PEDIATRICS®

The Association Between Adverse Childhood Experiences and Adolescent Pregnancy, Long-Term Psychosocial Consequences, and Fetal Death

Susan D. Hillis, Robert F. Anda, Shanta R. Dube, Vincent J. Felitti, Polly A. Marchbanks and James S. Marks

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://www.pediatrics.org/cgi/content/full/113/2/320

American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN™

The Association Between Adverse Childhood Experiences and Adolescent Pregnancy, Long-Term Psychosocial Consequences, and Fetal Death

Susan D. Hillis, PhD, MS*; Robert F. Anda, MD, MS*; Shanta R. Dube, MPH*;
Vincent J. Felitti, MD, FACP‡; Polly A. Marchbanks, PhD*; and James S. Marks, MD, MPH*

ABSTRACT. *Objectives.* Few reports address the impact of cumulative exposure to childhood abuse and family dysfunction on teen pregnancy and consequences commonly attributed to teen pregnancy. Therefore, we examined whether adolescent pregnancy increased as types of adverse childhood experiences (ACE score) increased and whether ACEs or adolescent pregnancy was the principal source of elevated risk for long-term psychosocial consequences and fetal death.

Design, Setting, and Participants. A retrospective cohort study of 9159 women aged ≥18 years (mean 56 years) who attended a primary care clinic in San Diego, California in 1995–1997.

Main Outcome Measure. Adolescent pregnancy, psychosocial consequences, and fetal death, compared by ACE score (emotional, physical, or sexual abuse; exposure to domestic violence, substance abusing, mentally ill, or criminal household member; or separated/divorced parent).

Results. Sixty-six percent (*n* = 6015) of women reported ≥1 ACE. Teen pregnancy occurred in 16%, 21%, 26%, 29%, 32%, 40%, 43%, and 53% of those with 0, 1, 2, 3, 4, 5, 6, and 7 to 8 ACEs. As the ACE score rose from zero to 1 to 2, 3 to 4, and ≥5, odds ratios for each adult consequence increased (family problems: 1.0, 1.5, 2.2, 3.3; financial problems: 1.0, 1.6, 2.3, 2.4; job problems: 1.0, 1.4, 2.3, 2.9; high stress: 1.0, 1.4, 1.9, 2.2; and uncontrollable anger: 1.0, 1.6, 2.8, 4.5, respectively). Adolescent pregnancy was not associated with any of these adult outcomes in the absence of childhood adversity (ACEs: 0). The ACE score was associated with increased fetal death after first pregnancy (odds ratios for 0, 1–2, 3–4, and 5–8 ACEs: 1.0, 1.2, 1.4, and 1.8, respectively); teen pregnancy was not related to fetal death.

Conclusions. The relationship between ACEs and adolescent pregnancy is strong and graded. Moreover, the negative psychosocial sequelae and fetal deaths commonly attributed to adolescent pregnancy seem to result from underlying ACEs rather than adolescent pregnancy per se. *Pediatrics* 2004;113:320–327; *adolescent pregnancy, child abuse, domestic violence, alcoholism, children of impaired parents, drug abuse.*

ABBREVIATIONS. ACE, adverse childhood experience; RR, relative risk; CI, confidence interval; OR, odds ratio.

Despite reductions in the rate of adolescent pregnancy and birthrates during the 1990s, adolescent pregnancy rates in the United States still exceed those of other developed countries by 2- to 15-fold.[1–4] Four of 10 adolescent girls get pregnant before the age of 20 in the United States, leading to ~900 000 teenage pregnancies each year.[3]

Consequences frequently attributed to adolescent pregnancy include long-term psychosocial and economic disadvantages for the teen mother, as characterized by lower educational attainment, lower income, and a greater risk of being a single parent.[5,6] The impact of adolescent pregnancy on the fetus or infant is multifaceted: many studies report an increased risk of fetal death, and infants born to teen mothers have an increased tendency to be low birth weight or premature and are apt to have poorer cognitive development, lower educational attainment, more frequent criminal activity, and higher risk of abuse, neglect, abandonment, and behavioral problems during childhood.[5,7–14] Whether adolescent pregnancy itself leads to adverse reproductive outcomes or whether these outcomes are consequences of the same environment of adversity that led to the teen pregnancy has been the focus of debate.[15] The association between adolescent pregnancy and long-term consequences seems to be attenuated when factors that precede and increase the risk of adolescent pregnancy are controlled.[15,16]

Previous reports have identified a host of community-, family-, school-, and individual-level antecedents of adolescent pregnancy,[3,17–30] including restrictive contraceptive marketing laws, higher unemployment rates, higher violent crime rates, higher adolescent suicide rates, higher high school drop-out rates, lower parental education, inadequate health care insurance coverage, having an older sister or friend who was an adolescent parent, minority race/ethnicity, lower family connectedness, physical or sexual abuse, general maltreatment by family, greater number of sex partners, alcohol use, substance use, and attempted suicide.[17–30] Despite the plethora of antecedents, none of them have been shown to account independently for a large portion of the teen pregnancy risk.[3,17–30] The more effective adolescent pregnancy-prevention programs, which have focused broadly on youth development,[3,31–33] may ameliorate several of these risk factors.

Although 2 decades of research have been dedicated to identifying antecedents and consequences of adolescent pregnancy, little attention has been devoted to the role of adverse childhood experiences (ACEs) as either an antecedent or a factor that may influence those consequences commonly attributed to teen pregnancy. Numerous reports have used the ACE Study (conducted in California in 1995–1997) to address the association between major causes of death and disability in the United States and ACEs. Indicators included in the ACE measure, each of which is given a score of 1 (maximum: 8) are self-reported household exposure during childhood to emotional abuse, physical abuse, sexual abuse, intimate partner violence, living with a substance-abusing, mentally ill, or criminal household member, or having a parent who was divorced or separated. Published reports have demonstrated strong, graded associations between the ACE score and many unfavorable outcomes including suicide attempts, smoking, alcohol abuse, high-risk sexual behavior, sexually transmitted diseases, and unintended pregnancy in adult women.[34–42]

We used the ACE Study to evaluate whether exposure to ACEs increased the risk of adolescent pregnancy. We hypothesized that each category of ACE would independently increase the risk of adolescent pregnancy and that, as the ACE score rose, the risk of adolescent pregnancy would increase. In terms of unfavorable consequences of adolescent pregnancy, we examined whether ACEs or adolescent pregnancy itself was the principal source of elevated risk for long-term psychosocial consequences including serious family problems, job problems, financial problems, high stress, and uncontrollable anger in adulthood. We also examined whether ACEs or adolescent pregnancy per se was associated with negative effects on viability of the infant, as measured by fetal death.

