Physical illness, social disadvantage, and risky sexual behavior in adolescence and young adulthood

Jenna Alley, MS
University of Utah
Department of Psychology
jenna.alley@psych.utah.edu

Rebecca Y. Owen, MS
University of Utah
Department of Sociology
r.owen@utah.edu

Sarah E. Wawrzynski, BSN
University of Utah
College of Nursing
s.wawrzynski@utah.edu

Lauren Lasrich, MS
University of Utah
Department of Family & Consumer Studies
lauren.lasrich@utah.edu

Zobayer Ahmmad, MS
University of Utah
Department of Sociology
zubi.ahmmad@utah.edu

Rebecca Utz, PhD
University of Utah
Department of Sociology
rebecca.utz@soc.utah.edu

*Corresponding Author
Daniel E. Adkins, PhD
University of Utah
Department of Sociology & Psychiatry
daniel.adkins@soc.utah.edu
380 S 1530 E, Salt Lake City, UT 84112, USA.
Office: (801) 581-6153
Fax: (801) 585-3784

Abbreviations:

LHT- Life history theory; SES- Socioeconomic status; DNA- Deoxyribonucleic acid; STD- Sexually transmitted disease; STI- Sexually transmitted infection; MI- Multiple Imputation

Acknowledgements:

We thank the Consortium of Families and Health Research (C-FAHR) at the University of Utah for the graduate fellowship support that led to the development of this paper. We are grateful to Cynthia A. Berg for critical editorial input, Claudia Geist for methodological guidance, and Bruce Ellis for intellectual inspiration. This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this analysis.

Conflicts of interest:

None.

Abstract

Purpose

This study investigates the influence of physical illness on sexual risk-taking behaviors in adolescence and young adulthood, both directly and through moderation of the impact of social disadvantage. We hypothesize positive effects for social disadvantage and physical illness on sexual risk behavior, consistent with the development of fast life history behavioral strategies among young people facing greater life adversity.

Methods

Using the first two waves of the National Longitudinal Study of Adolescent Health, we develop a mixed effects multinomial logistic regression model predicting sexual risk behavior (nonmonogamous sex v. monogamous sex, and nonmonogamous sex v. no sex) by social characteristics, physical illness, interactions thereof, and control covariates. Analyses use multiple imputation to address a modest amount of missing data.

Results

Subjects reporting higher levels of physical illness had lower odds of abstinence v. engaging in nonmonogamous sex (OR=0.79, $p<0.001$), and lower odds of having monogamous sex v. engaging in nonmonogamous sex (OR=0.89, $p<0.001$), compared to individuals in better health. Physical illness significantly moderated the gender (OR=0.85, $p<0.001$), race/ethnicity (e.g., OR=1.23, $p<0.001$), and childhood SES (OR=0.95; $p<0.05$) effects for the abstinent v. nonmonogamous sex comparison. All substantive findings were robust across waves and in sensitivity analyses.

Conclusions

These findings offer general, but not universal, support for the predictions of life history theory. Physical illness and various social disadvantages are generally associated with increased sexual risk behavior in adolescence and young adulthood. Further, analyses show that the buffering effects of several protective social statuses against sexual risk-taking are substantially eroded by physical illness.

Implications and Contribution

Various social disadvantages and physical illness are positively associated with adolescent and young adult nonmonogamous sex. Additionally, the protective effects of female gender, white identity, and socioeconomic advantage against sexual risk-taking in adolescence and young adulthood are substantially reduced by physical illness. Findings elucidate the powerful influence of physical illness on adolescent and young adult sexual behavior.

Keywords: adolescent behavior, risk-taking, burden of illness, sexuality, physical health, trauma, stress, gender

Introduction

Understanding the predictors and process of risky sexual behavior has important public health implications given associations between sexual risk-taking and negative health and quality of life outcomes, including STD/STI and unintended pregnancy[1,2]. Previous research has shown childhood adversity to be a powerful predictor of increased risk-taking during adolescence and young adulthood, including risky sexual behavior[3,4]. This developmental period is of particular interest in the study of sexual risk behavior, as it is characterized by critical transitions including puberty, sexual debut, and sexual identity formation[5]. Related to these developmental changes, there is a well-documented normative increase in risk-taking during adolescence[5]. However, these increases are not evenly distributed across social groups, with comparatively large increases observed among males and disadvantaged groups[6]. Life history theory (LHT) may offer a useful lens for investigating this process, as it provides a comprehensive theoretical framework with testable predictions regarding the influence of adversity, broadly defined, on risky sexual behavior in adolescence and young adulthood[3].

LHT posits that in response to various types of adversity, individuals make biobehavioral trade-offs in their allocation of time and energy to maximize the primary evolutionary outcomes of survival and reproduction (i.e., fitness)[3]. These trade-offs manifest as developmental adaptions to one's environment, which promote fitness via behavioral strategies calibrated by early social environmental experiences, particularly early rearing environment[7]. Of particular interest here is the LHT trade-off of mating v. parenting effort, which may be measured by behavioral indicators such as earlier sexual debut and a greater number of sexual partners[8,9]. Specifically, LHT posits that experiencing more unpredictable/harsh early environments predispose individuals to maximize short term mating, which can readily be achieved in adolescence by earlier sexual debut and engaging in nonmonogamous sex, at the expense of longer term parental investment[10].

