Reproductive Outcomes Among Women with Eating Disorders or Disordered Eating Behavior: Does Methodological Approach Shape Research Findings?

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Abstract

Background: There is a well-documented link between eating disorders (EDs) and adverse health outcomes, including fertility difficulties. These findings stem largely from clinical data or samples using a clinical measure (e.g., diagnosis) of EDs, which may limit our understanding of how EDs or disordered eating behaviors (DEBs) shape female fertility.

Methods: We compared reproductive outcomes from two longitudinal data sources, clinical and population-based data from the Utah Population Database (UPDB) ($N=6,046$), and nonclinical community-based data from the National Longitudinal Study of Adolescent to Young Adult Health (Add Health) ($N=5,951$). We examined age at first birth using Cox regression and parity using negative binomial regression.

Results: Using the UPDB data, women with diagnosed ED experienced later ages of first birth (hazard rate ratio [HRR] = 0.38; $p < 0.01$) and lower parity (incidence rate ratio [IRR] = 0.38; $p < 0.01$) relative to women without EDs. Using the Add Health sample, women who self-reported DEB experienced earlier age of first birth (HRR = 1.16; $p < 0.05$) and higher parity (IRR = 1.17; $p < 0.01$) relative to women without DEB.

Conclusions: Conflicting results suggest two sets of mechanisms, physical/biological (sex specific) and social/behavioral (gender specific), may be simultaneously shaping the reproductive outcomes of women with histories of EDs or DEB. Discipline-specific methodology likely shapes Women's Health research outcomes.

Keywords: eating disorders, reproductive health, age at first birth, parity, disordered eating behaviors, fertility

Introduction

AMONG GENDER SCHOLARS, it is preferred to distinguish between biological sex (male/female) and gender as a social construct (man/woman).¹ Similarly, within Women's Health research, both sex—the biological aspects of being female assigned at birth, and gender—the social norms, cultural expectations, normative behaviors of being a woman, play important, independent, and interdependent roles in the health of women.² Mainstream language conventions, however, continue to conflate sex and gender, such as in national surveys³ and the use of “male/female to female/male” when denoting transgender identities.⁴ We approach this study of women's reproductive outcomes with careful attention to the difference between sex and gender.

A clinical framework is commonly used in research examining eating disorders (EDs) and disordered eating behavior (DEB; the behavioral symptoms of ED that may or not meet diagnostic thresholds) among women, particularly when fertility is the outcome. Overwhelmingly, clinical research of reproductive outcomes among women with ED or DEB examines female sex, rather than gender, emphasizing woman's biological and physical ability to complete pregnancy successfully. This literature highlights the well-documented link between EDs, reduced fecundability (probability of pregnancy in a menstrual cycle), and infertility (inability to achieve pregnancy).^{5–8} Clinical studies of EDs generally examine fertility as a discrete event due to clinical sampling techniques and/or the use of a single fertility event as an outcome; however, women's reproductive years span many decades.

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Clinical studies, despite being of short duration, provide incredible insights into the potential biological disruptions of female fertility by an ED^{4,9}; however these studies often omit the importance of gender, and decontextualize fertility from broader social contexts. Female fertility is both a biological and a social process; reproduction is a social behavior tied to individual and societal family preferences,^{10,11} with motherhood representing a gender-specific rite of passage normatively expected of women.¹² Therefore, a social science framework approach to studying EDs and women's reproductive outcomes can enrich our understanding of how EDs shape long-term fertility. Yet, few studies examine the relationship between EDs and fertility beyond a clinical framework.

This study addresses limitations in the literature by comparing two different samples of women that were drawn using two different scientific approaches. We use two data sources: (1) a nationally-representative sample of women that measures DEB in addition to self-reported diagnoses from the National Longitudinal Study of Adolescent to Young Adult Health (Add Health), a data set widely used by social scientists, and (2) a population-based sample of the State of Utah that includes clinical measures of EDs (diagnosis) from the Utah Population Database (UPDB) which was developed by biomedical scientists. We examine age at first birth and parity (number of children) of women to assess the influence of ED or DEB on long-term reproduction. Comparing analyses from these two samples will improve understandings of how disciplinary approach may shape research findings. Awareness of how methodology shapes assumptions is vital to provide a more complete understanding of women's health across the life course and to better address their healthcare needs.

