The impact of adverse childhood experiences on health problems: evidence from four birth cohorts dating back to 1900

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Abstract

Background. We examined the relationship of the number of adverse childhood experiences (ACE score) to six health problems among four successive birth cohorts dating back to 1900 to assess the strength and consistency of these relationships in face of secular influences the 20th century brought in changing health behaviors and conditions. We hypothesized that the ACE score/health problem relationship would be relatively “immune” to secular influences, in support of recent studies documenting the negative neurobiologic effects of childhood stressors on the developing brain.

Methods. A retrospective cohort study of 17,337 adult health maintenance organization (HMO) members who completed a survey about childhood abuse and household dysfunction, as well as their health. We used logistic regression to examine the relationships between ACE score and six health problems (depressed affect, suicide attempts, multiple sexual partners, sexually transmitted diseases, smoking, and alcoholism) across four successive birth cohorts: 1900–1931, 1932–1946, 1947–1961, and 1962–1978.

Results. The ACE score increased the risk for each health problem in a consistent, strong, and graded manner across four birth cohorts (P < 0.05). For each unit increase in the ACE score (range: 0–8), the adjusted odds ratios (ORs) for depressed affect, STDs, and multiple sexual partners were increased within a narrow range (ORs: 1.2–1.3 per unit increase) for each of the birth cohorts; the increase in risk for suicide attempts was stronger but also in a narrow range (ORs: 1.5–1.7).

Conclusions. Growing up with ACEs increased the risk of numerous health behaviors and outcomes for 20th century birth cohorts, suggesting that the effects of ACEs on the risk of various health problems are unaffected by social or secular changes. Research showing detrimental and lasting neurobiologic effects of child abuse on the developing brain provides a plausible explanation for the consistency and dose–response relationships found for each health problem across birth cohorts, despite changing secular influences.

Keywords: Childhood abuse; Household dysfunction; Health behaviors; Secular trends

Over the past century, advances in medicine and public health reduced morbidity and increased life expectancy in the United States [1,2]. In the early half of the 1900s, infectious diseases such as tuberculosis and pneumonia were the leading causes of death; currently, chronic diseases account for the majority of deaths in the United States [3]. From about 1950 to the present, research about health behaviors, which are risk factors for multiple types of chronic diseases, and many infectious diseases that are currently public health problems [2,4–7] has proliferated. Although these efforts have provided vast information on lifestyle and behavioral risk factors for disease, the underlying determinants of such risk factors and lifestyles remain poorly understood. For example, most persons are aware that smoking causes lung cancer and heart disease, but why do some people become smokers and what explains differences in ability to quit [8]?

An expanding body of research suggests that childhood abuse can lead to a variety of negative health outcomes and behaviors, such as substance abuse, suicidal behaviors, and
depressive disorders [9–17]. The Adverse Childhood Experiences (ACE) Study is a large-scale epidemiological study that assesses the impact of various forms of childhood abuse and household dysfunction on a wide variety of health behaviors and outcomes from adolescence to adulthood. Because these types of experiences rarely occur in isolation and are highly interrelated [18,19] the ACE Study uses a cumulative stressor model to assess the relationship between the total number of these childhood exposure (ACE score) and various health outcomes. Recent publications from this study have repeatedly shown a strong, graded relationship between the ACE score and high-priority health and social problems such as smoking [19], unintended pregnancies [20], sexually transmitted diseases [21], male involvement in teen pregnancy [22], adult alcohol problems [23,24], attempted suicides [25], illicit drug use [26], and leading causes of death in the United States [18].

Many health behaviors and outcomes that are important in medicine and public health and that have been a focus of the ACE Study have been influenced by secular, social, and/or economic changes, as well as evolving medical and public health initiatives to alter them. For instance, certain psychiatric disorders have increased in prevalence over the past several decades [27–30]; suicides and suicide attempts have increased among younger cohorts [30,31] as is the case when examined cross-culturally [32]. Economic [30,32] and societal influences [28], as well as campaigns to increase screening for depressive disorders [33] have been postulated as reasons for these secular trends.

Sexual behaviors have also changed due to social influences [34]. Over the past several decades a trend for younger age at first intercourse [34,35], later age for first marriage [34,36], and higher rates of divorce [36,37] have emerged. These trends likely contributed to increases in the number of sexual partners over a lifetime as well as the increased risk for STDs, especially among adolescents [34,36].

The prevalence of alcohol and tobacco use have changed considerably over the past century [38]. For example, the NLAES study also found that alcohol use in early adulthood (ages 20 through 24) was less than 50% for those born between 1874 and 1937 (pre-World War II), whereas it was 75% for those born between 1968 and 1974 (Vietnam War era) [39]. Trends in cigarette smoking are among the most notable of these secular influences, due to factors such as changing attitudes about the social desirability of smoking, sales taxes, and the dissemination of information about their health effects. Per capita cigarette consumption in the United States was very low from 1900 until 1917, peaked in 1960, and has declined significantly since the Surgeon General’s report in 1964 [40,41].