The sum of one's life history trade-offs across development may be conceptualized as a *life*

history strategy[3]. Life history strategies are typically conceptualized as a continuum of "fast" to "slow", calibrated by one's time horizon as indicated by the level of unpredictability/harshness (i.e., adversity) in one's early environment[11]. Previous research has shown that early experiences of adverse environments are indeed strong predictors of a fast life history strategy[4,10]. This includes unpredictable environments, including parental job loss, divorce/conflict, and frequent changes in household composition, as well as harsh environmental exposures, such as childhood maltreatment, low socioeconomic status (SES), and racial discrimination[12,13]. Individuals exposed to harsh or unpredictable types of social disadvantage have been shown to, on average, exhibit faster life history strategies than those who are not exposed to such environments[4]. Thus, LHT theorizes that early experiences of social disadvantage signal a shorter time horizon with increased risk of mortality, which encourages the biobehavioral development of fast strategies[1,10,14].

This signal of increased mortality risk is not restricted to external environmental cues. Rather, a burgeoning perspective within LHT has proposed that internal states of health and illness can also signal shorter time horizons with increased risk of mortality, and thus trigger developmental shifts toward fast life history strategies[8,9]. Proposed indictors of such adverse internal states include chronic illness, functional limitations, and disability[8,10]. Thus, if an internal predictor (e.g., illness or disability) signals increased risk of early mortality, this could foster biobehavioral adaptations toward fast LH strategies, including maximizing short term mating at the expense of longer term parental investment[15,16]. The current study aims to elaborate on this growing area of research and represents a notable extension of LHT, as little research to date has empirically considered the impact of physical illness and disability on life history strategies.

We hypothesize: *H1:* Individuals who experience more social disadvantage (e.g., severe childhood adversity [e.g., sexual abuse], racial/ethnic disadvantage, low SES) will be more likely to engage in nonmonogamous sex than those who experience greater social advantage. *H2:* Physical illness

and functional limitations will be positively associated with engaging in nonmonogamous sex. *H3:* Physical illness and functional limitations will increase the impact of social disadvantage on sexual risk-taking.

Methods

Sample

We use data from the first two waves of the National Longitudinal Study of Adolescent Health (Add Health). Add Health is the largest nationally representative contemporary sample of adolescents to young adults in the United States, and includes an over-sampling of racial/ethnic minorities. Sample features have been previously described (http://www.cpc.unc.edu/projects/addhealth/design).[17,18] Briefly, a sample of 20,745 adolescents in grades 7–12 were initially assayed for data collection in 1994–1995 (Wave 1), and then again 18 months later in 1996 (Wave 2). In Wave 1, a questionnaire was administered to a selected residential parent of each adolescent, from which some covariates in the present analysis were derived (e.g., childhood household income, parental educational attainment).

Measures

Sexual risk-taking: We examined two items from Add Health, at each of the two analyzed waves, to examine causal sex as compared to monogamous sex and abstinence. The first question asked whether the individual has ever had sex, and the second asked if the individual has ever had sex outside of a romantic relationship, in the past 12 months. Individuals reporting yes to both items were coded as "nonmonogamous sex"; those reporting that they have had sex, but not sex outside of a relationship in the past 12 months, were coded as "monogamous sex"; and those reporting that they had not had sex were coded as "no sex" (i.e., "abstinent"). Logical inconsistences between items were coded missing and

addressed by multiple imputation (see below).

Physical illness: Physical illness was estimated as a latent variable using the indicators *self-rated health* (single 5-level item), *functional limitations* (≥4 item count), *ability to climb stairs* (single dichotomous item), and *chronic conditions* (dichotomous, any of ≥6 conditions) using confirmatory factor analysis (CFA) (see Supplemental Methods for details). Model fit for the physical illness latent variable was excellent as assessed by all considered fit indices (RMSEA=0.01, CFI=1.00, TLI=0.99). A factor score was output based on the physical illness CFA, and was standardized in all analyses. Given that this construct includes aspects of both physical illness and disability, it is challenging to find a single, succinct umbrella term to describe it[19]. We opt for the term "physical illness" as self-rated health has the strongest standardized loading, by a substantial margin, of the CFA indicators (see Supplemental Table S1), suggesting that physical health/illness is indeed the dimension captured by the CFA.

Severe childhood adversity: Measures of childhood adversity comprised five dichotomous indicators: *sexual abuse, physical abuse, neglect, drugs/alcohol accessible at home, and parental incarceration,* occurring in adolescence. These five severe childhood adversities are described in detail in Supplemental Methods. Briefly, each of the five childhood adversities was retrospectively assessed and queried childhood and adolescent experiences using multiple questionnaire items. A cumulative adversity count ranging from 0 to 5 was used in the primary analysis; sensitivity analyses examining the effects of the individual childhood adversities are presented in Supplemental Table S6.

Sociodemographic characteristics: All presented models included a set of sociodemographic covariates characterizing the adolescent's social background. These included household SES measures for parental education and household income assessed at Wave 1. These measures have been previously described[17], and are described in detail in Supplemental Methods. Both parental education and income were standardized in the analysis. Self-reported race/ethnicity (coded Black, Asian, Hispanic,

White (ref), and Other), nativity (i.e., first-generation immigrant status), and age were also included as covariates in all presented models.