EDs, DEBs, and fertility

EDs, defined as a range of psychological disorders of abnormal or disturbed eating habits by the DSM-5,¹³ often begin in adolescence, with an average age of diagnosis at 17–18 years.^{14,15} Although an estimated 2.7 per 100,000 adolescents between the ages of 13 and 18 in the United States have an ED¹⁶—anorexia (AN), bulimia (BN), and binge eating disorder (BED) being most common—many youth go undiagnosed. EDs often persist chronically into adulthood and approximately two-thirds of adults fully recover.¹⁶

DEB includes the behavioral symptoms of ED, such as purging, binge eating, fasting, and is more common than diagnosed ED.¹⁶ DEBs are clinically relevant independent of whether they evolve into an ED based on diagnostic criteria.¹⁶ The high prevalence of adolescent DEB and their correlation with adverse health outcomes illustrates that DEB has long-lasting impacts on well-being.^{17,18}

The etiology of EDs and DEB includes both social-familial¹⁹ and biological risk factors,^{20,21} with gender representing the strongest single predictor.²⁰ EDs and DEB are associated with a multitude of deleterious health outcomes²²; in addition to increased morbidity and mortality among women of childbearing age,^{23,24} a history of EDs is linked to lifetime fertility issues.⁶ Women with a history of EDs, including AN, BN, BED, and related behavior, may experience low and high body weight and irregular or absent menstruation.^{6,22,25–28} Some research indicates that EDs are associ-

ated with reduced parity and greater likelihood of being childless.⁷ Although the physical consequences of EDs are associated with reduced fecundability and fertility,^{6,22,29} women with EDs often can still conceive.³⁰

While there are numerous adverse physical health outcomes of EDs, fertility is also a socially influenced behavior.^{11,12} Parenthood represents a culturally valued marker of adulthood^{31,32} that shapes women's identities. Social processes across the life course may influence women with histories of EDs or DEB and their reproductive health; community studies indicate that women with histories of AN, BN, BED, and related behaviors are more likely to identify pregnancies as unplanned and have multiple children in early life.^{9,33} This is unsurprising given evidence that women with EDs—particularly BED and related behavior—are more likely to engage in risk-taking behaviors commonly associated with unplanned pregnancies and early fertility.^{18,31,34,35} This indicates a need for research on the long-term reproductive outcomes of EDs and DEB outside of the clinical setting.

Disciplinary framework and research outcomes: clinical versus nonclinical sampling and measurement

In “The Clinician's Illusion,” Cohen and Cohen³⁶ articulate that varying findings between clinicians and researchers are tied to differences in sampling methodologies. Sample type influences the generalizability of a research study, yet clinical research is often generalized to nonclinical populations.³⁶ Clinical sampling typically involves populations currently suffering from a disease (a prevalence sample), including individuals seeking care who are clinically diagnosed as exhibiting symptomatology. Studies relying on clinical samples have limited generalizability.³⁵ Community or population samples commonly involve populations who have ever experienced the disease (an incidence sample), who may not be seeking care, and often utilize self-reports.³⁵

Women's Health research examining the fertility consequences of EDs has mostly examined clinical samples.^{10,37} Those studies that do use population or community samples³⁸ often still utilize clinical measurements of EDs, particularly diagnosis. Largely, EDs are underdiagnosed.³⁹ For example, racial/ethnic minorities are equally likely to present ED symptomatology, including DEB, yet may be less likely to be diagnosed.^{40,41} This reduces the generalizability of study findings that may utilize population or community samples, yet rely on clinical measurements.

It is difficult to examine the influence of clinical EDs within community or population samples, given that there are small subsamples of individuals diagnosed with an ED. This has led scholars to use proxy measures, such as scales measuring risk factors or DEB.^{18,42} Measuring DEBs indicative of EDs in community-based surveys moves beyond utilization of medical services. This method may capture underrepresented individuals in clinical samples, such as impoverished women or women of color.⁴³ A problem with using DEB as proxy measures, even though they have clinical relevancy and may affect one's life trajectory,^{31,33,42} is that DEB may not be as severe or long term as diagnosed EDs. Furthermore, individual self-reports often generate higher scores for DEB than clinical interviews.⁴⁴ Therefore, both clinical and nonclinical sampling designs and measurements may shape inferences researchers make about the effect of EDs on female reproduction.