In this study we examine the graded relationship between the ACE score and six health problems among four successive birth cohorts dating back to 1900 to assess the strength and consistency of this relationship in the face of powerful secular influences. Because of recent studies documenting the negative and lasting neurobiologic effects of childhood trauma/stressors on the developing brain [42,43] we hypothesized that the ACE score/health problem relationship would be resistant or “immune” to secular influences, such as changing social attitudes and the dissemination of health information that primarily exert their influence on adolescents and adults. Specifically, we assessed the strength and consistency of the relationships between the ACE score and depressed affect, suicide attempts, multiple sexual partners, sexually transmitted diseases smoking, and alcoholism for the following successive birth cohorts: 1900–1931, 1932–1946, 1947–1961, and 1962–1978. We reasoned that if the ACE score increased the risk for each of these health problems across birth cohorts in a consistent and graded manner, this would suggest that these types of childhood experiences exert an effect irrespective of the social, economic, political, and secular influences specific to the era in which these persons were born and raised.

Methods

The ACE Study is a collaboration between Kaiser Permanente’s Health Appraisal Center (HAC) in San Diego, California, and the U.S. Centers for Disease Control and Prevention. The ACE Study was approved by the institutional review boards of Kaiser Permanente, Emory University, and the U.S. Department of Health and Human Services. Potential participants were sent letters that accompanied the ACE Study questionnaire informing them that their participation was voluntary and their answers would held in strictest confidence and would never become part of their medical record.

Study population

The study population was drawn from the HAC, which provides standardized medical, psychosocial, and preventive health evaluations to adult members of Kaiser Health Plan in San Diego County. In any 4-year period, 81% of the adult members obtain this service and over 50,000 members are evaluated yearly. Thus, data from the HAC represents the experiences and health of a majority of adult Kaiser members in San Diego. Additionally, their visit to the HAC is primarily for the purposes of complete health assessments, rather than symptom or illness-based care.

All persons evaluated at the HAC complete a standardized questionnaire, which includes health histories and health-related behaviors, a medical review of systems, and psychosocial evaluations. This information was included in the ACE Study database.

Two weeks after their HAC evaluation, each person who received the ACE Study questionnaire by mail. The questionnaire collected detailed information about ACEs (e.g.,
abuse), household dysfunction (e.g., domestic violence), and health-related behaviors from adolescence to adulthood. Wave 2 respondents were asked detailed questions about health topics than analysis of wave 1 data had shown to be important [18]. The response rate for both survey waves combined was 68%, for a total of 18,175 responses.

We excluded 754 respondents who coincidentally underwent examinations during the time frames for both survey waves. The unduplicated total number of respondents was 17,421. After exclusion of 17 respondents with missing information about race and 67 with missing information about educational attainment, the final study sample included 95% of the respondents (17,337/18,175) (Wave I=8,708, Wave II=8,629).

Assessment of representativeness, and response or reporting bias

In Wave I, the HAC questionnaire data were abstracted for both respondents and nonrespondents to the ACE Study questionnaire, enabling a detailed assessment of the representativeness of respondents in terms of demographic characteristics and health-related issues. Results of this analysis have been published elsewhere [44]. Briefly, nonrespondents tended to be younger, less educated, or from racial/ethnic minority groups. After controlling for demographic differences, health behaviors such as smoking, alcohol or drug abuse, and health conditions like heart disease, hypertension, obesity, and chronic lung disease did not differ between respondents and nonrespondents. Thus, there was no evidence that the general health of respondents and nonrespondents differed.

In addition, questions allowed assessment of the strength of the relationship between childhood sexual abuse and health behaviors, diseases, and psychosocial problems; the strength of these relationships was virtually identical for respondents and nonrespondents [44]. Thus, there was no evidence that respondents to the ACE Study questionnaire were biased toward attributing their health problems to childhood experiences such as sexual abuse [44].

Definitions of adverse childhood experiences (ACEs)

Questions used to define ACEs are listed in Table 1. All questions about ACEs pertained to the respondents’ first 18 years of life (≤18 years of age). For questions adapted from the Conflict Tactics Scale (CTS) [45] there were five response categories: “never,” “once or twice,” “sometimes,” “often,” or “very often.” We defined three types of childhood abuse: emotional abuse (two questions), physical abuse (two questions), or contact sexual abuse (four questions) by Wyatt [46]. In addition, we defined five exposures to household dysfunction during childhood: exposure to substance abuse (defined by two questions) [47], mental illness (two questions), violent treatment of mother or stepmother (four questions) [45], criminal behavior in the household (one question), and parental separation or divorce (one question). Respondents were defined as exposed to a category if they responded “yes” to one or more of the questions in that category. The total number of these exposures (range: 0–8) was summed to create the ACE score. Due to small sample sizes, ACE scores of 4 or more were combined into one category (≥4). Thus, analyses were conducted with the summed score as four dichotomous variables (yes/no) with 0 experiences as the referent.

Definition of birth cohorts

The four birth cohort groups were based on respondents’ age at entry into the study using 15-year increments, with the exception of the cohort dating from 1900 to 1931. Due to small sample sizes we combined 1900–1915 and 1919–1931. The four cohort groups have been used in prior reports from the ACE Study that examined the associations of the ACE score on male involvement in teen pregnancy and illicit drug use by birth cohort [26,48].

