Child Abuse and Other Traumatic Experiences, Alcohol Use Disorders, and Health Problems in Adolescence and Young Adulthood

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Objective We prospectively examined the health effects of child abuse and other traumatic events, with objective health indicators and consideration of alcohol use disorders (AUD). **Methods** Adolescents ($n = 668$) were recruited from clinical and community sources. At baseline, we examined child abuse and other traumas, AUD, health-related symptoms, physical findings, and blood assays. Subjects were assigned to Trauma Classes (TC), including witnessing violence, physical abuse, and sexual abuse. Health outcomes were again determined at 1-year and young adult follow-up. **Results** In adolescence, higher TC severity was associated with more health-related symptoms, increased age-adjusted body mass index, and stress-response immune system indices. In adolescence and young adulthood, the relationships between TC and health-related symptoms were mediated by anxiety. AUD was associated with liver injury, and cigarette smoking with heart/lung symptoms. **Conclusions** Child abuse predicted persistently elevated health-related symptoms primarily attributable to anxiety, and early signs of liver disease were attributable to AUD.

Key words adolescents; alcohol use; child abuse; health.

Child abuse has been found to predict mental disorders, substance-related problems, and health risk behaviors (Gilbert et al., 2009). Retrospective studies with adults have suggested that child abuse leads to major physical health problems (Brown, Young, Anda, Felitti, & Giles, 2006). Child abuse has been systematically related to diminished subjective health quality and obesity (Thomas, Hypponen, & Power, 2008). With some specific conditions excepted (e.g., sexually transmitted diseases: Wilson & Widom, 2009); however, few prospective studies have examined whether child abuse leads to physical health problems. In addition, consideration of the relationship between child abuse and later substance use disorders is important for understanding health outcomes.

A few studies have examined the relationship between child maltreatment and global health status. Hussey and colleagues (Hussey, Chang, & Kotch, 2006) studied a large sample of adolescents who, when follow-up in young adulthood, completed a retrospective child abuse questionnaire. Child abuse reports were associated with poorer subjective health quality in adolescence. Among 378 adolescent enrolled in addictions treatment (Stevens, Murphy, & McKnight, 2003), PTSD-like symptoms were positively associated with subjective health symptoms. Among 1041 children at high risk for child abuse and neglect (Flaherty et al., 2006), child maltreatment at age 4 years predicted poorer overall child health at age 6 as well as an increased incidence of illnesses requiring medical attention.

Child abuse has been associated with overweight status in some studies. In the Hussey study (2006), physical abuse, but not sexual abuse, was found to be associated with overweight status by BMI in late adolescence. Among 782 community subjects (Johnson, Cohen, Kasen, & Brook, 2002), childhood sexual abuse ($n = 22$) was not associated with adolescent or young adulthood obesity. In a prospective study of female children with sexual abuse ($n = 84$) and a comparison sample ($n = 102$), those with a sexual abuse history showed a more rapid increase in BMI during adolescence and a higher obesity rate in young adulthood (Noll, Zeller, Tricket, & Putnam, 2007). Among over 9,000 children followed to middle adulthood (Thomas et al., 2008), physical abuse, but not sexual abuse, predicted increased BMI and higher rates of

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obesity in middle adulthood. In the latter study, child abuse did not predict type 2 diabetes in middle adulthood. Comorbid obesity, high blood pressure, lipid abnormalities, increased blood glucose, and diabetes mellitus have been termed ''metabolic syndrome'' (Steinberger et al., 2009). Given a relationship between child abuse and obesity, one might expect that child abuse would predict elevations in other metabolic syndrome indicators. This possibility has not been studied.

Stressors have been found to induce changes in immune functioning, and immune system indicators may be relevant to understanding child abuse and health. In the laboratory, immunoglobulin increases have been demonstrated in response to acute stressors (Endresen et al., 1991). Immunoglobulin increases have also been observed in response to natural stressors. In young adults, school exams predicted increases in plasma immunoglobulins IgM, IgG, and IgA (Glaser, Mehl, Penn, & Speicher, 1986). A study comparing 14 girls with sexual abuse and 13 control girls (De Bellis, Burke, Trickett, & Putnam, 1996) did not observe significant group differences on plasma antinuclear antibody titers. Immunoglobulin levels have not been previously studied in association with child abuse.

While individuals with child abuse have not been reported to have diagnosed medical diseases in adolescence and young adulthood, studies in later adulthood have noted an association between child abuse and some specific medical disease outcomes. Adults with child maltreatment histories have been reported to show elevated rates of liver disease, lung cancer and heart disease (Dong, Dube, Felitti, Giles, & Anda, 2003; Dong et al., 2004; Brown et al., 2006). These medical diseases may be an indirect result of risky health behaviors, particularly substance use disorders.

Childhood abuse has been found to be associated with or to predict adolescent substance use disorders. Among 3,559 students in grades 7 through 12, Hamburger, Leeb, and Swahn (2008) found that sexual abuse, physical abuse, and witnessing violence were associated with increased preteen alcohol use. Using data from the National Longitudinal Study of Adolescent Health ($n = 12{,}748$), Shin, Edwards, and Heeren (2009) found physical abuse and sexual abuse were associated with binge drinking. In a subset of the subjects described here, sexual abuse, physical abuse, and other stressors were more common among adolescents with AUD than among control adolescents (Clark, Lesnick, & Hegedus, 1997a). Physical or sexual abuse accelerated the onset of AUD and accounted for the relationship between AUD and major depressive disorder (Clark, De Bellis, Lynch, Cornelius, & Martin, 2003).