Prior research has primarily examined fertility at a discrete time point, rather than across the life span.⁹ Clinical research largely focuses on the influence of EDs on the biology of fertility (menstruation, fecundability, *etc.*), which illustrates an emphasis on the sex-specific health implications of an ED or DEB. However, it is important to study the overall reproductive experiences of women with EDs, given that fertility and reproduction involve both biological sex-specific characteristics and gender-specific behaviors across woman's reproductive years. Long-term reproductive outcomes of women with EDs, including birth timing and parity, have implications for life opportunities, including employment and education,^{45,46} their future health and well-being,³⁵ and their children's health.⁴⁷

Current study

We examine fertility timing and parity of women within two separate longitudinal samples: community-based Add Health data and population-based UPDB data. We anticipate that the measurement and sampling differences between the datasets will result in varying outcomes of the association between EDs or DEB and reproductive outcomes. Because the UPDB sample is clinically based and includes a clinical ED measurement, we anticipate that EDs will be associated with later ages at first birth and lower parity, similar to findings in other clinical studies. However, we anticipate a weaker, or nonpresent, association between self-reported ED or DEB and reproductive outcomes among women in the Add Health, community-based, sample. Given the use of a proxy measure (DEBs) and ED self-reports in Add Health, their ED or DEB may be less severe/prolonged compared to women diagnosed with EDs.

Method

Data sources

Add Health. We utilized the restricted-use full sample of the Add Health survey, Waves I (1994–1995), III (2001–2002), and IV (2008–2009). Add Health followed American adolescents over 14 years at four “waves.” Wave I sampled 132 schools which were nationally representative for region, urbanity, size, and ethnicity between 1994 and 1995. A total of 20,074 adolescents in grades 7–12 at Wave I were surveyed using self-administered in-school questionnaires. In-home interviews were conducted with participants at Wave III and Wave IV. Due to attrition, Wave IV included ~15,700 of the original sample. The full study design is described elsewhere.⁴⁸

The UPDB. The UPDB has comprehensive coverage of the Utah population and is based on linked vital records into multigenerational family trees, medical records from the University of Utah Hospitals and Clinics (UUHC), statewide hospitalization records from the Utah Department of Health (UDOH), and height, weight, and residence information from the Driver's License Division (DLD). Medical, hospitalization, and DLD records are available from 1995 to present. The use of UPDB data was approved by the University of Utah's Resource for Genetic and Epidemiologic Research and its institutional review board.

The analytic UPDB data include women over the age of 15 treated for an ED between the ages of 12 and 32. There are 2,933 women captured in the UPDB treated for an ED between 1995 and 2015. These individuals were matched to three randomly selected individuals in UPDB who were never treated for an ED and who are not biologically related, but match on five key sociodemographic characteristics (*i.e.*, age, sex, race, ethnicity, and religious affiliation) to generate a comparison population of 8,799 women, leaving a final sample of 11,732 women.

Analytic samples

Add Health. Of those participating in Waves I, III, and IV, 8,352 respondents were female. Of these 8,352 women, 2,400 had missing values on key variables, such as race/ethnicity ($n=642$), body mass index (BMI, $n=1,387$), or ED or DEB ($n=1,296$). After list-wise deletion, 5,951 women remained, including 1,430 women who self-report ED diagnosis or engage in DEB (at Wave III) and a group of 4,521 female peers without self-reported ED or DEB.^a In the second set of analyses examining age at first birth, women who gave birth before Wave III are excluded to address potential violations of time-order causality (~16% of the sample). This yields an analytic subsample of 4,061 women, 911 of who self-report an ED or DEB and 3,150 peers who do not report ED or DEB.

Utah population database. Of the original 2,933 women diagnosed with an ED, 1,208 had missing values on key covariates, such as BMI and household income. After list-wise deletion, an analytic sample of 1,725 women diagnosed with an ED and 4,321 general-population matches without an ED was generated ($n=6,046$).^b In the second set of analyses examining age at first birth, women who had a child/right censored before age 18 (8%), or who were diagnosed with an ED after the birth of their first child (5%), are excluded for comparison with the Add Health sample. This yields a subsample of 1,231 women who were diagnosed with an ED and 2,515 general population matches. Figure 1 provides a flow chart for analytic sample selection.

Measures

The following section describes the measures from both samples. Table 1 presents measurement similarities and differences across the two samples.

Add Health

Dependent variables. Parity was measured at Wave IV (when respondents were an average age of 28 [range: 24–33]) and is a count of total live births.

Age at first birth was measured in years. For Cox proportional hazard models, we exclude women whose first birth occurred before Wave III to establish time-order causality. Right censoring is set to age of subject at Wave IV for childless subjects.