METHODS

The epidemiologic methods used for the ACE Study have been described elsewhere.[34–42] Briefly, a retrospective cohort study was conducted among adult enrollees in a large health maintenance organization (Kaiser Permanente Medical Care Program) in San Diego, California. The protocol was approved by the institutional review boards of Emory University and Kaiser Permanente and by the office of Human Research Protection, Department of Health and Human Services (formerly Office of Protection from Research Risks, National Institutes of Health).

Each year >50 000 adult plan members underwent a standardized health evaluation. In a typical 4-year period in the 1990s, >80% of continuously enrolled members obtained this service, which included a detailed history, psychosocial evaluation, lab studies, and physical examination. Within 2 weeks after this evaluation, participants received a mailed ACE questionnaire that assessed self-reported exposure to childhood abuse or household dysfunction.

Data were collected in 2 survey waves, August to November 1995 to March 1996 (wave 1) and January to October 1997 (wave 2). The overall response rate was 68% (wave 1: 70%; wave 2: 65%). Of the 17 421 respondents, 9420 (54.1%) were female. After excluding women who denied having had sexual intercourse ($n = 208$) and who had missing data on race or education ($n = 53$), our sample included 9159 women.

Definitions of ACEs, Adolescent Pregnancy, Fetal Death, and Psychosocial Consequences

All measures of ACEs were ascertained by the ACE questionnaire and pertained to experiences during the respondent's first 18 years of life (Table 1). Each category of abuse and dysfunction has been described in detail.[34–42] Questions regarding verbal and physical abuse, intimate partner violence,[43] contact sexual abuse during childhood,[44] and household substance abuse[45] were adapted from previously used scales.

Information about adolescent pregnancy, defined as a pregnancy reported from ages 11 to 19 years, was also obtained through the ACE questionnaire. During wave 2, the question was direct: "How old were you the first time you became pregnant?" During wave 1, the occurrence of a first pregnancy during adolescence was estimated from questions measuring the age when first pregnancy ended. The first question was: "During what month and year did your first pregnancy end?" Age when the first pregnancy ended was computed by subtracting the participant's date of birth from the date the first pregnancy ended. A combination of age when first pregnancy ended and outcome of first pregnancy (live birth, stillbirth/miscarriage, tubal pregnancy, or elective abortion) was used to impute the age when the participant first became pregnant. For live births, the age when the participant first became pregnant was assumed to have been 8 months before the age when pregnancy ended. For elective abortions or ectopic pregnancies, age at first pregnancy was assumed to have been 2 months before the age when the first pregnancy ended. For reporting fetal death (defined as stillbirth/miscarriage), age at first pregnancy was assumed to be 4 months before the pregnancy ended. Because both direct and estimated variables were available for wave 2, we evaluated the validity of our imputation by using data from that wave. We found that 89.2% of women from wave 2 who would have been classified by the estimated variable as having had an adolescent pregnancy were identified correctly. The assumption of pregnancy beginning 8 months before live birth, 4 months before stillbirth/miscarriage, and 2 months before abortion or ectopic pregnancy, was more accurate than alternate assumptions (such as pregnancy beginning 9 months earlier for births and 3 months earlier for abortions and ectopic pregnancies; data not shown). The frequency of adolescent pregnancy was nearly identical in the 2 waves (23.2% in wave 1 using the estimated variable and 23.8% in wave 2 using the direct variable).

Information regarding psychosocial consequences was obtained from the Kaiser Health Appraisal questionnaire. These questions defined family problems, job problems, and financial problems: "Are you now having serious or disturbing problems with your: family (yes/no), job (yes/no), financial matters?" The question used to define high stress was "Please fill in the circle that best describes your stress level (high/medium/low)." Finally, the question defining fear of uncontrollable anger was "Have you ever had reason to fear your anger getting out of control?"

Statistical Analyses

Persons with incomplete information about an ACE were considered not to have had that experience. Analyses using this approach were almost identical to analyses that excluded participants with incomplete information. The unadjusted association between each of the 8 categories of ACEs and adolescent pregnancy was estimated by using relative risks (RRs) and 95% confidence intervals (CIs).[46] The Mantel-Haenszel χ^2 test for linear trend in proportions was used to evaluate whether the risk of adolescent pregnancy increased as the number of categories of ACEs (ACE score, classified as 0, 1, 2, 3, 4–5, 6, or 7–8) increased.[47] Separate logistic regression models were used to obtain adjusted odds ratios (ORs) (as estimates of RR) and 95% CIs for the association between ACE score and adolescent pregnancy, long-term psychosocial consequences, and fetal death.[46] We used the population attributable risk percent to estimate the proportion of cases of adolescent pregnancy attributable to ACEs.[48] For the examination of whether adolescent pregnancy was an independent predictor of each of the long-term psychosocial sequelae, maximum-likelihood ratio tests[46] demonstrated highly significant differences according to whether participants had been exposed to ACEs; therefore, the associations between adolescent pregnancy and long-term sequelae are reported separately for those with (ACE score: ≥1) and without (ACE score: 0) ACEs. Finally, we examined the association between ACEs and each of our major outcomes