Definition of selected health behaviors and health outcomes

Mental health
Depressed affect. Depressed affect was defined as a “yes” response to the question that was included in both ACE Study survey waves “Have you had or do you now have depression or feel down in the dumps?” We compared the measure of depressed affect to a validated screening tool developed by the Rand Corporation for lifetime prevalence of major depression or dysthymia (which was available for ACE survey Wave I only) [49]. In this comparison (2 × 2 table), lifetime depressed affect was significantly associated with the validated measure [49] (X² = 1476, df = 1; P < 0.0001); the sensitivity, specificity, and predictive value positive for lifetime depressed affect were 83, 60, and 87%, respectively.

Lifetime suicide attempt (ever attempting suicide). Attempted suicide was defined as a “yes” response to the question “Have you ever attempted to commit suicide?” Using data available from Wave II only, the mean number of suicide attempts (standard deviation) was 1.6 (91); the range was 1–4 times; 75th and 95th percentiles were 2 and 4, respectively. Number of attempts did not differ between men and women or according to the ACE Score.

Sexual health
Multiple sexual partners. Defined as having 30 or more sexual partners over a lifetime.

Sexually transmitted diseases. A “yes” response to the HAC question: “Have you ever been treated for or told you had any venereal disease?”
Substance use and abuse

Ever been a cigarette smoker. The following question defined this behavior: “Have you smoked at least 100 cigarettes in your entire life?” A “yes” response defined ever being a smoker. The median age at initiation for smoking was 18 years for birth cohorts 1900–1931, 1932–1946, and 1947–1961 and 17 years for 1962–1978.

Self-reported alcoholic. A “yes” response to the question “Have you ever considered yourself to be an alcoholic?” defined self-reported alcoholism [47]. “Assessment of the methodological studies indicate that for the general population, self-reports of alcohol use are fairly accurate” [50]. Furthermore, assuring respondents of the confidentiality of their responses (which was part of the ACE Study protocol) and providing responses in a private setting (mail survey in the home for the ACE Study) also enhance the accuracy of self-reported alcohol abuse [50,51]. The prevalence we found for self-reported alcoholism, 6.5%, is similar to previously reported data on alcohol dependence [52].

Statistical analysis

All analyses were conducted using SAS (version 8.2). Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were obtained from multiple logistic regression models that estimated the likelihood of the selected health behavior or outcome with the ACE score stratified by birth cohort. Covariates in all models were included on a priori reasoning rather than using stepwise selection and included age (continuous variable), sex, race, and education (high school diploma, some college, or college graduate versus less than high school). We had no a priori hypotheses about interaction between demographic variables and the ACEs to examine. Using SAS regression diagnostics, we found no evidence of collinearity. In the data that we present herein,
persons with incomplete information about an ACE were considered not to have had that experience. To assess the potential effect of this assumption, we repeated our analysis after excluding any respondent who had missing information on any ACE and found no substantial difference in the results.

To test for a trend (graded relationship) between the ACE score and the risk of each selected health outcome by birth cohort, we entered ACE score as an ordinal variable into logistic models, with adjustment for the demographic covariates (sex, age, race, and education). We used this test to assess the consistency of the association between the ACE score and the risk of each selected health outcome by birth cohort, we entered ACE score as an ordinal variable.

### Results

#### Characteristics of study population
The study population included 9367 women (54%) and 7970 men (46%). The mean age (standard deviation) was 56 (15.2) years. Seventy-five percent of participants were white, 39% were college graduates, 36% had some college education, and 18% were high school graduates. Only 7% had not graduated from high school.

#### Adverse childhood experiences
The prevalence of each individual ACE and of the ACE scores is shown in Table 1. Women were more likely than men to report ACEs, with exception of physical abuse (Table 1). Sixty-four percent of respondents reported at least 1 ACE. We found no substantial difference in prevalence of ACEs between Waves 1 and 2; after adjusting for base age, educational attainment, sex, and race the mean ACE score was 1.5 for both waves.

#### Risk for health behaviors and outcomes by birth cohort
In Table 2 we present data for mental health outcomes for each of four birth cohorts. As the ACE score increased, there was a graded increase in the likelihood of reporting depressed affect and suicide attempts for all cohorts. Compared to 0 ACEs there was a 4-fold increased likelihood of depressed affect for any of the cohorts that reported 4 or more ACEs (p < .05). For suicide attempts there was a 9-fold to 13-fold increase for any of the cohorts if persons had reported 4 or more ACEs compared to persons with no ACEs (p < .05).

### Table 2

| ACE Score | 1900–1931 | 1932–1946 | 1947–1961 | 1962–1978 |
|----------|-----------|-----------|-----------|-----------|
|          | N  | %   | Odds ratio | N  | %   | Odds ratio | N  | %   | Odds ratio | N  | %   | Odds ratio |
| Depressed affect | | | | | | | | | | | | |
| 0        | 2625 | 16.7 | 1.0  | 1936 | 17.8 | 1.0  | 1279 | 21.6 | 1.0  | 415 | 21.6 | 1.0  |
| 1        | 1540 | 19.2 | 1.2  (1.1–1.4) | 1473 | 26.3 | 1.7  (1.4–2.0) | 1082 | 31.3 | 1.6  (1.3–1.9) | 419 | 27.2 | 1.3  (1.0–1.8) |
| 2        | 765  | 26.9 | 1.9  (1.6–2.3) | 901  | 36.9 | 2.7  (2.2–3.2) | 776  | 37.5 | 2.1  (1.7–2.6) | 316 | 34.4 | 1.9  (1.4–2.7) |
| 3        | 356  | 28.1 | 2.0  (1.5–2.5) | 555  | 38.9 | 2.8  (2.3–3.5) | 526  | 45.1 | 2.7  (2.2–3.4) | 213 | 41.3 | 2.4  (1.7–3.4) |
| ≥4       | 302  | 43.1 | 3.8  (2.9–4.9) | 669  | 47.5 | 3.7  (3.1–4.6) | 831  | 51.5 | 3.5  (2.9–4.3) | 358 | 51.1 | 3.6  (2.6–5.0) |
| Totalb   | 5588 | 20.9 | 1.3  (1.3–1.4) | 5534 | 28.9 | 1.3  (1.3–1.4) | 4494 | 35.0 | 1.3  (1.2–1.3) | 1721| 34.1 | 1.3  (1.2–1.3) |