^aSupplemental analyses (not shown) indicate that Hispanics were more likely to be excluded from the analytic sample.

^bThe analytic sample was not significantly different from the original sample based on sociodemographic characteristics.

FIG. 1. Flowchart of analytic samples: Utah Population Database and Add Health.

Primary independent variable. Self-identified ED diagnosis was assessed with a single yes/no question, “have you ever been diagnosed with an eating disorder?” To select individuals who report engaging in DEBs, we first identified individuals participating in unhealthy weight compensatory behaviors. Respondents were asked, “During the past seven days what did you do to keep from gaining weight?” For example, individuals who reported, “made yourself vomit, fasted or skipped meals, took laxatives, took diet pills, or diuretics,” were coded as having DEBs. Those who identified, “eaten so much in a short period of time that [they] would have been embarrassed if others had seen them do it, in the past seven days” were also coded as having a DEB. Questions related to ED diagnosis and DEBs were only asked in Wave III.

Covariates. Race and ethnicity compare self-identified Non-Hispanic Whites, Hispanics, and Non-Hispanic other.

BMI was calculated from height and weight data measured at Wave III.

Household Income of respondents at Wave III is an ordinal measure of household income ranging from 1 “<\$10,000” to 8 “+\$75,000.”

UPDB

Dependent variables. Parity was a count of total live births by age 32 taken from birth certificates. Parity cutoff age was set to 32 years of age to match the Add Health data at Wave IV.

Age at first birth was measured in years, with right censoring set at age 32 for childless subjects or age of death/age of emigration from Utah for childless subjects under age 32. We excluded women whose first birth was before age 18 for comparison with the Add Health sample and preserve time order causality.

Primary independent variable. ED was measured using hospitalization (UDOH) and outpatient (UUHC) medical records. Individuals with records indicating primary diagnosis of ICD-9 code 307.1 (AN), ICD-9 Code 307.51 (BN),

and ICD-9 307.50 (ED not otherwise specified [EDNOS]) were identified as having been diagnosed with an ED. We restricted the ED sample to those diagnosed between ages 12 and 32 years (average age at first diagnosis = 21.8 years, standard deviation [SD] = 5.32 years).

Covariates. Race and ethnicity is a categorical variable comparing Non-Hispanic Whites, Hispanics, and Non-Hispanic Other, taken from vital records.

BMI was calculated using height and weight information from individuals’ hospital records (at time of ED diagnosis) or using earliest available height and weight information of driver’s license issued after the age of 16 or before age 32 years (average age at measurement = 21.16, SD = 4.98).

We adjusted for average household income at the census block level using available driver’s license information. Household income is an ordinal measure of eight average estimated categories ranging from 1 “<\$10,000” to 8 “+\$75,000”. This information was only available at a single time point for each respondent (year 2000; average age at measurement = 21.74 years, SD = 5.86) and was transformed into ordinal categories to match the household income measurement within Add Health.

Analytic strategy

First, we present descriptive statistics for both the Add Health and UPDB samples. Second, we assess the association between ED or DEB and Parity for both samples using negative binomial regression. Negative binomial regression is appropriate for count variables with overdispersion (such as number of births).⁴⁹ Third, we assess the relationship between ED or DEB and Age at first birth for both analytic subsamples (limited to births after Wave III for Add Health and after age 18 for UPDB) using Cox proportional hazard modeling. Cox regression is appropriate for event analyses (such as time until first birth).⁵⁰ We stratify models by birth year to adjust for potential confounding cohort effects. Regression and hazard analyses are presented in series of three step-wise models starting with a

