions targeting WM could help reduce rates of impulsive sexual risk taking during adolescence. The relative malleability of WM also makes it a strong candidate for preventive interventions that can be delivered at young ages (Diamond & Lee, 2011). Initial studies documenting positive gains with WM training indicate the promise of this approach in improving cognitive control over impulsive tendencies in both children and adolescents (Holmes, Gathercole, & Dunning, 2009; Klingberg, 2010). The fact that WM also mediated the influence of several social address variables (e.g., race, SES) further enhances its appeal as an intervention target. Finally, parents can also influence their children's self-control and risk taking apart from limitations in WM; this could provide an additional strategy for intervening, especially for families with children that exhibit weakness in self-control.

In sum, our findings demonstrate that poor self-control associated with weak WM can make some adolescents more vulnerable to risky sexual choices, especially during mid-

adolescence. WM has previously been linked to adolescent drug use and early sexual initiation, but this is the first time that its association with condom non-use has been examined. The protective influence of WM was found to operate independently of the strong parental effects on adolescent sexual behaviors. These findings highlight the promise of interventions that target WM and associated self-regulatory abilities as an alternative mechanism by which the high costs associated with adolescent sexual risk taking can be reduced.

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References

- Abma, JC., Martinez, GM., Copen, CE. Vital and Health Statistics. Washington, DC: U.S. Department of Health and Human Services; 2010. Teenagers in the United States: Sexual activity, contraceptive use, and childbearing: National Survey of Family Growth 2006–2008 (No. Series 23, Number 20); p. 1-78. Retrieved from http://www.cdc.gov/nchs/data/series/sr_23/sr23_030.pdf
- Bickel WK, Yi R, Landes RD, Hill PF, Baxter C. Remember the future: Working memory training decreases delay discounting among stimulant addicts. *Biological Psychiatry*. 2011; 69:260–265. DOI: 10.1016/j.biopsych.2010.08.017 [PubMed: 20965498]
- Brückner H, Martin A, Bearman PS. Ambivalence and pregnancy: Adolescents' attitudes, contraceptive use and pregnancy. *Perspectives on sexual and reproductive health*. 2004; 36:248–257. DOI: 10.1111/j.1931-2393.2004.tb00029.x [PubMed: 15687083]
- Craver, K., Joyner, K., Udry, JR. National estimates of adolescent romantic relationships. In: Florsheim, P., editor. *Adolescent romantic relations and sexual behavior: theory, research, and practical implications*. Mahwah, NJ: Lawrence Erlbaum Associates; 2003. p. 291-329.
- Crone EA, Dahl RE. Understanding adolescence as a period of social–affective engagement and goal flexibility. *Nature Reviews Neuroscience*. 2012; 13:636–650. DOI: 10.1038/nrn3313 [PubMed: 22903221]
- D'Esposito M, Postle BR, Ballard D, Lease J. Maintenance versus manipulation of information held in working memory: An event-related fMRI study. *Brain and Cognition*. 1999; 41:66–86. DOI: 10.1006/brcg.1999.1096 [PubMed: 10536086]
- Deptula DP, Henry DB, Schoeny ME. How can parents make a difference? Longitudinal associations with adolescent sexual behavior. *Journal of Family Psychology*. 2010; 24:731–739. DOI: 10.1037/a0021760 [PubMed: 21171771]
- Diamond A, Lee K. Interventions shown to aid executive function development in children 4 to 12 years old. *Science*. 2011; 333:959–964. DOI: 10.1126/science.1204529 [PubMed: 21852486]
- Dittus P, Michael S, Becasen J, Gloppen K, Guilamo-Ramos V. Protective effects of parental monitoring knowledge and enforcement of rules on adolescent sexual behavior: A meta-analysis. *Journal of Adolescent Health*. 2013; 52(s14)doi: 10.1016/j.jadohealth.2012.10.037
- Donohew L, Zimmerman R, Cupp PS, Novak S, Colon S, Abell R. Sensation seeking, impulsive decision-making, and risky sex: Implications for risk taking and design of interventions. *Personality and Individual Differences*. 2000; 28:1079–1091. DOI: 10.1016/S0191-8869(99)00158-0
- Duckworth AL, Seligman MEP. Self-discipline outdoes IQ in predicting academic performance of adolescents. *Psychological Science*. 2005; 16:939–944. DOI: 10.1111/j.1467-9280.2005.01641.x [PubMed: 16313657]
- Engle RW. Working memory capacity as executive attention. *Current Directions in Psychological Science*. 2002; 11:19–23. DOI: 10.1111/1467-8721.00160
- Evans GW, Schamberg MA. Childhood poverty, chronic stress, and adult working memory. *Proceedings of the National Academy of Sciences*. 2009; 106:6545–6549. DOI: 10.1073/pnas.0811910106