Analytical strategy

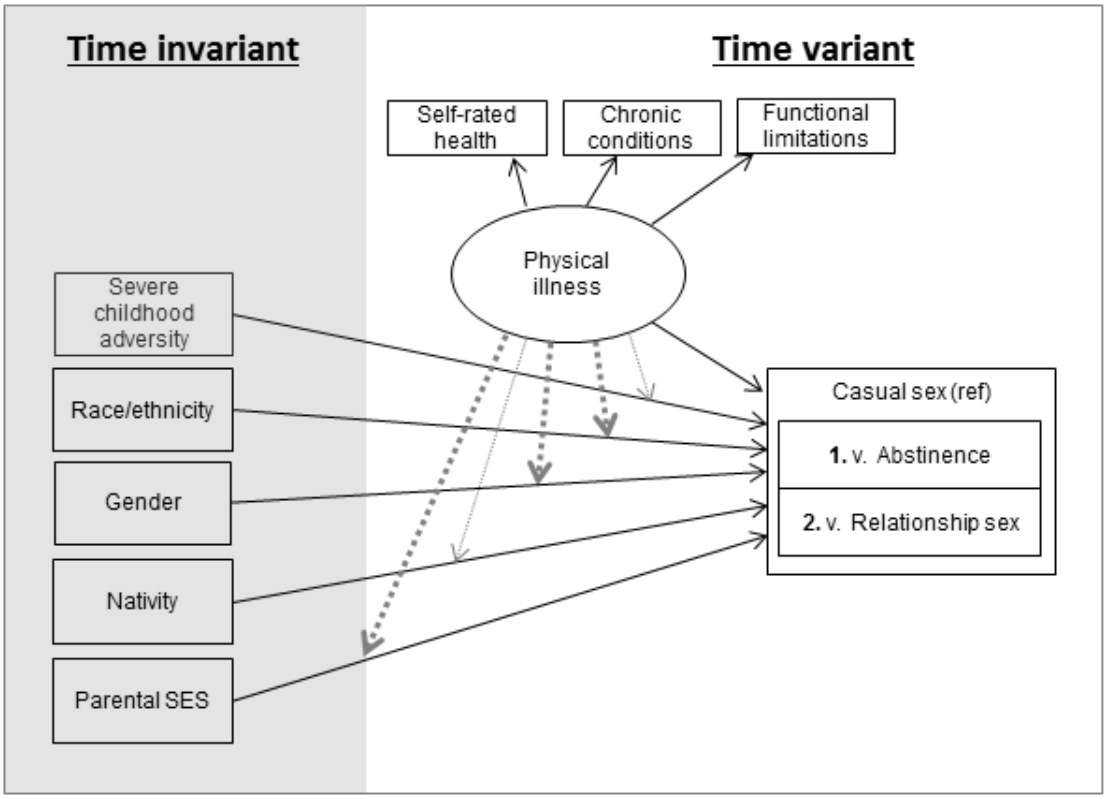
There was a small-to-moderate amount of missing data at both waves (<30% missing for all analysis variable and <4% for most; see Supplemental Table S2). We addressed missing data using the multiple imputation (MI) method, *multiple imputation with chained equations* (*MICE)*.[20,21] We used a conservative 30 imputations in all MI analyses[22]. Additional MI details are presented in Supplemental Methods.

Our primary analytical approach pools data from Waves 1 and 2 to maximize statistical power (wave-specific sensitivity analyses are also reported in Supplemental Tables S3 and S4). Thus, primary inferential analyses use mixed effects multinomial logistic regression. Mixed effects multinomial logistic regression is used to model a polytomous outcome variable with *X* categories (three categories, here), with *X*-1 logistic comparisons (nonmonogamous sex v. monogamous sex, and nonmonogamous sex v. abstinence) using a common reference category (nonmonogamous sex). In each of these comparison, the logged odds of the outcomes are predicted by a linear combination of the predictor variables, with a random intercept at the cluster level specified to account for the multilevel data structure in which observations are nested within subjects. Among other benefits, the random intercept corrects standard errors and coefficients for the nonindependence of observations within subjects.[23]

The model was developed sequentially, beginning with (1) a baseline model of social disadvantage and sociodemographic controls predicting the (logged odds of) trichotomous sexual risk outcome. We follow this baseline model with a (2) second model adding the physical illness construct as a predictor. Next, in order to test whether illness moderates the effects of the social factors, we (3) add interaction between illness and all of the significantly associated social factors from Model 2. In the final

model, (4) we trim nonsignificant (p≥0.05) interactions from the model. The full model is visualized as a simplified path diagram in Figure 1.

Figure 1. Simplified path diagram of the proposed model of moderation, by physical illness, of the effects of social disadvantage on sexual risk behavior. Bold dotted lines represent statistically significant moderation effects, thin dotted lines represent nonsignificant tested moderation effects.



To test the robustness of our substantive results we conducted several sensitivity analyses. These are briefly discussed in the results and documented in Supplementary Materials. Sensitivity analyses include the following: (A) the primary model sequence using complete case analysis (rather than multiple imputation). (B) The primary model sequence stratified by wave. (C) A model substituting the five individual severe childhood adversities (i.e., dichotomous indicators of sexual abuse, physical abuse, neglect, drugs/alcohol in the home, parental incarceration) for the cumulative childhood adversity index.)

Results

Descriptive statistics for the analysis variables are detailed in Table 1. Briefly, abstention was the modal sexual risk category in both waves I (56%) and II (49%). The monogamous sexual risk category increased in frequency notably across waves from 17% (WI) to 32% (WII), while the nonmonogamous category became less frequent (WI: 26%; WII: 19%), but still not uncommon. Illness was standardized in both waves, as was parental educactio nand logged household income. Demographic proportions are reflective of Add Health's minority oversample. The age range across the two waves was approximately 11.6-22.5, with a mean of 16.7. Finally, the mean number of severe childhood adversities was 0.7 and ranged from 0 to 5.