TABLE 1. COMPARISON OF ADD HEALTH AND UTAH POPULATION DATABASE MEASURES

	<i>Add Health</i>	<i>UPDB</i>	<i>Parity or timing analyses</i>
Key independent variables			
Eating disorder psychopathology	Includes women who self-report ever having been diagnosed or engage in at least one eating disorder behavior between the ages of 18 and 26 (Wave III). Comparison group includes women without eating disorder or disordered eating behavior.	Includes women who have at least one outpatient or inpatient (hospitalization) record of eating disorder diagnosis of anorexia nervosa, bulimia nervosa, or eating disorder-not otherwise specified, after the age of 12, but before the age of 32. Comparison group is a random sample of women matched on age and race/ethnicity who have not had an eating disorder diagnosis or event. Timing analysis excludes individuals diagnosed with an eating disorder after the birth of their first child/right censor.	Both
Dependent variables			
Number of children	Total number of children born to women within the sample by ages 24–32 (Wave IV).	Total number of children born to women within the sample by age 32.	Parity analysis only
Age at first birth	Measured in years at first birth by Wave IV (ages 24–32). Excludes women whose first birth occurred before Wave III (ages 18–26). Right censoring is set to age at Wave IV for childless women.	Measured in years at first by age 32. Excludes women whose eating disorder diagnosis occurred before birth of first child. Right censoring is set to age 32 or age last known to be in Utah for childless women.	Timing analysis only
Covariates			
Race/ethnicity	Compares Non-Hispanic Whites to Hispanics and non-Hispanic other.	Compares Non-Hispanic Whites to Hispanics and non-Hispanic other.	Both
Married before first child	Compares those who were not married before the birth of their first child or age at censor to women who were born before the birth of their first child or age at censor.	Compares those who were not married before the birth of their first child or age at censor to women who were born before the birth of their first child or age at censor.	Timing analysis
Marital status	Compares those who were married by Wave IV (ages 24–32) to those who were not married by Wave IV.	Compares those who were married by age 32 to those who were not married by age 32.	Parity analysis
Body mass index	Calculated using measured height and weight information at Wave III (ages 18–26). For those with missing measurement data, self-reported height and weight information provided at Wave III was utilized.	Calculated using clinically measured height and weight information at first eating disorder measurement by age 32. For those missing clinical measurement of height and weight, driver's license data were utilized. Timing	Both

(continued)

TABLE 1. (CONTINUED)

<i>Add Health</i>		<i>UPDB</i>	<i>Parity or timing analyses</i>
		analysis excludes individuals whose only known height and weight information was gathered after first birth or right censoring.	
Household income	Household income ordinal category of self-reported annual household income ranges: 1 “<\$10,000” 2 “\$10,000–\$14,999” 3 “\$15,000–\$19,999” 4 “\$20,000–\$29,999” 5 “\$30,000–\$39,999” 6 “\$40,000–\$49,999” 7 “\$50,000–\$74,999” 8 “+\$75,000.” Measured at Wave III.	Household income ordinal category of eight average annual household income ranges measured at the census block level: 1 “<\$10,000” 2 “\$10,000–\$14,999” 3 “\$15,000–\$19,999” 4 “\$20,000–\$29,999” 5 “\$30,000–\$39,999” 6 “\$40,000–\$49,999” 7 “\$50,000–\$74,999” 8 “+\$75,000”.	Both

UPDB, Utah population database.

TABLE 2. DESCRIPTIVE STATISTICS OF SAMPLES

	<i>Add Health</i>		<i>Utah population database</i>	
	<i>Analytic sample</i> (N = 5,951) % or Mean (SD)	<i>Analytic subsample^a</i> (n = 4,061) % or Mean (SD)	<i>Analytic sample</i> (N = 6,046) % or Mean (SD)	<i>Analytic subsample^a</i> (n = 3,746) % or Mean (SD)
Key independent variables				
Eating disorder (ED) ^{b,c}	3.65%	3.60%	28.53%	32.86%
Disordered eating behavior (DEB)	21.72%	20.12%	N.A.	N.A.
ED or DEB	24.03%	22.43%	N.A.	N.A.
Dependent variables				
Number of children ^d	1.08 (1.19)	0.58 (0.89)	1.48 (1.69)	1.14 (1.56)
Age at first birth	22.16 (3.69)	24.75 (2.82)	22.68 (3.85)	23.72 (3.49)
Covariates				
Race/ethnicity				
Non-Hispanic White	61.96%	65.23%	86.26%	89.00%
Hispanic	8.67%	7.73%	9.59%	7.02%
Non-Hispanic other	29.37%	27.04%	4.15%	3.98%
Married ^d	38.27%	55.21%	38.74%	32.35%
Body mass index (BMI) ^b	26.90 (7.05)	26.45 (7.03)	22.64 (6.02)	22.30 (6.48)
Household income ^{b,e}				
<\$10,000	31.54%	33.00%	0.02%	0.03%
\$10,000–14,999	8.55%	7.98%	0.07%	0.05%
\$15,000–19,999	7.91%	7.14%	1.01%	0.93%
\$20,000–29,999	12.80%	11.40%	10.12%	9.77%
\$30,000–39,999	9.78%	8.62%	21.24%	19.75%
\$40,000–49,999	7.38%	7.12%	20.51%	20.05%
\$50,000–74,999	11.19%	11.80%	36.67%	37.75%
\$75,000+	10.85%	12.95%	10.37%	11.67%

Data come from Add Health and from the UPDB.