- Eysenck SBG, Easting G, Pearson PR. Age norms for impulsiveness, venturesomeness and empathy in children. *Personality and Individual Differences*. 1984; 5:315–321. DOI: 10.1016/0191-8869(84)90070-9
- Farah MJ, Betancourt L, Shera DM, Savage JH, Giannetta JM, Brodsky NL, Hurt H. Environmental stimulation, parental nurturance and cognitive development in humans. *Developmental Science*. 2008; 11:793–801. DOI: 10.1111/j.1467-7687.2008.00688.x [PubMed: 18810850]
- Fergusson DM, Horwood JL, Ridder EM. Show me the child at seven II: Childhood intelligence and later outcomes in adolescence and young adulthood. *Journal of Child Psychology and Psychiatry*. 2005; 46:850–858. DOI: 10.1111/j.1469-7610.2005.01472.x [PubMed: 16033633]
- Finer LB. Unintended pregnancy among U.S. adolescents: Accounting for sexual activity. *Journal of Adolescent Health*. 2010; doi: 10.1016/j.jadohealth.2010.02.002
- Finn PR, Justus A, Mazas C, Steinmetz JE. Working memory, executive processes and the effects of alcohol on Go/No-Go learning: testing a model of behavioral regulation and impulsivity. *Psychopharmacology*. 1999; 146:465–472. [PubMed: 10550497]
- Gerrard M, Gibbons FX, Houlihan AE, Stock ML, Pomery EA. A dual-process approach to health risk decision making: The prototype willingness model. *Developmental Review*. 2008; 28:29–61. DOI: 10.1016/j.dr.2007.10.001
- Gottfredson, MR., Hirschi, T. A general theory of crime. Stanford, CA: Stanford University Press; 1990.
- Green L, Fry AF, Myerson J. Discounting of delayed rewards: A life-span comparison. *Psychological Science*. 1994; 5:33–36. DOI: 10.1111/j.1467-9280.1994.tb00610.x
- Halpern CT, Joyner K, Udry JR, Suchindran C. Smart teens don't have sex (or kiss much either). *Journal of Adolescent Health*. 2000; 26:213–225. DOI: 10.1016/S1054-139X(99)00061-0 [PubMed: 10706169]
- Hinson JM, Jameson TL, Whitney P. Impulsive decision making and working memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition*. 2003; 29:298–306. DOI: 10.1037/0278-7393.29.2.298
- Hirschi, T. Causes of Delinquency. Piscataway, NJ: Transaction Publishers; 2001.
- Hollingshead, AB. Four factor index of social status. New Haven, CT: Yale University Press; 1975.
- Holmes J, Gathercole SE, Dunning DL. Adaptive training leads to sustained enhancement of poor working memory in children. *Developmental Science*. 2009; 12:F9–F15. DOI: 10.1111/j.1467-7687.2009.00848.x [PubMed: 19635074]
- Hoyle RH, Stephenson MT, Palmgreen P, Lorch EP, Donohew RL. Reliability and validity of a brief measure of sensation seeking. *Personality and Individual Differences*. 2002; 32:401–414. DOI: 10.1016/S0191-8869(01)00032-0
- Huebner AJ, Howell LW. Examining the relationship between adolescent sexual risk taking and perceptions of monitoring, communication, and parenting styles. *Journal of Adolescent Health*. 2003; 33:71–78. DOI: 10.1016/S1054-139X(03)00141-1 [PubMed: 12890597]
- Jarvis, B. Medialab (Version 2004.2.87). New York, NY: Empirisoft; 2004.
- Kane, MJ., Conway, ARA., Hanbrick, DZ., Engle, RW. Variation in working memory capacity as variation in executive attention and control. In: Conway, ARA, Jarrold, C, Kane, MJ, Miyake, A., Towse, JN., editors. Variation in working memory. New York, NY: Oxford University Press; 2007. p. 21–48.
- Kerr M, Stattin H, Burk WJ. A reinterpretation of parental monitoring in longitudinal perspective. *Journal of Research on Adolescence*. 2010; 20:39–64. DOI: 10.1111/j.1532-7795.2009.00623.x
- Khurana A, Romer D, Betancourt LM, Brodsky NL, Giannetta JM, Hurt H. Early experimentation vs. progression in adolescent drug use: A test of an emerging neurobehavioral imbalance model. *Development and Psychopathology*. 2014:1–13. FirstView.
- Khurana A, Romer D, Betancourt LM, Brodsky NL, Giannetta JM, Hurt H. Working memory ability predicts trajectories of early alcohol use in adolescents: The mediational role of impulsivity. *Addiction*. 2013; 108:506–515. DOI: 10.1111/add.12001 [PubMed: 23033972]
- Khurana A, Romer D, Betancourt L, Brodsky NL, Giannetta JM, Hurt H. Early adolescent sexual debut: The mediating role of working memory ability, sensation seeking and impulsivity. *Developmental Psychology*. 2012; 48:1416–1428. DOI: 10.1037/a0027491 [PubMed: 22369334]