| Suicide attempts | | | | | | | | | | | | |
| Odds ratio in this row represents test for trend (P < .05), with ACE score as an ordinal variable. | | | | | | | | | | | | |

a All odds ratios adjusted for sex, race, education, and age at survey.
b Odds ratio in this row represents test for trend (P < .05), with ACE score as an ordinal variable.
Prevalence and adjusted odds ratios\(^a\) for the relationship between ACE score and multiple sexual partners and sexually transmitted diseases, stratified by birth cohort

| ACE Score | N    | %    | Odds ratio | N    | %    | Odds ratio | N    | %    | Odds ratio | N    | %    | Odds ratio |
|-----------|------|------|------------|------|------|------------|------|------|------------|------|------|------------|
|           | 1900–1931 |      |            | 1932–1946 |      |            | 1947–1961 |      |            | 1962–1978 |      |            |
| 0         | 2625  | 2.1  | 1.0 (referent) | 1936  | 4.9  | 1.0 (referent) | 1279  | 6.3  | 1.0 (referent) | 415  | 3.6  | 1.0 (referent) |
| 1         | 1540  | 4.4  | 1.9 (1.3–2.7) | 1473  | 5.4  | 1.1 (0.8–1.6) | 1082  | 6.8  | 1.1 (0.8–1.5) | 419  | 3.6  | 0.9 (0.4–1.9) |
| 2         | 765   | 5.9  | 2.7 (1.8–4.1) | 901   | 8.8  | 2.0 (1.4–2.7) | 776   | 7.7  | 1.3 (0.9–1.9) | 316  | 7.0  | 2.1 (1.1–4.1) |
| 3         | 356   | 4.5  | 2.1 (1.2–3.8) | 555   | 11.2 | 2.9 (2.1–4.2) | 526   | 9.9  | 1.9 (1.3–2.8) | 213  | 7.0  | 2.4 (1.1–5.1) |
| ≥4        | 302   | 6.6  | 3.6 (2.1–6.2) | 669   | 10.0 | 3.1 (2.2–4.4) | 831   | 14.2 | 3.7 (2.7–5.0) | 358  | 8.1  | 2.8 (1.4–5.5) |
| Total\(^b\) | 5588  | 3.6  | 1.3 (1.2–1.4) | 5534  | 6.9  | 1.3 (1.2–1.4) | 4494  | 8.6  | 1.3 (1.2–1.4) | 1721 | 5.6  | 1.2 (1.1–1.4) |

Multiple sexual partners (≥30 over a lifetime)

| ACE Score | N    | %    | Odds ratio | N    | %    | Odds ratio | N    | %    | Odds ratio | N    | %    | Odds ratio |
|-----------|------|------|------------|------|------|------------|------|------|------------|------|------|------------|
| 0         | 2625  | 3.0  | 1.0 (referent) | 1936  | 4.7  | 1.0 (referent) | 1279  | 9.6  | 1.0 (referent) | 415  | 9.6  | 1.0 (referent) |
| 1         | 1540  | 3.3  | 1.1 (0.8–1.6) | 1473  | 7.7  | 1.8 (1.3–2.4) | 1082  | 13.0 | 1.4 (1.1–1.8) | 419  | 15.0 | 1.6 (1.1–2.5) |
| 2         | 765   | 4.8  | 1.6 (1.1–2.5) | 901   | 7.4  | 1.7 (1.2–2.4) | 776   | 15.3 | 1.7 (1.3–2.2) | 316  | 17.7 | 2.0 (1.3–3.2) |
| 3         | 356   | 5.3  | 2.0 (1.2–3.3) | 555   | 11.5 | 2.9 (2.0–4.0) | 526   | 15.8 | 1.8 (1.3–2.4) | 213  | 25.8 | 3.3 (2.1–5.2) |
| ≥4        | 302   | 8.6  | 3.4 (2.1–5.5) | 669   | 10.2 | 2.6 (1.9–3.7) | 831   | 21.9 | 2.9 (2.2–3.7) | 358  | 22.1 | 2.7 (1.8–4.1) |
| Total\(^b\) | 5588  | 3.8  | 1.3 (1.2–1.4) | 5534  | 7.3  | 1.2 (1.2–1.3) | 4494  | 14.4 | 1.2 (1.2–1.3) | 1721 | 17.0 | 1.2 (1.1–1.3) |

Sexually transmitted diseases

\(\text{a All odds ratios adjusted for sex, race, education, and age at survey.}\)

\(\text{b Odds ratio in this row represents test for trend (P < .05), with ACE score as an ordinal variable.}\)