^aExcludes women whose first birth occurred before observation period (Add Health) or whose BMI measurement or first birth occurred before age 18 and/or age of ED diagnosis (UPDB).^bMeasured at Wave III, when respondents were between the ages of 18 and 24 years of age (Add Health); between the age of 12 and 32 (UPDB).^cIncludes women diagnosed with an eating disorder (ICD-9 codes 307.1, 307.5–307.52; ICD 10 codes f50–f50.9) (UPDB) or women who self-identify being diagnosed with an eating disorder (Add Health).^dMeasured at Wave IV, when respondents were between the ages of 24 and 32 years (Add Health); by age 32 (UPDB).^eMeasured at household level (Add Health); at census-block level (UPDB).

BMI, body mass index; SD, standard deviation; N.A., not applicable.

TABLE 3. NEGATIVE BINOMIAL REGRESSION RESULTS (INCIDENCE RATE RATIOS)

Number of children	Add Health sample					UPDB sample		
	Model 1a	Model 1b	Model 2a	Model 2b	Model 3a	Model 3b	Model 4	Model 5
Primary independent variable								
Eating disorder (ED) ^a	1.07 (0.08)		1.08 (0.08)		1.11 (0.07)		0.34*** (0.01)	0.38*** (0.01)
Disordered eating behavior (DEB) ^b		1.18*** (0.04)		1.17*** (0.04)		1.13*** (0.04)		
Covariates								
Birth year	0.91*** (0.01)	0.91*** (0.01)	0.94*** (0.01)	0.94*** (0.01)	0.94*** (0.01)	0.94*** (0.01)	0.93*** (0.00)	0.96*** (0.00)
Household income ^c			0.96*** (0.01)	0.96*** (0.01)	0.96*** (0.01)	0.96*** (0.01)		0.93*** (0.01)
Race/ethnicity ^d								
Hispanic			1.20*** (0.05)	1.19*** (0.05)	1.19*** (0.05)	1.17*** (0.05)		1.83*** (0.07)
Non-Hispanic other			1.39*** (0.04)	1.37*** (0.04)	1.36*** (0.04)	1.35*** (0.04)		1.17 (0.08)
Marital status ^e			2.11*** (0.07)	2.11*** (0.07)	2.12*** (0.07)	2.12*** (0.07)		2.18*** (0.07)
Body mass index (BMI)					1.01*** (0.00)	1.01*** (0.00)		1.00 (0.00)
N	5,951	5,951	5,951	5,951	5,951	5,951	6,046	6,046

Data come from Add Health and from the UPDB. Robust standard errors in parentheses. Incidence rate ratios reported.

^aCompares those who self-identify as having been diagnosed with an ED to peers without ED diagnosis (reference group) within the Add Health sample; compares those diagnosed with an ED (ICD-9 codes 307.1, 307.5–307.51) to women without an ED diagnosis (UPDB sample).

^bCompares those who self-identify as engaging in a DEB in the past 7 days to peers without DEB (reference group) within the Add Health sample.

^cHigher scores indicate higher household income level (ordinal category) measured at the individual level within the Add Health sample and at the census block level within the UPDB sample.

^dReference group is non-Hispanic White.

^eCompares those who have never been married (reference group), those who have been married at least once by Wave IV (Add Health sample) or age 32 (UPDB sample).

*** $p < 0.001$.

TABLE 4. COX PROPORTIONAL HAZARD RESULTS (HAZARD RATE RATIOS)