The adverse health consequences associated with AUD, reflected by organ pathology and disease history, have been more systematically studied in adults than in adolescents. In adults, abnormalities found to be caused by chronic alcohol dependence include elevated liver injury indices (Allen, Fertig, Litten, Sillanaukee, & Anton, 1997), elevated immunoglobulins (Mili, Flanders, Boring, Annest, & DeStefano, 1992), elevated erythrocyte mean cell volume (MCV) (Seppa, Sillanaukee & Koivula, 1992), and decreased electrolytes, such as calcium, magnesium, phosphate, and potassium (Elisaf, Bairaktari, Kalaizidia, & Siamopoulos, 1998). The medical records of 417 adolescents with substance use disorders and 2082 demographically matched subjects, those with substance use disorders had more abdominal pain, sleep disorders, and asthma (Mertens, Fisher, Fleming, & Weisner, 2007). In some of the subjects described here (Clark, Lynch, Donovan, & Block, 2001), 128 adolescents with AUD (compared with 131 controls) showed more health-related symptoms (HS), laboratory tests indicating liver injury, and some physical exam abnormalities. Thus, some health problems reported by adolescents with AUD have been verified by objective findings and may be attributable to toxic alcohol effects on the liver and other organs.

While adolescents with AUD evidence objective indicators of some health problems, their subjective HS seem disproportionately elevated. These HS may, to some extent, reflect somatic anxiety symptoms (Ginsburg, Riddle, & Davies, 2006). In prior reports on the present sample (Clark et al., 1997b), adolescents with AUD were observed to have elevated rates of anxiety disorders, including PTSD. In our study of health problems among adolescents with AUD (Clark et al., 2001), we found that an index of negative emotionality was highly correlated with HS, mediated the relationship between AUD and HS, and was not correlated with serum liver enzyme levels or physical exam abnormalities. Among adult women (Lang et al., 2008), child abuse was observed to lead to mental and physical health difficulties through anxiety disorder symptoms. Child abuse may induce HS, at least in part, through anxiety.

Prior research has had several shortcomings. In most studies, the relationship between child abuse and health outcomes has been determined exclusively with cross-sectional or retrospective methods. In some studies, decades have passed between recalled childhood events and the adulthood assessment, amplifying the potential for recall bias. The available studies on adolescents have

typically assessed health status with only a few global and subjective questions. Some studies with large community samples have included few subjects with child abuse histories. Studies on child abuse have rarely examined the effects of other traumatic experiences. Substance use that may adversely influence health outcomes has typically not been concurrently examined. The extent to which HS may be attributable to anxiety has also been neglected in child abuse studies.

The present study addresses several of these shortcomings. We examined relationships among child abuse, AUD, and physical health problems in adolescence, and prospectively assessed later adolescent and young adult health outcomes. This is the first study to examine these relationships with a comprehensive physical health assessment. The evaluation included questions on 136 health symptoms, laboratory studies of blood including liver injury and other objective indicators, and physical examinations including blood pressure and body mass index (BMI) measurements. Child abuse was considered in the context of other traumatic experiences. We concurrently measured AUD and cigarette smoking. The study conducted 1-year follow-up and young adult outcome evaluations. We hypothesized that child abuse and other traumatic experiences would be associated with HS, being overweight, and stress-related laboratory findings, while AUD would be associated with liver injury. We expected the relationship between child abuse and HS to be mediated by anxiety.

Methods

Participants

Subjects were 668 adolescents (ages 12- to 18-years old) participating in a longitudinal study at the Pittsburgh Adolescent Alcohol Research Center recruited from clinical ($n = 455$) and community ($n = 213$) sources. Clinical sources included hospital-based out-patient and in-patient addictions and psychiatric programs, free-standing addictions programs, and residential programs for youth with family difficulties. Community subjects were randomly selected from the local area using survey methods. More details on these methods have been presented in prior publications (Clark et al., 2001). The subjects were 48% female ($n = 317$), 16.2 ± 1.5 years old, and 81% whites ($n = 541$) and 19% African Americans ($n = 127$). Six subjects from other races were excluded here. By Hollingshead Two-Factor Index of Social Position (1975), socioeconomic status (SES) determined by combining weighted education and job status scores was 37.5 ± 13.5. SES scores range from 11 to 77, and middle class families score from 28 to 43 (Hong, Nelesen, Krohn, Mills, & Dimsdale, 2006).

The 1-year follow-up assessment was completed with 555 of these 668 subjects (83%). Subjects missing the 1-year follow-up, compared to those completing the 1-year follow-up assessment, were more likely to be male (68% vs. 49% for subjects who missed the visit and those who completed the visit, respectively; $\chi^2 = 13.3$, $df = 1$, $p < .001$), not different on age at baseline (16.3 ± 1.5 vs. 16.2 ± 1.5, $F = 0.0$, $df = 1{,}666$, $p = .9$), not different on race (22% African American vs. 18% African American, $df = 1$, $\chi^2 = 0.9$), lower on SES (32.6 ± 14.7 vs. 38.6 ± 13.0, $F = 19.0$, $df = 1{,}666$, $p < .001$), less likely to have been recruited from the community (18% vs. 35%, $\chi^2 = 12.6$, $df = 1$, $p < .001$), and more likely to have had adolescent AUD (56% vs. 44%; $\chi^2 = 5.4$, $df = 1$, $p = .02$). The young adult assessment was completed with 439 of these 668 subjects (66%). Subjects missing the young adult assessment, compared to those completing the visit, were more likely to be male (65% vs. 46%; $\chi^2 = 23.5$, $df = 1$, $p < .001$), were older at baseline (16.5 ± 1.5 vs. 16.1 ± 1.5, $F = 7.2$, $df = 1{,}666$, $p = .008$), more likely to be African American (24% vs. 17%, $df = 1$, $\chi^2 = 0.03$), lower on SES (35.2 ± 14.2 vs. 38.7 ± 13.0, $F = 10.3$, $df = 1{,}666$, $p = .001$), less likely to have been recruited from the community (23% vs. 37%, $\chi^2 = 13.5$, $df = 1$, $p < .001$), and more likely to have had adolescent AUD (53% vs. 42%; $\chi^2 = 6.9$, $df = 1$, $p = .008$).

Procedures

Subjects were paid $125 in gift certificates for participating in a protocol that characterized trauma history, substance use disorders, other mental disorders, health status, and other variables. The subject's biological mother or other caretaker confirmed the adolescent's health history as well as other information, and was paid $50 for participation. The study was approved by the University of Pittsburgh Human Subjects Institutional Review Board. Active participant and parental consent were required. The follow-up assessments examined here were at 1-year after the baseline and at age 25-years old.