Statistical test for a trend between ACE score and health outcomes in each birth cohort

We performed a test for trend between the ACE score and each health problem for persons born in each cohort: 1900–1931, 1932–1946, 1947–1961, and 1962–1978. The four ORs for depressed affect were 1.3 for each cohort (Table 2). These results suggest that for every increase in the ACE score the risk of depressed affect increased by

\(\text{Table 4}\)

Prevalence and adjusted odds ratios\(^a\) for the relationship between substance use/problems and the ACE score, stratified by birth cohort

| ACE Score | 1900–1931 |      | Odds ratio | 1932–1946 |      | Odds ratio | 1947–1961 |      | Odds ratio | 1962–1978 |      | Odds ratio |
|-----------|-----------|------|------------|-----------|------|------------|-----------|------|------------|-----------|------|------------|
| 0         | 2625      | 2.0  | 1.0 (referent) | 1936      | 3.1  | 1.0 (referent) | 1279      | 3.1  | 1.0 (referent) | 415      | 3.1  | 1.0 (referent) |
| 1         | 1540      | 3.7  | 1.8 (1.2–2.6) | 1473      | 5.9  | 1.9 (1.4–2.7) | 1082      | 6.3  | 2.0 (1.3–3.0) | 419      | 4.1  | 3.1 (1.2–8.8) |
| 2         | 765       | 4.8  | 2.4 (1.5–3.6) | 901       | 9.7  | 3.3 (2.4–4.7) | 776       | 8.6  | 2.8 (1.9–4.2) | 316      | 4.4  | 3.5 (1.2–9.9) |
| 3         | 356       | 7.6  | 4.0 (2.5–6.5) | 555       | 11.5 | 4.3 (2.9–6.1) | 526       | 12.9 | 4.5 (3.0–6.9) | 213      | 7.0  | 6.0 (2.1–17.0) |
| ≥4        | 302       | 13.9 | 5.1 (3.1–8.3) | 669       | 16.4 | 7.0 (5.0–9.9) | 831       | 17.6 | 7.3 (5.0–10.6) | 358      | 12.9 | 10.9 (4.2–28.1) |
| Total\(^b\) | 5588      | 3.6  | 1.5 (1.3–1.6) | 5534      | 7.4  | 1.5 (1.4–1.5) | 4494      | 8.7  | 1.4 (1.3–1.5) | 1721     | 5.6  | 1.4 (1.3–1.6) |

\(\text{a All odds ratios adjusted for sex, race, education, and age at survey.}\)

\(\text{b Odds ratio in this row represents test for trend, with ACE score as an ordinal variable.}\)
30%. For suicide attempts, the ordinal ORs were 1.5 to 1.7; the risk of suicide attempts increased by 50–70% for every unit increase in the ACE score. For multiple sexual partners and sexually transmitted diseases the ordinal ORs for the test for trend was between 1.2 and 1.3; a 20 to 30% increased likelihood for these two outcomes exists for every increase in the ACE score, in all four birth cohorts (Table 3).

The test for trend showed a 20–30% increased risk for smoking and a 40–50% increased risk for self-reported alcoholism when examining all cohorts (Table 4).

There was no significant difference in the ordinal ORs between each of the four birth cohorts for any of the health problems that we examined. Thus, the test for trend assessed the strength of the relationship between the ACE score and health problems for each birth cohort; the overlapping 95% CIs indicated that there was no meaningful difference in these relationships according to birth cohort.

Discussion

The risk of depressed affect, suicide attempts, multiple sexual partners, sexually transmitted diseases, ever smoking cigarettes, and alcoholism increased in a graded manner as the ACE score increased in each of four successive birth cohorts from 1900 to 1978. Furthermore, the strength of these graded relationships did not differ substantially or in a statistically significant manner between successive birth cohorts. The difference in the overall prevalence for health behaviors observed in each birth cohort is consistent with varied secular influences. However, the focus of the present analyses was to examine how increasing ACE scores affect the risk (adjusted OR) of these behaviors within successive birth cohorts. The remarkably consistent relationships between the ACE score and the risk of a variety of health behaviors and outcomes suggest that the mechanisms by which these stressful experiences exert their effect are resistant to, or unaffected by, the many and changing influences on health and behavior such as those that occurred throughout the 20th century.

The graded or “dose–response” relationship between the burden of ACEs and the risk of the six health outcomes for successive birth cohorts dating back to 1900 is consistent with emerging information about the neurobiological effects of early traumatic experiences on the developing brain of infants and young children. Neurological development during early childhood is the foundation on which experiences, positive or negative, are organized and processed. Home and family environments and the characteristics of the parents and persons to whom children are exposed are powerful determinants of emotional, behavioral, cognitive, social, and physiologic functioning later in life [42,43]. Specifically, child abuse and neglect can adversely affect the developing brain in ways that result in emotional, social, and cognitive impairments, increasing the risk for substance abuse, depression, suicide, and a variety of other problems [54–57]. Thus, the strong association in successive 20th century birth cohorts between the ACE score and the six health outcomes offers compelling evidence that the impact of ACEs on multiple types of health problems is a consistent phenomenon that may have its roots in the inherent biologic effects of traumatic stressors (ACEs) on the developing nervous system of children.