- King KM, Lengua LJ, Monahan KC. Individual differences in the development of self-regulation during pre-adolescence: Connections to context and adjustment. *Journal of Abnormal Child Psychology*. 2013; 41:57–69. DOI: 10.1007/s10802-012-9665-0 [PubMed: 22865096]
- Klingberg T. Training and plasticity of working memory. *Trends in Cognitive Sciences*. 2010; 14:317–324. DOI: 10.1016/j.tics.2010.05.002 [PubMed: 20630350]
- Lansford JE, Yu T, Erath SA, Pettit GS, Bates JE, Dodge KA. Developmental precursors of number of sexual partners from ages 16 to 22. *Journal of Research on Adolescence*. 2010; 20:651–677. DOI: 10.1111/j.1532-7795.2010.00654.x [PubMed: 20823951]
- MacKinnon DP, Lockwood CM, Williams J. Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate behavioral research*. 2004; 39:99–128. DOI: 10.1207/s15327906mbr3901_4 [PubMed: 20157642]
- Madden, GJ., Bickel, WK., editors. *Impulsivity: the behavioral and neurological science of discounting*. Washington, DC: American Psychological Association; 2010.
- Magid V, MacLean MG, Colder CR. Differentiating between sensation seeking and impulsivity through their mediated relations with alcohol use and problems. *Addictive Behaviors*. 2007; 32:2046–2061. DOI: 10.1016/j.addbeh.2007.01.015 [PubMed: 17331658]
- Miller EK, Cohen JD. An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*. 2001; 24:167–202. DOI: 10.1146/annurev.neuro.24.1.167
- Milner B. Interhemispheric differences in the localization of psychological processes in man. *British Medical Bulletin*. 1971; 27:272–277. [PubMed: 4937273]
- Moffitt TE, Arseneault L, Belsky D, Dickson N, Hancox RJ, Harrington H, Caspi A. A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences*. 2011; 108:2693–2698. DOI: 10.1073/pnas.1010076108
- O'Donnell L, O'Donnell CR, Stueve A. Early sexual initiation and subsequent sex-related risks among urban minority youth: The Reach for Health Study. *Family Planning Perspectives*. 2001; 33:268–275. DOI: 10.2307/3030194 [PubMed: 11804436]
- Owen AM, Downes JJ, Sahakian BJ, Polkey CE, Robbins TW. Planning and spatial working memory following frontal lobe lesions in man. *Neuropsychologia*. 1990; 28:1021–1034. DOI: 10.1016/0028-3932(90)90137-D [PubMed: 2267054]
- Pedlow CT, Carey MP. Developmentally appropriate sexual risk reduction interventions for adolescents: Rationale, review of interventions, and recommendations for research and practice. *Annals of Behavioral Medicine*. 2004; 27:172–184. DOI: 10.1207/s15324796abm2703_5 [PubMed: 15184093]
- Pfeifer JH, Allen NB. Arrested development? Reconsidering dual-systems models of brain function in adolescence and disorders. *Trends in Cognitive Sciences*. 2012; 16:322–329. DOI: 10.1016/j.tics.2012.04.011 [PubMed: 22613872]
- Picot J, Shepherd J, Kavanagh J, Cooper K, Harden A, Barnett-Page E, Frampton GK. Behavioural interventions for the prevention of sexually transmitted infections in young people aged 13–19 years: A systematic review. *Health Education Research*. 2012; 27:495–512. DOI: 10.1093/her/cys014 [PubMed: 22350195]
- Poulin C, Graham L. The association between substance use, unplanned sexual intercourse and other sexual behaviours among adolescent students. *Addiction*. 2001; 96:607–621. DOI: 10.1046/j.1360-0443.2001.9646079.x [PubMed: 11300964]
- Raine A, Reynolds C, Venables PH, Mednick SA. Stimulation seeking and intelligence: A prospective longitudinal study. *Journal of Personality and Social Psychology*. 2002; 82:663–674. DOI: 10.1037/0022-3514.82.4.663 [PubMed: 11999930]
- Raver CC, McCoy DC, Lowenstein AE, Pess R. Predicting individual differences in low-income children's executive control from early to middle childhood. *Developmental Science*. 2013; 16:394–408. DOI: 10.1111/desc.12027 [PubMed: 23587038]
- Raykov T. Evaluation of scale reliability for unidimensional measures using latent variable modeling. *Measurement and Evaluation in Counseling and Development*. 2009; 42:223–232. DOI: 10.1177/0748175609344096
- Reyna VF, Farley F. Risk and Rationality in Adolescent Decision Making. *Psychological Science in the Public Interest*. 2006; 7:1–44. DOI: 10.1111/j.1529-1006.2006.00026.x [PubMed: 26158695]