Measures

Trauma Classes

A structured trauma interview was added to the Schedule for Affective Disorders and Schizophrenia for School Age Children (K-SADS) for DSM-IV (Kaufman et al., 1997). Trauma was defined by PTSD Criterion A of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV: American Psychiatric Association, 1994).

The interview included structured questions to determine trauma characteristics, including sexual features, injuries, and perpetrators. Sexual abuse was defined as forced or illicit genital fondling, or oral, vaginal or anal intercourse in a familial context. Similar experiences outside the family context were labeled rape. Physical abuse was defined as maltreatment in the family context with serious injury or multiple instances with bruises. Violent victimization was defined as traumatic injury sustained by interpersonal violence outside the familial context. Witnessing violence was indicated when the subject was not the victim. Non-interpersonal traumatic events indicated accidents, including motor vehicle accidents, where injuries were not the result of interpersonal violence. After determining their presence or absence, we examined the relationships between PTSD symptoms and each of these traumatic experiences to determine their relative severity. This procedure identified seven Trauma Classes (TCs) and each subject was assigned to a TC group. Subjects with multiple traumas were assigned to the highest applicable TC severity. The TC labels, PTSD symptom counts, and sample sizes were as follows: (a): TC-0: No traumatic experiences: (0 by definition; $n = 100$); (b) TC-1: Non-interpersonal trauma: (0.5 ± 1.7; $n = 139$); (c) TC-2: Witnessing violence: (1.0 ± 2.3; $n = 122$); (d) TC-3: Violent victimization: (1.1 ± 2.4; $n = 87$); (e) TC-4: Physical abuse: (1.7 ± 3.1; $n = 87$); (f) TC-5: Sexual abuse: (3.4 ± 4.6; $n = 81$); (g) TC-6: Rape: (4.3 ± 5.1; $n = 52$). To serve as a reference group, TC-0 included only community-recruited subjects with neither traumatic experiences nor AUD. Excluding TC-0, TC was confirmed to be significantly associated with PTSD symptoms ($F = 18.5$; $df = 5{,}562$; $p < .001$). TC and AUD were also significantly associated ($\chi^2 = 29.6$, $df = 5$, $p < .001$).

Substance Use Disorders

Information about substance use disorders, including AUD, was gathered by revised sections of the Structured Clinical Interview for DSM (Martin, Pollock, Lynch, & Bukstein, 2000). Diagnoses were considered to be present if the subject met the diagnostic criteria in the 6 months prior to the interview. Cigarette use was determined by the average number of cigarettes per day in the prior month.

Health-related Symptoms

HS were determined by a checklist (Arria, Dohey, Mezzich, Bukstein, & Van Thiel, 1995). This questionnaire assessed 136 self-reported symptoms in 15 areas: General health status (9 items), Sleep (5 items), Eating (10 items), Skin (9 items), Eyes and Vision (11 items), Ears and Hearing (4 items), Mouth (4 items), Nose (6 items), Throat and Neck (7 items), Heart and Lungs (14 items), Abdomen (26 items), Bleeding and Metabolism (10 items), Muscles and Joints (6 items), Neurological (10 items), and Urinary (5 items). One point was scored for each affirmative response and the items were summed to construct the HS Total Score (Clark et al., 2001).

Laboratory Tests

Blood was collected in the morning after an overnight fast. Assays were performed by a commercial or medical center laboratory. Serum assays with abbreviations and normal ranges were: (a) liver injury tests: gamma-glutamyl transpeptidase (gamma-GTP: normal range: females 5–29; males 5–38 U/l); glutamic-oxaloacetic transaminase (sgot, a.k.a., aspartate aminotransaminase or AST: females: 9–25; males: 10–40 U/l); glutamic-pyruvic transaminase (sgpt, a.k.a., alanine aminotransaminase or ALT: females 7–30; males 10–55 U/l); (b) immunoglobulins: IGM (56–352 mg/dl); IGA (70–312); IGG (639–1349); (c) metabolic syndrome indicators: glucose (<100 mg/dl); triglycerides (<150 mg/dl); (d) electrolytes: sodium (Na: 135–145 mEq/l); chloride (Cl: 96–106); potassium (K: 3.5–5.0); phosphorous (PO4: 2.4–4.1 mg/dl); calcium (Ca: 8.5–10.5 mg/dl); magnesium (Mg: 1.5–2.0 mEq/l); (e) hematologic indicators: cell counts (red: $4.1–6.1 \times 10^6/mm^3$; white: $4.5–10.5400 \times 10^3/mm^3$; platelets: $150–400 \times 10^3/mm^3$); WBC differential (monocytes: 2–8%; lymphocytes: 20–40%; neutrophils: 40–60%); hematocrit (female 37–48%; males 42–52%); mean red cell corpuscular hemoglobin (MCH: 28–33 pg/cell); mean red cell corpuscular hemoglobin concentration (MCHC: 32–36 g/dl). Part of a research project component completed in the initial phase of the overall project, the laboratory tests were conducted at baseline with a subject subset ($n = 395$).

Physical Examination

Physical abnormalities were determined by a health care practitioner examination performed by a physician, nurse practitioner, or physician's assistant. The exam results were classified as normal or abnormal in 15 areas: (a) general appearance, (b) eyes (including fundoscopic exam), (c) ears (including otoscopic exam), (d) nose and pharynx, (e) mouth (including dentition and oral mucosa), (f) neck (including thyroid and lymph node exam), (g) respiratory, (h) cardiovascular, (i) abdomen, (j) neurologic exam (including cranial nerves, motor exam, sensory exam, cerebellar exam, involuntary movements, reflex exam), (k) skin, (l) head, (m) thorax, (n) back, and (o) extremities. Along with the laboratory tests, the health care practitioner examinations were conducted at the baseline assessments

with a subject subset. Height and weight, along with systolic and diastolic blood pressures, were collected at each assessment. For the adolescent measurements, the age-adjusted BMI percentile was calculated using the Centers for Disease Control computer program. For the young adult assessment, age adjustment was not necessary. A health history interview inquired about diagnosed health problems, emergency department visits, medical hospitalizations, and surgeries.