Physiological and anatomical changes in the brains of individuals who experienced childhood abuse are progressively being documented and provide biological plausibility for our findings. For example, Teicher et al. conducted electroencephalograms (EEGs) to measure limbic irritability [58] and found the percentage of clinically significant brain-wave abnormalities to be higher among individuals who had a history of early trauma versus those who did not experience early trauma [58]. Magnetic resonance imaging (MRI) has revealed reductions in hippocampal volumes among severely sexually abused women and reductions in the intracranial and cerebral volumes among maltreated children compared to nonabused individuals [59,60]. Although the effects cannot be defined to any specific area of the brain, it has been shown that the limbic system, which is responsible for emotional response, is adversely affected. Studies such as these document the anatomic and functional neurological changes among persons who experienced one or more forms of abuse compared to nonabused individuals [54,58–60]. Because ACEs rarely occur in isolation [18,19], the cumulative effect of multiple ACEs likely has an even more powerful negative effect on a young child’s developing brain via repeated and/or chronic activation of the stress response. We speculate that in this scenario, the inherent human stress response [61] and effects of the adrenal release of catecholamines and corticosteroids on developing neurons and neural networks may provide a biological explanation for these findings.

Other explanations for the strong and consistent relationships found between adverse childhood experiences and multiple health problems across birth cohorts may be that childhood abuse and neglect result in negative psychological and social consequences. For example, abuse and neglect in childhood were shown to be associated with poor self-esteem, conflicted relationships, and severe life events in adulthood, in turn, acting as risk factors for depression and other adverse health consequences [62].

There are several potential limitations with retrospective reporting of childhood experiences and self-reporting of the outcome measures. In both cases, underreporting of ACEs and the health outcomes may have occurred. For example, respondents may have difficulty recalling certain events or may choose not to disclose health-related behaviors. Longitudinal follow-up of adults whose childhood abuse was documented has shown that their retrospective reports of childhood abuse are likely to underestimate actual occurrence [63,64]. Difficulty recalling childhood events likely results in misclassification (classifying persons truly ex-
posed to ACEs as unexposed) that would bias our results toward the null [52]. Thus, this potential weakness probably resulted in underestimates of the true strength of the relationships between ACEs and the six outcomes we examined.

Our data cannot provide certainty about the temporal relationship between the exposure (ACEs) and the six health outcomes studied. For some respondents it is possible that the outcomes may have occurred before the ACEs. However, the majority of suicide attempts (68%) occurred during adulthood [25]. Alcoholism typically takes multiple years to become fully manifest [65]. Cumulating 30 or more sexual intercourse partners would likely require multiple years of sexual activity. Our assessment of depressed affect included a lifetime history, which makes it likely that most reports included problems with depressive disorders during adulthood. Based on the finding presented, future studies may be able to build on the knowledge about depressed affect by including detailed instruments that focus on the assessment of affective symptoms in adulthood. Finally, the median age of onset of smoking was in the late teens for all birth cohorts, making it likely that ACEs antedated the initiation of smoking for most respondents. Thus, for the six health outcomes studied it is likely that the ACEs occurred before the onset of the health problems.

The prevalence of childhood exposures we report is nearly identical to those reported in surveys of the general population. We found that 16% of the men and 25% of the women met the case definition for contact sexual abuse; a national telephone survey of adults in 1990 conducted by Finkelhor et al. using similar criteria estimated that 16% of men and 27% of women had been sexually abused [66]. As for physical abuse, 28% of the men from our study had experienced this as boys, which closely parallels the percentage found (31%) in a recent population-based study of Ontario men that used questions from the same scales [67]. The similarity of the estimates from the ACE study to those of population-based studies suggests that our findings are likely to be applicable in other settings.

Our data offers compelling evidence that behaviors known to increase the risk of chronic diseases have had their origins in childhood regardless of the changing influences of the 20th century. Recent findings from the neurosciences provide biologic plausibility for both the “dose–response” relationship of the ACE score to health outcomes and the apparent “immunity” of this relationship to powerful and varied secular influences on health and behavior. Studies that have shown the social and psychological consequences due to the effects of early adverse experience also support our findings. This information challenges the disciplines of medicine and public health to address both the primary prevention and secondary prevention of ACEs in the 21st century. Primary prevention of child abuse using early life family-based intervention has shown promise [68]. Because these experiences are common, early detection and intervention may be an effective means in preventing or mitigating the myriad sequelae of ACEs. Furthermore, educating health care providers about the long-term effects of early stressful or traumatic childhood experiences on adolescents and adults may increase their ability to help patients recover from the effects of these childhood experiences.