Table 1. Descriptive statistics of analysis variables (MI, m=30; N_j=20774)

Variable	Mean	SD	min	max
Wave I: nonmonogamous sex	0.26	0.44	0	1
Wave II: nonmonogamous sex	0.19	0.39	0	1
Wave I: abstain	0.56	0.5	0	1
Wave II: abstain	0.49	0.5	0	1
Wave I: monogamous	0.17	0.38	0	1
Wave II: monogamous	0.32	0.47	0	1
Wave I illness	-0.11	0.83	-1.82	4.15
Wave II illness	-0.08	0.88	-3.21	4.72
Immigrant	0.08	0.27	0	1
Hispanic	0.17	0.38	0	1
Black	0.22	0.42	0	1
Asian	0.07	0.26	0	1
Other Race	0.03	0.17	0	1
Female	0.51	0.5	0	1
Household Income	0	1	-4.27	4.13
Parental Education	0	1	-3.49	3.02
Age	16.72	1.87	11.64	22.5
Cumulative Severe Adversity	0.7	0.95	0	5

Primary inferential analyses comprise four nested mixed effects multinomial logistic regressions comparing the odds of engaging in monogamous sex (Comparison 1) and abstaining from sex

(Comparison 2), versus the reference category—engaging in nonmonogamous sex. Given the large number of parameters estimated by these multinomial logistic regressions, we primarily focus discussion on the coefficients directly relevant to our hypotheses and avoid the discussion of most nonsignificant coefficients (which are, however, fully documented in Table 2).

Model 1: Social Disadvantage

The first, baseline model in the series includes sociodemographic characteristics (i.e., nativity, race/ethnicity, gender, childhood SES) and cumulative severe childhood adversity, in order to map the effects of social disadvantage on sexual risk behavior. As shown in Table 2, Model 1, first-generation immigrants exhibited increased odds of abstaining rather than engaging in nonmonogamous sex by 119.6% (odds ratio [OR]=2.196, $p<0.001$), relative to US natives; the association was not statistically significant in the monogamous versus nonmonogamous sex comparison. The effects of race/ethnicity on sexual risk-taking were mixed. Compared to their white counterparts, black young people and those who coded as other race/ethnicity (primarily multiracial young people) were less likely to engage in monogamous sex (OR=0.671, $p<0.001$; OR=0.744, $p<0.05$, respectively), and also less likely to abstain (OR=0.382, $p<0.001$; OR=0.690, $p<0.01$, respectively), than to engage in nonmonogamous sex, compared to the white reference group. Conversely, Asian young people were more likely than their white counterparts to engage in monogamous sex (OR=1.259, $p<0.05$) and abstain (OR=2.065, $p<0.001$) compared to having nonmonogamous sex. Females were markedly less likely to engage in nonmonogamous sex than males in both comparisons ($p<0.001$).

Parental educational attainment was significant in Comparison 2—for each standard deviation increase in parental educational attainment, the odds of abstaining, versus engaging in nonmonogamous sex, increased by 18.4% ($p<0.001$). As expected, cumulative severe childhood adversity was positively associated with nonmonogamous sex. For each additional childhood adversity

Table 2. Multinomial Logistic Regression of pooled Wave I & Wave II Data: Monogamous Sex v. Nonmonogamous Sex and Abstaining v. Nonmonogamous Sex (MI, m=30)

		Monogamous	v. Nonmonogamous			Abstinent	v. Nonmonogamous	
Main effects	Model 1	Model 2	Model 3	Model 4	Model 1	Model 2	Model 3	Model 4
Immigrant	1.184	1.153	1.149	1.152	2.196***	2.083***	2.084***	2.077***
	(1.72)	(1.45)	(1.42)	(1.45)	(9.18)	(8.61)	(8.58)	(8.58)
Hispanic	1.018	1.023	1.022	1.023	1.014	1.023	1.025	1.025
	(0.28)	(0.35)	(0.34)	(0.36)	(0.22)	(0.38)	(0.41)	(0.41)
Black	0.671***	0.664***	0.665***	0.666***	0.382***	0.374***	0.385***	0.386***
	(-7.21)	(-7.43)	(-7.35)	(-7.33)	(-18.93)	(-19.38)	(-18.68)	(-18.67)
Asian	1.259*	1.292*	1.289*	1.293*	2.065***	2.182***	2.197***	2.207***
	(2.29)	(2.54)	(2.50)	(2.56)	(7.99)	(8.61)	(8.65)	(8.73)
Other Race	0.744*	0.755*	0.749*	0.755*	0.690**	0.708**	0.708**	0.711**
	(-2.33)	(-2.23)	(-2.28)	(-2.22)	(-3.21)	(-3.00)	(-3.00)	(-2.96)
Female	1.970***	2.021***	2.026***	2.026***	1.847***	1.938***	1.919***	1.920***
	(15.49)	(15.96)	(15.98)	(15.99)	(15.37)	(16.52)	(16.21)	(16.22)
Household Income	0.947	0.942*	0.942*	0.942*	1.044	1.033	1.032	1.032
	(-1.84)	(-2.04)	(-2.06)	(-2.05)	(1.65)	(1.24)	(1.21)	(1.22)
Parental Education	1.051	1.040	1.040	1.039	1.184***	1.161***	1.154***	1.154***
	(1.87)	(1.47)	(1.45)	(1.45)	(7.36)	(6.52)	(6.23)	(6.23)
Age	1.126***	1.128***	1.128***	1.128***	0.649***	0.650***	0.650***	0.650***
	(7.23)	(7.35)	(7.39)	(7.39)	(-28.35)	(-28.25)	(-28.23)	(-28.21)
Severe Child Adversity	0.834***	0.844***	0.847***	0.845***	0.605***	0.617***	0.620***	0.619***
	(-8.09)	(-7.62)	(-7.46)	(-7.57)	(-23.13)	(-22.38)	(-22.12)	(-22.26)
Illness		0.872***	0.906*	0.887***		0.767***	0.797***	0.789***
		(-5.86)	(-2.46)	(-3.52)		(-12.03)	(-6.03)	(-7.54)
Interaction effects								
Illness*Immigrant			0.961				1.004	
			(-0.42)				(0.05)	
Illness*Black			1.040	1.035			1.236***	1.225***
			(0.79)	(0.71)			(4.51)	(4.41)
Illness*Asian			1.026				1.063	
			(0.23)				(0.63)	
Illness*Other Race			1.089				1.027	
			(0.71)				(0.22)	
Illness*Female			0.932	0.929			0.847***	0.845***
			(-1.60)	(-1.68)			(-4.05)	(-4.15)
Illness*Parental Education			0.977	0.983			0.947*	0.952*
			(-0.97)	(-0.76)			(-2.44)	(-2.30)
Illness*Severe Child Adversity			0.972				0.979	
			(-1.23)				(-0.96)	
Intercept	0.183***	0.174***	0.172***	0.172***	5686.0***	5242.8***	5213.3***	5209.6***
	(-6.04)	(-6.28)	(-6.32)	(-6.32)	(34.25)	(34.00)	(34.02)	(33.96)
Random intercept variance	5.942***	5.663***	5.622***	5.621***	5.942***	5.663***	5.622***	5.621***
	(15.18)	(15.01)	(14.98)	(14.97)	(15.18)	(15.01)	(14.98)	(14.97)
N	41548	41548	41548	41548	41548	41548	41548	41548

