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The mediating role of psychological distress in the relationship between adverse childhood experiences and adult smoking

Tara Wynn Strine
Walden University

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COLLEGE OF HEALTH SCIENCES

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Tara Wynn Strine

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Review Committee

Dr. Morton Wagenfeld, Committee Chairperson, Public Health Faculty

Dr. Sandra Rasmussen, Committee Member, Public Health Faculty

Dr. Angela Prehn, School Representative, Public Health Faculty

Chief Academic Officer

David Clinefelter, Ph.D.

Walden University
2010

Abstract

The Mediating Role of Psychological Distress in the Relationship Between Adverse
Childhood Experiences and Adult Smoking

by

Tara Wynn Strine, MPH

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

in Public Health

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Abstract

While research has indicated that impaired mental health partially mediates the relationship between adverse childhood experiences (ACEs) and alcohol and illicit drug use, little research has examined potential mediators in the relationship between ACEs and smoking, the number one cause of preventable mortality in the United States. Accordingly, this study examined the potential mediating effect of psychological distress on the relationship between ACEs and smoking using data from Wave II of the ACE Study, a cross-sectional study completed between June and October of 1997 on a sample of adult health maintenance organization members (N = 7,211). The theoretical underpinnings for this study were grounded in the developmental psychopathological perspective which examines both environmental and biological influences as they interact to promote or impede social, emotional, and behavioral development. Mediation modeling employing both linear and logistic regression techniques indicated that, after adjusting by select covariates, psychological distress (as assessed using the SF-36 Mental Component Summary score) partially mediated the relationship between several of the ACEs examined and smoking in women. These same relationships were not found in men. This research contains several key findings with social change implications. First, additional research should be conducted to examine the causes, developmental paths, and critical points that link ACEs and psychological distress to smoking among women. Second, given the gender differences in the association between ACEs and smoking, gender-specific intervention programs that build resiliency, increase positive social support, and provide tools for developing alternative coping strategies may be important adjuncts to smoking cessation programs, particularly for women with a history of ACEs.

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Dedication

This dissertation is dedicated to my mother, Karen Strine, who unexpectedly passed away on July 25, 2009 while traveling with me to Minnesota for my final Walden residency.

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Chapter 1: Introduction to the Study

Introduction

Adverse childhood experiences (ACEs) are surprisingly common (Flaherty et al., 2009) and have been viewed as a major public health problem (Margolin & Gordis, 2000). In 2007, according to the Administration on Children, Youth, and Families, the rates of maltreatment were extremely high for infants 1 year of age and younger (21.9 per 1,000 infants) and decreased with increasing age, 5.4 per 1,000 among children 16 to 17 years old (U.S. Department of Health and Human Services, 2009a). Approximately 794,000 U.S. children were victims of maltreatment (rate of 10.6 per 1,000 children) and 1,760 children died from abuse or neglect—most of them less than 4 years of age (rate 2.4 per 100,000 children). Fifty-nine percent of these victims experienced neglect, 10.8% were victims of physical abuse, 7.6% were sexually abused, 4.2% were psychologically maltreated, and 13.1% experienced multiple forms of maltreatments. Nearly 80% of child maltreatment perpetrators were parents, and another 6.6% were other relatives of the victim.

Over the last several decades, research has begun to elucidate the long-term negative impact of ACEs (e.g., abuse, neglect, and household dysfunction) on the emotional, behavioral, and cognitive development of children (Arias, 2004; Repetti, Taylor, & Seeman, 2002; Taylor, Lerner, Sage, Lehman, & Seeman, 2004). This deleterious impact may be due to an unhealthy environment that impedes the resolution of early life developmental issues (Sroufe & Rutter, 1984) as well as actual modifications in brain anatomy and functioning during important developmental periods (McEwen &

Stellar, 1993). These disruptions in the normal developmental process can result in the adoption of unhealthy coping behaviors throughout the lifespan (Leitenberg, Gibson, & Novy, 2004; Stevens, Colwell, Smith, Robinson, & McMillan, 2005) as well as psychological distress. Psychological distress is defined as temporary or permanent maladaptive psychological functioning as the result of stressful life events ranging in severity from temporary situational distress to long-term mental illness (Ridner, 2004).

Current research has consistently linked ACEs to later life illicit drug use (Douglas et al., 2010; Dube et al., 2003; Simpson & Miller, 2002; Widom, Marmorstein, & White, 2006; Wu, Schairer, Dellor, & Grella, 2010) and alcohol use and abuse (Anda et al., 2002; Dube et al., 2006; Koss et al., 2003; Langeland & Hartgers, 1998; Rothman, Edwards, Heeren, & Hingson, 2008; Timko, Sutkowi, Pavao, & Kimerling, 2008; Young, Hansen, Gibson, & Ryan, 2006). Several studies have suggested that mental illnesses such as posttraumatic stress disorder (PTSD), depression, anxiety, and antisocial behavior may mediate this relationship (DeWit, MacDonald, & Offord, 1999; Douglas et al., 2010; Lo & Cheng, 2007; Simpson & Miller, 2002; White & Widom, 2008). Notably, none of these studies examined nicotine use.

Research is only beginning to elucidate the magnitude and complexity of the relationship between ACEs and smoking, the number one cause of preventable mortality in the United States (Centers for Disease Control and Prevention, 2005; Mokdad, Marks, Stroup, & Gerberding, 2004). Current research indicates that ACEs are significantly associated with early smoking initiation, smoking maintenance, and heavy smoking (Acierno, Kilpatrick, Resnick, Saunders, & Best, 1996; Anda et al., 1999; Csoboth,

Birkas, & Purebl, 2003; Diaz, Simantov, & Rickert, 2002; Edwards, Anda, Gu, Dube, & Felitti, 2007; Nichols & Harlow, 2004; Simantov, Schoen, & Klein, 2000; van Loon, Tijhuis, Surtees, & Ormel, 2005). Research also indicates that depression, personality disorders, and anxiety—conditions often associated with ACEs—are consistently linked to smoking behavior and dependence (Dierker & Donny, 2008). Given its anxiolytic and sedative properties (e.g., ability to modify mood, manage dysphoria, regulate negative affect), it is posited that smoking may be viewed as a viable coping option to compensate for emotional, social, and behavioral deficiencies in functioning (Carmody, 1992; Escobedo, Reddy, & Giovino, 1998; Hughes, 1988; Kassel, Stroud, & Paronis, 2003; Koval, Pederson, Mills, McGrady, & Carvajal, 2000; Mermelstein, 1999; Pomerleau & Pomerleau, 1984; Repetti et al., 2002).

Given the pervasive effect of ACEs throughout the life course and the deleterious effect of smoking on health, this dissertation examined the potential mediating effect of psychological distress on the relationship between individual ACEs and smoking. Notably, this study added to existing research by examining a wide array of ACEs as well as the cumulative impact of multiple ACEs. Most current ACE research has examined one or a few types of ACEs and thus fails to account for the cumulative effect of multiple stressors, underestimates the burden of victimization, and may lead to inaccurate conclusions about the relationship between a specific ACE and a given outcome (Anda et al., 1999; Dong, Anda, Dube, Giles, & Felitti, 2003; Felitti et al., 1998; Finkelhor, Ormrod, Turner, & Hamby, 2005). Moreover, as current research suggests that child abuse and neglect may affect men and women differently (Widom et al., 2006) and that

stressors that lead to smoking initiation and maintenance may vary by gender (Byrne & Mazanov, 1999), these relationships were further explored by gender.

Theoretical Framework

Macmillan (2009) suggested that child maltreatment theory should incorporate the biological, social, and psychological aspects of abuse and neglect in order to fully understand their impact. The developmental psychopathological perspective examines both environmental and biological influences as they interact to promote or impede development (Sameroff, 2000; Sroufe & Rutter, 1984). It is not a single theory, but rather an overarching perspective that includes multiple theories and multiple domains of development, for example, cognitive, social, genetic, neurobiological (Cicchetti & Toth, 2005), with an emphasis on the interplay between normal and abnormal development, risk and protective factors, and internal and external influences (Cicchetti & Toth, 1995).

Developmental psychopathology posits that each developmental stage builds on the previous stage (Sroufe & Rutter, 1984). Given this hierarchical and integrated structure, successful resolution of early-life issues increases the likelihood of subsequent successful adjustment, whereas failure increases the probability of continued difficulties (Sroufe & Rutter, 1984). Unresolved issues compound over time and increase the risk of psychological distress and subsequent maladaptive coping behaviors such as smoking.

Neurobiology is another important component of developmental psychopathology (Cicchetti & Toth, 2005). While many parts of the brain are somewhat malleable throughout the lifespan, the most active and vulnerable time for brain development is during childhood and adolescence (Twardosz & Lutzker, 2010). During this time, the

brain uses environmental cues to develop and form important synaptic connections (Twardosz & Lutzker, 2010). Abuse, neglect, or maltreatment during this important developmental process, known as experience-expectant development (Andersen, 2003; Black & Grennough, 1998; Greenough, Black, & Wallace, 1987; Twardosz & Lutzker, 2010), can lead to modifications in brain anatomy and functioning (Anda et al., 2006; McEwen & Stellar, 1993). While it is possible to generate new synaptic connections and modify existing connections later in life, known as experience-dependent development, this type of development is less automatic and requires repetitive routines and teaching—stability not often found in abusive households (Teicher et al., 2004). Given the important emotional and neurobiological development that occurs during this critical period, persons who experienced ACEs often have emotional, physical, social, and behavioral deficits (Repetti et al., 2002). These deficits can lead to psychological distress, chronic mental illness (Heim & Nemeroff, 2001), and suboptimal coping skills (Gibson & Leitenberg, 2001), thus providing a potential mechanism linking ACEs to psychological distress and subsequent smoking.

Statement of the Problem

Persons who experience ACEs are disproportionately affected by mental illness and drug use. A potential causal chain of events may be responsible for this relationship. Current research suggests that ACEs can lead to emotional, cognitive, physical, and behavioral deficits by modifying the hormones and brain circuitry that regulate stress (McEwen, 2003) and disrupting stage-salient developmental tasks (Sroufe & Rutter, 1984). These decrements may lead to ineffective coping strategies and a desire to self-

medicate. Given that smoking has anxiolytic and sedative properties, it may be viewed as a viable alternative coping strategy (Escobedo et al., 1998; Kassel et al., 2003; Koval et al., 2000; Mermelstein, 1999). While there are a number of other potential explanations for the increased prevalence of smoking among persons who experienced ACEs—for example, inadequate parent knowledge, supervision, and support (Biglan, Duncan, Ary, & Smolkowski, 1995; Wills & Cleary, 1996); increased peer influence (Biglan et al., 1995; Mounts & Steinberg, 1995)—psychological distress may be an important mediator in this relationship.

Numerous studies have reported associations between ACEs and illicit drug use (Simpson & Miller, 2002) and have suggested that the psychological distress associated with mental illnesses may mediate this association (DeWit et al., 1999; Douglas et al., 2010; Lo & Cheng, 2007; Simpson & Miller, 2002; White & Widom, 2008). While research indicates that ACEs are associated with smoking (Acierno et al., 1996; Anda et al., 1999; Csoboth et al., 2003; Diaz et al., 2002; Edwards et al., 2007; Nichols & Harlow, 2004; Simantov et al., 2000; van Loon et al., 2005)—the number one cause of preventable mortality in the United States (Centers for Disease Control and Prevention, 2005; Mokdad et al., 2004)—it is notable that few studies have examined potential mediators in this relationship. Given this important gap in the literature, the analyses in this study examined the potential mediating effect of psychological distress on the association between ACEs and smoking. In addition, the study determined if this relationship varied by type of ACEs, total number of ACEs, and gender.

Purpose of the Study

The purpose of this study was to quantitatively examine whether psychological distress mediated the relationship between ACEs and smoking in adulthood. This study also sought to determine if psychological distress played a different role in the relationship between ACEs and smoking by type of ACE, cumulative number of ACEs, and gender.

Brief Definition of Terms

Key terms will briefly be described below. More detailed definitions can be found in Chapter 3.

Psychological distress was assessed using the SF-36 algorithms developed to calculate individual (Ware, Snow, Kosinski, & Gandek, 1993) and Mental Component Summary (MCS) scores (Ware, 2000; Ware, Kosinski, & Keller, 1994). Psychological distress is defined as temporary or permanent maladaptive psychological functioning as the result of stressful life events (Ridner, 2004). Used as a continuous variable in this study, the MCS scores range in severity from absence of psychological distress to chronic mental illnesses (Ware, 2000). The MCS score has been shown to be useful in screening persons with psychiatric disorders. For example, a cutoff score of 42 has a 74% sensitivity and 81% specificity for detecting persons diagnosed with depression (Ware, Kosinski et al., 1994).

Types of abuse included verbal (Straus & Gelles, 1990), physical (Straus & Gelles, 1990), and sexual (Wyatt, 1985).

Types of neglect included emotional and physical (Bernstein et al., 1994).

Types of household dysfunction included violence against the mother (Straus & Gelles, 1990), substance abuse in the household (Schoenborn, 1995), mental illness in the household, parental separation or divorce, and incarcerated household member.

ACE score was defined as cumulative exposure to abuse, neglect, and household dysfunction (Anda et al., 1999; Anda et al., 2006; Dong et al., 2004; Felitti et al., 1998).

Smoking status was assessed using the question: “Do you smoke cigarettes now?” This question was adopted from several national surveys including the Behavioral Risk Factor Surveys (Siegel, Frazier, Mariolis, Brackbill, & Smith, 1993) and the Third National Health and Nutrition Examination Survey (Crespo, Keteyian, Heath, & Sempos, 1996).

Research Questions and Hypotheses

The following research questions and hypotheses have been developed from a review of the ACE and smoking literature. A more detailed description of the study can be found in chapter 3.

Research question 1.

What is the nature of the relationships between ACEs and smoking and ACEs and psychological distress?

Hypothesis 1.

Null Hypothesis (H_{01}): There is not a relationship between ACEs (abuse, neglect, and household dysfunction) and smoking or ACEs and psychological distress (as assessed by the SF-36 Mental Component Summary Scale) among members of a

Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a1}): Among members of a Kaiser Permanente HMO in San Diego, California, ACEs increase the risk of psychological distress (as assessed using the SF-36 Mental Component Summary score) as well as adult smoking.

Research question 2.

Does the relationship between ACEs and smoking and ACEs and psychological distress vary by type of ACE?

Hypothesis 2.

Null Hypothesis (H_{o2}): There is no difference in the effects of different types of ACEs on the subsequent risk of psychological distress (as assessed by the SF-36 Mental Component Summary Scale) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a2}): Different types of ACEs (abuse, neglect, and household dysfunction) have varying affects on the subsequent risk of psychological distress (as assessed using the SF-36 Mental Component Summary score) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research question 3.

As the cumulative number of ACEs increases does the risk of psychological distress and smoking increase?