Age at first birth	Add Health subsample				UPDB subsample				
	Model 1a	Model 1b	Model 2a	Model 2b	Model 3a	Model 3b	Model 4	Model 5	Model 6
Primary independent variable									
Eating disorder (ED) ^a	1.15 (0.15)		1.15 (0.15)		1.15 (0.14)		0.34*** (0.02)	0.39*** (0.02)	0.38*** (0.02)
Disordered eating behavior (DEB) ^b		1.12* (0.07)		1.16* (0.07)		1.16* (0.07)			
Covariates									
Household income ^c			1.00 (0.01)	1.00 (0.01)	1.00 (0.00)	1.00 (0.00)		0.88*** (0.02)	0.88*** (0.02)
Race/ethnicity ^d									
Hispanic			0.99 (0.09)	0.97 (0.09)	0.99 (0.14)	0.98 (0.09)		1.81*** (0.17)	1.82*** (0.17)
Non-Hispanic other			1.16** (0.07)	1.15* (0.07)	1.16** (0.07)	1.15* (0.07)		0.85 (0.11)	0.86 (0.11)
Marital status ^e			2.25*** (0.12)	2.26*** (0.12)	2.25*** (0.12)	2.26*** (0.12)		2.50*** (0.14)	2.48*** (0.14)
Body mass index (BMI)					1.00 (0.00)	1.00 (0.00)			0.99* (0.01)
N	4,061	4,061	4,061	4,061	4,061	4,061	3,746	3,746	3,746

Data come from Add Health. Robust standard errors in parentheses; hazard ratios reported; hazard estimates are stratified by birth year. Right censoring set to age of death/age of emigration for childless women under 32, and age 32 for childless subject within the UPDB sample. Right censoring is set to age at Wave IV for childless women in the Add Health sample.

^aCompares those who self-identify as having been diagnosed with an ED to peers without ED diagnosis (reference group) within the Add Health sample; compares those diagnosed with an ED (ICD-9 codes 307.1, 307.5–307.51) to women without an ED diagnosis (UPDB sample).

^bCompares those who self-identify as engaging in a DEB in the past seven days to peers without DEB (reference group) within the Add Health sample.

^cHigher scores indicate higher household income level (ordinal category) measured at the individual level within the Add Health sample and at the census block level within the UPDB sample. Reference group is non-Hispanic white

^eCompares those who have never been married reference group, those who have been married at least once by Wave IV (Add Health sample) or age 32 (UPDB sample).

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

baseline model, baseline model with sociodemographic covariates, and a full model, including BMI.

Results

Table 2 reports descriptive statistics of the analytic samples. Approximately 3.7% of the Add Health sample self-reports an ED diagnosis and 22% a DEB. Fifteen percent of the UPDB sample includes women diagnosed with an ED between the ages of 12 and 32 (this is due to the matched-sampling design and is not representative of the proportion of women diagnosed with ED). The median number of children born to women in the Add Health sample by early adulthood was one compared to two in the UPDB sample. The average age at first birth of women in the Add Health subsample was 24.7 years ($SD=2.78$) compared to age 24.5 ($SD=4.31$) in the UPDB subsample.

Negative binomial regression results from Add Health data indicate that self-reported ED diagnosis is not significantly associated with parity (Table 3; models 1a–3a), but that self-reported DEB associates with increased parity (incidence rate ratio [IRR]=1.13 [95% confidence interval=1.07–1.21]; $p<0.001$) (models 1b–3b). Negative binomial regression results from UPDB data (model 6) indicate that prior ED diagnosis associates with reduced parity (IRR=0.38 [0.35–0.41]; $p<0.001$).

Cox proportional hazard results from Add Health data indicate that self-reported ED diagnosis is not significantly associated with age at first birth (Table 4; models 1a–3a), but that self-reported DEB is associated with earlier age at first birth (hazard rate ratio [HRR]=1.16 [1.03–1.30]; $p<0.05$) (models 1b–3b). Results from UPDB data (models 4–6) indicate that ED diagnosis is associated with later ages at first birth (HRR=0.38 [0.34–0.43]; $p<0.001$).

Discussion

This study illustrates that while EDs or DEB may be associated with reproductive timing and parity, sampling design and ED measurement shape inferences made about the relationship between EDs and reproductive outcomes. Although steps were taken to transform UPDB and Add Health data into comparable samples (we constrained UPDB analytic samples to match the age structure and measurement timing of the Add Health samples), there were important differences in measurement of EDs across these two types of data. These differences arguably shaped the results of this study. Women with an ED diagnosis in the UPDB sample were more likely to experience later age at first birth and lower parity at 32 years old. Meanwhile, women with self-reported DEB from Add Health were more likely to experience earlier age at first birth and higher parity by ages 24–32 years.