TABLE 1. Definitions of ACEs

Questions	Defining Responses
Verbal abuse: Sometimes parents or other adults hurt children. While you were growing up, that is, in your first 18 years of life, how often did a parent, stepparent, or adult living in your home: 1) swear at you, insult you, or put you down? 2) threaten to hit you or throw something at you but didn't do it?	"often" or "very often" to either question
Physical abuse: Sometimes parents or other adults hurt children. While you were growing up, that is, in your first 18 years of life, how often did a parent, stepparent, or adult living in your home: 1) push, grab, slap, or throw something at you? or 2) hit you so hard that you had marks or were injured?	"often" or "very often" to question 1 or "sometimes," "often," or "very often" to question 2
Sexual abuse: Some people, while they are growing up in their first 18 years of life, had a sexual experience with an adult or someone at least 5 years older than themselves. These experiences may have involved a relative, family friend, or stranger. During the first 18 years of life, did an adult, relative, family friend, or stranger ever: 1) touch or fondle your body in a sexual way, 2) have you touch their body in a sexual way, 3) attempt to have any type of sexual intercourse with you (oral, anal, or vaginal), or 4) actually have any type of sexual intercourse with you (oral, anal, or vaginal)?	"yes" to any of the 4 questions
Intimate partner violence: Sometimes physical blows occur between parents. While you were growing up in your first 18 years of life, how often did your father (or stepfather) or mother's boyfriend do any of these things to your mother (or stepmother): 1) push, grab, slap, or throw something at her, 2) kick, bite, hit her with a fist, or hit her with something hard, 3) repeatedly hit her over at least a few minutes, or 4) threaten her with a knife or gun or use a knife or gun to hurt her?	"sometimes," "often," or "very often" to at least 1 of the first 2 questions or any response other than "never" to at least one of the third and fourth questions
Household substance abuse: During your first 18 years of life, did you live with anyone who was a problem drinker or alcoholic? During the first 18 years of life, did you live with anyone who used street drugs?	"yes" to either question
Mental illness in household: During your first 18 years of life, was anyone in your household depressed or mentally ill? During your first 18 years of life, did anyone in your household attempt to commit suicide?	"yes" to either question
Incarcerated household member: During your first 18 years of life, did anyone in your household go to prison?	"yes"
Parental separation or divorce: Were your parents ever separated or divorced?	"yes"

(adolescent pregnancy, fetal death, and psychosocial consequences) in analyses restricted to participants in the youngest birth cohort (birth years 1962–1978) and thus aged ≤34 years at interview.

RESULTS

Our study population tended to be ≥50 years of age at interview (62%), to have attended college (72%), and to be white (77%). We found that 66% (6015 of 9159) of participants reported exposure to ≥1 categories of childhood adversity (Table 1). Compared with women who did not report ACEs, those who reported ≥1 ACEs were significantly more likely to be <35 years of age at interview, to be black or Hispanic, and to report certain risk behaviors in adolescence, including street drug use, alcohol use, and ≥1 suicide attempts as well as having had ≥5 sex partners in their lifetimes (Table 2).

Exposure to each of the 8 types of ACEs was associated with an increased risk of adolescent pregnancy (Table 3). Compared with women who did not report the specific ACEs, RRs showed increases in adolescent pregnancy: 90% for those who, as children, had an incarcerated family member; 60% for those who experienced childhood sexual abuse; 60% for those reporting childhood emotional abuse; 50% for those reporting physical abuse; 60% for those reporting household substance abuse; 20% for those who lived with a mentally ill family member during childhood; 60% for those exposed to intimate partner violence; and 60% for those whose parents were divorced or separated.

After adjustment for age at interview, education, and race, ORs for adolescent pregnancy increased significantly and incrementally as exposure (the ACE score) increased (Table 4). The adjusted ORs for adolescent pregnancy were 1.0, 1.4, 1.8, 2.2, 2.4, 3.2, 3.4, and 5.5 among those with 0, 1, 2, 3, 4, 5, 6, and 7 to 8 ACEs, respectively. Whereas 16.0% of those who experienced no ACEs reported their first pregnancy occurring during adolescence, this risk rose to 53.0% among women who experienced 7 to 8 ACEs. Our findings regarding the dose-gradient effect of ACEs on adolescent pregnancy were essentially unchanged by adjusted analyses restricted to wave 2 (wave 2 included expanded behavioral data), which allowed us to control for additional potential confounders, including multiple sex partners, drinking during adolescence, drug use during adolescence, daughter of an adolescent mother, and suicide attempt during adolescence. If one assumes that exposure to ACEs causes adolescent pregnancy, then ACEs lead to a population attributable risk percent of 33%, indicating that one third of adolescent pregnancies could be prevented by eliminating exposure to ACEs.

We observed that women who were exposed during childhood to even modest levels of ACEs (ACE score: 1–2) were significantly more likely to report at interview (mean age at interview: 55.8 years) that they were having current "serious or disturbing" family problems, job problems, financial problems, or high levels of stress or that they had experienced fear of uncontrollable anger (Table 5). As exposure to

TABLE 2. Distribution of Demographic and Behavioral Characteristics by ACEs

	N	≥1 ACEs % (n) N = 6015	No. ACEs % (n) N = 3144
Race or ethnicity*			
White	7033	77.1 (4639)	76.2 (2394)
Black	437	5.5 (333)	3.3 (104)
Hispanic	489	5.9 (352)	4.4 (137)
Asian	718	5.5 (329)	12.4 (389)
American Indian	36	0.47 (28)	0.25 (8)
Other	446	5.6 (334)	3.6 (112)
Education*			
Some high school	722	7.0 (420)	9.6 (302)
High school graduate	1852	19.3 (1160)	22.0 (692)
Some college	3444	40.7 (2449)	31.7 (995)
College graduate	3141	33.0 (1986)	36.7 (1155)
Age at interview, y*			
19–34	1042	13.5 (810)	7.4 (232)
35–49	2483	30.7 (1846)	20.3 (637)
50–64	2860	31.7 (1909)	30.3 (951)
≥65	2774	24.1 (1450)	42.1 (1324)
Daughter of adolescent mother†			
Yes	656	15.3 (463)	12.7 (193)
No	3902	84.7 (2574)	87.3 (1328)
Smoked during adolescence*			
Yes	2391	30.2 (1819)	18.2 (572)
No	6768	69.8 (4196)	81.8 (2572)
Drank alcohol during adolescence*			
Yes	2204	54.6 (1659)	35.8 (545)
No	2354	45.4 (1378)	64.2 (976)
Used street drugs during adolescence*			
Yes	522	15.0 (454)	4.5 (68)
No	4036	85.0 (2583)	95.5 (1453)
Suicide attempt during adolescence*			
Yes	111	3.5 (105)	0.39 (6)
No	4447	96.5 (2932)	99.6 (1515)
≥5 lifetime sex partners*			
Yes	2652	39.3 (2156)	17.4 (496)
No	5684	60.7 (3331)	82.6 (2353)

* $P < .0001$; † $P < .05$. Data for daughter of adolescent mother, drank alcohol during adolescence, used street drugs during adolescence, and suicide attempt during adolescence were available for the second wave only (total = 4558).