Hypothesis 3.

Null Hypothesis (H_{03}): There is not a cumulative effect of multiple ACEs (abuse, neglect, and household dysfunction) on the risk of subsequent psychological distress (as assessed by the SF-36 Mental Component Summary Scale) or smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a3}): As the cumulative number of ACEs (abuse, neglect, and household dysfunction) increases, so does the risk of subsequent psychological distress (as assessed by the SF-36 Mental Component Summary Scale) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research question 4.

Do the relationships between ACEs and psychological distress and ACEs and smoking vary by gender?

Hypothesis 4.

Null Hypothesis (H_{04}): There is not a difference in the relationships between ACEs (abuse, neglect, and household dysfunction) and psychological distress (as assessed by the SF-36 Mental Component Summary Scale) or ACEs and smoking by gender among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a4}): The relationships between ACEs (abuse, neglect, and household dysfunction) and psychological distress (as assessed by the SF-36

Mental Component Summary Scale), and ACEs and smoking are stronger for female (versus male) members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research question 5.

Does psychological distress mediate the relationship between ACEs and smoking?

Hypothesis 5.

Null Hypothesis (H_{05}): Psychological distress (as assessed by the SF-36 Mental Component Summary Scale), does not mediate the relationship between ACEs (abuse, neglect, and household dysfunction) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a5}): Psychological distress (as assessed by the SF-36 Mental Component Summary Scale), mediates the relationship between ACEs (abuse, neglect, and household dysfunction) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Significance of the Study

This study contributes to existing research by examining the long-term consequences of ACEs on smoking—the number one cause of preventable mortality in the United States (Centers for Disease Control and Prevention, 2005; Mokdad et al., 2004). Many of the contemporary researchers in the field of child abuse and neglect

indicate that the major problem with ACE research is that studies usually focus on one or a few forms of victimization (Finkelhor et al., 2005; Green et al., 2000). To date, the majority of recent research has concentrated primarily on the impact of childhood sexual abuse on adult health and behaviors (Felitti et al., 1998) and has ignored the potential deleterious effects of childhood physical and emotional abuse or neglect (Widom, DuMont, & Czaja, 2007) and household dysfunction on these outcomes. This research fails to account for the cumulative effect of multiple stressors, underestimates the burden of victimization, and possibly leads to inaccurate assumptions about the relationships between specific ACEs and negative outcomes (Anda et al., 1999; Dong, Anda et al., 2003; Felitti et al., 1998; Finkelhor et al., 2005). This dissertation examined some of the gaps in the literature by examining the potential relationships among smoking, psychological distress, and a broad range of ACEs as well as the cumulative impact of multiple ACEs.

Moreover, research indicates that the stress associated with ACEs and type of ACE may affect men and women differently (Byrne & Mazanov, 1999; Widom et al., 2006). Given that this may warrant gender-specific prevention and intervention strategies, this study also examined the associations of ACEs, psychological distress, and smoking by gender.

Implications for Social Change

ACEs are common in the United States and evoke long-term consequences on social, emotional, and behavioral development, often leading to chronic mental illness and unhealthy coping behaviors, including smoking. Current research indicates that

persons who report 5 or more ACEs are 3 times more likely to have ever smoked and 2 times more likely to be a current smoker than those with no reported ACEs (Anda et al., 1999). Smoking, an established risk factor for a number of chronic diseases, is the number one cause of preventable mortality in the United (Centers for Disease Control and Prevention, 2005; Mokdad et al., 2004). Given these facts, determining potential mediating effects on the relationship between ACEs and smoking could lead to positive social change by providing knowledge useful for program developers, educators, public health professionals, mental and physical health professionals, and other specialists searching for direction in improving prevention and intervention programs to combat the high rate of smoking in this population.

Assumptions and Limitations

It is assumed that the participants completed the questionnaires truthfully and to the best of their ability.

It was possible that there were significant differences in characteristics between those who did and did not chose to participate in the ACE Study. Notably, analysis comparing responders to non-responders were conducted in Wave I of the ACE Study (Felitti et al., 1998). Completed medical evaluations for every person eligible for the study were abstracted to compare respondents' and non-respondents' medical history, laboratory results, and physical findings (Felitti et al., 1998). Respondent and non-respondent groups were similar with regard to sociodemographic characteristics (e.g., percentages of women, mean years of education, marital status), self-rated health, engagement in adverse health behaviors (e.g., smoking and other substance abuse), and

presence of chronic diseases such as heart attack, stroke, chronic obstructive lung disease, hypertension, and diabetes (Felitti et al., 1998). While respondents were older and more likely to be White than the non-respondents, the actual magnitude of the differences was small (Felitti et al., 1998).

Given the cross-sectional nature of the study and the retrospective nature of the childhood victimization questions, recall bias was possible due to forgotten or nondisclosed abuse as well as false recollections of abuse that served to provide meaning to current distress and illness (Cicchetti & Toth, 2005; Raphael & Cloitre, 1994; Schraedley, Turner, & Gotlib, 2002). Furthermore, there may be differences in reporting retrospective information about childhood abuse by gender. For example, in an article by Widom and Morris (1997), among persons with a history of documented sexual abuse in childhood, fewer men than women later considered the event sexual abuse. Notably, longitudinal follow-up studies of adults with documented childhood abuse suggested that retrospective reports of childhood abuse often underrepresented actual events (Hardt & Rutter, 2004; Widom & Shepard, 1996; Widom & Morris, 1997) and that recall may not be as inaccurate as originally anticipated (Bernstein et al., 1994; Brewin, Andrews, & Gotlib, 1993; Dill, Chu, Grob, & Eisen, 1991). Most importantly, Edwards, Anda et al. (2001) found no evidence of response rate bias (i.e., persons who did not participate in the ACE Study experienced childhood sexual abuse at the same rate as those who agreed to participate). Moreover, those who participated in the study were equally as likely as those who did not to attribute childhood sexual abuse to current mental and physical health problems (Edwards, Anda et al., 2001).

Test-retest reliability was also conducted in the ACE Study. In both Wave I and Wave II, 658 participants completed the ACE survey (Dube, Williamson, Thompson, Felitti, & Anda, 2004). The test-rest reliability of adult reports of childhood sexual, physical, and emotional abuse, as well as forms of household dysfunction (i.e., mental illness in household, substance abuse in household, parental discord or divorce, incarcerated household member, and domestic violence) were assessed using Cohen's kappa (Dube et al., 2004). The kappa coefficients ranged from good to substantial agreement (range: 0.46-0.86), as defined by Fleiss (1981) and Landis and Koch (1977), for each category of childhood abuse and household dysfunction (Dube et al., 2004).

Smoking status was also self-reported. Studies indicate that self-reported estimates may underestimate true smoking prevalence (Gorber, Schofield-Hurwitz, Hardt, Levasseur, & Tremblay, 2009; Lewis et al., 2003). Moreover, Anda et al. (1999) found that the prevalence of smoking in the ACE Study was lower than the average population of California and surmised that this may be due to the older and more educated sample in the study. Given this, the authors examined Wave I data by adjusting for differences in the demographics (age, sex, race, educational attainment) between the study population and the 1995 census population estimates for California (Anda et al., 1999). After accounting for sociodemographic differences, 14.4% of the study population currently smoked compared to 15.5% of California residents (Centers for Disease Control and Prevention, 1996), suggesting that the sociodemographic characteristics of the study population accounts for the lower prevalence of smoking (Anda et al., 1999).

Given that persons in the ACE Study are older and more educated than the general population, the generalizability of the study may be limited. Notably, ACE Study estimates are similar to population-based surveys (Dong, Anda et al., 2003). In two population-based surveys of adults (Finkelhor, Hotaling, Lewis, & Smith, 1990; MacMillan et al., 1997), 16% of men had been sexually abused and 31% were physically abused; similar to the 16% and 30% of men, respectively, in Wave I and Wave II of the ACE Study (Dong, Anda et al., 2003). Similarly, 27% of women were sexually abused in the Finkelhor, Hotaling, Lewis, & Smith (1990) study compared to the 25% in the ACE Study (Dong, Anda et al., 2003).

Given that the data are cross-sectional, the temporal relationships between ACEs, psychological distress, and smoking are more difficult to determine. However, most research to date suggests that, in general, ACEs increase the risk of psychological distress, and psychological distress increases the risk of smoking (Wills, Sandy, & Yaeger, 2002). However, there are studies that suggest that smoking precedes psychological distress (Breslau, Novak, & Kessler, 2004; Choi, Patten, Gillin, Kaplan, & Pierce, 1997; Goodman & Capitman, 2000; Johnson et al., 2000; McGee, Williams, & Stanton, 1998; Munafo, Hitsman, Rende, Metcalfe, & Niaura, 2008; Pohl, Yeragani, Balon, Lycaki, & McBride, 1992; Steuber & Danner, 2006), that the relationship between smoking and psychological distress may result from common environmental (e.g., alcohol use, parental or peer smoking) or genetic factors (Kendler et al., 1993), and that the relationship between smoking and psychological distress may be bidirectional in

nature (Chaiton, Cohen, O'Loughlin, & Rehm, 2009; Kendler et al., 1993; Paperwalla, Levin, Weiner, & Saravay, 2004).

Research in the area of child victimization would benefit from additional questions addressing timing, chronicity, and relationship of victim to perpetrator—information that was not available in the ACE Study (Cicchetti & Toth, 1995; Kaplow & Widom, 2007). According to a study conducted by Jun et al. (2008), there is a strong dose-response relationship between early smoking initiation and severity and chronicity of abuse among adolescent females. The timing of the event(s) is also important. According to Thornberry, Ireland, and Smith (2001), abuse experienced during adolescents has stronger and more negative behavioral consequences than abuse experienced in childhood alone. A study by Kaplow and Widom (2007) also suggests that maltreatment experienced later in childhood is predictive of more behavioral problems in adulthood. The ACE Study would also benefit from additional information on the participant's past and current environmental circumstances. While the ACE Study assesses stress and trauma within the family environment during childhood, broader environmental exposures (e.g., social, economic, neighborhood, social support) could impact psychological and behavioral development. Moreover, research suggests that persons abused as children are at increased risk of victimization as adults due to environmental factors and poor relationships and coping skills (Coid et al., 2001; McNutt, Carlson, Persaud, & Postmus, 2002; Messman-Moore, & Long, 2003; Schaaf & McCanne, 1998). Finally, few studies have examined potential protective factors that

may mitigate the adverse effects of ACEs (e.g., religious participation, quality of relationship with primary caregiver, social support, resiliency).

Summary

According to developmental psychopathology, ACEs can lead to deleterious modification in brain development and functioning and leave stage-salient developmental issues unresolved which can produce deficits in emotional processing, social competence, and behavioral self-regulation. This can lead to psychological distress and ineffective coping strategies throughout the life course. Self-medication through smoking to manage negative feelings may be seen as a viable coping strategy. While research suggests that psychiatric illness may be an important mediator between ACEs and drug and alcohol use, little research has examined the potential mediating variables that link ACEs and smoking. Given this gap in the current scientific literature, this dissertation examined the potential mediating effect of psychological distress on the relationship between ACEs and smoking by individual ACE, total number of ACEs, and gender.

Chapter 2 reviews the existing literature and the relationships between ACEs and smoking, ACEs and psychological distress, and psychological distress and smoking to lay the foundation for the research questions. The chapter also provides biological and behavioral explanations for these relationships using developmental psychopathology theories. In addition to describing the lifetime effect of ACEs on child development and adult health, the chapter provides an overview of the deleterious impact of smoking on U.S. children, adolescents, and adults, and provides an overview of gender differences in smoking initiation, maintenance, and cessation. This chapter also includes a discussion of

literature that challenges the temporal relationships that form the foundation of this dissertation.

Chapter 3 includes a description of the sample, data collection, measures, and analysis of the data. The chapter also provides a detailed discussion of the SF-36 scale (Ware, Kosinski et al., 1994; Ware et al., 1993) as well as a description of why linear and logistic regression and mediation models are appropriate for this study.

Chapter 4 quantitatively examines the potential mediating effect of psychological distress on the relationship between ACEs and adult smoking by individual ACE, total ACE score, and gender.

Finally, chapter 5 includes a brief explanation for why the study was conducted, an interpretation of the findings, recommendations for future research, implications for social change, and limitations of the study.

Chapter 2: Literature Review

Introduction

While several recent articles have suggested that persons who have experienced ACEs are more likely to smoke, the exact mechanism(s) linking ACEs with smoking has not been fully elucidated. This dissertation examined the potential mediating effect of psychological distress on the relationship between ACEs and smoking in adulthood. The review offers both biological and behavioral explanations for these potential relationships using theories from developmental psychopathology.

A search of literature was conducted through psychology and medical databases such as PUBMED, PsycINFO, PscyARTICLES, and MEDLINE. The list of search terms used to conduct the literature review included smoking, psychological distress, stress, depression, anxiety, PTSD, adverse childhood experiences, physical abuse, sexual abuse, emotional abuse, child maltreatment, family violence, neurobiology, amygdala, hippocampus, prefrontal cortex, experience-expectant and experience-dependent development, allostatic load, developmental psychopathology, and brain development. Literature from the past five years on each topic was examined and additional references were selected from articles of interest. Multiple books were also examined to provide information on the history and scoring of the SF-36, statistical procedures, ACEs definitions, and the anatomy of the brain.

In order to lay the groundwork for the proposed hypotheses in this dissertation, this chapter provides an overview of literature that addresses developmental psychopathology—the theoretical underpinnings for this research—as well as current

knowledge about the relationship between ACEs and smoking, ACEs and psychological distress, and psychological distress and smoking.

Current Mediation Research and Theoretical Underpinnings

Current Mediation Research

The proposed study was adapted from research examining the potential mediating effect of various mental illnesses on the relationship between specific ACEs and drug use (Douglas et al., 2010; Lo & Cheng, 2007; Simpson & Miller, 2002; White & Widom, 2008). In a review article conducted by Simpson and Miller (2002), the authors indicated that childhood abuse may be a factor in the development of substance use problems among women, but that the relationship is probably mediated by psychiatric conditions. Lo and Cheng (2007) examined the relationship between type (physical or sexual) and persistence of abuse and substance use (current abuse of alcohol, marijuana, or other drugs) among 762 persons using the first five waves of the National Youth Survey as well as wave 7. Their research suggested that childhood physical abuse was related to current substance abuse and that depression partially mediated this relationship (Lo & Cheng, 2007). White and Widom (2008) interviewed 582 women with documented cases of early childhood abuse at two points in time, early and middle adulthood, as well as matched controls. This study included questions regarding perceived stability of home life, childhood trauma, household drug use, and potential protective factors, including religious participation, amount of contact with relatives, and quality of relationship with the primary caregiver (White & Widom, 2008). The authors noted that PTSD mediated the relationship between early childhood maltreatment and illicit drug use (White &

Widom, 2008). Finally, Douglas et al. (2010) conducted a secondary analysis of 2061 persons with a lifetime diagnosis of alcohol or drug dependence and 449 controls from pooled genetics substance-dependence studies. They concluded that mood and anxiety disorders partially mediated the relationship between ACEs and substance dependence (Douglas et al., 2010). Notably, none of these studies examined nicotine use.