Anxiety

Anxiety was measured by the Hamilton Anxiety Rating Scale (HARS: Hamilton, 1959). With a subset of the present sample, the HARS was found to be a reliable and valid measure of anxiety in adolescents (Clark & Donovan, 1994). The HARS Total Score was used as an indicator of anxiety.

Data Analyses

The HS scales, laboratory assays, and other continuous variables were analyzed by ANCOVAs. Where applicable, the analysis first examined the total score as the overall model. Where multiple independent indicators examined a construct (e.g., liver injury), a multivariate ANCOVA was conducted. Where the overall model was statistically significant, subscales were examined. Dichotomous variables were analyzed by logistic regression analyses. We examined the main effects of TC and AUD in statistical models including demographic characteristics as covariates. The extent to which anxiety mediated the relationship between TC and HS was examined. The tested model specified that TC (independent variable) caused anxiety (mediating variable) and that anxiety causes HS (outcome variable). For the model to be supported (Baron & Kenny, 1986; MacKinnon & Luecken, 2008), TC must be significantly related to anxiety, anxiety must be significantly related to HS, and the relationship between TC and HS must have changed when anxiety was added.

Results

Demographic Characteristics

The demographic characteristics of the seven TC groups are presented in Table I. Higher TC severity was significantly associated with older age, lower SES, female gender, and African-American ethnicity. In analyses excluding TC-0, TC severity was associated with the presence of AUD and clinical recruitment source. The presence of AUD was significantly associated with older age, male gender, and white ethnicity. Excluding TC-0, AUD was associated with clinical recruitment source. Subsequent analyses included these demographic characteristics as covariates.

Health-related Symptoms

The main effects of TC and AUD on HS at the baseline assessment are presented in Table II. At baseline, highly significant effects of TC were seen for the HS Total Score and for many subscales. The effect of AUD on HS Total Score was not significant. A significant effect of AUD was noted for only one scale, HS Heart/Lung subscale ($\chi^2 = 92.6$, $p < .001$). Examining health symptoms at the one-year follow-up, TC did ($F = 2.6$, $df = 6,476$, $p = .02$)

Table I. Baseline Demographic Characteristics of Adolescents by TC

	Trauma classes							Main effects	
	TC-0 ($n = 100$) *M (SD)*	TC-1 ($n = 139$) *M (SD)*	TC-2 ($n = 122$) *M (SD)*	TC-3 ($n = 87$) *M (SD)*	TC-4 ($n = 87$) *M (SD)*	TC-5 ($n = 81$) *M (SD)*	TC-6 ($n = 52$) *M(SD)*	TC *F*	AUD *F*
Age (years)	15.5 (1.5)	16.3 (1.4)	16.3 (1.4)	16.8 (1.1)	16.5 (1.4)	16.0 (1.7)	16.2 (1.4)	3.6**	26.9***
SES	44.5 (11.9)	40.1 (12.2)	34.7 (13.5)	37.9 (13.0)	35.6 (12.8)	32.4 (13.3)	35.7 (15.2)	8.7***	0.1
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	$\chi 2$	$\chi 2$
Gender									
Female	58 (58)	55 (40)	51 (42)	18 (21)	29 (33)	60 (74)	46 (89)	99.5**	28.3***
Male	42 (42)	84 (60)	71 (58)	69 (79)	58 (67)	21 (26)	6 (11)		
Ethnicity									
White	84 (84)	121 (87)	94 (77)	61 (70)	76 (87)	59 (73)	46 (89)	19.5**	7.9**
African American	16 (16)	18 (13)	28 (23)	26 (30)	11 (13)	22 (27)	6 (11)		
Source									
Clinical	0 (0)	89 (58)	95 (78)	74 (85)	76 (87)	79 (97)	51 (98)	74.9***	119***
Community	100 (100)	59 (42)	27 (22)	13 (15)	11 (13)	2 (3)	1 (2)		

*$p < .05$; **$p < .01$; ***$p < .001$.