Two sets of mechanisms, physical (sex based) and social-behavioral (gender based), may be simultaneously shaping these women's reproductive outcomes. Individuals often go undiagnosed with an ED for prolonged periods and oftentimes only after their DEB has become severe.⁵¹ Approximately 40% of the UPDB sample with ED was first diagnosed at the time of hospitalization, and 30% experienced an additional ED-related medical event. Women with ED history within the UPDB sample may be experiencing known physical health complications such as loss of menstruation that disrupt fertility. Women within the Add Health sample with self-reported ED or DEB may not have experienced the same physical health conse-

quences because their DEB was less severe and shorter. This assumption is partially supported by our finding that Add Health women with EDs or DEB had higher average BMIs (28.78, $SD=7.67$) relative to women with EDs within UPDB (22.64, $SD=6.02$). Although our samples permitted only the comparison of BMI across the ED groups in each (given availability of comparable data across the two sources), it is assumed that other physical health outcomes would also likely differ. Thus, sex-based mechanisms that link an ED diagnosis with biological or physical health outcomes may be more relevant in clinical samples or those using clinical measures.

Conversely, gender-based explanations might be more useful in explaining why we saw increased and early fertility of women with DEB within Add Health. Previous research has found that women with histories of EDs or DEB may be more likely to engage in risky sexual behaviors,³³ including utilizing inadequate forms of birth control,^{34,52} which may result in unintended pregnancy.²⁷ Because unintended pregnancy increases the likelihood for subsequent pregnancies,⁵³ women engaging in DEB, especially those without physical health complications associated with severe and clinically diagnosed cases of EDs, may face early entry into parenthood and experience multiple births at younger ages.⁴⁹

Finally, women with ED may have different attitudes toward motherhood that may shape their reproductive behavior. Research suggests that women with EDs experience conflicted feelings about being pregnant, particularly during their first pregnancy.⁵⁴ Women with prior AN are more likely to identify a pregnancy as unwanted or unplanned relative to women without ED history or histories of BN,^{7,29,55} which may illustrate important differences in pregnancy attitudes based on ED type. Thus, researchers and clinicians should consider the fertility intentions and motherhood attitudes of women with histories of ED or DEB in addition to their physical health status or their engagement in risky sexual behaviors when studying and/or addressing their reproductive health.

The samples used in these analyses are representative of different populations: a nationally representative sample (Add Health) and a sample derived from diagnosed ED cases and their matched peers in the state of Utah (UPDB). Utah has the highest total fertility rate in the United States, estimated at 2.3 births per woman.⁵⁶ While results from Add Health are therefore more generalizable to American women, analyses on the clinically oriented sample (UPDB), with more limited generalizability, reflect common findings regarding EDs and fertility. A common strategy for ED screenings and prevention is interventions targeted toward college-age women on United States campuses.^{57,58} Clinicians should be sensitive to how clinical outcomes may not be generalizable to women in nonclinical settings, particularly women exhibiting DEB but who do not meet diagnostic thresholds.

This study does not account for complete fertility histories of women due to data constraints (Add Health Wave IV was completed at ages 18–32 years). This study concerns the reproductive health of women in early adulthood.^c Given the trend toward delayed fertility, particularly increased fertility

^cIt is important to note, however, in supplementary analyses (not shown) of the Utah Population Database samples; results do not substantially change when using full completed fertility (15–50 years of age).

after the age of 40,⁵⁹ future studies should examine complete fertility histories of women with a history of ED or DEB.

Although it is likely that age at first birth and parity vary by type-specific ED, fertility outcomes likely vary by diagnosis (e.g., AN, BN, and EDNOS)⁶⁰ or DEB; this analysis did not examine specific forms of EDs or DEB separately given the limitations in Add Health's instrumentation and small sample sizes of each diagnosis or type of behavior. In addition, this study cannot assess the reproductive outcomes of women of color; UPDB data contained very few minority women, and Hispanics were more likely to have missing Add Health data. Future research should account for the reproductive health of racial/ethnic minorities and explore whether reproductive outcomes vary by ED type.

Conclusion

It is well known that EDs negatively influence women's reproductive health. However, we illustrate that methodology can shape the inferences we make about the association between EDs and reproductive health. Both physical (sex specific) and social-behavioral (gender specific) mechanisms may influence the fertility outcomes of women with histories of EDs or DEB. Additional research should identify which social-behavioral mechanisms shape the reproductive health of women with histories of EDs or DEB. Disciplinary framework often determines whether gender effects are accounted for when we make inferences about sex-specific health outcomes. We may be generating a "clinician's illusion"³⁵ when we emphasize sex over gender and when we take a strictly clinical approach to reproduction. Awareness of how research outcomes are influenced by disciplinary framework is vital to a comprehensive understanding of women's health across the life course.

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