Exponentiated coefficients; t statistics in parentheses

***p<0.001; **p<0.01; *p<0.05

experienced, the odds of engaging in monogamous sex decreased by 16.6% ($p<0.001$), and the odds of abstaining from sex decreased by 39.5% ($p<0.001$), relative to nonmonogamous sex.

Model 2: Physical Illness Main Effects

Model 2 introduced a physical illness term to Model 1. The direction and magnitude of the sociodemographic and cumulative severe adversity effects remained similar to the baseline model. The results from both Comparison 1 and Comparison 2 indicate that illness is a significant risk factor for engaging in nonmonogamous sex. That is, for each standard deviation increase in physical illness, the odds of abstaining decreased by 23.3% ($p<0.001$), and the odds of engaging in monogamous sex decreased by 12.8% ($p<0.001$), compared to the nonmonogamous reference group.

Model 3: Moderation of Social Disadvantage by Physical Illness

Model 3 includes interaction effects of illness to all of the significant social factors—race/ethnicity, gender, SES, and cumulative severe adversity. In the comparison of monogamous versus nonmonogamous sex (Comparison 1), the direction and magnitude of the effects of the sociodemographic variables and cumulative severe adversity remained substantively unchanged from Model 2 and no interactions were significant. In the comparison of abstinence versus nonmonogamous sex (Comparison 2), all of the main effects for the sociodemographic variables, as well as cumulative severe adversity and illness, remained substantively unchanged. Additionally, a number of interactions were significant. Being ill and having black identity increases the odds of abstaining versus engaging in nonmonogamous sex by 23.6% ($p<0.001$). Being both female and ill decreases the odds of abstaining ($OR=0.847$, $p<0.001$). This implies that the protective factor of being female is attenuated by illness, increasing the probability of engaging in nonmonogamous sex. Similarly, the protective factor of increased parental education is also attenuated by illness, decreasing the probability of abstaining ($OR=0.947$, $p<0.05$) compared to engaging in nonmonogamous sex.

Model 4: Final Model

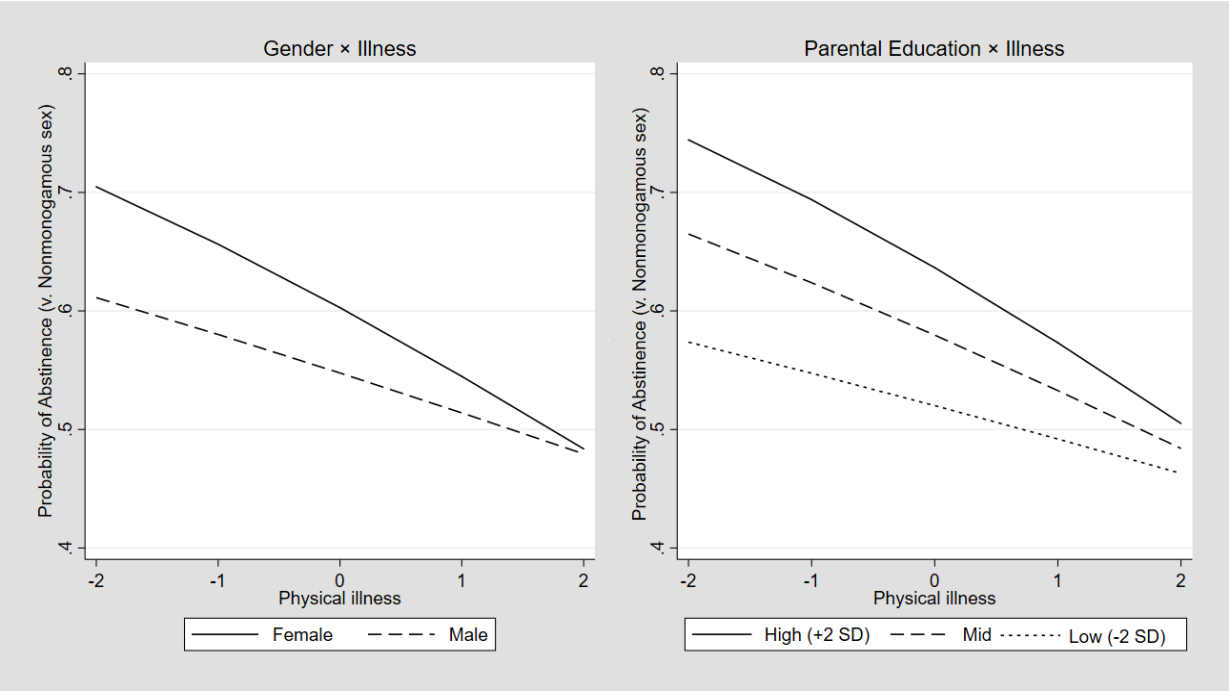
The fourth and final model includes all sociodemographic variables, cumulative severe adversity, illness, and the three significant illness interactions from Model 3. All noted findings from Model 3 where substantively unchanged. Thus, in addition to significant positive associations of several social disadvantages and physical illness to nonmonogamous sex, three interactions were significant in the abstinence versus nonmonogamous sex comparison (Comparison 2). Hence, being both ill and female decreased the odds of abstaining by 15.5% ($p<0.001$; see Figure 2, first panel). Similarly, being ill with higher parental educational attainment decreased the odds of abstaining by 4.8% ($p<0.05$). Thus, again, the protective factor of higher parental education (OR=1.154, $p<0.001$) was diminished by illness (OR=0.789, $p<0.001$; Figure 2, second panel). Finally, black adolescents who were also ill had increased odds of abstaining by 22.5% ($p<0.001$). As with the other two significant interactions visualized in Figure 2, this is another case of physical illness attenuating the protective influence of an advantaged social status, white racial identity in this case.