Developmental Psychopathology

The research in this dissertation posits that emotional and neurobiological development is disrupted during critical periods of development among children who experience ACEs resulting in emotional, physical, social, and behavioral deficits (Repetti et al., 2002). These deficits often lead to psychological distress (Heim & Nemeroff, 2001), ineffective coping strategies (Gibson & Leitenberg, 2001) and maladaptive patterns of behavior, such as smoking (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997). This pathway can be explained using developmental psychopathology (Sameroff, 2000; Sroufe & Rutter, 1984), an overarching perspective that includes multiple theories and examines the influence of both environmental and biological factors on development (Cicchetti & Toth, 2005).

Stage-salient issues. Developmental psychopathology posits that each developmental stage builds on the previous stage (Sroufe & Rutter, 1984). Each task is hierarchically organized and integrated therefore successful resolution of early-stage-salient tasks increases the likelihood of subsequent successful adjustment whereas failure on an early task increases the risk of subsequent maladaptation (Sroufe & Rutter, 1984).

These difficulties compound over time and increase the risk of psychological distress and subsequent maladaptive coping behaviors such as smoking (Kazdin et al., 1997).

There are a number of stage salient developmental issues throughout the lifespan however most research has concentrated on issues related to childhood and adolescence. One issue in infancy is the ability to regulate and interpret affective experience (Cicchetti & Toth, 1995, 2005). Given that early communication occurs primarily between a caregiver and the child, disruptions in the development of affect regulation in children who experience maltreatment is likely. A number of studies have noted that maltreated infants and toddlers are more likely than those who had not been mistreated to be angry, frustrated, noncompliant, depressed, and ambivalent while attempting to accomplish a task (Cicchetti & Toth, 1995). Many of these characteristics carry over into childhood, adolescence, and adulthood and result in behavioral dysregulation (Cummings, Hennessy, Rabideau, & Cicchetti, 1994; Hennessy, Rabideau, Cicchetti, & Cummings, 1994; Maughan & Cicchetti, 2002), difficulties processing social information (Pollak, Cicchetti, Hornung, & Reed, 2000; Pollak & Kistler, 2002; Pollak & Sinha, 2002), and hypervigilance to hostile cues (Dodge, Pettit, Bates, & Valente, 1995).

During the first year of life, a primary task is establishing a secure attachment relationship with the caregiver (Cicchetti & Toth, 1995, 2005). This relationship lays the foundation for future relationships and provides the groundwork for the understanding of self (Sroufe, 1979). Given this, maltreated children often develop insecure attachment relationships (Cicchetti & Barnett, 1991; Lynch & Cicchetti, 1991) and experience depressive symptoms throughout the lifespan (Toth & Cicchetti, 1996).

Following the development of attachment relationships is the development of self-system processes (Cicchetti & Toth, 1995, 2005) or the sense of self as autonomous from the caregiver (Sroufe, 1979). Maltreated children often display neutral or negative affect when they see themselves in the mirror, often have difficulties talking about internal states and feelings of self and others (Beeghly & Cicchetti, 1994; Cicchetti, Rogosch, Maughan, Toth, & Bruce, 2003; Toth, Cicchetti, Macfie, & Emde, 1997; Toth, Cicchetti, Macfie, Rogosch, & Maughan, 2000), and can even experience dissociation (Macfie, Cicchetti, & Toth, 2001).

Given the difficulties with early stage-salient issues of development, maltreated children often have elevated aggression towards or withdrawal from peers (Cicchetti & Toth, 2005). Research has also noted other antisocial behavior such as meanness, bullying, and disruptiveness (Klimes-Dougan & Kistner, 1990; Rogosch & Cicchetti, 1994; Salzinger, Feldman, Hammer, & Rosario, 1993; Shields & Cicchetti, 2001). These behaviors create problems in the school environment and often result in academic failure (Eckenrode, Laird, & Doris, 1993), difficulties in peer adjustment and self-perception, and depression (Okun, Parker, & Levendosky, 1994).

Neurobiological implications. Neurobiology is another important component of developmental psychopathology (Cicchetti & Toth, 2005). While the brain is continually molded by experiences throughout the lifespan, it is particularly sensitive (i.e., plastic) during various times in childhood and adolescents (Andersen, 2003; Huttenlocher, 1979; Twardosz & Lutzker, 2010). Different parts of the brain play different roles in social, behavioral, and emotional development and they mature during different developmental

periods in a hierarchical fashion (Joseph, 1982, 1992, 1999). For example, the regulatory brainstem, responsible for reflexive motor and vital functions, is fully developed at birth whereas the more complex regions such as the limbic system, responsible for emotions and behaviors, and the cortical regions, responsible for analytic thinking, mature during later stages of development (Figure 1).

Experience-expectant development (Andersen, 2003; Black & Grennough, 1998; Greenough et al., 1987; Twardosz & Lutzker, 2010), which is dominant during childhood, is a time when the brain adapts by incorporating environmental information permanently into the brain (Andersen, 2003). During this time, different parts of the brain are highly responsive to particular types of environmental stimuli and these regions of the brain are flooded with synapses (Twardosz & Lutzker, 2010). The brain creates, strengthens, and discards neuronal pathways and synapses in response to environmental stimuli (Barnekow & Kraemer, 2005; Duman, Heninger, & Nestler, 1997; Gould & Tanapat, 1999; Joseph, 1982, 1992, 1999; Read, Perry, Moskowitz, & Connolly, 2001; Teicher et al., 2003; Walsh, 1980, 1981; Walsh, Budtz-Olsen, Penny, & Cummins, 1969). Repeated experiences strengthen neural pathways creating a memory that shapes perceptions and responses to the environment (U.S. Department of Health and Human Services, 2009b).

Repeated stress in a child's environment during critical periods of brain development can lead to modifications in brain anatomy and functioning (Anda et al., 2006; McEwen & Stellar, 1993). Growth in each region of the brain depends on stimulation to activate the region. Chronic stimulation in one area of the brain can over-

develop certain neural pathways in that region and cause under-development in other regions of the brain (U.S. Department of Health and Human Services, 2009b). For example, chronic stimulation of the brain's fear response may activate this region of the brain and limit development in regions of the brain responsible for intellectual, perceptual, social, and emotional characteristics (Cicchetti & Tucker, 1994; Geyer, Wilkinson, Humby, & Robbins, 1993; Joseph, 1982, 1992, 1999; Joseph & Casagrande, 1980; Joseph & Gallagher, 1980).

Once this critical developmental period is completed, the brain area is less plastic. Changes that occur at this point, experience-dependent development (Andersen, 2003; Black & Grennough, 1998; Greenough et al., 1987; Twardosz & Lutzker, 2010), are an attempt by the brain to compensate for changes in the environment (Andersen, 2003). This involves the generation of new neural connections or the modification of existing ones and is encoded in the brain through consistent routines, interactions, and specific teaching; conditions often not present in households where maltreatment occurs (Twardosz & Lutzker, 2010). Given the important neurobiological development that is occurring during this critical time period, persons who experienced ACEs often have emotional, physical, social, and behavioral deficits (Repetti et al., 2002). These deficits can lead to psychological distress (Heim & Nemeroff, 2001), suboptimal coping skills (Gibson & Leitenberg, 2001), and subsequent maladaptive coping behaviors such as smoking (Kazdin et al., 1997).

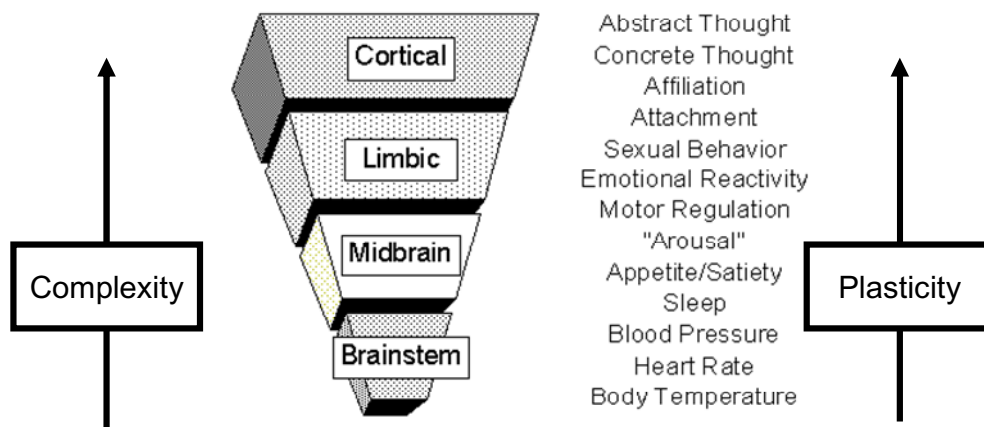


Figure 1. Hierarchy of brain development. Adapted from "Applied Principles of Neurodevelopment to Clinical Work With Maltreated and Traumatized Children: The Neurosequential model of Therapeutics" by B. Perry, 2006, *Working with Traumatized Youth in Child Welfare*, p 44. Copyright 2006 by the Guilford Press. Retrieved May 17, 2010 from http://childtraumaacademy.org/Documents/NeurosequentialModel_06.pdf. Reprinted with permission.

Smoking

Smoking harms nearly every major organ of the body (U.S. Department of Health and Human Services, 2004). In the United States, it causes more deaths than alcohol, illegal drugs, car accidents, suicide, homicide, and AIDS combined (Centers for Disease Control and Prevention, 2005; McGinnis & Foege, 1993). In fact, recent research has identified smoking as the leading cause of preventable mortality; accounting for nearly one in five deaths each year in the United States (Centers for Disease Control and Prevention, 2005; Mokdad et al., 2004). Half of all long-term smokers die prematurely from smoking-related disease (U.S. Department of Health and Human Services, 2004);

on average, dying 14 years earlier than nonsmokers (Centers for Disease Control and Prevention, 2002).

Nearly 90% of persons who smoke have their first cigarette before the age of 18 years (U.S. Department of Health and Human Services, 1994). In fact, approximately 20% of teens are current smokers (Eaton et al., 2008) and, on average, are addicted to nicotine before the age of 13 years (Elders & Perry, 1994). Notably, most persons who start smoking at a young age often become heavy, nicotine-dependent smokers in adulthood (Kandel, Chen, Warner, Kessler, & Grant, 1997; Woolf, 1997). Current research suggests that potential risk factors for adolescent smoking include low socioeconomic status, peer smoking, psychological distress, exposure to violence or abuse, and family stress (Ary & Biglan, 1988; Conrad, Flay, & Hill, 1992; Covey, Glassman, & Stetner, 1990; Dembo, Dertke, Borders, Washburn, & Schmeidler, 1988; Simantov et al., 2000). Most of these factors were considered in this dissertation.

Notably, many chronic diseases exhibit a dose-response relationship with smoking. For example, the risk of smoke-related cancers is related to total lifetime exposure to cigarette smoke (e.g., number of cigarettes smoked each day, age at smoking initiation, number of years smoked) (American Cancer Society, 2003). In a study conducted by Weintraub, Klein, Seelaus, Agarwal, and Helfant (1985), total pack-years was a significant independent risk factor for coronary artery disease. In a study conducted by Bhat et al. (2008), there was a strong dose-response relationship between smoking and ischemic stroke risk in women aged 15 to 49 years (current smokers versus never smokers, $OR = 2.6$; number of cigarettes per day, $OR = 2.2$ for 1 to 10; $OR = 2.5$ for 11 to

20; $OR = 4.3$ for 21 to 39; and $OR = 9.1$ for 40 or more cigarettes/day). Despite these alarming statistics, smoking cessation always produced health benefits. For example, smoking cessation by age 35 reduces the risk of premature death by 90%. Notably, the risk of premature death is also substantially decreased if one quits by age 50 (American Cancer Society, 2003).

Gender differences in smoking

There are several differences in smoking patterns by gender. First, negative affect, including depression, is related to smoking among both men and women but the relationship is much stronger for women (Brandon & Baker, 1991; Husky, Mazure, Paliwal, & McKee, 2008; McKee, Maciejewski, Falba, & Mazure, 2003). In fact, recent research suggests that stressful childhood life events may disproportionately influence a women's decision to use drugs (Simpson & Miller, 2002; Widom et al., 2006). Notably, in stratified analysis conducted by Patton et al. (1996), an association between regular smoking and psychiatric morbidity was found among teenage girls in Grades 7 through 11, however, the association was only found among males in Grade 7.

Second, women seem to be less dependent on nicotine than men (Bjornson et al., 1995; Gritz et al., 1998; Perkins, Jacobs, Sanders, & Caggiula, 2002; Royce, Corbett, Sorensen, & Ockene, 1997; Ward, Klesges, Zbikowski, Bliss, & Garvey, 1997), they are less likely to be heavy smokers (Giovino et al., 1994), and have lower concentrations of cotinine; a metabolite (byproduct) of nicotine (Bjornson et al., 1995; Etter & Perneger, 2000; Etter, Vu Duc, & Perneger, 2000; Glassman et al., 1993; Ward et al., 1997). Notably, however, studies have consistently found that women have lower quit rates than

men (Perkins & Scott, 2008; Royce et al., 1997; Wetter et al., 1999), have lower confidence in their ability to quit (Etter, Prokhorov, & Perneger, 2002), and often experience worse withdrawal symptoms during smoking cessation attempts (Perkins, Donny, & Caggiula, 1999; Royce et al., 1997). In fact, recent research suggests that the smoking rates for adolescent and adult women may actually be increasing (De Von Figueroa-Moseley, Landrine, & Klonoff, 2004).

The rate of smoking among women is particularly disconcerting because, as compared to men, women who smoke have higher rates of myocardial infarction (Prescott, Hippe, Schnohr, Hein, & Vestbo, 1998; Prescott, Scharling, Osler, & Schnohr, 2002), impaired lung functioning (Dransfield, Davis, Gerald, & Bailey, 2006), and lung cancer (Olak & Colson, 2004). Additionally, babies born to women who smoke during pregnancy are more likely to be born with a low birth weight, have a 30% higher odds of being born prematurely, and are significantly more likely to die due from sudden infant death syndrome (DiFranza, Aligne, & Weitzman, 2004; U.S. Department of Health and Human Services, 2009c). Recent research also indicated that smoking during pregnancy increases the risk that the child may become a subsequent smoker (Buka, Shenassa, & Niaura, 2003).