Table II. HS at Baseline in Adolescents by TC

	Trauma classes							Main effects	
	TC-0 ($n = 100$) *M (SD)*	TC-1 ($n = 139$) *M (SD)*	TC-2 ($n = 122$) *M (SD)*	TC-3 ($n = 87$) *M (SD)*	TC-4 ($n = 87$) *M (SD)*	TC-5 ($n = 81$) *M (SD)*	TC-6 ($n = 52$) *M (SD)*	TC *F*	AUD *F*
HS Total Score	5.5 (6.1)	10.2 (11.3)	9.3 (8.7)	9.5 (11.7)	12.8 (13.9)	18.2 (15.9)	19.6 (13.9)	7.6***	0.5
General	0.2 (0.6)	0.6 (1.2)	0.8 (1.1)	0.5 (1.0)	0.9 (1.4)	1.4 (1.7)	1.1 (1.5)	5.7***	0.2
Sleep Habits	0.3 (0.6)	0.6 (1.0)	0.9 (0.9)	0.6 (0.9)	1.2 (1.2)	1.2 (2.2)	1.3 (1.2)	6.5***	2.4
Eating Habits	0.7 (0.9)	1.0 (1.3)	1.0 (1.2)	0.9 (1.1)	1.2 (1.3)	1.6 (1.4)	1.9 (1.3)	5.2***	0.1
Skin	0.8 (1.2)	0.8 (1.1)	0.9 (1.2)	0.8 (1.1)	1.1 (1.4)	1.3 (1.6)	1.9 (1.6)	3.5**	0.7
Eyes & vision	0.4 (0.8)	0.6 (1.2)	0.5 (1.0)	0.6 (1.1)	0.6 (1.0)	1.0 (1.6)	1.1 (1.2)	1.4	1.6
Ears & Hearing	0.1 (0.4)	0.2 (0.5)	0.2 (0.4)	0.2 (0.5)	0.3 (0.7)	0.4 (0.8)	0.2 (0.5)	1.8	0.2
Mouth	0.1 (0.4)	0.3 (0.7)	0.2 (0.6)	0.3 (0.6)	0.3 (0.6)	0.6 (0.9)	0.6 (0.9)	2.6*	0.2
Nose	0.6 (0.9)	0.8 (1.2)	0.6 (1.1)	0.8 (1.3)	0.8 (1.4)	1.0 (1.4)	1.0 (1.3)	1.7	2.8
Throat & Neck	0.3 (0.7)	0.5 (0.8)	0.6 (0.9)	0.6 (1.0)	0.6 (1.2)	1.0 (1.4)	0.9 (1.3)	2.3*	1.3
Heart/Lungs	0.4 (0.9)	1.0 (1.6)	0.9 (1.5)	1.0 (1.7)	1.2 (1.9)	1.5 (1.9)	1.6 (1.6)	3.3**	5.5*
Abdomen	0.3 (0.8)	0.9 (2.0)	0.6 (1.3)	0.9 (2.0)	1.1 (1.9)	2.4 (3.4)	2.4 (3.1)	7.3***	0.3
Metabolism & Bleeding	0.4 (0.9)	0.7 (1.3)	0.5 (1.0)	0.6 (1.3)	0.9 (1.4)	1.3 (1.7)	1.7 (1.9)	6.1***	0.2
Muscles & Joints	0.2 (0.6)	0.7 (1.2)	0.4 (0.8)	0.5 (0.9)	0.05 (1.0)	0.9 (1.3)	1.0 (1.4)	4.3***	2.1
Neurological	0.3 (0.9)	1.0 (1.7)	1.1 (1.9)	1.4 (2.2)	1.6 (2.2)	2.3 (2.6)	2.3 (2.4)	6.0***	3.7
Urinary	0.2 (0.4)	0.4 (0.7)	0.2 (0.5)	0.3 (0.8)	0.5 (0.9)	0.6 (1.1)	0.9 (1.0)	3.8**	1.1

ANCOVA (*F*) results shown with covariates including gender, age, ethnic group, SES.
*$p < .05$; **$p < .01$; ***$p < .001$.

and AUD did not ($F = 0.5$, $df = 1,476$, $p = 0.5$) predict HS Total Score. Examining health symptoms at the young adult follow-up, TC did ($F = 4.1$, $df = 6,420$, $p = .001$) and AUD did not ($F = 0.0$, $df = 1,420$, $p = 0.8$) predict HS Total Score.

Mediation Analyses

At baseline, TC and HARS Total Score were statistically associated ($F = 16.9$, $df = 6,655$, $p < .001$) after accounting for demographic characteristics and AUD. In a model including demographic characteristics and AUD, HARS Total Score was ($F = 205.8$, $df = 1,593$; $p < .001$) and TC was not ($F = 1.2$, $df = 6,593$, $p = .3$) associated with HS Total Score. At 1-year follow-up, HARS Total Score at baseline predicted ($F = 98.6$, $df = 1,475$; $p < .001$) and TC did not predict ($F = 0.7$, $df = 6,475$, $p = .7$) 1-year follow-up HS Total Scores. At the young adult assessment, HARS Total Score at baseline predicted ($F = 23.4$, $df = 1,341$; $p < .001$) and TC did not predict ($F = 1.4$, $df = 5,341$, $p = .2$) young adult HS Total Scores. These analyses demonstrated that HARS Total Score fully mediated the relationships between TC and HS Total Score at baseline, 1-year, and young adult assessments.

We examined whether cigarette use mediated the relationship between AUD and heart/lung symptoms. Controlling for demographic characteristics, TC was significantly associated with cigarette use ($F = 10.7$, $df = 6,673$, $p < .001$). In the mediation model, cigarette use was ($F = 11.3$, $df = 1,581$; $p < .001$) and AUD was not ($F = 3.5$, $df = 1,581$; $p = .06$) significantly associated with the HS Heart/Lung subscale. These analyses demonstrated that cigarette use fully mediated the relationship between AUD and heart/lung symptoms.

Health History

In the overall sample, the most common health problems noted by history at baseline were fractures (i.e., broken bones) in 225 or 668 cases (34%), head injury ($n = 48$ or 7%) and asthma ($n = 106$ or 16%). The proportion of subjects with these health problems is presented in Table III. Other noted health problems were relatively rare, occurring at rates of <5%. Logistic regression analyses were conducted with these outcomes as dependent variables, and demographic characteristics as covariates. At baseline, neither TC nor AUD were significantly associated with fractures, head injures, or asthma. When added to this model, cigarette use did not account for significant variance on asthma. Similarly, at the 1-year follow-up, neither TC nor AUD predicted fractures, head injury, or asthma. Again, cigarette use at baseline did not predict asthma at the 1-year follow-up. At the young adult follow-up, again, neither TC nor AUD predicted fractures, head injury, or asthma. Cigarette use at baseline did not predict asthma at the young adult follow-up.

Table III. Health History and Physical Exam Indicators at Baseline for Adolescents by TC

	TC-0 ($n=100$) *n* (%)	TC-1 ($n=139$) *n* (%)	TC-2 ($n=122$) *n* (%)	TC-3 ($n=87$) *n* (%)	TC-4 ($n=87$) *n* (%)	TC-5 ($n=81$) *n* (%)	TC-6 ($n=52$) *n* (%)	Main Effects TC $\chi2$	Main Effects AUD $\chi2$
Fractures	23 (23)	50 (36)	40 (33)	33 (38)	33 (38)	29 (36)	17 (33)	5.4	0
Head Injury	1 (1)	11 (8)	8 (7)	8 (9)	9 (10)	9 (11)	2 (4)	1.8	0.1
Asthma	13 (13)	22 (16)	18 (15)	14 (16)	13 (15)	14 (17)	12 (23)	3.2	0.6
	($n=85$)	($n=98$)	($n=81$)	($n=47$)	($n=56$)	($n=55$)	($n=39$)		
Overweight	33 (39)	23 (24)	29 (36)	10 (22)	17 (30)	15 (28)	20 (53)	12.8*	1.3
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*F*	*F*
Age-adjusted BMI %ile	69 (27)	58 (29)	67 (28)	61 (30)	62 (28)	62 (29)	76 (25)	2.2*	1.4
Blood pressure								2.3**	2.6
systolic	112 (11)	112 (11)	113 (12)	113 (10)	112 (10)	109 (11)	108 (11)	1.1	1.9
diastolic	72 (10)	70 (11)	73 (11)	68 (8)	69 (10)	69 (10)	66 (10)	4.4**	5.0*
# PE abnormalities	1.2 (0.9)	1.4 (1.9)	1.6 (1.6)	0.8 (1.2)	1.1 (1.4)	1.4 (1.2)	1.5 (1.3)	0.7	1.9