Various sensitivity analyses were conducted, and the results uniformly support the robustness of our primary analysis (see Supplemental Methods and Supplemental Table S3-S6 for details). Specifically, sensitivity analysis comparing pooled data to wave-specific cross-sectional analyses supported robustness of the model in both mid and late adolescence (Supplemental Tables S3 and S4); comparison of complete case analysis to MI analyses supported our use of multiple imputation (Supplemental Table S5); and analyzing the five severe childhood adversity indicators separately, supported their aggregation into a cumulative index (Supplemental Table S6).

Discussion

This study's primary aim was to examine the interactive impact of social disadvantage and physical

Figure 2. Marginal predicted probabilities of abstinence (v. nonmonogamous sex) visualizing gender and parental education interactions with physical illness



illness on risky sexual behavior using the largest nationally representative sample of US adolescents and young adults. The analysis was guided by life history theory, which suggests that increased risk-taking may serve as an adaptive response to social disadvantage and physical illness, as individuals adapt to potentially abbreviated time horizons to maximize fitness via earlier, and more active, sexual behavior, at the expense of longer term parental investment strategies[4,24,25]. The current results generally support these hypotheses, as the study found that adolescents who report higher levels of physical illness and social disadvantage are, indeed, more likely to engage in nonmonogamous sex compared to either monogamous sex or abstinence. Further, this study found that physical illness attenuates the protective effects of higher SES, female gender, and white identity. Indeed, the most interesting aspect of the analysis was the finding that at high levels of illness, adolescents are relatively likely to engage in nonmonogamous sexual behavior regardless of occupying privileged race, class, and gender statuses. Given that severe illness is one of the strongest and most honest signals of increased mortality risk, it is,

according to the logic of life history theory, unsurprising that we should observe physical illness pervasively eroding the protective effects of social advantages.

Results are consistent with previous research demonstrating that various social disadvantages and adversities are positively associated with sexual risk-taking behavior[2,4,25,26]. Thus, supporting the notion that high levels of environmental uncertainty or harshness can promote fast life history strategies, including heightened sexual risk-taking[3,10]. Moreover, we found several social advantages to be protective against sexual risk-taking. These include parental education—in accordance with previous literature, this study found that adolescents with highly educated parents were less likely to engage in nonmonogamous sex[3,10,27]. Female gender was also found to be highly protective against sexual risk-taking. Females were noted to be less likely to engage in nonmonogamous sex when compared to both monogamous sex and abstinence. This finding was expected, as previous research has consistently indicated that women are less receptive to offers of nonmonogamous sex, and more likely to engage in monogamous sex, than men[28,29]. This is likely due to both social influences constraining young women's sexual behavior, as well as evolutionary influence related to sex differences in required parental investment in the evolutionary environment.

The second substantial finding in this study is the association of physical illness to risky sexual behavior, as measured by nonmonogamous sex, in adolescence and the transition to adulthood. Previous research has examined the differences in sexual behaviors of adolescents with chronic conditions and disability as compared to their healthier and more able-bodied peers. Much of the research conducted on this topic to date has found that adolescents with physical ailments engage is as much or more sex than their healthy counterparts[30-32] LHT suggests that this is because the honest signal of physical illness on mortality risk should be a potent trigger of biobehavioral adaptation toward fast life history strategies, particularly in adolescence and young adulthood. That is, illness signals to the organism that their mortality risk is elevated, and thus encourages the biobehavioral adaptation of fast

life history strategy to maximize fitness via effortful reproduction[10,7,33]. That is not say, however, that there is a general association of health conditions to increased sexual activity. For instance, Kahn and Halpern recently found that among individuals in their 30's, those with physical disabilities were less likely to report having any sex compared to those without disabilities[34]. Future research will be needed to square these findings, and contrasting the current study with Kahn and Halpern[34] indicates several potentially high impact topics for future research including exploring (a) the differential impacts of disability and illness, (b) possible heterogeneity in our nonmonogamous sex finding across developmental periods (e.g., adolescence versus mature adulthood), and (c) the importance of disaggregating the sexually active population into finer grained categories such as monogamous and nonmonogamous sexual activity.

Most importantly, this study found a coherent pattern of attenuation of the protective effects of social advantages by physical illness. Thus, across privileged racial/ethnic, gender, and socio-economic statuses, the protective effects of social advantage are progressively eroded as physical illness increases. While future research will be needed to replicate and extend these findings, they strongly indicate that the influence of physical illness on risky sexual behavior is pervasive and powerful, at least during adolescence and the transition to adulthood.