TABLE 3. ACEs and Risk of Adolescent Pregnancy

	Adolescent Pregnancy	
	% (n)	RR (95% CI)
Household substance abuse		
Yes	32.3 (872/2698)	1.6 (1.5–1.8)
No	19.8 (1266/6390)	Referent
Household mental illness		
Yes	27.3 (583/2135)	1.2 (1.1–1.3)
No	22.4 (1555/6953)	Referent
Exposed to intimate partner violence		
Yes	34.0 (427/1255)	1.6 (1.4–1.7)
No	21.8 (1711/7833)	Referent
Incarcerated family member		
Yes	42.4 (200/472)	1.9 (1.7–2.1)
No	22.5 (1938/8616)	Referent
Verbal abuse		
Yes	34.5 (416/1207)	1.6 (1.4–1.7)
No	21.9 (1722/7881)	Referent
Physical abuse		
Yes	30.4 (753/2476)	1.5 (1.3–1.6)
No	21.0 (1385/6612)	Referent
Sexual abuse		
Yes	32.2 (728/2262)	1.6 (1.4–1.7)
No	20.7 (1410/6826)	
Parents divorced/separated		
Yes	32.5 (727/2240)	1.6 (1.5–1.7)
No	20.6 (1411/6848)	

Total sample size decreased due to missing data on adolescent pregnancy.

TABLE 4. Categories of ACEs and Adolescent Pregnancy

Categories of Exposure to ACEs	%*	Adolescent Pregnancy	
		Unadjusted OR (95% CI)	Adjusted OR† (95% CI)
0 (3101)	16.0	1.0 (referent)	1.0 (referent)
1 (2227)	21.2	1.4 (1.2–1.6)	1.4 (1.2–1.6)
2 (1410)	25.6	1.8 (1.5–2.1)	1.8 (1.5–2.1)
3 (945)	29.1	2.2 (1.8–2.5)	2.2 (1.8–2.5)
4 (661)	32.4	2.5 (2.1–3.0)	2.4 (2.0–3.0)
5 (400)	39.5	3.4 (2.7–4.3)	3.2 (2.6–4.1)
6 (212)	42.5	3.9 (2.9–5.2)	3.6 (2.6–4.8)
7–8 (132)	53.0	6.0 (4.1–8.4)	5.6 (3.9–8.2)
P for trend	<.000001		

* Percent of those with the listed number of categories of ACEs who experienced an adolescent pregnancy.
† All ORs are adjusted for race, education, and age at interview.

ACEs rose from zero to 1 to 2, 3 to 4, and ≥5, the adjusted ORs, respectively, for serious family problems (1.0, 1.5, 2.2, and 3.3), job problems (1.0, 1.4, 2.3, and 2.9), financial problems (1.0, 1.6, 2.3, and 2.4), high stress (1.0, 1.4, 1.9, and 2.2), or uncontrollable anger (1.0, 1.6, 2.8, and 4.5) also increased steadily and consistently. In contrast, adolescent pregnancy was not associated with even 1 of these long-term outcomes among women who did not experience ACEs (ACE score: 0). For those exposed to childhood adversity (ACE score: ≥1), adolescent pregnancy was associated with modest increases in family and financial problems and in high stress and fear of uncontrollable anger.

Among women who had ever been pregnant, a strong and independent trend was observed for the association between ACE score and fetal death as the outcome of the first pregnancy (Table 6). This risk increased from 6.9% to 7.6%, 8.7%, and 10.2%, re-

spectively, for those with exposure to 0, 1 to 2, 3 to 4, or ≥5 ACEs. Among women who had a second pregnancy (n = 6789), the risk of fetal death as the outcome of the second pregnancy increased from 7.0% for those with no exposure to ACEs to 8.6%, 9.9%, and 10.3%, respectively, for those with 1 to 2, 3 to 4, and ≥5 types of ACEs. Women whose first pregnancy occurred during adolescence, however, did not have an elevated risk of fetal death (Table 6).

Finally, in analyses restricted to women aged ≤34 at interview (11% of our sample), we found that the significant trend persisted for the association between ACEs and adolescent pregnancy (occurring in 8.7%, 18.7%, 18,2%, 26.1%, 29.3%, 31.8%, 34.9%, and 51.9% of those with an ACE score of 0, 1, 2, 3, 4, 5, 6, and 7–8, respectively; P for trend < .0001). As the ACE score rose in these younger women, the risk of fetal death as the outcome of the first pregnancy

TABLE 5. Adolescent Pregnancy and ACEs: Long-Term Psychosocial Consequences*

	Current Serious Family Problems		Current Serious Job Problems		Current Serious Financial Problems		Current High Stress		Fear of Inability to Control Anger	
	% (n)*	Adjusted OR† (95% CI)	% (n)*	Adjusted OR† (95% CI)	% (n)*	Adjusted OR† (95% CI)	% (n)*	Adjusted OR† (95% CI)	% (n)*	Adjusted OR† (95% CI)
Adolescent pregnancy ACEs = 0										
No	7.4 (192)	Referent	4.1 (106)	Referent	7.3 (190)	Referent	11.2 (291)	Referent	3.1 (81)	Referent
Yes	9.4 (46)	1.2 (0.8–1.7)	3.9 (19)	0.8 (0.5–1.5)	7.7 (38)	1.0 (0.7–1.4)	12.6 (62)	1.1 (0.8–1.6)	3.1 (15)	1.0 (0.6–1.8)
Adolescent pregnancy ACEs > 1										
No	12.9 (563)	Referent	9.0 (392)	Referent	14.7 (639)	Referent	18.7 (813)	Referent	7.0 (305)	Referent
Yes	17.7 (288)	1.3 (1.1–1.6)	8.9 (145)	1.0 (0.8–1.2)	19.1 (311)	1.3 (1.1–1.5)	22.3 (363)	1.3 (1.1–1.4)	9.8 (160)	1.4 (1.1–1.7)
ACEs										
0	7.6 (240)	Referent	4.1 (128)	Referent	7.4 (231)	Referent	11.3 (354)	Referent	3.1 (96)	Referent
1–2	11.5 (419)	1.5 (1.3–1.8)	6.5 (236)	1.4 (1.1–1.7)	12.6 (462)	1.6 (1.3–1.9)	16.6 (606)	1.4 (1.2–1.6)	5.3 (192)	1.6 (1.3–2.1)
3–4	16.1 (259)	2.2 (1.8–2.6)	11.7 (188)	2.3 (1.8–2.9)	19.9 (321)	2.3 (1.9–2.8)	22.7 (366)	1.9 (1.6–2.2)	9.6 (155)	2.8 (2.1–3.7)
5+	23.6 (176)	3.3 (2.7–4.2)	15.5 (116)	2.9 (2.2–3.8)	22.8 (170)	2.4 (1.9–3.0)	27.7 (2070)	2.2 (1.8–2.8)	15.8 (118)	4.5 (3.4–6.1)