- Reynolds B, Penfold RB, Patak M. Dimensions of impulsive behavior in adolescents: Laboratory behavioral assessments. *Experimental and Clinical Psychopharmacology*. 2008; 16:124–131. DOI: 10.1037/1064-1297.16.2.124 [PubMed: 18489016]
- Reynolds B, Schiffbauer R. Delay of gratification and delay discounting: A unifying feedback model of delay-related impulsive behavior. *Psychological Record*. 2005; 55:439.
- Rhoades BL, Greenberg MT, Lanza ST, Blair C. Demographic and familial predictors of early executive function development: Contribution of a person-centered perspective. *Journal of Experimental Child Psychology*. 2011; 108:638–662. DOI: 10.1016/j.jecp.2010.08.004 [PubMed: 20828709]
- Romer D. Adolescent risk taking, impulsivity, and brain development: Implications for prevention. *Developmental Psychobiology*. 2010; 52:263–276. DOI: 10.1002/dev.20442 [PubMed: 20175097]
- Romer D, Betancourt L, Giannetta JM, Brodsky NL, Farah M, Hurt H. Executive cognitive functions and impulsivity as correlates of risk taking and problem behavior in preadolescents. *Neuropsychologia*. 2009; 47:2916–2926. DOI: 10.1016/j.neuropsychologia.2009.06.019 [PubMed: 19560477]
- Romer D, Betancourt L, Giannetta JM, Brodsky NL, Farah M, Hurt H. Does adolescent risk taking imply weak executive function? A prospective study of relations between working memory performance, impulsivity, and risk taking in early adolescence. *Developmental Science*. 2011; 14:1119–1133. DOI: 10.1111/j.1467-7687.2011.01061.x [PubMed: 21884327]
- Romer D, Duckworth AL, Sznitman S, Park S. Can adolescents learn self-control? Delay of gratification in the development of control over risk taking. *Prevention Science*. 2010; 11:319–330. DOI: 10.1007/s11121-010-0171-8 [PubMed: 20306298]
- Romer D, Hennessy M. A biosocial-affect model of adolescent sensation seeking: The role of affect evaluation and peer-group influence in adolescent drug use. *Prevention Science*. 2007; 8:89–101. DOI: 10.1007/s11121-007-0064-7 [PubMed: 17286212]
- Satterwhite CL, Torrone E, Meites E, Dunne EF, Mahajan R, Ocfemia MCB, Weinstock H. Sexually transmitted infections among US women and men. *Sexually Transmitted Diseases*. 2013; 40:187–193. DOI: 10.1097/OLQ.0b013e318286bb53 [PubMed: 23403598]
- Schneider, W., Eschman, A., Zuccolotto, A. E-Prime user's guide. Pittsburgh, PA: Psychology Software Tools, Inc; 2002.
- Shamosh NA, DeYoung CG, Green AE, Reis DL, Johnson MR, Conway ARA, Gray JR. Individual differences in delay discounting. *Psychological Science*. 2008; 19:904–911. DOI: 10.1111/j.1467-9280.2008.02175.x [PubMed: 18947356]
- Shearer DL, Mulvihill BA, Klerman LV, Wallander JL, Hovinga ME, Redden DT. Association of early childbearing and low cognitive ability. *Perspectives on Sexual and Reproductive Health*. 2002; 34:236–243. DOI: 10.2307/3097822 [PubMed: 12392216]
- Somerville LH, Casey B. Developmental neurobiology of cognitive control and motivational systems. *Current Opinion in Neurobiology*. 2010; 20:236–241. DOI: 10.1016/j.conb.2010.01.006 [PubMed: 20167473]
- Spear, L. *The Behavioral Neuroscience of Adolescence*. New York, NY: W. W. Norton & Company; 2010.
- Squeglia LM, Schweinsburg AD, Pulido C, Tapert SF. Adolescent binge drinking linked to abnormal spatial working memory brain activation: Differential gender effects. *Alcoholism: Clinical and Experimental Research*. 2011; 35:1831–1841. DOI: 10.1111/j.1530-0277.2011.01527.x
- Stautz K, Cooper A. Impulsivity-related personality traits and adolescent alcohol use: A meta-analytic review. *Clinical Psychology Review*. 2013; 33:574–592. DOI: 10.1016/j.cpr.2013.03.003 [PubMed: 23563081]
- Steinberg L. A dual systems model of adolescent risk taking. *Developmental Psychobiology*. 2010; 52:216–224. DOI: 10.1002/dev.20445 [PubMed: 20213754]
- Tanner JM, Whitehouse RH, Marubini E, Resele LF. The adolescent growth spurt of boys and girls of the Harpenden Growth Study. *Annals of Human Biology*. 1976; 3:109–126. DOI: 10.1080/03014467600001231 [PubMed: 1275435]
- Udry JR. Age at menarche, at first intercourse, and at first pregnancy. *Journal of Biosocial Science*. 1979; 11:433–441. DOI: 10.1017/S0021932000012517 [PubMed: 511871]