Finally, it bears explicitly stating that while we generally frame this analysis in the life history perspective, the findings support a more nuanced interpretation, including the influence of cultural and structural factors, as well as evolutionary ones. For example, our finding that first generation immigrants were less likely to engage in nonmonogamous sex than their native peers contradicts the naïve LHT prediction that individuals facing increased social adversity will be more likely to engage in risky sex. Obviously, cultural factors are at play in this instance, and in other features of the results, including the observed racial/ethnic differences. This suggests that future research would be well-served by integrating life history perspectives with established sociological paradigms including stigma theory[35],

the weathering hypothesis[36], and minority stress theory[37].

Limitations

A primary limitation of this study is the difficulty in adjudicating between theoretical explanations, specifically cumulative stress hypothesis and life history theory. While most of our result support LHT claims, these results may also be explained by cumulative stress/risk perspectives, which posit that the accumulation of stress (i.e., social disadvantage and illness) across development will increase externalizing behaviors (including sexual impulsivity) and lower well-being by eroding psychological resources[38]. However, cumulative stress perspectives do not provide strong predictions regarding sexual behaviors, which are not inherently pathological, rather the theoretical focus in the cumulative stress paradigm is primarily on health outcomes. Moreover, there is nothing in cumulative stress theory that would predict the strong moderating effect of illness on social advantages observed here[3,38]. Future research will be needed to more fully adjudicate between cumulative stress and life history perspectives on adolescent sexual behavior.

Additionally, due to the nature of retrospective self-report questionnaires, the current study is vulnerable to limitations such as recall bias, which could influence the analysis results. This is a universal limitation of observational research; however, relatively short windows for retrospection (typically 1 year or less), a very large sample size, and repeated assessments per individual ensure that the current study does not suffer disproportionately from this limitation. Another noteworthy limitation stemmed from inconsistencies in measurement across waves in Add Health, which needed to be harmonized to allow proper statistical analysis (e.g., the exact measures of functional limitations varied across waves). However, the strongest indicator of the physical illness latent variable, self-rated health, as well as some functional limitation items and chronic conditions were assessed at every wave. Psychometric best practices were employed to accommodate variations in the illness indicators data wave collection[39].

Despite these limitations, the current study provides important insight into how internal predictors, such as physical illness, affect sexual risk-taking behaviors in adolescence.

Conclusion

Research examining risk-taking behavior has emphasized the need to understand how social disadvantage and adversity influences risk-taking behavior. Here we have extended this literature by examining the interacting effects of social and physical adversity on sexual risk behavior. Findings from this study advance our understanding of how illness impacts risk-taking behavior, and notably supports and extends life history theorizing regarding sexual behavior trade-offs in adolescence[40,41]. These findings suggest the need for further research exploring the relationship between physical illness and risk-taking behaviors across the life course. Additionally, future work in this area should focus on mental health and specific childhood disease states, as well as the influence of culturally specific stressors on life history strategies and risk-taking. Being able to identify adolescents at increased risk can identify areas for intervention from parents, healthcare providers, and policy makers in order to support children's transition into healthy adult lives.

References

1. Edwards WM, Coleman E. Defining sexual health: a descriptive overview. *Archives of sexual behavior.* 2004;33(3):189.
2. Ryan RM, Mendle J, Markowitz AJ. Early Childhood Maltreatment and Girls' Sexual Behavior: The Mediating Role of Pubertal Timing. *Journal of Adolescent Health.* 2015;57(3):342-347.
3. Del Giudice MG, S.W., Kaplan, H.S. Life History and Evolutionary Psychology. In: Buss DM, ed. *The Handbook of Evolutionary Psychology.* Vol 1. 2nd ed. New York, NY: Wiley; 2015:88-114.
4. Simpson JA, Griskevicius V, Kuo SIC, Sung S, Collins WA. Evolution, Stress, and Sensitive Periods: The Influence of Unpredictability in Early Versus Late Childhood on Sex and Risky Behavior. *Developmental Psychology.* 2012;48(3):674-686.
5. Coley RL, Votruba-Drzal E, Schindler HS. Fathers' and Mothers' Parenting Predicting and Responding to Adolescent Sexual Risk Behaviors. *Child Development.* 2009;80(3):808-827.
6. Hagan J, Foster H. S/He's a rebel: toward a sequential stress theory of delinquency and gendered pathways to disadvantage in emerging adulthood. *Social Forces.* 2003;82(1):53-86.
7. Chisholm JS, Ellison PT, Evans J, et al. Death, Hope, and Sex: Life-History Theory and the Development of Reproductive Strategies [and Comments and Reply]. *Current Anthropology.* 1993;34(1):1-24.
8. Griskevicius V, Delton AW, Robertson TE, Tybur JM. Environmental Contingency in Life History Strategies: The Influence of Mortality and Socioeconomic Status on Reproductive Timing. *Journal of personality and social psychology.* 2011;100(2):241-254.
9. Ellis BJ, McFadyen-Ketchum S, Dodge KA, Pettit GS, Bates JE. Quality of Early Family Relationships and Individual Differences in the Timing of Pubertal Maturation in Girls: A Longitudinal Test of an Evolutionary Model. *Journal of personality and social psychology.* 1999;77(2):387-401.
10. Szepsenwol O, Griskevicius V, Simpson JA, Young ES, Fleck C, Jones RE. The effect of predictable early childhood environments on sociosexuality in early adulthood. *Evolutionary Behavioral Sciences.* 2017;11(2):131-145.
11. Kaplan HS, Gangestad SW. Life history theory and evolutionary psychology. *The handbook of evolutionary psychology.* 2005:68-95.
12. Belsky J, Schlomer GL, Ellis BJ. "Beyond cumulative risk: Distinguishing harshness and unpredictability as determinants of parenting and early life history strategy": Correction.(Special Section: Beyond Mental Health: An Evolutionary Analysis of Development Under Risky and Supportive Environmental Conditions)(Correction notice)(Author abstract). *Developmental Psychology.* 2012;48(3):686.
13. Schafer MH, Ferraro KF, Mustillo SA. Children of Misfortune: Early Adversity and Cumulative Inequality in Perceived Life Trajectories. *American Journal of Sociology.* 2011;116(4):1053-1091.
14. Ellis BJ, Oldehinkel AJ, Nederhof E. The adaptive calibration model of stress responsivity: An empirical test in the Tracking Adolescents' Individual Lives Survey study. *Development and Psychopathology.* 2017;29(3):1001-1021.
15. Hill S, Boehm G, Prokosch M. Vulnerability to Disease as a Predictor of Faster Life History Strategies. *Adaptive Human Behavior and Physiology.* 2016;2(2):116-133.
16. Waynforth D. Life-history theory, chronic childhood illness and the timing of first reproduction in a British birth cohort. *Proceedings Of The Royal Society B-Biological Sciences.* 2012;279(1740):2998--3002.
17. Adkins DE, Wang V, Dupre ME, van den Oord E, Elder GH. Structure and Stress: Trajectories of Depressive Symptoms across Adolescence and Young Adulthood. *Social Forces.* 2009;88(1):31-60.