* The percent of those with an adolescent pregnancy/no ACEs, adolescent pregnancy/≥1 ACEs, and ACEs who experienced each psychosocial consequence.
† All ORs are adjusted for age, race, and education; ACEs analyses are also adjusted for adolescent pregnancy.

TABLE 6. Adolescent Pregnancy and ACEs: Fetal Death as a Potential Consequence

	Fetal Death After First Pregnancy		Fetal Death After Second Pregnancy		Fetal Death After First or Second Pregnancy	
	% n*	Adjusted OR† (95% CI)	% n*	Adjusted OR† (95% CI)	% n*	Adjusted OR† (95% CI)
ACEs						
0	6.9 (216)	Referent	7.0 (161)	Referent	10.9 (341)	Referent
1–2	7.6 (279)	1.2 (0.96–1.4)	8.6 (235)	1.3 (1.04–1.6)	12.6 (460)	1.2 (1.03–1.4)
3–4	8.7 (140)	1.4 (1.1–1.7)	9.9 (116)	1.5 (1.2–1.9)	14.1 (228)	1.4 (1.2–1.7)
5+	10.2 (76)	1.8 (1.3–2.3)	10.3 (59)	1.7 (1.2–2.3)	15.8 (118)	1.7 (1.3–2.1)
Adolescent pregnancy						
No	8.2 (569)	Referent	8.8 (427)	Referent	12.7 (886)	Referent
Yes	6.7 (142)	0.8 (0.6–0.9)	7.5 (144)	1.1 (0.9–1.3)	12.3 (261)	0.9 (0.8–1.1)

* Percent of those with varying levels of ACEs or adolescent pregnancy who experienced fetal death.
† All ORs are adjusted for age, race, and education; ACEs analyses are also adjusted for adolescent pregnancy and adolescent pregnancy analyses for ACEs.

increased (occurring in 4.8%, 5.7%, 6.0%, and 11.3% of those with 0, 1–2, 3–4, and ≥5 ACEs, respectively; *P* for trend < .05). Each of the psychosocial consequences considered in this age group also increased incrementally as the ACE score increased from zero to 1 to 2, 3 to 4, and ≥5 (family problems: 4.3%, 9.3%, 12.4%, and 24.8% [*P* for trend < .0001]; job problems: 7.2%, 10.0%, 14.5%, and 22.6% [*P* for trend < .0001]; financial problems: 12.0%, 22.4%, 27.4%, and 27.1% [*P* for trend < .001]; difficulty controlling anger: 4.8%, 8.7%, 11.5%, and 19.6% [*P* for trend < .0001]; and current high stress: 12.0%, 17.0%, 27.8%, and 27.1% [*P* < .0001]).

DISCUSSION

We found a strong dose-response link between the number of ACEs and teen pregnancy: As the ACE score increased, the risk of adolescent pregnancy increased incrementally. This link is more potent than the relationships found for many other previously identified risk factors and accounts for a sizable fraction of the teen pregnancies, assuming that the relationship is causal. Incremental increases in

the ACE score were associated with enduring consequences reported decades after childhood, at a mean age of 56 years. A sizeable proportion of the adult participants who, as children, suffered ACEs, years later described their lives as beset by high stress, uncontrollable anger, and serious or disturbing problems with their families, jobs, and finances. We found that these adverse psychosocial sequelae so commonly thought to be related to the adolescent pregnancy actually seem to be due to being reared in families with difficult ACEs. When the family environment did not have ACEs, becoming pregnant as an adolescent did not raise the likelihood of these long-term, negative, psychosocial consequences.

We also found that as the ACE score rose, the risk of fetal death after both the first and second pregnancies increased. In contrast to previous reports,[8–10,13,49] however, adolescent pregnancy itself was not associated with an increased risk of fetal death. The strength, consistency, and gradient character of the associations between ACEs and adolescent pregnancy, long-term psychosocial sequelae,

and fetal death suggest that ACEs may be causal risk factors for each of these unfavorable outcomes.

ACEs were shown previously to increase the frequency of high-risk sexual behaviors that are associated with adolescent pregnancy, such as early onset of intercourse and high numbers of sexual partners.[34] Engagement in early, unprotected sexual activity that leads to adolescent pregnancy may represent a misguided attempt to achieve the interpersonal connectedness, support, and hope[50] that may have been lacking in childhood among those exposed to ACEs.[51–56] Although reports have demonstrated that several of the individual ACEs, such as sexual abuse and physical abuse, increase the risk of adolescent pregnancy,[26,30,32] few reports have addressed the cumulative impact of exposure to multiple types of adversity during childhood[57,58] on adolescent pregnancy. This limitation is salient, because abuse rarely takes place as an isolated event but typically occurs within a broader social context of multiple adversities including family dysfunction and disconnectedness.[42]

Previous reports have suggested that long-term psychosocial disadvantages are common and costly consequences of teen pregnancy.[5,6] However, findings of such studies may be biased by their failure to adequately account for differences in the family environments of those teens who become pregnant and those teens who delay pregnancy and childbearing. In contrast to reports not accounting for familial variation, a study comparing teens who gave birth with their sisters who delayed childbearing found that family background accounted for many of the disadvantages commonly attributed to adolescent pregnancy per se.[59,60] Furthermore, a study comparing teens from similar environments who gave birth with those who delayed childbearing found no long-term psychosocial consequences of early childbearing.[15] In the present study, we found no evidence to support an association between adolescent pregnancy and long-term psychosocial consequences (high stress, uncontrollable anger, and serious or disturbing problems with their families, jobs, and finances) in women who had not suffered ACEs.