ACEs and Smoking

Smoking among persons with ACEs is of public health concern for several reasons. First, childhood victimization has repeatedly been shown to increase the risk of adverse health behaviors such as alcoholism, excessive drug use, risky sexual behaviors, and eating disorders into adulthood (Bulik, Prescott, & Kendler, 2001; De Von Figueroa-

Moseley et al., 2004; Nelson et al., 2006; Repetti et al., 2002; Rodgers et al., 2004; Simpson & Miller, 2002; White & Widom, 2008; Widom et al., 2007; Widom & Hiller-Sturmhofel, 2001). Research is only beginning to examine the relationship between ACEs and smoking and findings to date suggests that ACEs are significantly associated with early smoking initiation, smoking maintenance, heavy smoking, and nicotine dependence (Acierno et al., 1996; Anda et al., 1999; Csoboth et al., 2003; De Von Figueroa-Moseley et al., 2004; Diaz et al., 2002; Edwards et al., 2007; Nichols & Harlow, 2004; Simantov et al., 2000; van Loon et al., 2005); many of the characteristics found among chronic nicotine users.

Second, the nature of this relationship and the factors that lead from ACEs to drug use are not well understood (Simpson & Miller, 2002). To date, the majority of research in this area has focused on illicit drug use (Douglas et al., 2010; Dube et al., 2003; Simpson & Miller, 2002; Widom et al., 2006; Wu et al., in press) and alcohol use and abuse (Anda et al., 2002; Dube et al., 2006; Heffernan et al., 2000; Koss et al., 2003; Rothman et al., 2008; Timko et al., 2008; Young et al., 2006). Several of these studies have suggested that the relationship between ACEs and drug use may be mediated through symptoms of PTSD, depression, antisocial behavior, social phobia, and stressful life events (DeWit et al., 1999; Douglas et al., 2010; Lo & Cheng, 2007; Simpson & Miller, 2002; White & Widom, 2008). Additional research to further elucidate the relationship between ACEs and substance use and abuse may lead to effective prevention and intervention strategies for this at-risk population.

ACEs and Psychological Distress

Over the last several decades, research has begun to elucidate the deleterious long-term impact of ACEs on the emotional, cognitive, and behavioral development of children (Arias, 2004; Repetti et al., 2002; Taylor et al., 2004). According to developmental psychopathology, there are a series of important age- and stage-salient issues that one must master throughout the lifespan in order to lead a healthy life. Efforts to achieve these goals can be hampered by internal and external forces (Sroufe & Rutter, 1984). For example, the environment can have a negative effect on emotional regulation because of lack of stimulation and learning opportunities (Schatz, Smith, Borkowski, Whitman, & Keogh, 2008; Twardosz & Lutzker, 2010). Due to inadequate nurturing, children who live in abusive households often do not develop the ability to control intense feelings or identify and label emotions in themselves or others (Camras et al., 1988; Repetti et al., 2002; Taylor, Way et al., 2006; Twardosz & Lutzker, 2010). Moreover, feelings of being safe and worthy of love are often replaced by feelings of being unworthy, incompetent, powerless, helpless, interpersonally dependent, or bad, which increases the risk of psychological distress (Coffey, Leitenberg, Henning, Turner, & Bennett, 1996; Finkelhor & Browne, 1985; Harris, Brown, & Bilfulco, 1990; Liem & Boudewyn, 1999; Wright, Crawford, & Del Castillo, 2009).

In addition to environmental factors, recurrent exposure to stress associated with ACEs can lead to potentially irreversible changes in the circuits of the brain that regulate stress (McEwen, 2006; Twardosz & Lutzker, 2010). Research is beginning to elucidate a number of areas in the brain that are potentially modified by stress. These include, but are

not limited to, the hippocampus, amygdala, and prefrontal cortex (Anda et al., 2006; Bremner, 2003; Glaser, 2000; Heim & Nemeroff, 1999, 2001, 2002; Heim et al., 2000; McEwen, 2007; McFarlane et al., 2005; Nemeroff, 2004; Nemeroff & Vale, 2005; Penza, Heim, & Nemeroff, 2003; Roozendaal, McEwen, & Chattarji, 2009; Shea, Walsh, Macmillan, & Steiner, 2004; Sullivan et al., 2006; Teicher et al., 2003; Van Voorhees & Scarpa, 2004). Each will be described briefly below not as an exhaustive description of the brain regions, their anatomy, or functioning but rather as a means of providing several examples of structural and functional changes in the brain in response to stress. The three brain regions selected act in unison therefore modifications in one region may affect the functions of the other regions (McEwen, 2007). Notably, these areas regulate the activity of the hypothalamic-pituitary-adrenal (HPA) axis which is responsible for controlling reactions to stress (Herman et al., 2003) and is implicated in the development of mental health conditions such as major depression and PTSD (Shea et al., 2004).

Hippocampus

The hippocampus, part of the limbic system, is located deep in the forebrain and helps regulate emotion, learning, memory, and the retrieval of episodic information (Salloway & Blitz, 2002). It contains a high density of glucocorticoid receptors and is therefore vulnerable to stress hormones, for example, cortisol and norepinephrine (Gould & Tanapat, 1999; McEwen, 1999; Sapolsky, Uno, Rebert, & Finch, 1990). Unlike many regions of the brain, the hippocampus has the capacity to generate new neurons in adulthood (Diamond, Fleshner, Ingersoll, & Rose, 1996; Luine, Villegas, Martinez, & McEwen, 1994). However, stress-related steroids affect the hippocampus by reducing the

excitability of some hippocampal neurons (Anda et al., 2006; Teicher et al., 2003), inhibiting the development of new neurons, causing atrophy in the electrical circuitry of the hippocampus, and diminishing memory function (Diamond, Fleshner, Ingersoll, & Rose, 1996; Luine, Villegas, Martinez, & McEwen, 1994; McEwen, 1999; McEwen, 2007). These structural modifications can increase the risk of a number of psychiatric conditions including PTSD (Shin et al., 2004), schizophrenia (Harrison, 2004), and severe depression (Campbell & Macqueen, 2004).

Amygdala

The amygdala, part of the limbic system, is almond shaped and located deep within the temporal lobes adjacent to the hippocampus (Salloway & Blitz, 2002). It plays a crucial role in formulating and storing memories associated with emotional events, responding to threats, controlling aggression and sexual behaviors, and comprehending social cues (Hariri, Tessitore, Mattay, Fera, & Weinberger, 2002; LeDoux, 2000; Ochsner et al., 2004; Roozendaal et al., 2009; Salloway & Blitz, 2002; Taylor, Eisenberger, Saxbe, Lehman, & Lieberman, 2006; Teicher et al., 2003). Unlike the hippocampus, which exhibits decreased functionality during chronic stress, stress actually increases neural growth and synaptic connectivity in the amygdala often causing permanent alterations in this brain region (Czeh, Perez-Cruz, Fuchs, & Flugge, 2008). Not surprisingly, these alterations can lead to anxiety disorders such as PTSD; often characterized by flashbacks and hyperarousal (Yehuda, 2002).

Prefrontal Cortex

The prefrontal cortex, part of the cortical system, carries out executive function such as predicting outcomes and determining consequences of actions, working towards a goal, and monitoring social interactions (Hyafil, Summerfield, & Koehlin, 2009). The prefrontal cortex also has a relatively high density of glucocorticoid receptors (Czeh et al., 2008; Diorio, Viau, & Meaney, 1993). Several studies have indicated substantial neuronal loss and dysfunction in this region among children who have experienced ACEs (Arnsten, 1999; Carrion et al., 2001; De Bellis, Keshavan, Spencer, & Hall, 2000). Notably, these changes are plastic and not degenerative in nature (Czeh et al., 2008). Many disorders, such as schizophrenia, bipolar disorder, and attention deficit hyperactivity disorder (ADHD) have been related to dysfunction of this portion of the brain (Almeida et al., 2009; Benetti et al., 2009; Pakkenberg, Scheel-Kruger, & Kristiansen, 2009; Pennington et al., 2008; Rubia et al., in press; Shaw & Rabin, 2009; Stahl, 2009).

It is not surprising, then, that research consistently indicates that early life stress is a major risk factor for subsequent mental disorders (Arnold, 2004; Cohen, Brown, & Smaile, 2001; Heim & Nemeroff, 2001). For example, persons who experienced ACEs suffer disproportionately from depressive symptoms (Anda et al., 2006; Arnold, 2004; Batten, Aslan, Maciejewski, & Mazure, 2004; Chapman, Dube, & Anda, 2007; Chapman et al., 2004; Fletcher, 2009; Gibb, Butler, & Beck, 2003; Hill et al., 2001; Keller, Neale, & Kendler, 2007; Kendler, Kuhn, & Prescott, 2004; Korkeila et al., 2005; Lu, Mueser, Rosenberg, & Jankowski, 2008; MacMillan et al., 2001; Molnar, Buka, & Kessler, 2001;

Nicolaidis, Curry, McFarland, & Gerrity, 2004; Schilling, Aseltine, & Gore, 2007) suicidality (Afifi, Boman, Fleisher, & Sareen, 2009; Bernet & Stein, 1999; Chapman et al., 2007; Dube et al., 2001; Enns et al., 2006), hallucinations (Anda et al., 2006; Whitfield, Dube, Felitti, & Anda, 2005), sleep disturbances (Anda et al., 2006; Brodsky & Stanley, 2008), memory disturbances (Anda et al., 2006; Brown et al., 2007; Edwards, Fivush, Anda, Felitti, & Nordenberg, 2001), personality disorders (Battle et al., 2004; Johnson et al., 2001; MacMillan et al., 2001; Molnar et al., 2001; Schilling et al., 2007; Tyrka, Wyche, Kelly, Price, & Carpenter, 2009; Widom, 1999), and various forms of anxiety disorders including PTSD (Anda et al., 2006; Chapman et al., 2007; Heim & Nemeroff, 2001; Kessler, Davis, & Kendler, 1997; Rodriguez, Ryan, Vande Kemp, & Foy, 1997; Spertus, Yehuda, Wong, Halligan, & Seremetis, 2003; Suliman et al., 2009). Given these facts, maltreated children are more likely to rely on unsophisticated coping responses to stressful situations such as tension reduction, distraction, avoidance, and escape (Johnson & Kenkel, 1991; Leitenberg, Greenwald, & Cado, 1992; Schatz et al., 2008; Stern & Zevon, 1990).

Psychological Distress and Smoking

Psychiatric disorders are one of the most cited risk factors for nicotine dependence (Dierker & Donny, 2008). Longitudinal studies have suggested that depression (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Dierker, Avenevoli, Merikangas, Flaherty, & Stolar, 2001; Fergusson, Goodwin, & Horwood, 2003; Kandel & Davies, 1986; Patton et al., 1998; Wills et al., 2002), behavioral disorders (Breslau, 1995), and anxiety (Patton et al., 1998), particularly PTSD (Feldner, Babson, &

Zvolensky, 2007), may increase the risk of subsequent smoking. Research has also implicated psychiatric conditions such as schizophrenia (de Leon et al., 1995; Hughes, Hatsukami, Mitchell, & Dahlgren, 1986; Williams & Ziedonis, 2004; Ziedonis, Kosten, Glazer, & Frances, 1994) and ADHD (Chilcoat & Breslau, 1999; Milberger, Biederman, Faraone, Chen, & Jones, 1997; Pomerleau, Downey, Stelson, & Pomerleau, 1995; Riggs, Mikulich, Whitmore, & Crowley, 1999) as risk factors for smoking.

While the general population has experienced a dramatic reduction in tobacco use during the past several decades, there has been little reduction in smoking among persons with mental disorders (Ziedonis, Williams, & Smelson, 2003). Epidemiologic studies have shown that people with mental disorders are two to three times more likely to be nicotine dependent than the general population; they constitute about 44.3% of all smokers; and they consume about half of all cigarettes in the U.S. (Grant, Hasin, Chou, Stinson, & Dawson, 2004; Lasser et al., 2000; Mykletun, Overland, Aaro, Liabo, & Stewart, 2008; Ziedonis et al., 2008). The quit rates for persons with mental illness are also lower (Anda et al., 1990; Glasgow, Klesges, Mizes, & Pechacek, 1985; Glassman et al., 1990; Lasser et al., 2000) placing those with psychiatric disorders at increased risk for tobacco-related morbidity and mortality (Goldman, 2000). This is evidenced by the rates of cardiovascular disease, respiratory diseases, and cancer among persons with serious mental illness, which are double that of age-matched controls (Ziedonis et al., 2003).

Aside from the added risk of illness and death in this population, tobacco increases the rate at which many widely used psychiatric medications are metabolized, which often results in higher dosage requirements, increased healthcare costs, and side-

effects (Williams & Ziedonis, 2006; Ziedonis et al., 2003). In fact, tobacco use can result in a 40% reduced serum level for some medications (Williams & Ziedonis, 2006).

Moreover, nicotine evokes dopamine release (satisfaction, pleasure), serotonin release (mood), norepinephrine (attention and response), acetylcholine (activates muscles, causes excitatory actions), vasopressin (vasoconstriction, memory), growth hormone, adrenocorticotrophic hormone (response to biological stress), and beta-endorphin (anxiety and pain perception) (Balfour & Fagerstrom, 1996; Benowitz, 1988); many of which can exacerbate mental illness symptoms (Balfour & Fagerstrom, 1996; Ziedonis et al., 2003).

Research indicates that persons who experience psychological distress may smoke as a means of regulating affect and coping (Pomerleau, Marks, & Pomerleau, 2000; Shiffman et al., 1986). Smoking may be one method to compensate for deficiencies in social and emotional development as well as a way to self-medicate biological dysregulations produced by abuse or neglect (Balfour & Fagerstrom, 1996; Glassman et al., 1990; Lerman et al., 1996; Penny & Robinson, 1986; Repetti et al., 2002). Studies of adolescent populations have found that stress is associated with smoking (Carmody, 1992; Castro, Maddahian, Newcomb, & Bentler, 1987; Patton et al., 1996; Sussman et al., 1993; Whalen, Jamner, Henker, & Delfino, 2001; Wills et al., 2002) and may, in fact, be predictive of its onset and escalation (Dugan, Lloyd, & Lucas, 1999; Kandel, Davies, Karus, & Yamaguchi, 1986; Seltzer & Oechsli, 1985; Sussman & Dent, 2000; Wills, 1986).

Smoking may be viewed as a viable coping option because of its perceived anxiolytic and sedative properties – for example, its ability to modify mood, manage

dysphoria, regulate negative affect, control situational anxiety and improve concentration (Escobedo et al., 1998; Kassel et al., 2003; Koval et al., 2000; Mermelstein, 1999; West, 1993). Evidence links smoking to enhanced serotonergic (Balfour & Fagerstrom, 1996; Repetti et al., 2002; Ribeiro, Bettiker, Bogdanov, & Wurtman, 1993) and dopaminergic activity (Anda et al., 2006; Volkow, Fowler, & Wang, 2003) and has been shown to regulate negative mood states (Jamner, Shapiro, & Jarvik, 1999). For example, studies have shown that nicotine reduces anger in both smokers and nonsmokers with high-hostility (Jamner et al., 1999; Whalen et al., 2001) and depressive symptoms in both nonsmokers and smokers with depression (Covey, Glassman, & Stetner, 1997; Covey & Tam, 1990; Glassman, Covey, Stetner, & Rivelli, 2001).