Logistic regression (Wald $\chi2$) or ANCOVA (F) statistics are shown with covariates including gender, age, ethnic group, SES.
*$p < .05$; **$p < .01$; ***$p < .001$.

Blood Assays

Liver Injury

For liver injury indicators overall at baseline (Table IV), AUD was ($F=3.3$, $df=3,312$, $p=.02$) and TC was not significantly associated with liver injury ($F=0.9$, $df=15,942$, $p=.5$). For individual indicators, the presence of AUD was associated with significantly higher γ-GTP, AST and ALT. At the 1-year follow-up assessment, AUD was significantly associated with liver injury overall ($F=5.3$, $df=3,237$, $p=.001$) and TC were not significantly associated with liver injury ($F=1.0$, $df=18,717$, $p=.5$). For individual indicators, AUD accounted for significant variance on γ-GTP ($F=10.3$, $df=1,250$, $p=.002$), and did not account for significant variance on AST ($F=0.0$, $df=1,250$, $p=.9$) and ALT ($F=0.2$, $df=1,250$, $p=.6$). At the young adult follow-up assessment, neither AUD ($F=1.7$, $df=3,178$, $p=.2$) nor TC ($F=1.1$, $df=18,540$, $p=.3$) were significantly associated with liver injury overall.

Immunoglobulins

At baseline (Table IV), TC and AUD were significantly associated with overall immunoglobulin levels. For individual indicators, TC was significantly associated with IgM and IgG and was not significantly associated with IgA. More severe TC tended to be associated with higher IgM and IgG. For individual indicators, AUD was significantly associated with IgM and IGA and was not significantly associated with IgG. The presence of AUD was associated with higher IgM and IgA levels. At the 1-year follow-up, neither TC ($F=1.0$, $df=18,414$, $p=.4$) nor AUD ($F=1.3$, $df=3,136$, $p=.3$) were significantly associated with overall immunoglobulin levels. Immunoglobulins were not measured at the young adult assessment.

Metabolic Syndrome Indicators

At baseline (Table IV), TC was and AUD was not significantly associated with overall metabolic syndrome indicators. For individual indicators, TC was associated with glucose levels and was not significantly associated with triglyceride levels. At the one-year follow-up, neither TC ($F=1.3$, $df=12,444$, $p=.2$) nor AUD ($F=0.1$, $df=2,221$, $p=.9$) were significantly associated with overall immunoglobulin levels. Metabolic syndrome indicators were not measured at the young adult assessment.

Electrolytes

At baseline (Table IV), TC was and AUD was not significantly associated with overall electrolyte levels. For individual indicators, TC was significantly associated with chloride and calcium levels, and was not associated with levels of sodium, potassium, phosphorous, or magnesium. At the 1-year follow-up, neither TC ($F=1.3$, $df=36,1296$, $p=.1$) nor AUD ($F=1.7$, $df=6,211$, $p=.1$) were significantly associated with overall electrolyte levels. Electrolyte levels were not measured at the young adult assessment.

Hematologic Indicators

At baseline (Table IV), neither TC nor AUD were significantly associated with overall blood cell counts. Neither TC nor AUD were significantly associated with WBC differential counts. Neither TC nor AUD were significantly