An increase in adverse pregnancy outcomes including fetal death, prematurity, and low birth weight has been observed more frequently in studies comparing younger teen mothers ($\leq$17 years) with mothers aged 20 to 34.[6,8–10,14,49,61,62] We found no increased risk of fetal death among adolescent mothers even in analyses comparing young adolescent mothers with mothers aged 20 to 34 years (data not shown). Both behavioral and physiologic mechanisms may be operative in increasing the risk of fetal death among those exposed to ACEs. Those who, as children, experience adversity and dysfunction are known to be at greater risk for alcohol abuse,[41] smoking,[36] and sexually transmitted diseases,[37] all of which may increase the risk of fetal loss.[11,49,63–66] Prolonged exposure to deficient or dysfunctional environments during childhood may lead to long-term physiologic alterations including neuroendocrine disruption and abnormalities in immunologic regulation.[55,56,67] Our findings suggest that the cumulative and chronic exposure to stress associated with ACEs during childhood led to an increased risk of nonviability for the infant, persisting through both the first and second pregnancies.

We considered limitations that may have biased our findings. Because exposure to ACEs was measured by self-report, it is likely that these childhood exposures were underreported. Any such underreporting, however, would likely have led to underestimation of the strength of association between ACEs and adolescent pregnancy. Similarly, our classifying persons with incomplete information about an ACE as having not had that experience would lead to a conservative estimate of the relationship between ACEs and adolescent pregnancy. Because information was not available regarding the time exposure to ACEs began, we were unable to evaluate the number of participants who had their initial exposure to ACEs during adolescence, possibly at the same time as the teen pregnancy. However, because the focus of most questions regarding ACEs was on events occurring during childhood, it is probable that the majority of cases of reported adolescent pregnancies followed, rather than preceded, the onset of exposure to various categories of ACEs. Future studies would be strengthened by measuring the age during childhood when exposure to each ACE first occurred and by including in their study population women with more variable sociodemographic characteristics. Although the generalizability of our findings cannot be guaranteed, their persisting significance for the youngest birth cohort suggests that the observed impact of ACEs is not a historical phenomenon that disappeared along with known changes in sexual behaviors and values that occurred during the latter part of the 20th century.

In light of the magnitude of the adolescent pregnancy problem and its consequences, the National Campaign to Prevent Teen Pregnancy proposed to reduce adolescent pregnancy rates by 30% by 2005, with programs strategically focused on youth development, changing sexual practices, or a combination of the two.[3,33] Similarly, *Healthy People 2010* includes as one of its primary national objectives the lowering of pregnancy rates for 15- to 17-year-olds by ~35% by 2010.[68] From our findings, it seems that the proposed reductions in teen pregnancy could be achieved by including programs successful in preventing or ameliorating family dysfunction. Olds et al[69,70] demonstrated that interventions directed toward preventing exposure to familial violence and household dysfunction by having public health nurses make home visits to high-risk families during the early years of a child's life can be successful. Although explanations for the success of youth development programs in preventing adolescent pregnancy have been somewhat elusive,[3,33] it is reasonable that successful programs may have components that help ameliorate the detrimental effects of prolonged exposure to family dysfunction. Effective youth development programs seem to build competence and confidence by promoting supportive relationships with peers and mentors; by strengthening education, decision-making, and autonomy; and by

providing opportunities for community service.[3,31,33] Programs that successfully ameliorate the effects of family dysfunction may have the potential to prevent not only adolescent pregnancy but also fetal death and long-term psychosocial sequelae.

The essential contribution of family connectedness to promoting adolescent health was demonstrated clearly and convincingly at the end of the millennium, largely through the National Longitudinal Study on Adolescent Health.[50,71,72] Findings from this national survey of >90 000 students indicated that for adolescents, family connectedness was protective against emotional distress, suicidal thoughts and behaviors, violence, cigarette use, alcohol use, marijuana use, and young age at sexual debut.[50] Our analysis supports and extends previous reports by demonstrating that family dysfunction has enduring and unfavorable health consequences for women during the adolescent years, the childbearing years, and beyond. The implications of our findings for public health and societal policies are profound. If we are to reduce both adolescent pregnancy and long-term negative psychosocial outcomes, we must help families avoid these most difficult personal circumstances.