Other possible pathways

While most psychiatric disorders found to be associated with smoking are reported to occur prior to smoking initiation, other pathways have been suggested (Costello, Erkanli, Federman, & Angold, 1999; Moolchan, Ernst, & Henningfield, 2000). A brief review of some additional hypotheses is found below.

Depression. Some research suggests that smoking may actually increase susceptibility to depression (Choi et al., 1997; Goodman & Capitman, 2000; Klungsoyr, Nygard, Sorensen, & Sandanger, 2006; Martini, Wagner, & Anthony, 2002; Munafo et al., 2008; Pasco et al., 2008; Steuber & Danner, 2006) due to its the potential deleterious effects on neurochemical pathways (Munafo et al., 2008; Pomerleau & Pomerleau, 1984). Research also suggests that smoking and depression may not have a causal relationship but, in fact, result from common environmental (e.g., alcohol use, parental or peer

smoking) or genetic factors (Kendler et al., 1993). Several researchers have also hypothesized that there is a bi-directional relationship between mental illness and smoking (Chaiton et al., 2009; Kendler et al., 1993; Paperwalla et al., 2004).

Anxiety. While not true of all anxiety disorders, some research suggests that smoking may increase an individual's risk of developing panic attacks and panic disorder (Johnson et al., 2000; McGee et al. 1998; Pohl et al., 1992). In fact, in a study conducted by Breslau et al. (2004), there was a significant association between preexisting daily smoking and the onset of panic disorder and agoraphobia after controlling for sociodemographic characteristics and preexisting psychiatric disorders.

Methods

Data for these analyses came from the Adverse Childhood Experiences (ACE) Study, one of the largest investigations ever conducted on the links between childhood maltreatment and later-life health and well-being. The ACE study is a cross-sectional study that collects retrospective data on a large variety of ACEs and other childhood experiences. The questions that comprised these ACEs were developed from well-established scales such as the Conflicts Tactics Scale (CTS) and the Childhood Trauma Questionnaire (CTQ); the smoking question was adopted from several national surveys including the Behavioral Risk Factor Surveys (Siegel et al., 1993) and the Third National Health and Nutrition Examination Survey (Crespo et al., 1996); and psychological distress was assessed using the SF-36 Mental Component Summary Score (Ware, Kosinski et al., 1994). Data from Wave II of the study were utilized because it contained information on psychological distress.

The statistical method employed in this study is referred to as mediation modeling. Its purpose is to attempt to identify and explain the relationship between an independent and dependent variable based on the inclusion of an explanatory variable. In other words, rather than suggesting that there is a direct causal relationship between an independent and dependent variable, it is hypothesized that the independent variable causes the mediation variable and the mediating variable, in turn, causes the dependent variable. More specifically, rather than suggesting that ACEs directly cause smoking, this study hypothesizes that ACEs cause psychological distress which subsequently causes smoking. This type of analysis assumes that the independent, dependent, and mediation variables are causally related; a major weakness of cross-sectional data. While the nature of the questions in the ACE Study indicates that ACEs come before adult smoking, the relationships between ACEs and psychological distress, and psychological distress and smoking are less clear and, in this study, are supported solely by the literature. Despite this limitation, several other studies have used ACE Study data to examine potential variables that mediate the relationship between ACEs and another variable of interest [e.g., mediators between ACEs and liver disease (Dong, Dube et al., 2003) and ACEs and prescription drug use (Anda, Brown, Felitti, Dube, & Giles, (2008))]. Longitudinal life-course data would better clarify the temporal relationship among the variables of interest as well as minimize recall bias, however this type of data collection is time and cost prohibitive.

Summary

The current review explored research in the areas of ACEs, psychological distress, and smoking. According to developmental psychopathology, there are important developmental stages, particularly during childhood, that build on one another in a hierarchical fashion and are greatly impacted by the internal and external environment. Age- and stage-salient issues include affect regulation, development of attachment, development of the self system, and development of peer relationships. Failure to grasp one or more of these concepts often leads to psychological distress. During this time, the brain is also developing. Chronic stress during important stages of development can modify neurological pathways in the brain, many of which are responsible for stress management and coping such as the hippocampus, amygdala, and prefrontal cortex. This creates additional deficits in emotional, physical, social, and behavioral functioning, further increasing the risk of psychological distress. Persons who experience psychological distress often develop ineffective coping strategies and may rely on self-medicating irresolvable feelings and emotions. Given its anxiolytic and sedative properties, nicotine may be seen as a viable coping option as has been consistently demonstrated by the high prevalence of smoking among persons with mental illness.

This dissertation hypothesizes that the relationship between ACEs and smoking may be mediated by psychological distress. The design for this study was chosen based upon a careful review of existing behavioral and psychological literature in the areas of ACEs, psychological distress, and smoking.

Chapter 3 includes a description of the sample, data collection, measures, and analysis of the data. The chapter will also provide a detailed discussion of the SF-36 scale (Ware, Kosinski et al., 1994; Ware et al., 1993) as well as a description of why linear and logistic regression and mediation models are appropriate for this study.

Chapter 3: Research Methods

Introduction

Chapter 3 includes a description of the sample, data collection, measures, and analysis of the data. The chapter also provides a detailed discussion of the SF-36 scale (Ware, Kosinski et al., 1994; Ware et al., 1993) as well as a description of why linear and logistic regression and mediation models are appropriate for this study.

Purpose of the Study

The purpose of this study was to quantitatively examine whether psychological distress mediated the relationship between ACEs and adult smoking. This study also determined if psychological distress played a different role in the relationship between ACEs and smoking by type of ACE, number of ACEs, and gender.

Research Design and Approach

This study sought to better understand the relationships between ACEs, psychological distress, as assessed by the SF-36 Mental Component Summary Score (Ware, Kosinski et al., 1994), and adult smoking. The study used logistic and linear regression to investigate the relationship between (a) ACEs and psychological distress, (b) psychological distress and smoking, and (c) ACEs and smoking in order to assure that a mediation model was appropriate. When appropriate, mediation techniques were implemented and Sobel tests were conducted to determine if psychological distress significantly mediated the relationship between ACE(s) and smoking (Sobel, 1982).

Mediation models were first introduced by Baron and Kenny in 1986. The purpose of this technique is to attempt to identify and explain the relationship between an independent and dependent variable based on the inclusion of an explanatory variable. In this study, the independent variable was ACEs (individual and cumulative ACE score), the dependent variable was adult smoking, and the explanatory variable was psychological distress (Figure 2)

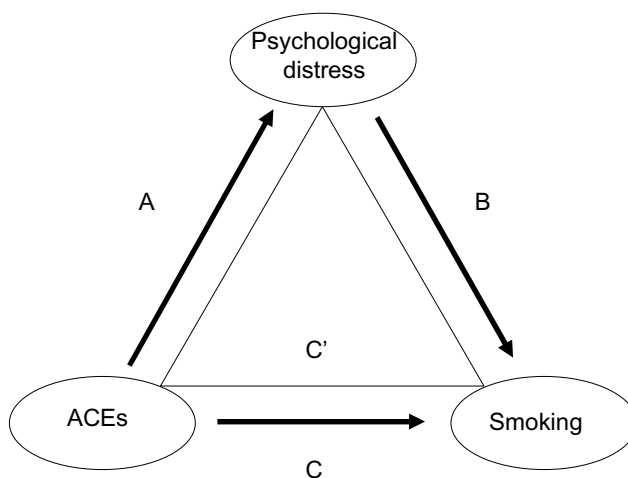


Figure 2. Generic mediation model being tested.
 C = direct pathway from ACE(s) to smoking.
 C' = indirect or mediating pathway from ACE(s) to smoking through psychological distress.

Several criteria must be satisfied in order for mediation analysis to be valid. First, the independent variable (ACEs) must be significantly associated with the mediating variable (psychological distress); the mediating variable (psychological distress) must be significantly associated with the dependent variable (smoking); and the independent variable (ACEs) must be significantly associated with the dependent variable (smoking) (MacKinnon, 2008). Second, the independent variable (ACEs) must be known to cause

the mediation variable (psychological distress), which in turn causes the dependent variable (smoking) (MacKinnon, 2008). Finally, the sample size must be large enough to assume that the data are normally distributed.

Logistic models that include both psychological distress and ACEs (individual or cumulative score) as independent variables and smoking as the dependent variable treat psychological distress as a potential mediating variable (Baron & Kenny, 1986; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). The Sobel test was used to determine whether the indirect effect of the independent variable (ACEs) on the dependent variable (smoking) through the mediator (psychological distress) was significant (Sobel, 1982). Given that the dependent variable (smoking) and the independent variable (ACEs) are dichotomous, and the mediating variable (psychological distress) is continuous, the coefficients in the mediation analyses were on two different scales. In order to make the coefficients compatible, techniques developed by MacKinnon and Dwyer (1993) were utilized to calculate the Sobel statistic as well as the percentage of the total effect that was mediated (Table 1).

Table 1

*Sobel Test (Mediation with a Dichotomous Outcome and a Continuous Mediator)*Variables

X = causal variable
M = mediating variable
Y = outcome

a = path from X to M
b = path from M to Y (controlling for X)
c = direct path from X to Y
c' = path from X to Y (controlling for M)

Mediation is calculated using three equations

$Y' = cX + E1$ (effect of X on Y ignoring M)
 $M' = aX + E2$ (effect of X on M)
 $Y'' = bM + c'X + E3$ (effect of both X and M on Y)

Formula for making coefficients comparable across equations

comp a = $a * SD(X) / SD(M')$
comp b = $b * SD(M) / SD(Y'')$
comp c = $c * SD(X) / SD(Y')$
comp c' = $c' * SD(X) / SD(Y'')$

Note: SD = standard deviation

where

$Var(Y') = c^2 * Var(X) + \pi^2 / 3$
 $Var(M') = a^2 * Var(X) + \pi^2 / 3$
 $Var(Y'') = c'^2 * Var(X) + b^2 * Var(M) + 2 * b * c' * Cov(X,M) + \pi^2 / 3$

Notes: Var = variance

SD = sqrt(variance)

$\pi^2 / 3$ is the variance of the standard logistic distribution

Table - (Continued)

$$\begin{aligned} \text{SE}(\text{comp a}) &= \text{SE}(a) * \text{SD}(X) / \text{SD}(M') \\ \text{SE}(\text{comp b}) &= \text{SE}(b) * \text{SD}(M) / \text{SD}(Y'') \\ \text{SE}(\text{comp c}) &= \text{SE}(c) * \text{SD}(X) / \text{SD}(Y') \\ \text{SE}(\text{comp c}') &= \text{SE}(c') * \text{SD}(X) / \text{SD}(Y'') \end{aligned}$$

Sobel test

$$\begin{aligned} \text{Sobnum} &= \text{comp a} + \text{comp b} \\ \text{Sobden} &= \sqrt{(\text{comp b}^2 * \text{SE}(\text{comp a})^2 + \text{comp a}^2 * \text{SE}(\text{comp b})^2)} \end{aligned}$$

$$\text{Sobel} = \text{sobnum} / \text{sobden}$$

% mediated

$$\begin{aligned} \text{New a} &= a \\ \text{New b} &= b / \text{SD}(Y'') \\ \text{New c} &= c / \text{SD}(Y') \\ \text{New c}' &= c' / \text{SD}(Y'') \end{aligned}$$

$$\text{Permed} = (\text{new a} * \text{new b}) / (\text{new a} * \text{new b} + \text{new c}')$$

Each model was run unadjusted and then adjusted by age group (18-34, 35-54, 55-74, 75+), race (White, Black, Asian, Native American, other), education (no high school diploma, high school/General Educational Development, some college/technical school, college graduate), parental smoking during childhood (yes/no), and alcohol use in the previous month (yes/no). All covariates were left in each model regardless of their significance. This was imperative given that four models must be run to determine the appropriateness of conducting mediation analysis, three that contain smoking as the dependent variable and one that contains psychological distress as the dependent variable. Each of the models needed to be comparable in order to accurately conduct the Sobel test

and determine the percent of the relationship between ACE(s) and smoking that was mediated through psychological distress.

Setting and Sample

Data for this study were drawn from the Adverse Childhood Experiences Study, Wave I, one of the largest studies of ACEs in a community sample (Edwards, Anda, Felitti, & Dube, 2004). The purpose of the original study was to examine the relationship between multiple categories of childhood trauma (ACEs) and health and behavioral outcomes later in life (Figure 3).

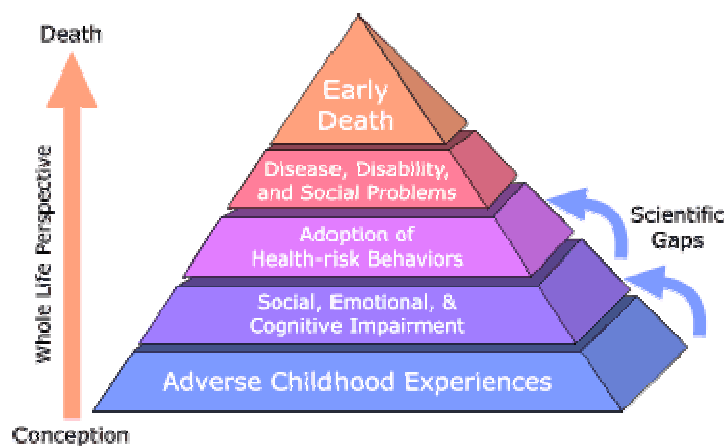


Figure 3. Adverse Childhood Experiences Study Pyramid. Department of Health and Human Services. Centers for Disease Control and Prevention. Retrieved May 17, 2010 from <http://www.cdc.gov/nccdphp/ace/pyramid.htm>. Reprinted with permission.

The ACE Study was made possible by a collaborative agreement between the CDC and Kaiser Permanente's Health Appraisal Clinic (HAC) in San Diego, California. In general, persons who are enrolled in the Kaiser Permanente Health Maintenance Organization (HMO) in San Diego are older and more educated than the general

population (Dong, Anda et al., 2003). According to Felitti et al. (1998), among those continuously enrolled in the HMO between 1992 and 1995, 81% of persons 25 years and older had been evaluated in the HAC. The ACE Study protocol was approved by the Institutional Review Boards (IRB) of the Southern California Permanente Medical Group (Kaiser Permanente IRB protocol #1790), the Office of Protection from Research Risks, National Institutes of Health (T-S44-10/10), and Centers for Disease Control and Prevention (CDC IRB #2825).