Table IV. Laboratory Blood Assays at Baseline for Adolescents by TC

	Trauma class							Main Effects	
	TC-0 ($n=70$) *M (SD)*	TC-1 ($n=86$) *M (SD)*	TC-2 ($n=66$) *M (SD)*	TC-3 ($n=38$) *M (SD)*	TC-4 ($n=52$) *M (SD)*	TC-5 ($n=46$) *M (SD)*	TC-6 ($n=37$) *M (SD)*	TC *F*	AUD *F*
Liver Injury								0.9	3.7*
γ-GTP	14.0 (9.0)	16.0 (9.4)	17.4 (7.2)	18.0 (10.1)	16.7 (7.0)	18.3 (13.2)	16.9 (8.2)	0.7	5.7*
AST	19.8 (5.3)	20.0 (12.9)	23.6 (22.7)	21.5 (7.8)	19.9 (5.2)	19.8 (8.9)	18.2 (6.0)	0.9	5.1*
ALT	12.9 (5.8)	15.9 (30.3)	21.0 (37.4)	18.3 (13.0)	15.4 (10.0)	15.1 (14.7)	16.7 (10.0)	0.5	8.8**
Immunoglobulins								2.0**	5.3***
IGM	172 (79)	160 (80)	197 (83)	173 (64)	145 (72)	161 (62)	210 (96)	2.2*	8.4**
IGA	162 (70)	203 (97)	184 (48)	215 (88)	188 (61)	201 (58)	175 (69)	1.1	9.6**
IGG	1192 (310)	1353 (475)	1279 (235)	1310 (289)	118 (212)	1226 (288)	1236 (283)	3.2**	2.6
Metabolic								1.8*	0.4
Glucose	86.0 (17.0)	84 (11)	90 (12)	91 (12)	88 (13)	87 (10)	88 (7.5)	2.4*	0.8
triglycerides	96.4 (47.2)	103 (53)	90 (43)	118 (55)	111 (58)	103 (51)	88 (39)	1.3	0.1
Electrolytes								1.7**	0.5
Na	140 (2.1)	141 (2.3)	141 (2.4)	141 (2.3)	141 (2.3)	141 (2.5)	140 (1.7)	1.4	1.6
Cl	103.7 (2.3)	102.4 (2.5)	103.7 (2.7)	103.5 (2.6)	104.0 (2.4)	104.1 (2.8)	104.3 (2.8)	2.9**	0.0
K	4.2 (0.6)	4.4 (0.5)	4.3 (0.4)	4.3 (0.4)	4.2 (0.4)	4.5 (0.7)	4.1 (0.4)	1.8	0.0
PO4	4.0 (0.8)	4.0 (0.7)	4.1 (0.7)	4.1 (0.6)	4.0 (0.7)	4.1 (0.6)	4.1 (0.6)	1.8	0.7
Ca	9.7 (0.7)	9.8 (0.6)	9.8 (0.7)	9.8 (0.5)	9.8 (0.6)	10.1 (0.7)	10.0 (0.7)	2.2*	0.0
Mg	1.8 (0.2)	1.9 (0.3)	1.8 (0.2)	1.8 (0.2)	1.8 (0.2)	1.9 (0.3)	1.8 (0.3)	1.0	0.0
	($n=47$)	($n=62$)	($n=58$)	($n=34$)	($n=37$)	($n=31$)	($n=24$)		
Cell counts								1.1	0.0
RBC	4.7 (0.4)	5.0 (0.4)	5.0 (0.4)	5.0 (0.4)	5.0 (0.5)	4.9 (0.5)	4.6 (0.4)		
WBC	6.3 (1.4)	6.8 (1.7)	6.8 (1.8)	6.8 (1.7)	6.8 (1.2)	6.9 (2.1)	7.2 (2.2)		
platelets	364 (55)	264 (64)	233 (48)	250 (45)	255 (50)	267 (61)	262 (50)		
WBC Differential								1.9	0.4
Monocytes (%)	9.0 (4.4)	4.5 (2.3)	7.4 (2.2)	7.6 (1.9)	7.9 (2.7)	7.1 (2.0)	6.5 (1.7)		
Lymphocytes (%)	31.8 (6.9)	32 (9.2)	33 (8.9)	57 (9.6)	57 (9.8)	56 (10.9)	62.8 (7.4)		
Neutrophils (%)	57 (9.0)	57.6 (9.9)	56 (11)	57 (9.6)	57 (9.8)	58 (10.9)	63 (7.4)		
RBC Indices								1.6	1.4
hematocrit	41 (3.3)	43 (3.3)	42.6 (3.6)	44.4 (3.5)	43.1 (4.0)	41 (4.1)	39.9 (42.3)		
MCH	30 (1.6)	29.5 (2.0)	29.9 (1.3)	30 (1.3)	29 (1.8)	28.9 (3.4)	29.6 (2.0)		
MCHC	34.2 (0.6)	33.9 (0.7)	34 (0.7)	34 (0.7)	33.8 (0.7)	33.7 (0.9)	33.9 (0.7)		

ANCOVA (*F*) statistics are shown with covariates including gender, age, ethnic group, SES.
*$p < .05$; **$p < .01$; ***$p < .001$.

associated with RBC indices. These indices were not collected at subsequent visits.

Physical Examination

BMI

At baseline (Table III), TC was and AUD was not significantly associated with age-adjusted BMI percentile. By CDC criteria, 33% of subjects were classified as overweight. TC was and AUD was not associated with overweight status. At 1-year follow-up, neither TC ($F=1.2$, $df=6,326$, $p=.3$) nor AUD ($F=0.1$, $df=1,326$, $p=.7$) were significantly associated with age-adjusted BMI percentile. At 1-year follow-up, 32% of the subjects were overweight by CDC criteria. At 1-year follow-up, neither TC (Wald $\chi^2=10.2$, $df=6$, $p=.1$) nor AUD were significantly associated with overweight status (Wald $\chi^2=0.2$, $df=1$, $p=.6$). At young adult follow-up, neither TC ($F=0.4$, $df=6,245$, $p=.9$) nor AUD ($F=0.0$, $df=1,245$, $p=.9$) were significantly associated with age-adjusted BMI percentile. At young adult follow-up, 58% of the subjects were overweight by CDC criteria. At young adult follow-up, neither TC (Wald $\chi^2=5.7$, $df=6$, $p=.5$) nor AUD were significantly associated with overweight status (Wald $\chi^2=0.9$, $df=1$, $p=.3$).

Blood Pressure

At baseline (Table III), TC was and AUD was not associated with overall blood pressure. Neither TC nor AUD were significantly associated with systolic blood pressure, whereas both TC and AUD were associated with diastolic blood pressure. TC paired comparisons revealed that this result was due to subjects in TC2 (i.e., Witnessing Violence) having significantly higher diastolic blood pressure than subjects in TC3, TC4 or TC6. Blood pressure readings meeting medical criteria for hypertension were present in only one case. At 1-year follow-up, neither TC ($F = 1.6$, $df = 6,270$, $p = .2$) nor AUD ($F = 0.6$, $df = 1,270$, $p = .4$) were significantly associated with systolic blood pressure, and TC ($F = 2.1$, $df = 6,270$, $p = .049$) but not AUD ($F = 0.0$, $df = 1,270$, $p = .99$) were significantly associated with diastolic blood pressure. At 1-year follow-up, TC paired comparisons revealed that this result was due to subjects in TC2 (i.e., Witnessing Violence) having significantly higher diastolic blood pressure than subjects in TC0, TC3, TC4, or TC6. Hypertension was rare, with a systolic reading over 150 in 0.4% of subjects and a diastolic reading over 90 in 5% of subjects. At young adult follow-up, neither TC ($F = 1.0$, $df = 6,240$, $p = .4$) nor AUD ($F = 0.6$, $df = 1,240$, $p = .4$) were significantly associated with systolic blood pressure and, similarly, neither TC ($F = 0.4$, $df = 6,240$, $p = .9$) nor AUD ($F = 0.6$, $df = 1,240$, $p = .4$) were significantly associated with diastolic blood pressure. At the young adult assessment, hypertension was uncommon, with a systolic reading over 150 in 2% of subjects and a diastolic reading over 90 in 7% of subjects.