References

[1] CDC. Ten great public health achievements—United States, 1900–1999. Morbid Mortal Weekly Rep 1999;48(12):241–3.
[2] CDC. Achievements in public health, 1900–1999: changes in the public health system. Morbid Mortal Weekly Rep 1999;48(50):1141–7.
[3] McKenna MT, Taylor WR, Marks JS, Koplan JP. Current issues and challenges in chronic disease control. In Brownson RC, editor. Chronic disease epidemiology and control. Washington, DC: APHA, 1998. p. 1–26.
[4] U.S. Department of Health and Human Services. Healthy People 2010. 2nd ed. Understanding and improving health and objectives for improving health. 2 vols. Washington, DC: U.S. Government Printing Office, November 2000.
[5] Singer M, Stopka T, Siano C, et al. The social geography of AIDS and hepatitis risk: qualitative approaches for assessing local differences in sterile-syringe access among injection drug users. Am J Public Health 2000;90:1049–56.
[6] Thorpe LE, Ouellet LJ, Levy JR, Williams IT, Monterroso ER. Hepatitis C virus infection: prevalence, risk factors, and prevention opportunities among young injection drug users in Chicago, 1997–1999. J Infect Dis 2000;182:1588–94.
[7] McGinnis JM, Foege WH. Actual causes of death in the United States. J Am Med Assoc 1993;270:2207–12.
[8] Anda RF, Williamson DF, Escobedo LG, Mast EE, Giovino GA, Remington PL. Depression and the dynamics of smoking: a national perspective. J Am Med Assoc 1990;264:1541–5.
[9] Brodsky BS, Malone KM, Ellis SP, Dulit RA, Mann JJ. Characteristics of borderline personality disorder associated with suicidal behavior. Am J Psychiat 1997;154:1715–9.
[10] Kingree JB, Thomason MP, Kaslow NJ. Risk factors for suicide attempts among low-income women with a history of alcohol problems. Addict Behav 1999;24:583–7.
[11] van der Kolk BA, Perry JC, Herman JL. Childhood origins of self-destructive behavior. Am J Psychiat 1991;148:1665–71.
[12] Kendall-Tackett KA, Williams LM, Finklehor D. Impact of sexual abuse on children: a review and synthesis of recent empirical studies. Psychol Bull 1993;113:164–80.
[13] Osofsky JD. The impact of violence on children. Future Child 1999;9(3):33–49.
[14] Mullen PE, Martin JL, Anderson JC, Romans SE, Herbison GP. Childhood sexual abuse and mental health in adult life. Br J Psychiat 1993;163:721–32.
[15] Heffernan K, Cloitre M, Tardiff K, Marzuk PM, Portera L, Leon AC. Childhood trauma as a correlate of lifetime opiate use in psychiatric patients. Addict Behav 2000;25:797–803.
[16] Kendler KS, Bulik CM, Silberg J, Hettema JM, Myers J, Prescott CA. Childhood sexual abuse and adult psychiatric and substance abuse disorders in women: an epidemiological and cotwin control analysis. Arch Gen Psychiat 2000;57:953–9.
[17] Rohsenow DJ, Corbett R, Devine D. Molested as children: a hidden contribution to substance abuse. J Subst Abuse Treat 1988;5:129.
[18] Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of 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;14:245–58.
Anda RF, Croft JB, Felitti VJ, et al. Adverse childhood experiences and smoking during adolescence and adulthood. J Am Med Assoc 1999;282:1652–8.

Dietz PM, Spitz AM, Anda RF, et al. Unintended pregnancy among adult women exposed to abuse or household dysfunction during their childhood. J Am Med Assoc 1999;282:1359–64.

Hillis SD, Anda RF, Felitti VJ, Nordenberg D, Marchbanks P. Adverse childhood experiences and sexually transmitted diseases in men and women: a retrospective study. Pediatrics 106:E11.

Anda RF, Felitti VJ, Chapman DP, et al. Abused boys, battered mothers, and male involvement in teen pregnancy. Pediatrics 2001;107:E19.

Dube SR, Anda RF, Felitti VJ, Edwards VJ, Croft JB. Adverse Childhood Experiences and personal alcohol abuse as an adult. Addict Behav 2002;7:713–25.

Anda RF, Whitfield CL, Felitti VJ, Chapman D, Edwards VJ, Dube SR, Williamson DF. Alcohol-impaired parents and adverse childhood experiences: the risk of depression and alcoholism during adulthood. J Psychiatr Serv 2002;53: In press.

Dube SR, Anda RF, Felitti VJ, Chapman D, Williamson DF, Giles WH. Childhood abuse, household dysfunction and the risk of attempted suicide throughout the life span: Findings from Adverse Childhood Experiences Study. J Am Med Assoc 2001;286:3089–96.

Dube SR, Anda RF, Felitti VJ, Chapman DP, Giles WH. Child abuse, neglect and household dysfunction and the risk of illicit drug use: The Adverse Childhood Experience Study. Pediatrics. 2003; in press.

Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S, Wittchen HU, Kendler K. Arch Gen Psychol 1994;51:8–19.

Twenge JM. The age of anxiety? Birth cohort change in anxiety and neuroticism, 1952–1993. J Pers Soc Psychol 2000;79:1007–21.

Klerman GL, Weissman MM. Increasing rates of depression. J Am Med Assoc 1989;261:2229–35.

McIntosh JL. Generational analyses of suicide: baby boomers and 13ers. Suicide Life Threat Behav 1994;24:334–42.

Kessler RC, Borges G, Walters EE. Prevalence of and risk factors for lifetime suicide attempts in the National Comorbidity Survey. Arch Gen Psychiatr 1999;56:617–26.

Diekstra RFW. The epidemiology of suicide and parasuicide. Acta Psychiatr Scand 1993;371(Suppl):9–20.

Olsof M, Marcus SC, Druss B, Elinson L, Tanielian MA, Pincus HA. National trends in the outpatient treatment of depression. J Am Med Assoc 2002;287:203–9.