The data for the ACE Study were collected in two waves. During each Wave, adult members of the Kaiser Permanente Medical Care Program in San Diego, California were offered a free comprehensive medical examination through the HAC (Felitti et al., 1998). Wave I was collected between August and November of 1995 and between January and March of 1996. Data for Wave II were collected between April and October of 1997. This research utilized data from Wave II because Wave I data did not contain information on psychological distress. All 13,330 Kaiser Health Plan members who completed standardized medical evaluations at the HAC between April and October of 1997 were eligible to participate in the ACE Study. ACE questionnaires were completed for 8,667 respondents. Respondents were excluded from analysis due to missing data on sociodemographic characteristics (race and education ($n = 38$), MCS score ($n = 1,241$), smoking status ($n = 96$), and drinking status ($n = 81$)) leaving 7,211 respondents (54% of the original population) available for analysis.

Each Kaiser member attending the San Diego HAC completed a standardized medical questionnaire (Health Appraisal Questionnaire [HAQ]) prior to their

appointment date. When they arrived at their appointment, they turned in the HAQ and filled out the SF-36 questionnaire, which was used as an assessment of functional health and well-being (Ware, Kosinski et al., 1994; Ware et al., 1993). During their appointment they received a standardized, biopsychosocial medical examination that included lab tests, audiometry, chest X-ray, electrocardiogram, and mammogram. This information was collected to garner additional information about the health of the individual and did not constitute an intervention. After the physical exam, patients were mailed a study questionnaire that asked questions about health behaviors and adverse childhood experiences (Family Health History [FHH]). Participation was voluntary and the patients were assured that the FHH would not become part of their medical record.

The FHH is a 168-item questionnaire designed to capture a broad range of childhood exposures and current health behaviors. Questions for this survey were extracted from existing surveys (Table 2). Questions from the Conflicts Tactics Scale (CTS) were used to define psychological and physical abuse during childhood and to define violence against the respondent's mother (Straus & Gelles, 1990). Four questions from the Wyatt Sexual History Questionnaire (WSHQ) (Wyatt, 1985) were used to define sexual abuse during childhood. Questions about exposure to alcohol or drug abuse during childhood were taken from the 1988 National Health Interview Survey (Centers for Disease Control and Prevention, n.d.) and questions about health-related behaviors and health problems were taken from the Behavioral Risk Factor Surveys (Siegel et al., 1993) and the Third National Health and Nutrition Examination Survey (Crespo et al., 1996). Finally, physical and emotional neglect were assessed using questions from the

Childhood Trauma Questionnaire (CTQ) short form (Bernstein et al., 2003). In a recent study conducted by Bernstein et al. (2003) of 286 drug and alcohol dependent adults, the internal consistency (Cronbach's alpha) for emotional and physical neglect was .92 and .79, respectively, and the test-retest reliability was .83 and .80 respectively. Only portions of the CTS and WSHQ questionnaires were used in this study and validity and reliability studies specifically using these questions could not be found. Moreover, validity and reliability studies regarding the questions about exposure to alcohol and drugs during childhood were not found.

The following ACEs were derived from responses to the questionnaire: (a) verbal abuse, (b) physical abuse, (c) sexual abuse, (d) emotional neglect, (e) physical neglect, (f) violence against mother, (g) alcoholic or drug-abusing family member, (h) mentally ill household member, (i) parents separated or divorced, and (j) incarcerated household member. The FHH also contains the smoking question that was used in this study: "Do you smoke cigarettes now?" Notably, a similar question is found in the Behavioral Risk Factor Surveillance System and has been shown to have high reliability and validity when compared to studies with the same or similar questions (Nelson, Holtzman, Bolen, Stanwyck, & Mack, 2001; Nelson, Powell-Griner, Town, & Kovar, 2003).

Instrumentation

Types of Abuse

Verbal abuse. Verbal abuse was determined from answers to the following two questions from the Conflict Tactics Scale [CTS]; (Straus & Gelles, 1990): (a) "How often did a parent, stepparent, or adult living in your home swear at you, insult you or put you

down?"; and (b) "How often did a parent, stepparent, or adult living in your home threaten to hit you or throw something at you, but did not do it?" Potential responses included "never," "once or twice," "sometimes," "often," and "very often." Persons who responded "often" or "very often" to either item were considered to have experienced verbal abuse during childhood.

Physical abuse. Physical abuse was determined from answers to the following two questions from the CTS (Straus & Gelles, 1990): "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: (a) push, grab, slap, or throw something at you?; or (b) hit you so hard that you had marks or were injured?" Potential responses included "never," "once or twice," "sometimes," "often," and "very often." Persons who responded "sometimes," "often," or "very often" to the first question or responded at least "once or twice" to the second question were considered to have been physically abused during childhood.

Sexual abuse. Sexual abuse was determined from four questions (Wyatt, 1985): "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 five 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: (a) touch or fondle your body in a sexual way?, (b) have you touch their body in a sexual way?, (c) attempt to have any type of sexual intercourse with you (oral, anal, or vaginal), or (d) actually have any type of sexual intercourse with you (oral, anal, or vaginal)?" Persons who

responded “yes” to any one of the four questions were considered to have experienced sexual abuse during childhood.

Types of Neglect

Emotional neglect. Emotional neglect was determined from five CTQ questions (Bernstein et al., 1994) with possible responses “never true,” “rarely true,” “sometimes true,” “often true,” and “very often true.” The responses were scored on a Likert scale ranging from 1 to 5, respectively. The following questions were reverse coded and the five responses added: (a) “There is someone in my family who helped me feel important or special,” (b) “I felt loved,” (c) “People in my family looked out for each other,” (d) “People in my family felt close to each other,” and (e) “My family was a source of strength and support.” A score of 15 and higher (moderate to extreme on the CTQ clinical scale) signified childhood emotional neglect (Bernstein et al., 1994).

Physical neglect. Physical neglect was determined from five CTQ questions (Bernstein et al., 1994) with possible responses “never true,” “rarely true,” “sometimes true,” “often true,” and “very often true.” The responses were scored on a Likert scale ranging from 1 to 5, respectively. Items two and five were reverse coded and the 5 responses were added: (a) “you did not get enough to eat,” (b) “you knew there was someone to take care of you and protect you,” (c) “your parents were too drunk or high to take care of the family,” (d) “you had to wear dirty clothes,” and (e) “there was someone to take you to the doctor if you needed it.” Scores of ten or higher (moderate to extreme on the CTQ clinical scale) signified childhood physical neglect (Bernstein et al., 1994).

Types of Household Dysfunction

Violence against mother. Violence against mother was determined from four questions from the CTS (Straus & Gelles, 1990). “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)?: (a) push, grab, slap, or throw something at her?; (b) kick, bite, hit her with a fist, or hit her with something hard?; (c) repeatedly hit her over at least a few minutes?; and (d) threaten her with a knife or gun to hurt her?” Potential responses include “never,” “once or twice,” “sometimes,” “often,” and “very often.” Persons who responded “sometimes,” “often,” or “very often” to at least one of the first two questions or had any response other than “never” to at least one of questions three or four were considered to have experienced violence against their mother during childhood.

Household substance abuse. Household substance abuse was determined from two questions: (a) “During the first 18 years of life did you live with anyone who was a problem drinker or alcoholic?” (Schoenborn, 1995); and (b) “During the first 18 years of life did you live with anyone who used street drugs?” Persons who responded “yes” to either question were considered to have experienced substance abuse in the household during childhood.

Mental illness in the household. Mental illness in the household was assessed using two questions: “During the first 18 years of life: (a) was anyone in household depressed or mentally ill? or b) did anyone in the household attempt or commit suicide?”

Persons who responded “yes” to either question were considered to have experienced mental illness in the household during childhood.

Parental separation or divorce. Parental separation or divorced was assessed by the question: “Were your parents ever separated or divorced?” Persons who responded “yes” to the question were considered to have experienced parental separation or divorce during childhood.

Incarcerated household member. Incarcerated household member was assessed using the question: “During the first 18 years of life, did anyone in your household go to prison?” Persons who responded “yes” were considered to have had an incarcerated household member during childhood.

ACE score

ACE score is a measure of cumulative exposure to abuse, neglect, and household dysfunction (Anda et al., 1999; Anda et al., 2006; Dong et al., 2004; Felitti et al., 1998). Exposure to any ACE counts as one point. The points were summed for a total score between 0 and 10 points. The ACE score indicates, in summary form, the amount of trauma the child or adolescent experienced across the ten categories. Research has confirmed that the number of respondents with high ACE scores are significantly higher ($p < 0.0001$) than would be expected if the ACEs were independent (Dong et al., 2004).

Table 2

Definitions of Abuse, Neglect, and Household Dysfunction

Type of maltreatment	Definitions
Verbal abuse	Often or very often a parent or other adult in the household swore at you, insulted you, or put you down and/or often or very often acted in a way that made you think that you might be physically hurt.
Physical abuse	Sometimes, often, or very often pushed, grabbed, slapped, or had something thrown at you and/or ever hit so hard that you had marks or were injured.
Sexual abuse	An adult or person at least five years older ever touched or fondled you in a sexual way, and/or had you touch their body in a sexual way, and/or attempted oral, anal, or vaginal intercourse with you and/or actually had oral, anal, or vaginal intercourse with you.
Emotional neglect	<p>Five Childhood Trauma Questionnaire (CTQ) questions (Bernstein et al., 1994) with possible responses “never true,” “rarely true,” “sometimes true,” “often true,” and “very often true.” Responses were scored on a Likert scale ranging from 1 to 5, respectively. The questions were reverse coded and the five responses were added:</p> <ol style="list-style-type: none"> 1. There is someone in my family who helped me feel important or special. 2. I felt loved. 3. People in my family looked out for each other. 4. People in my family felt close to each other. 5. My family was a source of strength and support. <p>A score of 15 and higher (moderate to extreme on the CTQ clinical scale) signify childhood emotional neglect (Bernstein et al., 1994).</p>

Table - (Continued)

Physical neglect	<p>Five Childhood Trauma Questionnaire (CTQ) questions (Bernstein et al., 1994) with possible responses “never true,” “rarely true,” “sometimes true,” “often true,” and “very often true.” Responses were scored on a Likert scale ranging from 1 to 5, respectively. Items two and five were reverse coded and the five responses were added:</p> <ol style="list-style-type: none"> 1. you did not get enough to eat. 2. you knew there was someone to take care of you and protect you. 3. your parents were too drunk or high to take care of the family. 4. you had to wear dirty clothes. 5. there was someone to take you to the doctor if you needed it. <p>Scores of ten or higher (moderate to extreme on the CTQ clinical scale) signify childhood physical neglect (Bernstein et al., 1994).</p>
Violence against Mother	<p>Your mother or stepmother was sometimes, often, or very often pushed, grabbed, slapped, or had something thrown at her and/or ever kicked, bitten, hit with a fist, or hit with something hard, and/or ever repeatedly hit over at least a few minutes and/or ever threatened or hurt by a knife or gun.</p>
Household substance abuse	<p>Lived with anyone who was a problem drinker or alcoholic and/or lived with anyone who used street drugs.</p>
Household mental illness	<p>A household member was depressed or mentally ill and/or a household member attempted suicide.</p>
Parental separation or divorce	<p>Parents were ever separated or divorced.</p>
Incarcerated household member	<p>A household member went to prison.</p>

Note: Events must have occurred before the age of 19 years.

Psychological distress (Table 3)

Psychological distress was assessed using the Mental Component Summary (MCS) score, calculated from the SF-36. The SF-36 is a generic, multipurpose, short-form, health survey with 36 questions and eight subscales (Ware, & Sherbourne, 1992; Ware et al., 1993): (a) physical functioning (10 items) – “a measure of the extent that one can perform normal activities”; b) role physical (4 items) – “a measure of how much work or other daily activities are affected by physical health”; c) bodily pain (2 items) – “a measure of the extent to which somatic symptoms interfere with enjoyment of life”; d) general health (5 items) – “measures a person’s overall assessment of the state of his or her health”; e) vitality (4 items) – “a measure of one’s energy and activity”; f) social functioning (2 items) – “a measure of how much one’s physical or emotional problems interfere with social activities”; g) role emotional (3 items) – “a measure of the extent to which emotional problems interfere with work or other activities”; and h) mental health (5 items) – “a measure of anxiety, depression, loss of behavioral/emotional control, and psychological well-being” (Ware et al., 1993, p. 3:4-3:9) (Table 3).

The eight scales form two distinct higher-ordered clusters, physical and mental health, which account for 80-85% of the variance in the eight scales (Fukuhara, Ware, Kosinski, Wada, & Gandek, 1998; Ware, Gandek, & Group, 1994). All eight scales comprise the MCS score but three scales (mental health, role emotion, and social functioning) correlate most highly and contribute most to the scoring (Ware, Kosinski et al., 1994). Table 4 provides an example of the calculation for one of the eight scales, physical functioning, using SAS software (Ware, Kosinski et al., 1994, p. C6). Table 5 is

the algorithm used to calculate the overall MCS score. As the mean score decreases, psychological distress increases. The general U.S. population mean norm MCS score for males is 50.73 and for females is 49.33 (Ware, Kosinski et al., 1994, pp. 7:2, 8:14).