Health Care Practitioner Examination

At baseline, most of the abnormalities reported for the health care practitioner exam were observed for the skin (53%), with the main problem being acne; the ears (14%), with the main problem being cerumen blockage or excessive ear wax; and the mouth (11%), with the main problem being severe caries or poor oral hygiene. At baseline, neither TC ($F = 0.7$, $df = 6,216$, $p = .6$) nor AUD ($F = 1.9$, $df = 1,216$, $p = .2$) were significantly associated with the sum of abnormalities on the health care practitioner exam. The Health Care Practitioner examination was not conducted at subsequent visits.

Discussion

In this study, HS were strongly associated with TC severity at baseline, 1-year follow-up and young adult assessments. Examining a comprehensive assessment of HS organized into 15 dimensions, we found that the total score, as well as 12 of 15 dimensions, were significantly and systematically associated with TC severity. While few HS were reported in the TC groups of lower severity, the two TC groups with traumatic experiences involving sexual events had average symptom counts in the 18–20 range. For adolescents, this indicates a relatively high symptom level. The relationship between TC and HSs persisted into young adulthood. The results supported a mediation model in which child abuse and other traumatic experiences lead to anxiety and anxiety leads to HSs. The prominence of symptoms commonly noted among adolescents with anxiety disorders, such as abdominal complaints (Ginsburg et al., 2006), along with the absence of objective findings that would explain these symptoms, further validate the interpretation that these symptoms are primarily somatic accompaniments of anxiety.

In some prior studies, physical abuse compared to sexual abuse has shown a stronger relationship with overweight status. That distinction was not replicated here. In the present study, baseline age-adjusted BMI and overweight status were significantly associated with TC. The pattern of results did not suggest that those with physical abuse, contrasted with those with sexual abuse, showed a greater tendency toward being overweight. Furthermore, the outcomes at 1-year and young adult follow-up assessments did not evidence a relationship between TC and overweight status. A significant relationship between glucose level and TC was noted at baseline, with the two less severe TC groups showing lower glucose. In general, however, a propensity toward metabolic syndrome was not associated with TC severity. We have previously noted that adolescents with AUD, compared to controls, show less regular exercise and poor eating habits (Thatcher & Clark, 2006). Chronic effects of such lifestyle problems may become more evident later in life.

Some laboratory tests and physical exam findings may have reflected stress-related responses. TC severity was associated with higher immunoglobulin levels, a finding consistent with that observed in prior studies of laboratory induced and naturalistic stress. Among adolescents, exposure to violence has been previously reported to be associated with elevated blood pressure (Murali & Chen, 2005). Here, elevated diastolic blood pressure was observed in the group of adolescents who reported witnessing violence.

The observed effects of AUD included elevated liver enzymes, elevated immunoglobulins, and HSs on the heart/lung dimension. Alcohol induces acute liver injury, so this finding was expected. Some studies have suggested that higher levels of alcohol consumption may be associated with elevated immunoglobulins

(Gonzalez-Quintela et al., 2007) as was the case here. While we observed an association between AUD and HSs referenced to the heart and lungs, cigarette use mediated this relationship. These findings provide some support for the hypothesis that the association between child abuse and liver, heart and lung diseases may, as suspected, be mediated by AUD and cigarette use. While few cases were found to have medical diseases in this study, AUD and cigarette use lead to medical diseases that become evident later in adulthood.

The sampling approach taken in this study has advantages and disadvantages. The inclusion of the community sample of adolescent with neither traumatic experiences nor AUD as a reference group afforded the opportunity to examine the extent to which the health problems evident in adolescents with traumatic experiences and/or AUD were above expected levels. For adolescents with traumatic experiences or AUD, we elected to include subjects from both community and clinical sources. In designing the study, we recognized that selection bias is often present in clinical samples. The relatively low base rates of AUD and severe child abuse among adolescents in the general population, however, result in their being present in small numbers in representative community samples. The recruitment plan did not recruit directly from child protective services agencies and may not have included a substantial representation of adolescents with the most severe forms of child abuse. In this study, the characteristics of interest, TC and AUD, were highly co-linear with recruitment source, making inclusion of recruitment source in multivariate models problematic. The extent to which these findings generalize to adolescents recruited by other methods will need to be determined in subsequent studies.

This study had other limitations. The assessment did not include an evaluation of neglect. We recognize that neglect would have been important to take into consideration in this study, in that neglect experiences are among the most common and severe forms of child maltreatment (Dubowitz & Bennett, 2007). Future research may be enhanced by obtaining records from child protective services to identify neglect and provide other supplementary information (Runyan et al., 2005). In addition, variation in alcohol use proximal to the time of the assessment may have influenced the results. In some adolescents, laboratory blood assays may have reverted to normal after a period of alcohol abstinence in the days prior to testing. This study did not provide interventions or systematic referrals for treatment. Over the course of the study, however, many subjects received medical, psychological, and/or addictions treatment. These treatments may have mitigated the effect of child maltreatment or other traumas on examined outcomes.

These findings have several clinical implications. HS associated with child abuse were, to some extent, attributable to anxiety. Health care practitioners providing services to adolescents with histories of child abuse should consider anxiety disorders in the differential diagnosis of such symptoms. Nevertheless, conscientious medical care dictates that the presentation of HS should be thoroughly investigated. Child abuse was associated with being overweight at the initial assessment, and some adolescents will need assistance in this area. Among overweight adolescents, monitoring of metabolic syndrome indicators, such as blood pressure and glucose, may facilitate the early identification of hypertension and diabetes. Chronic AUD and cigarette use may lead to liver, cardiac or lung diseases. The subclinical abnormalities that were observed here may, in some cases, preface the onset of more serious organic pathology and medical diseases in middle adulthood. Early intervention with children having abuse histories has been shown to reduce adulthood medical disease rates (Kessler et al., 2008). We recommend that health care practitioners seeing adolescents with child abuse histories regularly screen for alcohol, cigarette and other drug use, monitor for weight gain, and provide treatments for co-morbid mental disorders.

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