- Unnever JD, Cullen FT, Pratt TC. Parental management, ADHD, and delinquent involvement: Reassessing Gottfredson and Hirschi's general theory. *Justice Quarterly*. 2003; 20:471–500. DOI: 10.1080/07418820300095591
- Wechsler, D. Wechsler Intelligence Scale for Children® — Fourth Edition (WISC®-IV). San Antonio, TX: The Psychological Corporation; 2003.
- Willoughby T, Good M, Adachi PJC, Hamza C, Tavernier R. Examining the link between adolescent brain development and risk taking from a social-developmental perspective. *Brain and Cognition*. 2013; 83:315–323. [PubMed: 24128659]
- Winstanley CA, Olausson P, Taylor JR, Jentsch JD. Insight into the relationship between impulsivity and substance abuse from studies using animal models. *Alcoholism: Clinical and Experimental Research*. 2010; 34:1306–1318. DOI: 10.1111/j.1530-0277.2010.01215.x

Figure 1.

Potential Pathways of Influence of Working Memory and Parental Variables

Note. Dashed pathways represent negative associations, while bold lines represent positive effects. ‘Parental influences’ were assessed using three separate variables: parental involvement, parental monitoring, and parental SES. ‘Self-control’ was assessed using two separate variables - ‘acting without thinking’ and ‘inability to delay gratification’.

Figure 2.

Final SEM model with standardized coefficients corresponding to key significant pathways of influence.

Note. All paths are significant at $p < 0.05$, unless denoted by [†] $p < 0.06$. Model fit: Chi sq (248) = 330.44, $p < 0.001$, RMSEA (90% CI) = 0.03 (0.02, 0.04), CFI = 0.95, TLI = 0.93. All exogenous predictors were assessed at T1, while the mediators and outcomes were assessed at T2 and T3. Dashed pathways represent negative associations, while bold lines represent positive effects. The effect of age, gender, pubertal development, and race-ethnicity was covaried out. These paths are not shown for the sake of clarity. Path coefficients associated with the significant effects of key variables and covariates can be found in Table 2.