REFERENCES

1. Ventura SJ, Mathews TJ, Hamilton BE. Births to teenagers in the United States, 1940–2000. *Natl Vital Stat Rep*. 2001;49(10):1–23
2. Miller FC. Impact of adolescent pregnancy as we approach the new millennium. *J Pediatr Adolesc Gynecol*. 2000;13:5–8
3. Kirby D. *Emerging Answers: Research Findings on Programs to Reduce Teen Pregnancy*. Washington, DC: National Campaign to Prevent Teen Pregnancy; 2001
4. McElroy SW, Moore KA. Trends over time in teenage pregnancy and childbearing: the critical changes. In Maynard RA, Ed. *Kids Having Kids: Economic Costs and Social Consequences of Teen Pregnancy*. Washington, DC: Urban Institute Press; 1997:23–54
5. Maynard RA. The study, the context, and the findings in brief. In Maynard RA, Ed. *Kids Having Kids: Economic Costs and Social Consequences of Teen Pregnancy*. Washington, DC: Urban Institute Press; 1997: 1–22
6. Olausson PO, Haglund B, Weitoft GR, Cnattingius S. Teenage childbearing and long-term socioeconomic consequences: a case study in Sweden. *Fam Plann Perspect*. 2001;33:70–74
7. Moore DA, Morrison DR, Greene AD. Effects on the children born to adolescent mothers. In Maynard RA, Ed. *Kids Having Kids: Economic Costs and Social Consequences of Teen Pregnancy*. Washington, DC: Urban Institute Press; 1997:145–180
8. Satin AJ, Leveno KJ, Sherman ML, Reedy NJ, Lowe TW, McIntire DD. Maternal youth and pregnancy outcomes: middle school versus high school age groups compared with women beyond the teen years. *Am J Obstet Gynecol*. 1994;171:184–187
9. Strobino DM, Ensminger ME, Kim YJ, Nanda J. Mechanisms for maternal age differences in birth weight. *Am J Epidemiol*. 1995;142:504–514
10. Friede A, Baldwin W, Rhodes PH, et al. Young maternal age and infant mortality: the role of low birth weight. *Public Health Rep*. 1987;102: 192–199
11. Froen JF, Arnestad M, Frey K, Vege A, Saugstad OD, Stray-Pedersen B. Risk factors for sudden intrauterine unexplained death: epidemiologic characteristics of singleton cases in Oslo, Norway, 1986–1995. *Am J Obstet Gynecol*. 2001;184:694–702
12. Grogger J. Incarceration-related costs of early childbearing. Maynard RA, Ed. *Kids Having Kids: Economic Costs and Social Consequences of Teen Pregnancy*. Washington, DC: Urban Institute Press; 1997:231–256
13. Olausson PO, Cnattingius S, Haglund B. Teenage pregnancies and risk of late fetal death and infant mortality. *Br J Obstet Gynaecol*. 1999;106: 116–121
14. Jolly MC, Sebire N, Harris J, Robinson S, Regan L. Obstetric risks of pregnancy in women less than 18 years old. *Obstet Gynecol*. 2000;96: 962–966
15. Hotz VJ, McElroy SW, Sanders SG. The impacts of teenage childbearing
on the mothers and the consequences of those impacts for government. Maynard RA, Ed. *Kids Having Kids: Economic Costs and Social Consequences of Teen Pregnancy*. Washington, DC: Urban Institute Press; 1997: 55–94
16. Kirby D. Reflections on two decades of research on teen sexual behavior and pregnancy. *J Sch Health*. 1999;69:89–94
17. Lundberg S, Plotnick RD. Adolescent premarital childbearing: do economic incentives matter? *J Labor Econ*. 1995;13:177–200
18. Ku L, Sonenstein FL, Pleck JH. Neighborhood, family, and work: influences on the premarital behaviors of adolescent males. *Soc Forces*. 1993; 72:479–503
19. Moore KA, Blumenthal C, Sugland BW, Hyatt B, Snyder NO, Morrison DR. *State Variation in Rates of Adolescent Pregnancy and Childbearing*. Washington, DC: Child Trends, Inc; 1994
20. Singh S. Adolescent pregnancy in the United States: an interstate analysis. *Fam Plann Perspect*. 1986;18:210–220
21. Bearman PS, Bruckner H. *Power in Numbers: Peer Effects on Adolescent Girls' Sexual Debut and Pregnancy*. Washington, DC: National Campaign to Prevent Teen Pregnancy; 1999
22. East PL. The younger sisters of childbearing adolescents: their attitudes, expectations, and behaviors. *Child Dev*. 1996;67:267–282
23. Holden GW, Nelson PB, Velasquez J, Ritchie KL. Cognitive, psychosocial, and reported sexual behavior differences between pregnant and nonpregnant adolescents. *Adolescence*. 1993;28:557–572
24. Alan Guttmacher Institute. *Sex and America's Teenagers*. New York, NY: Alan Guttmacher Institute; 1994
25. Dittus PJ, Jaccard J. Adolescents' perceptions of maternal disapproval of sex: relationship to sexual outcomes. *J Adolesc Health*. 2000;26:268–278
26. Chandy JM, Harris L, Blum RW, Resnick MD. Female adolescents of alcohol misusers: sexual behaviors. *J Youth Adolesc*. 1994;23:695–709
27. Smith CA. The link between childhood maltreatment and teenage pregnancy. *Soc Work Res*. 1996;20:131–141
28. Pierre N, Shrier LA, Emans SJ, Durant RH. Adolescent males involved in pregnancy: associations of forced sexual contact and risk behaviors. *J Adolesc Health*. 1998;23:364–369
29. Springarn RW, DuRant RH. Male adolescents involved in pregnancy: associated health risk and problem behaviors. *Pediatrics*. 1996;98: 262–268
30. Raj A, Silverman JG, Amaro H. The relationship between sexual abuse and sexual risk among high school students: findings from the 1997 Massachusetts Youth Risk Behavior Survey. *Matern Child Health J*. 2000; 4:125–134
31. Allen JP, Kuperminc G, Philliber S, Herre K. Programmatic prevention of adolescent problem behaviors: the role of autonomy, relatedness, and volunteer service in the Teen Outreach Program. *Am J Community Psychol*. 1994;22:617–638
32. Allen JP, Philliber S, Hoggson N. School-based prevention of teen-age pregnancy and school dropout: process evaluation of the national replication of the Teen Outreach Program. *Am J Community Psychol*. 1990; 18:505–524
33. Nitz K. Adolescent pregnancy prevention: a review of interventions and programs. *Clin Psychol Rev*. 1999;19:457–471
34. Hillis SD, Anda RF, Felitti VJ, Nordenberg D, Marchbanks PA. Adverse childhood experiences and sexually transmitted diseases in men and women, a retrospective study. *Pediatrics*. 2000;106(1). Available at: http://www.pediatrics.org/cgi/content/full/106/1/e11
35. Dietz PM, Spitz AM, Anda RF, et al. Unintended pregnancy among adult women exposed to abuse or household dysfunction during their childhood. *JAMA*. 1999;282:1359–1364
36. Anda RF, Croft JB, Felitti VJ, et al. Adverse childhood experiences and smoking during adolescence and adulthood. *JAMA*. 1999;282:1652–1658
37. Hillis SD, Anda RF, Felitti VJ, Marchbanks PA. Adverse childhood experiences and sexual risk behaviors in women: a retrospective cohort study. *Fam Plann Perspect*. 2001;33:206–211
38. Felitti VJ, Anda RF, Nordenberg D, et al. The relationship of adult health status to childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *Am J Prev Med*. 1998;14:245–258
39. Anda RF, Felitti VJ, Chapman DP, et al. Abused boys, battered mothers, and male involvement in teen pregnancy: new insights for pediatricians. *Pediatrics*. 2001:107(2). Available at: http://pediatrics. aappublications.org/cgi/content/full/107/2/e19
40. Dube SR, Anda RF, Felitti VJ, Chapman DP, Williamson DF, Giles WH. Childhood abuse, household dysfunction, and the risk of attempted suicide throughout the life span: findings from the Adverse Childhood Experiences Study. *JAMA* 2001;286:3089–3096
41. Dube SR, Anda RF, Felitti VJ, Edwards VJ, Croft JB. Adverse childhood

experiences and personal alcohol abuse as an adult. *Addict Behav*. 2002; 27:713–725