Table 3

Abbreviated Content for Items in Mental Component Summary (MCS) Score

Scale	Abbreviated Item content	Response set
Physical functioning (PF) Does your health limit you in these activities?	Vigorous activities, such as running, lifting heavy objects, strenuous sports?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
	Moderate activities, such as moving a table, vacuuming, bowling?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
	Lifting or carrying groceries?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
	Climbing several flights of stairs?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
	Climbing one flight of stairs?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
	Bending, kneeling, or stooping?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
	Walking more than one mile?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all

Table - (Continued)

	Walking several blocks?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
	Walking one block?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
	Bathing or dressing?	1=yes, limited a lot 2=yes, limited a little 3=No, not limited at all
Role-physical (RF) During the past 4 years have you had any of the following problems with your work or usual activity as the result of your physical health?	Limited in the kind of work or other activities?	1=yes 2=no
	Cut down on the amount of time spent on work or other activities?	1=yes 2=no
	Accomplish less than would like?	1=yes 2=no
	Difficulty performing work or other activities?	1=yes 2=no
Bodily pain (BP) During the past 4 weeks	How much bodily pain have you had during the past 4 weeks?	1=none 2=very mild 3=mild 4=moderate 5=severe 6=very severe

Table – (Continued)

	How much did pain interfere with your normal work (including both work outside the home and housework)?	1=not at all 2=a little bit 3=moderately 4=quite a bit 5=extremely
General health (GH)	In general, how would you say your health is?	1=excellent 2=very good 3=good 4=fair 5=poor
	My health is excellent	1=yes 2=no
	I am as healthy as anybody I know	1=definitely true 2=mostly true 3=don't know 4=mostly false 5=definitely false
	I seem to get sick a little easier than other people	1=definitely true 2=mostly true 3=don't know 4=mostly false 5=definitely false
	I expect my health to get worse	1=definitely true 2=mostly true 3=don't know 4=mostly false 5=definitely false
Vitality (VT) During the past 4 weeks, did you	feel full of pep?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time

Table - (Continued)

	have a lot of energy?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time
	feel worn out?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time
	feel tired?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time
Social functioning (SF) During the past 4 weeks	To what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbors or groups?	1=not at all 2=a little bit 3=moderately 4=quite a bit 5=extremely
	How much of the time has your physical health or emotional problems interfered with your social activities?	1=all of the time 2=most of the time 3=some of the time 4=a little of the time 5=none of the time

Table - (Continued)

Role emotion (RE) During the past 4 years have you had any of the following problems with your work or other regular activities as the result of any emotional problems?	Cut down on the amount of time spent on work or other activities?	1=yes 2=no
	Accomplish less than would like?	1=yes 2=no
	Didn't do work or other activities as carefully as usual?	1=yes 2=no
Mental health (MH) These questions are about how you feel and how things have been going for you during the past 4 weeks.	Been a very nervous person?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time
	Felt so down in the dumps nothing could cheer you up?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time
	Felt calm and peaceful?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time

Table - (Continued)

	Felt downhearted and blue?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time
	Been a happy person?	1=all of the time 2=most of the time 3=a good bit of the time 4=some of the time 5=a little of the time 6=none of the time
Reported health transition (HT)	Compared to one year ago, how would you rate your health in general now?	1 = much better than one year ago 2=somewhat better than one year ago 3=about the same as one year ago 4=somewhat worse than one year ago 5=much worse than one year ago

Table 4

Algorithm to Calculate Physical Functioning Score.

pf01 = sf3; *vigorous activity;
 pf02 = sf4; *moderate activity;
 pf03 = sf5; *lift or carry;
 pf04 = sf6; *climb several flights;
 pf05 = sf7; *climb on flight;
 pf06 = sf8; *bend;
 pf07 = sf9; *walk more than 1 mile;
 pf08 = sf10; *walk more than 0.5 mile;
 pf09 = sf11; *walk one block;
 pf10 = sf12; *bath or dress;

Table - (Continued)

```

if pf01 = 3 and pf02 = . then pf02 = 3;
if pf01 = . and pf02 = 1 then pf01 = 1;
if pf01 = . and pf03 = 1 then pf01 = 1;

if pf04 = 3 and pf05 = . then pf05 = 3;
if pf04 = . and pf05 = 1 then pf04 = 1;

if pf07 = 3 and pf08 = . then pf08 = 3;
if pf08 = 3 and pf09 = . then pf09 = 3;
if pf07 = 3 and pf09 = . then pf09 = 3;
if pf07 = . and pf08 = 1 then pf07 = 1;
if pf08 = . and pf09 = 1 then pf08 = 1;
if pf07 = . and pf09 = 1 then pf07 = 1;

array sfvart(10) pf01 - pf10;

do i=1 to 10;
if sfvart{i} =4 or sfvart{i} =9 then sfvart{i} =.;
end;

ARRAY PFI(10) PF01 - PF10 ;
DO I = 1 TO 10 ;
IF PFI(I) < 1 OR PFI(I) > 3 THEN PFI(I) = . ;
END ;
PFNUM = N(OF PF01 - PF10) ;
PFMEAN = MEAN(OF PF01-PF10) ;
IF PFNUM GE 5 THEN
DO I = 1 TO 10 ;
IF PFI(I) = . THEN PFI(I) = PFMEAN ;
END ;
IF PFNUM GE 5 THEN RAWPF = SUM(OF PF01-PF10) ;
PF = ((RAWPF - 10)/(30 - 10)) * 100 ;

```

Table 5

Algorithm to Calculate Mental Component Summary (MCS) Score

$$\begin{aligned} \text{PF_ZD} &= (\text{PF} - 84.52404) / 22.89490; \\ \text{RP_ZD} &= (\text{RP} - 81.19907) / 33.79729; \\ \text{BP_ZD} &= (\text{BP} - 75.49196) / 23.55879; \\ \text{GH_ZD} &= (\text{GH} - 72.21316) / 20.16964; \\ \text{VT_ZD} &= (\text{VT} - 61.05453) / 20.86942; \\ \text{SF_ZD} &= (\text{SF} - 83.59753) / 22.37642; \\ \text{RE_ZD} &= (\text{RE} - 81.29467) / 33.02717; \\ \text{MH_ZD} &= (\text{MH} - 74.84212) / 18.01189; \end{aligned}$$

Raw MCS score

$$\text{mrawd} = (\text{PF_ZD} * -0.22999) + (\text{RP_ZD} * -0.12329) + (\text{BP_ZD} * -0.09731) + (\text{GH_ZD} * -0.01571) + (\text{VT_ZD} * 0.23534) + (\text{SF_ZD} * 0.26876) + (\text{RE_ZD} * 0.43407) + (\text{MH_ZD} * 0.48581);$$

Standardized MCS score

$$\text{MCS} = (\text{mrawd} * 10) + 50 ;$$

The SF-36 has been shown to be valid in general population surveys in the United States and other countries (Sabbah, Drouby, Sabbah, Retel-Rude, & Mercier, 2003; Wang et al., 2008; Ware, Keller, Gandek, Brazier, & Sullivan, 1995), as well as in young and old adult patients with specific diseases (Friedman, Heisel, & Delavan, 2005; Gandek, Sinclair, Kosinski, & Ware, 2004; Linde, Sorensen, Ostergaard, Horslev-Petersen, & Hetland, 2008; Lotus Shyu, Lu, & Chen, 2009; Sciolla, Patterson, Wetherell, McAdams, & Jeste, 2003; Teul, Baran, & Zbislawski, 2008; Ware et al., 1993). It has also been shown to estimate disease burden for a number of conditions including arthritis, cancer, cardiovascular disease, diabetes, spinal cord injuries, and depression when

compared to general population norms (Failde, Medina, Ramirez, & Arana, 2009; Forchheimer, McAweeney, & Tate, 2004; Huang et al., 2008; Turner-Bowker, Bartley, & Ware, 2002; Veehof, ten Klooster, Taal, van Riel, & van de Laar, 2008).

Extensive psychometric testing of the SF-36 has occurred in the United States (Garratt, Ruta, Abdalla, Buckingham, & Russell, 1993; Jenkinson, Coulter, & Wright, 1993; McHorney, Ware, Lu, & Sherbourne, 1994; Wagner et al., 1995) and other countries (Anagnostopoulos, Niakas, & Pappa, 2005; Augustovski, Lewin, Elorrio, & Rubinstein, 2008; Bullinger, 1995; Demiral et al., 2006; Hoopman, Terwee, Deville, Knol, & Aaronson, 2009; Lam, Tse, Gandek, & Fong, 2005; McCallum, 1995; Qu, Guo, Liu, Zhang, & Sun, 2009; Rampal, Martin, Marquis, Ware, & Bonfils, 1994; Sullivan, Karlsson, & Ware, 1995). The reliability of the eight scales and two summary scales has been estimated using both internal consistency and test-retest methods with the majority of the results for the eight scales exceeding 0.80 (McHorney et al., 1994; Ware et al., 1993) and the reliability estimates for the Physical and Mental Component Summary Scores generally exceeding 0.90 (Ware, Kosinski et al., 1994). These results have been replicated across 24 patient groups with different sociodemographic characteristics and diagnoses (McHorney et al., 1994; Turner-Bowker et al., 2002; Ware, Kosinski et al., 1994; Ware et al., 1993). In addition, the content validity of the SF-36 has been tested against other widely used generic health surveys. Results suggest that the scale contains the most frequently measured health concepts (Ware, Kosinski et al., 1995; Ware et al., 1993). Predictive studies of validity have linked SF-36 scales and summary measures to 180-day survival (Rumsfeld et al., 1999), five year survival (Ware, Gandek et al., 1994),

utilization of health care services (Ware, Gandek et al., 1994), the clinical course of depression (Beusterien, Steinwald, & Ware, 1996; Silveira et al., 2005; Weinstein, Berwick, Goldman, Murphy, & Barsky, 1989; Wells, Burnam, Rogers, Hays, & Camp, 1992), and loss of job within one year (Ware, Gandek et al., 1994). Moreover, clinical studies have shown that three of the scales with the most physical factor content are more responsive to pre and post- knee replacement (Busija, Osborne, Nilsson, Buchbinder, & Roos, 2008; Katz, Larson, Phillips, Fossel, & Liang, 1992), hip replacement (Busija et al., 2008; Kantz, Harris, Levitsky, Ware, & Davies, 1992; Shi, Mau, Chang, Wang, & Chiu, 2009), and heart valve surgery (Phillips & Lansky, 1992; Supino et al., 2009) while the three scales with the most mental factor content are more responsive to changes in depression severity (Beusterien et al., 1996; Ware, Keller et al., 1995) and drug treatment and interpersonal therapy for depression (Coulehan, Schulberg, Block, Madonia, & Rodriguez, 1997).

Covariates of interest

These variables were included in the adjusted linear and logistic models.

Age. 18 to <35 years, 35 to <55 years, 55 to <75 years, 75+ years.

Sex: male, female (total population model only)

Education. No high school diploma, high school diploma/GED, some college or technical school, college graduate

Race. White, Black, Hispanic, Asian, Native American, other

Parental smoking. Did mother or father smoke during your first 18 years? Yes,

No

Drink alcohol in the previous month. During the past month, did the respondent have any beer, wine, wine coolers, cocktails or liquor? Yes, No

Analyses

This study used linear and logistic regression analysis to examine the potential mediating effect of psychological distress on the relationship between ACEs and smoking. With the exception of the SF-36 Mental Component Summary Score (MCS) (Ware, Kosinski et al., 1994), which is a continuous variable, all other variables used in these analyses were categorical and therefore logistic regression analysis was conducted. For analyses where the MCS score (Ware, Kosinski et al., 1994) was the outcome variable of interest, linear regression was employed. Given that the models contain both dichotomous and continuous variables, the coefficients in the mediation analyses were on two different scales. In order to make the coefficients compatible, techniques developed by MacKinnon and Dwyer (1993) were utilized in order to obtain an accurate assessment of the significance of psychological distress in mediating the relationship between ACE(s) and smoking as well as the percent of the relationship mediated by psychological distress. The following research questions and the hypotheses reflect these types of analyses.

Research question 1.

What is the nature of the relationships between ACEs and smoking and ACEs and psychological distress?

Hypothesis 1.

Null Hypothesis (H_{01}): There is not a relationship between ACEs (abuse, neglect, and household dysfunction) and smoking or ACEs and psychological distress (as assessed by the SF-36 Mental Component Summary Scale) among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a1}): Among members of a Kaiser Permanente HMO in San Diego, California, ACEs increase the risk of psychological distress (as assessed using the SF-36 Mental Component Summary score) as well as adult smoking.

Research question 2.

Does the relationship between ACEs and smoking and ACEs and psychological distress vary by type of ACE?

Hypothesis 2.

Null Hypothesis (H_{02}): There is no difference in the effects of different types of ACEs on the subsequent risk of psychological distress (as assessed by the SF-36 Mental Component Summary Scale) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a2}): Different types of ACEs (abuse, neglect, and household dysfunction) have varying affects on the subsequent risk of psychological distress (as assessed using the SF-36 Mental Component Summary

score) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research question 3.

As the cumulative number of ACEs increases does the risk of psychological distress and smoking increase?

Hypothesis 3.

Null Hypothesis (H_{03}): There is not a cumulative effect of multiple ACEs (abuse, neglect, and household dysfunction) on the risk of subsequent psychological distress (as assessed by the SF-36 Mental Component Summary Scale) or smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a3}): As the cumulative number of ACEs (abuse, neglect, and household dysfunction) increases, so does the risk of subsequent psychological distress (as assessed by the SF-36 Mental Component Summary Scale) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research question 4.

Do the relationships between ACEs and psychological distress and ACEs and smoking vary by gender?

Hypothesis 4.

Null Hypothesis (H_{04}): There is not a difference in the relationships between ACEs (abuse, neglect, and household dysfunction) and psychological distress (as

assessed by the SF-36 Mental Component Summary Scale) or ACEs and smoking by gender among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a4}): The relationships between ACEs (abuse, neglect, and household dysfunction) and psychological distress (as assessed by the SF-36 Mental Component Summary Scale), and ACEs and smoking are stronger for female (versus male) members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research question 5.

Does psychological distress mediate the relationship between ACEs and smoking?

Hypothesis 5.

Null Hypothesis (H_{05}): Psychological distress (as assessed by the SF-36 Mental Component Summary Scale), does not mediate the relationship between ACEs (abuse, neglect, and household dysfunction) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

Research Hypothesis (H_{a5}): Psychological distress (as assessed by the SF-36 Mental Component Summary Scale), mediates the relationship between ACEs (abuse, neglect, and household dysfunction) and smoking among members of a Kaiser Permanente Health Maintenance Organization (HMO) in San Diego, California.

With the exception of the Sobel test and the analysis to determine the percent of the total relationship mediated (conducted in SPSS statistics 17.0 and Microsoft Office Excel 2003), all programming and analyses were conducted in SAS 9.2 (SAS Institute Inc., Cary, NC, 2008). The SF-36 individual and Mental Component Summary raw and transformed scores (linear transformation to transform scores to a mean of 50 and standard deviation of 10 using general U.S. population norms) were calculated using methods developed by Ware and colleagues (Ware et al., 1993; Ware, Kosinski et al., 1994). A relationship between ACEs (independent) and smoking (dependent) and psychological distress (independent) and smoking (dependent) were established using logistic regression. Given that the measure of psychological distress is continuous, establishing the relationship between ACEs (independent) and psychological distress (dependent) was conducted using linear regression. When a significant relationship was established for the three models listed above, a mediation logistic regression model (smoking = ACE psychological distress) was run to examine the potential mediating effect of psychological distress on the relationship between ACEs and smoking. These models were run for each individual ACE, total number of ACEs, and gender. Each model was run unadjusted and then adjusted by age group (18-34, 35-54, 55-74, 75+), race (White, Black, Asian, Native American, other), education (no high school diploma, high school/GED, some college/technical school, college graduate), parental smoking during childhood (yes/no), and alcohol use in the previous month (yes/no).

Protection of Patients' Rights

Names, appointment date, date of birth, and Kaiser member record numbers were obtained from electronic databases created by appointment clerks. A study identification number was assigned to each participant and a linkage between the study identification number and the Kaiser member record number was created. This linkage is confidential and available only to the principle investigator at Kaiser Permanente and select Kaiser employees who work under the principle investigator's supervision. Respondents returned the completed form to Kaiser Permanente. The data obtained from the Centers for Disease Control and Prevention (CDC) for this study contains no unique identifiers.