Gillmore MR, Schwartz P, Civic D. The social context of sexuality: the case of the United States. In: Holmes KK, Mardh PA, Sparling PF, et al, editors. Sexually Transmitted Diseases. New York: McGraw–Hill, 1999.

Leigh BC, Morrison DM, Trocki K, Temple MT. Sexual behaviors of American adolescents: results from a U.S. national survey. J Adolesc Health 1994;15:117–25.

Michael RT, Gagnon JH, Laumann EO, Kolata G. Sex in America: A Report of the Surgeon General. Atlanta, Georgia: U.S. Dept of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Office of Smoking and Health, 2000.

Perry BD, Pollard R. Homeostasis, stress, trauma, and adaptation—a neurodevelopmental view of childhood trauma. Child Adolesc Psychiatr Clin North Am 1998;7:33–51.

DeBellis MD, Baum AS, Birnaber MA, Keshavan MS, Eccard CH, Boring AM, Jenkins FJ, Ryan ND. A. E. Bennett Research Award. Developmental traumatology. Part I. Biological stress systems. Soc Biol Psychiatr 1999;45:1259–70.

Edwards VJ, Anda RF, Nordenberg DF, Felitti VJ, Williamson DF, Wright JA. Bias assessment for child abuse survey; factors affecting probability of response to a survey about childhood abuse. Child Abuse Negl 2001;25:307–12.

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.

Wyatt GA. The sexual abuse of Afro-American and white American women in childhood. Child Abuse Negl 1985;9:507–19.

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

Anda RF, Chapman DP, Felitti VJ, Edward VE, Williamson DF, Croft JP, Giles WH. Adverse childhood experiences and risk of paternalism in teen pregnancy. Obstet Gynecol 2002;100(1):37–45.

Burnam MA, Wells KB, Leake B, Landsverk J. Development of a brief screening instrument for detecting depressive disorders. Med Care 1988;26:775–89.

U.S. Dept. of Health and Human Services. Ninth Special Report to the US Congress) on Alcohol and Health. Rockville, MD: Author, 1997.

Clark W. Some comments on methods. In: Clark WB, Hilton ME, editors. Alcohol in America: drinking practices and problems. Albany, NY: State University of New York Press, 1991, p. 19–25.

Helzer JE, Burnam A, McEvoy LT. Alcohol abuse and dependence. In Robins LN, Regier DA, editors. Psychiatric disorders in America: The Epidemiologic Catchment Area Study. New York, NY: Free Press, 1991, 81–115.

Rothman KJ, Greenland S. Modern epidemiology. Philadelphia: Lippincott Raven, 1998.

Weiss MJS, Wagner SH. What explains the negative consequences of adverse childhood experiences on adult health? Am J Prev Med 1998;14:356–60.

Green AH, Voeller K, Gaines RW, Kubie J. Neurological impairment in maltreated Children. Child Abuse Neglect 1981;5:129–34.

Perry BD, Pollard RA, Blakely TL, Baker WL, Vigilante D. Child- hood trauma, the neurobiology of adaptation and use-dependent development of the brain; how states become traits. Infant Mental Health J 1995;16:271–91.

Van der Kolk BA, Fisler RE. Childhood abuse and neglect and loss of self-regulation. Bull Menninger Clin 1994;58:145–68 1994.

Teicher MH, Ito Y, Glod CA, Andersen SL, Dumont N, Ackerman E. Preliminary evidence for abnormal cortical development in physically and sexually abused children using EEG coherence and MRI. Ann N Y Acad Sci 1997;821:160–75.

Stein MB, Koverola C, Hanna C, Torchia MG, McClarty B. Hippocampal volume in women victimized by childhood sexual abuse. Psychol Med 27:951–9.

DeBellis MD, Keshavan MS, Clark DB, Casey BJ, Giedd JN, Boring AM, Frustaci K, Ryan ND. A. E. Bennett Research Award. Developmental traumatology. Part II. Brain development. Biol Psychiatr. 1999;45:1271–84.

Selye H. The stress of life. New York: McGraw–Hill, 1976.
[62] Bifulco A, Brown GW, Moran P, Ball C, Campbell C. Predicting depression in women: the role of past and present vulnerability. Psychol Med 1998;28:39–50.
[63] Femina DD, Yeager CA, Lewis DO. Child abuse: adolescent records vs adult recall. Child Abuse Negl 1990;14:227–31.
[64] Williams LM. Recovered memories of abuse in women with documented child sexual victimization histories. J Trauma Stress 1995;8:649–73.
[65] Vaillant GE. The natural history of alcoholism. Cambridge, MA: Harvard University Press, 1983.
[66] Finkelhor D, Hotaling G, Lewis IA, Smith C. Sexual abuse in a national survey of adult men and women: prevalence, characteristics, and risk factors. Child Abuse Negl 1990;14:19–28.
[67] MacMillan HL, Fleming JE, Trocmé N, Boyle MH, et al. Prevalence of child physical and sexual abuse in the community: results from the Ontario Health Supplement. J Am Med Assoc 1997;278:131–5.
[68] Olds DL, Robinson J, O’Brien R, Luckey DW, Pettitt LM, Henderson CR Jr, Ng RK, Sheff KL, Korfmacher J, Hiatt S, Talmi A. Home visiting by paraprofessionals and by nurses: a randomized, controlled trial. Pediatrics 2002;110:486–96.