42. Dube SR, Anda RF, Felitti VJ, Edwards VJ, Williamson DF. Exposure to abuse, neglect, and household dysfunction among adults who witnessed intimate partner violence as children: implications for health and social services. *Violence Vict*. 2002;17:3–17

43. Straus M, Gelles RJ. *Physical Violence in American Families: Risk Factors and Adaptations to Violence in 8,145 Families*. New Brunswick, NJ: Transaction Press; 1990

44. Wyatt GE. The sexual abuse of Afro-American and white-American women in childhood. *Child Abuse Negl*. 1985;9:507–519

45. Schoenborn CA. Exposure to alcoholism in the family: United States, 1988. *Adv Data*. 1991;205:1–13

46. Kleinbaum DK, Kupper LL, Morgensern H. *Epidemiologic Research*. New York, NY: Van Nostrand Reinhold Company; 1982:255–257

47. Fleiss JL. *Statistical Methods for Rates and Proportions*. New York, NY: John Wiley & Sons; 1981:143–146

48. Hennekens CH, Curing JE. *Epidemiology in Medicine*. Boston, MA: Little, Brown, and Company; 1987

49. McAnarney ER. Young maternal age and adverse neonatal outcome. *Am J Dis Child*. 1987;141:1053–1059

50. Resnick MD, Bearman PS, Blum RW, et al. Protecting adolescents from harm: findings from the National Longitudinal Study on Adolescent Health. *JAMA*. 1994;278:823–832

51. Strasburger VC, Donnerstein E. Children, adolescents, and the media: issues and solutions. *Pediatrics*. 1999;103:129–139

52. Peterson C, Seligman ME, Vaillant GE. Pessimistic explanatory style is a risk factor for physical illness: a thirty-five year longitudinal study. *J Pers Soc Psychol*. 1988;55:23–27

53. Seligman ME. *Learned Optimism*. New York, NY: Knopf; 1991

54. Institute of Medicine. Conclusions and recommendations. In: Brown SS, Eisenberg L, Eds. *The Best Intentions: Unintended Pregnancy and the Well-Being of Children and Families*. Washington, DC: National Academy Press; 1995:250–270

55. Cousins N. *Head First: The Biology of Hope*. New York, NY: E. P. Dutton; 1989

56. Diamond M, Hopson J. *Magic Trees of the Mind*. New York, NY: Plume; 1998

57. Coid JW. Relation between childhood sexual and physical abuse and risk of revictimisation in women: a cross-sectional study. *Lancet*. 2001; 358:450–454

58. Lee MC. Family and adolescent childbearing. *J Adolesc Health*. 2001;28: 307–312

59. Geronimus AT, Korenman S. Maternal youth or family background? On the health disadvantages of infants with teenage mothers. *Am J Epidemiol*. 1993;137:213–225

60. Geronimus A, Korenman S. The socioeconomic consequences of teen childbearing reconsidered. *Q J Econ*. 1992;107:1187–1214

61. Huang DY, Usher RH, Kramer MS, Yang H, Morin L, Fretts RC. Determinants of unexplained antepartum fetal deaths. *Obstet Gynecol*. 2000; 95:215–221

62. Scholl TO, Hediger ML, Huang J, Johnson FE, Smith W, Ances IG. Young maternal age and parity: influences on pregnancy outcomes. *Ann Epidemiol*. 1992;2:565–575

63. Raymond EG, Cnattingius S, Kiely JL. Effects of maternal age, parity, and smoking on the risk of stillbirth. *Br J Obstet Gynaecol*. 1994;101: 301–306

64. Winbo I, Serenius F, Dahlquist G, Kallen B. Maternal risk factors for cause-specific stillbirth and neonatal death. *Acta Obstet Gynecol Scand*. 2001;80:235–244

65. Mills JL, Graubard BI, Hurley EE, Rhoads GG, Berendes HW. Maternal alcohol consumption and birth weight: how much drinking during pregnancy is safe? *JAMA*. 1984;252:1875–1879

66. Winbo I. Smoking, alcohol, and reproduction. *Am J Public Health*. 1983; 73:115406

67. Allsworth JE, Zierler S, Krieger N, Harlow BL. Ovarian function in late reproductive years in relation to lifetime experiences of abuse. *Epidemiology*. 2001;12:676–681

68. US Department of Health and Human Services. *Healthy People 2010: Understanding and Improving Health*. 2nd Ed. Washington, DC: US Government Printing Office; 2000

69. Olds DL, Eckenrode J, Henderson CR Jr, et al. Long-term effects of home visitation on maternal life course and child abuse and neglect: fifteen year follow-up of a randomized trial. *JAMA*. 1997;278:637–643

70. Olds DL, Henderson CR Jr, Chamberlin R, Tatelbaum R. Preventing child abuse and neglect: a randomized trial of nurse home visitation. *Pediatrics*. 1985;78:65–78

71. Guijarro S, Naranjo J, Padilla M, Gutierez R, Lammers C, Blum RW. Family risk factors associated with adolescent pregnancy: study of a group of adolescent girls and their families in Ecuador. *J Adolesc Health*. 1999;25:166–172

72. Sieving RE, NcNeely CS, Blum RW. Maternal expectations, mother-child connectedness, and adolescent sexual debut. *Arch Pediatr Adolesc Med*. 2000;154:809–816

RECORDING CONSULTATIONS

"It is a wonderful idea to record a consultation. Everyone should. How can you really listen, really listen, as you sit in a strange room and dice over your own life with a doctor you have never met before?"

Gearin-Tosh M. *Living Proof*. Scribner; 2002

Submitted by Student

Updated Information & Services	including high-resolution figures, can be found at: http://www.pediatrics.org/cgi/content/full/113/2/320
References	This article cites 51 articles, 12 of which you can access for free at: http://www.pediatrics.org/cgi/content/full/113/2/320#BIBL
Citations	This article has been cited by 25 HighWire-hosted articles: http://www.pediatrics.org/cgi/content/full/113/2/320#otherarticles
Subspecialty Collections	This article, along with others on similar topics, appears in the following collection(s): **Adolescent Medicine** http://www.pediatrics.org/cgi/collection/adolescent_medicine
Permissions & Licensing	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://www.pediatrics.org/misc/Permissions.shtml
Reprints	Information about ordering reprints can be found online: http://www.pediatrics.org/misc/reprints.shtml

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™