Table 1

Correlations between key variables.

Variable Names	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Corsi block tapping T1 (1)	1.00																
Object two back T1 (2)	0.29	1.00															
Digit span backwards T1 (3)	0.20	0.15	1.00														
Spatial working memory T1 (4)	0.31	0.31	0.24	1.00													
Acting Without Thinking T2 (5)	-0.17	-0.07	-0.01	-0.10[†]	1.00												
Inability to Delay Gratification T2 (6)	-0.02	-0.06	-0.04	-0.06	0.16	1.00											
Sensation Seeking T2 (7)	0.05	0.04	0.10[†]	0.04	0.44	-0.04	1.00										
Acting Without Thinking T3 (8)	-0.22	-0.04	-0.06	-0.10[†]	0.64	0.15	0.26	1.00									
Inability to Delay Gratification T3 (9)	-0.10[†]	-0.15	-0.02	-0.06	0.16	0.52	-0.08	0.17	1.00								
Sensation Seeking T3 (10)	0.06	0.12	0.04	0.06	0.34	-0.04	0.71	0.31	-0.07	1.00							
Parental Monitoring T1 (11)	-0.01	-0.04	0.09	-0.03	-0.15	-0.14	-0.10[†]	-0.09	-0.08	-0.12	1.00						
Parental Involvement T1 (12)	-0.03	-0.10[†]	-0.07	-0.07	-0.19	0.02	-0.15	-0.06	-0.00	-0.08	0.37	1.00					
Parental SES (13)	0.08	0.11	0.14	0.01	-0.16	-0.15	-0.04	-0.13	-0.21	0.07	0.11	0.05	1.00				
Alcohol use T2 (14)	-0.03	0.01	-0.04	0.05	0.38	0.13	0.32	0.20	0.08	0.31	-0.16	-0.19	-0.12	1.00			
Alcohol use T3 (15)	0.09	0.06	-0.01	0.02	0.34	0.14	0.31	0.26	0.08	0.33	-0.07	-0.19	-0.05	0.55	1.00		
Virginity Status T2 (16)	-0.10[†]	-0.01	-0.15	-0.06	0.25	0.23	0.08	0.13	0.23	0.04	-0.14	-0.11	-0.13	0.31	0.25	1.00	
Sexual Risk Involvement T3 (17)	-0.06	-0.03	-0.06	-0.00	0.32	0.21	0.16	0.27	0.16	0.12	-0.17	-0.11	-0.21	0.39	0.44	0.63	1.00
Mean (SD)	6.44 (3.12)	77.18 (8.81)	7.68 (1.88)	24.42 (18.15)	0.41 (0.31)	49.28 (28.76)	2.39 (0.76)	0.36 (0.29)	47.99 (28.44)	2.42 (0.77)	3.68 (0.51)	3.19 (0.62)	47.4 (15.42)	0.66 (0.72)	0.74 (0.85)	0.15 (0.36)	0.36 (0.62)

Note. Values that are significant ($p < 0.05$) are listed in bold.
T1 = Time 1, T2 = Time 2, T3 = Time 3.