18. Adkins DE, Wang V, Elder GH. Stress processes and trajectories of depressive symptoms in early life: Gendered development. In: Turner HA, Schieman S, eds. *Advances in Life Course Research: Stress processes across the life course* Boston: Elsevier JAI; 2008:107-134.
19. Organization WH. *International Classification of Functioning, Disability and Health (ICF).* Geneva, Switzerland: World Health Organization; 2001.
20. White IR, Royston P, Wood AM. Multiple imputation using chained equations: issues and guidance for practice. *Statistics in medicine.* 2011;30(4):377-399.
21. Azur MJ, Stuart EA, Frangakis C, Leaf PJ. Multiple imputation by chained equations: what is it and how does it work? *International journal of methods in psychiatric research.* 2011;20(1):40-49.
22. von Hippel PT. How Many Imputations Do You Need? A Two-stage Calculation Using a Quadratic Rule. *Sociological Methods & Research.* 2018:0049124117747303.
23. Singer JD, Willett JB. Applied longitudinal data analysis: Modeling change and event occurrence. Oxford university press; 2003.
24. Hurst JE, Kavanagh PS. Life history strategies and psychopathology: the faster the life strategies, the more symptoms of psychopathology. *Evolution and Human Behavior.* 2017;38(1):1-8.
25. Hartman S, Li Z, Nettle D, Belsky J. External-environmental and internal-health early life predictors of adolescent development. *Development and psychopathology.* 2017;29(5):1839.
26. James J, Ellis BJ, Schlomer GL, Garber J. Sex-Specific Pathways to Early Puberty, Sexual Debut, and Sexual Risk-taking: Tests of an Integrated Evolutionary–Developmental Model. *Developmental Psychology.* 2012;48(3):687-702.
27. Ritterman Weintraub ML, Fernald LCH, Adler N, Bertozzi S, Syme SL. Perceptions of Social Mobility: Development of a New Psychosocial Indicator Associated with Adolescent Risk Behaviors. *Frontiers in Public Health.* 2015;3:62.
28. Grello CM, Welsh DP, Harper MS. No strings attached: The nature of casual sex in college students. *Journal of Sex Research.* 2006;43(3):255-267.
29. Clark RD. HE. Gender Differences in Receptivity to Sexual Offers. *Journal of Psycology and Human Sexuality.* 1989;2(1):39-55.
30. Suris J-C, Parera N. Sex, drugs and chronic illness: health behaviours among chronically ill youth. *The European Journal of Public Health.* 2005;15(5):484-488.
31. Surís J-C, Michaud P-A, Akre C, Sawyer SM. Health risk behaviors in adolescents with chronic conditions. *Pediatrics.* 2008;122(5):e1113.
32. Cheng MM, Udry JR. Sexual behaviors of physically disabled adolescents in the United States. *Journal of Adolescent Health.* 2002;31(1):48-58.
33. Anderson KG. Adverse Childhood Environment: Relationship With Sexual Risk Behaviors and Marital Status in a Large American Sample. *Evolutionary psychology : an international journal of evolutionary approaches to psychology and behavior.* 2017;15(2):1474704917710115.
34. Kahn NF, Halpern CT. Experiences of vaginal, oral, and anal sex from adolescence to early adulthood in populations with physical disabilities. *Journal of Adolescent Health.* 2018;62(3):294-302.
35. Link BG, Phelan JC. Conceptualizing Stigma. *Annual Review of Sociology.* 2001;27(1):363-385.
36. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis.* 1992;2(3):207-221.
37. Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans. A biopsychosocial model. *Am Psychol.* 1999;54(10):805-816.
38. Appleyard K, Egeland B, Dulmen MHM, Alan Sroufe L. When more is not better: the role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry.* 2005;46(3):235-245.

39. Bauer DJ, Hussong AM. Psychometric approaches for developing commensurate measures across independent studies: traditional and new models. *Psychol Methods.* 2009;14(2):101-125.
40. Ellis BJ, Del Giudice M. Beyond allostatic load: Rethinking the role of stress in regulating human development. *Development and Psychopathology.* 2014;26(01):1-20.
41. Ellis BJ, Del Giudice M, Dishion TJ, et al. The evolutionary basis of risky adolescent behavior: Implications for science, policy, and practice. *Developmental Psychology.* 2012;48(3):598-623.