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Table 2

Unstandardized path coefficients and standard errors corresponding to significant effects.

Model Pathways	B (SE)	p value
Direct effects on main outcome variable		
Virginity Status (T2) → Sexual Risk Involvement (T3)	0.94 (0.09)	<0.001
Alcohol use (T3) → Sexual Risk Involvement (T3)	0.18 (0.04)	<0.001
AWT (T3) → Sexual Risk Involvement (T3)	0.25 (0.10)	<0.01
Parental Monitoring (T1) → Sexual Risk Involvement (T3)	-0.07 (0.04)	=0.06
Hispanic ethnicity → Sexual Risk Involvement (T3)	0.21 (0.10)	<0.05
Socio-economic status → Sexual Risk Involvement (T3)	-0.004 (0.002)	=0.01
Direct effects on other outcomes and mediators		
Socio-economic status → WM (T1)	0.013 (0.005)	<0.01
WM (T1) → AWT (T2)	-0.08 (0.02)	<0.001
Parental Involvement (T1) → AWT (T2)	-0.06 (0.02)	<0.001
WM (T1) → AWT (T3)	-0.04 (0.02)	<0.01
AWT (T2) → AWT (T3)	0.58 (0.04)	<0.001
WM (T1) → IDG (T2)	-3.63 (1.91)	<0.06
WM (T1) → IDG (T3)	-4.00 (1.78)	<0.05
Socio-economic status → IDG (T3)	-0.18 (0.09)	<0.05
IDG (T2) → IDG (T3)	0.47 (0.05)	<0.001
Parental Involvement (T1) → Alcohol use (T2)	-0.09 (0.04)	<0.05
AWT (T2) → Alcohol use (T2)	0.77 (0.12)	<0.001
AWT (T3) → Alcohol use (T3)	0.48 (0.13)	<0.001
Alcohol use (T2) → Alcohol use (T3)	0.61 (0.05)	<0.001
AWT (T2) → Virginity Status (T2)	0.18 (0.07)	<0.01
IDG (T2) → Virginity Status (T2)	0.002 (0.001)	<0.01
Influence of covariates		
Age → WM (T1)	0.23 (0.10)	<0.05
Age → Virginity Status (T2)	0.10 (0.02)	<0.001
Age → Alcohol use (T2)	0.14 (0.04)	<0.001
Age → AWT (T2)	0.06 (0.02)	<0.01
Age → Parental Monitoring (T1)	-0.18 (0.08)	<0.05
Age → Parental Involvement (T1)	-0.28 (0.09)	<0.001
Female → Parental Monitoring (T1)	0.29 (0.12)	<0.05
Female → WM (T1)	-1.73 (0.93)	=0.06
Pubertal development X Female → WM (T1)	0.41 (0.22)	=0.06
Black racial identity → Virginity Status (T2)	0.12 (0.04)	<0.01
Black racial identity → WM (T1)	-0.77 (0.19)	<0.001
Black racial identity → Alcohol use (T3)	-0.19 (0.09)	<0.05
Total Indirect Effects (using bias-corrected bootstrap re-sampling)		Estimate (95% CI)
WM (T1) → Sexual Risk outcome (T3) (significant indirect pathways included AWT, IDG, and alcohol use as mediators)		-0.05 (-0.09, -0.02)

Model Pathways	B (SE)	p value
<i>WM (T1) → Virginity Status (T2)</i> (significant indirect pathways included AWT and IDG as mediators)	−0.02 (−0.04, −0.01)	
<i>Parental Involvement (T1) → Sexual Risk outcome (T3)</i> (significant indirect pathways included AWT and alcohol use as mediators)	−0.04 (−0.06, −0.02)	

Note. WM = Working Memory; AWT = Acting Without Thinking; IDG = Inability to Delay Gratification.