In order to gain access to this data, an Adverse Childhood Experiences (ACE) Study Collaboration Proposal Form was completed describing the specific analyses that would be conducted with the data. In order to secure rights to the data, this form was approved and signed by the Kaiser Permanente and the CDC project officers. All analyses were conducted on the CDC server which contains the state-of-the-art security and back-up systems. After completion of this study, personal access to the data will be terminated.

Chapter 4 quantitatively examines the potential mediating effect of psychological distress on the relationship between ACEs and adult smoking by individual ACE, total ACE score, and gender using Wave II of the Adverse Childhood Experiences Study.

Chapter 4: Results

Introduction

The purpose of the current study was to quantitatively examine whether psychological distress mediated the relationships between ACEs and adult smoking by type of ACE, total number of ACEs, and gender. Five hypotheses were tested using logistic and linear regression techniques and mediation methods.

Sample Demographics

Between April and October of 1997, 8,667 members Kaiser Permanente Health Maintenance Organization in San Diego, California participated in Wave II of the ACE Study. Among these, 7,211 (83.2%) respondents had complete information for the study variables and were included in the analyses - 3,895 females and 3,316 males (Table 6). The mean age of the population was 55.9 years, approximately three-quarters of the sample was White, 8.1% were current smokers (7.7% among women and 8.6% among men), over 75% had at least some college education, and the mean MCS score was slightly higher for men than women (53.2 versus 51.2, respectively, suggesting that women are slightly more likely than men to have psychological distress).

Table 6

Descriptive Characteristics of the Study Population

Characteristics	Total (<i>N</i> = 7,211) %	Females (<i>n</i> = 3,895) %	Males (<i>n</i> = 3,316) %
Age group (years)			
18-34	9.4	11.3	7.1
35-54	37.8	38.9	36.6
55-74	42.2	40.1	44.7
75+	10.6	9.7	11.7
Mean age (SD)	55.9 (15.0)	54.8 (15.4)	57.3 (14.4)
Race			
White	74.8	73.8	75.9
Black	4.0	4.1	4.0
Hispanic	10.7	11.0	10.3
Asian	8.0	9.1	6.8
Native American	0.4	0.3	0.4
Other	2.2	1.8	2.6
Education			
No high school diploma	7.2	7.9	6.5
High school/GED	14.6	16.8	12.1
Some college/technical school	40.9	42.8	38.6
College graduate	37.3	32.6	42.8
History of parental smoking	72.5	71.7	73.4
Smoking status			
Current smoker	8.1	7.7	8.6
Former smoker	41.5	33.3	51.2
Never smoker	50.4	59.1	40.2
Mean MCS score (SD)	52.1 (9.0)	51.2 (9.50)	53.2 (8.2)

Sample ACEs

Females were more likely than males to report emotional abuse and emotional neglect (11.7% versus 8.2%, and 16.4% versus 12.2%, respectively) and sexual abuse (24.2% versus 16.7%, respectively), while males were more likely to report physical abuse and physical neglect (28.6% versus 24.6%, and 10.5% versus 8.6%, respectively)

(Table 7). In general, women reported more household dysfunction than men, particularly with regard to mental illness in the household (25.0% versus 14.6%, respectively) and household substance abuse (29.9% versus 25.5%, respectively). Females were also more likely than males to report four or more ACEs (18.7% versus 13.0%, respectively).

Table 7

ACE Characteristics of Study Sample

ACE	Total (<i>N</i> = 7,211) %	Females (<i>n</i> = 3,895) %	Males (<i>n</i> = 3,316) %
Abuse			
Emotional	10.1	11.7	8.2
Physical	26.4	24.6	28.6
Sexual	20.8	24.2	16.7
Neglect			
Emotional	14.4	16.4	12.2
Physical	9.5	8.6	10.5
Household dysfunction			
Violence against mother	12.9	13.6	12.1
Parental separation or divorce	24.0	25.3	22.4
Mental illness in household	20.2	25.0	14.6
Household substance abuse	27.9	29.9	25.5
Incarcerated household member	6.0	6.9	4.8
Total number of ACES			
0	33.3	32.1	34.7
1	25.4	24.1	26.9
2	15.4	14.7	16.1
3	9.9	10.4	9.3
4+	16.1	18.7	13.0

Hypotheses 1 and 2

The first hypothesis predicted that persons who reported ACEs would be more likely than those without ACEs to smoke and to report an increased level of psychological distress. The second hypothesis predicted that the relationships between

ACEs and smoking and ACEs and psychological distress would vary by type of ACE. Logistic regression was used to examine the association between ACEs and smoking (Figure 2, line C, p. 45) and linear regression was performed to examine the relationship between ACEs and psychological distress (Figure 2, line A, p. 45).

ACEs and Smoking

In the unadjusted models, with the exception of sexual abuse, mental illness in the household, and persons who experienced one ACE (versus no ACEs), persons with ACEs were more likely to smoke than persons without ACEs (Table 8). After adjusting for sociodemographic characteristics (age group, race, education) and other covariates (parental smoking during childhood, alcohol consumption in the previous month), persons who experienced emotional and physical abuse, parental separation or divorce, or who had an incarcerated household member were significantly more likely to smoke than those who did not experience these ACEs (Table 8). These results suggest that many of the ACEs are associated with subsequent smoking but the magnitude of the association between ACEs and smoking differs by type of ACE.

ACEs and Psychological Distress

As described in Chapter 3, the lower the MCS score, the greater the level of psychological distress. As can be seen from the parameter estimates and p-values in Table 8, for each ACE in the unadjusted and adjusted models, persons with increased psychological distress were more likely to smoke.

Hypothesis 3

The third hypothesis predicted that as the cumulative number of ACEs (total ACE score) increased, the relationship between ACEs and smoking and ACEs and psychological distress would become stronger.

Cumulative Number of ACEs and Smoking

In the unadjusted model there appeared to be a dose-response relationship between the number of ACEs and the risk of smoking. Notably, however, after adjusting for sociodemographic characteristics (age group, race, education) and other covariates (parental smoking during childhood, alcohol consumption in the previous month) the association was attenuated and no longer significant (Table 8). This suggests that the relationship between number of ACEs and smoking is actually explained by the other variables in the model; in this case, education level and age (as education level and age decreased, the risk of smoking increased).

Cumulative Number of ACEs and Psychological Distress

As noted above, as the MCS score decreases, the level of psychological distress increases. As can be seen from the parameter estimates and p-values in Table 8, in both the unadjusted and adjusted models, as the cumulative number of ACEs increased, the risk of psychological distress significantly increased.

Table 8

Unadjusted and Adjusted Relationships Between ACEs and Smoking and ACEs and Psychological Distress-Total Population

ACE	Unadjusted			Adjusted		
	Smoking=ACE Odds ratio (95% CI)	PD=ACE Parameter estimate	Pr> t	Smoking=ACE Odds ratio (95% CI)	PD=ACE Parameter estimate	Pr> t
Abuse						
Emotional						
Yes	1.61 (1.26-2.05)*	-4.387	<0.0001	1.35 (1.05-1.73)*	-3.812	<0.0001
No	Referent			Referent		
Physical						
Yes	1.61 (1.35-1.93)*	-2.568	<0.0001	1.34 (1.11-1.61)*	-2.355	<0.0001
No	Referent			Referent		
Sexual						
Yes	1.20 (0.98-1.47)	-1.832	<0.0001	1.10 (0.89-1.35)	-1.455	<0.0001
No	Referent			Referent		
Neglect						
Emotional						
Yes	1.46 (1.17-1.81)*	-4.014	<0.0001	1.22 (0.97-1.52)	-3.561	<0.0001
No	Referent			Referent		
Physical						
Yes	1.46 (1.13-1.89)*	-2.419	<0.0001	1.28 (0.98-1.67)	-2.335	<0.0001
No	Referent			Referent		
Household dysfunction						
Violence against mother						
Yes	1.56 (1.25-1.95)*	-2.259	<0.0001	1.19 (0.94-1.49)	-1.724	<0.0001
No	Referent			Referent		

Table - (Continued)

Parental separation or divorce						
Yes	1.59 (1.32-1.90)*	-1.668	<0.0001	1.27 (1.05-1.53)*	-1.186	<0.0001
No	Referent			Referent		
Mental illness in the household						
Yes	1.17 (0.95-1.43)	-3.967	<0.0001	1.07 (0.87-1.32)	-3.468	<0.0001
No	Referent			Referent		
Household substance abuse						
Yes	1.40 (1.17-1.68)*	-2.314	<0.0001	1.03 (0.85-1.25)	-1.729	<0.0001
No	Referent			Referent		
Incarcerated household member						
Yes	2.16 (1.63-2.85)*	-1.067	0.0002	1.69 (1.26-2.26)*	-0.928	0.0359
No	Referent			Referent		
Total number of ACEs						
0	Referent	-2.265	<0.0001	Referent	-1.074	<0.0001
1	1.06 (0.83-1.35)			0.94 (0.73-1.21)		
2	1.60 (1.24-2.07)*			1.32 (1.01-1.71)*		
3	1.59 (1.18-2.14)*			1.18 (0.87-1.60)		
4+	1.90 (1.49-2.42)*			1.29 (1.00-1.68)		

Note. Adjusted odds ratios adjusted by age group, race, education, parental smoking during childhood, and alcohol use in past month; PD = psychological distress.

*p < 0.05

Hypothesis 4

The fourth hypothesis predicted that the relationships between ACEs and smoking and ACEs and psychological distress would vary by gender.

ACEs and Smoking

In the unadjusted models, with the exception of mental illness in the household, and persons with one ACE versus no ACEs, women with ACEs were more likely to smoke (Figure 4, Table 9). The profile for men looked markedly different (Figure 4, Table 10). Only physical abuse, emotional neglect, parental separation or divorce, household substance abuse, incarcerated household member, and two or four or more ACEs (versus no ACEs) were associated with smoking.

After adjusting for sociodemographic characteristics (age group, race, education) and other covariates (parental smoking during childhood, alcohol consumption in the previous month), many of the relationships between ACEs (emotional and physical abuse, physical neglect, violence against the mother, parental separation or divorce, and incarcerated household member) and smoking remained significant for women (Table 9). Notably, among men, after adjusting for selected covariates, all the associations between ACEs and smoking were attenuated and no longer significant (Table 10). This indicates that other variables in the model accounted for the majority of the relationship between ACEs and smoking in men. The strongest predictors of smoking in all of these models were educational attainment and age (as education level and age decreased, the risk of smoking increased). Given that there is not an association between ACEs and smoking

among men, mediation modeling is not appropriate (i.e., there is no relationship therefore there is nothing to mediate).

ACEs and Psychological Distress

In the unadjusted models for women, all associations between ACEs and psychological distress were significant (Figure 5, Table 9). With the exception of incarcerated household member, the same pattern was found in the unadjusted models for men (Figure 5, Table 10). After adjusting for selected covariates, the association between incarcerated household member and psychological distress became nonsignificant for women. In addition to incarcerated household member, parental separation or divorce also became non-significant for men after adjusting.

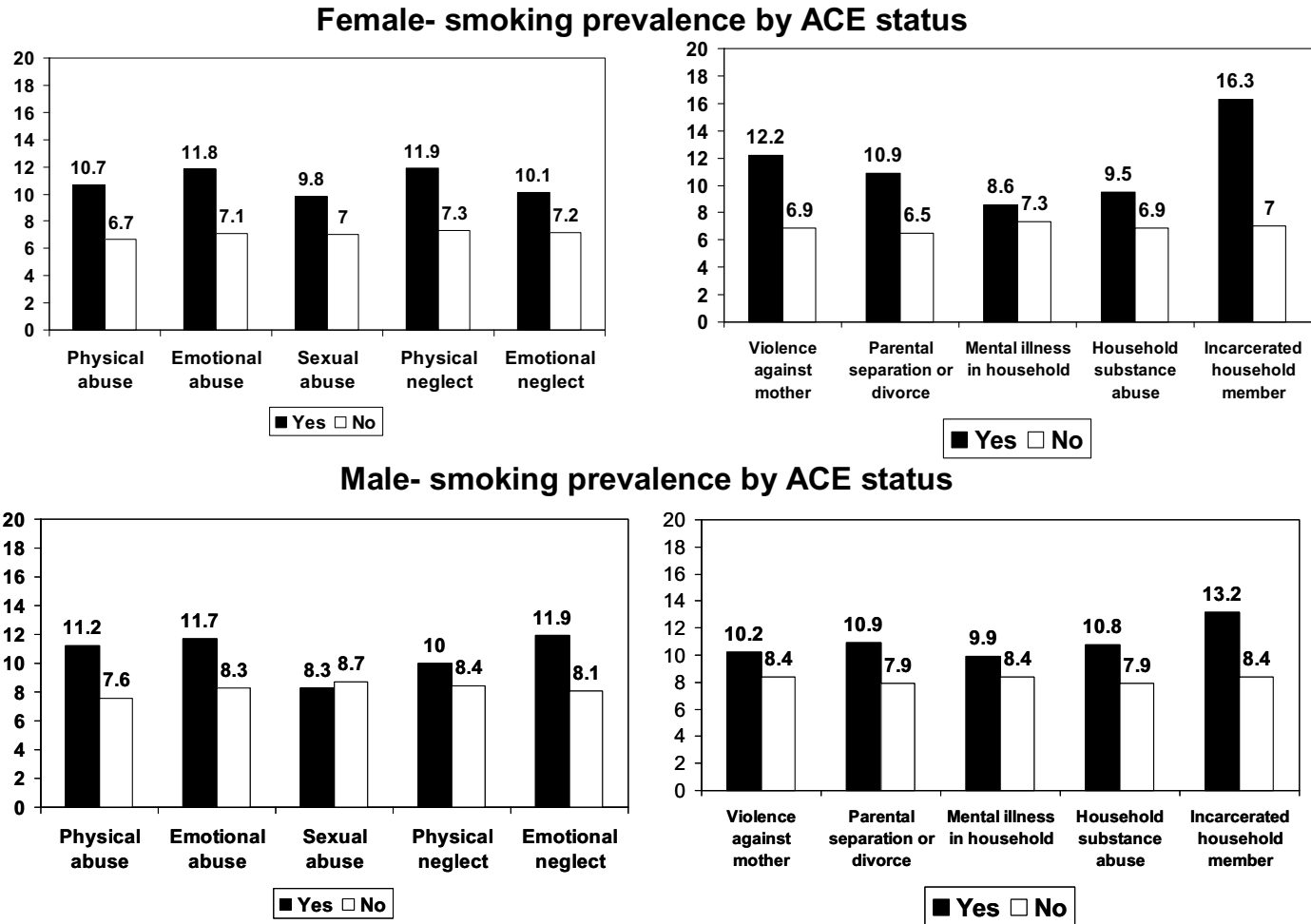


Figure 4. Smoking prevalence by ACE status and gender.

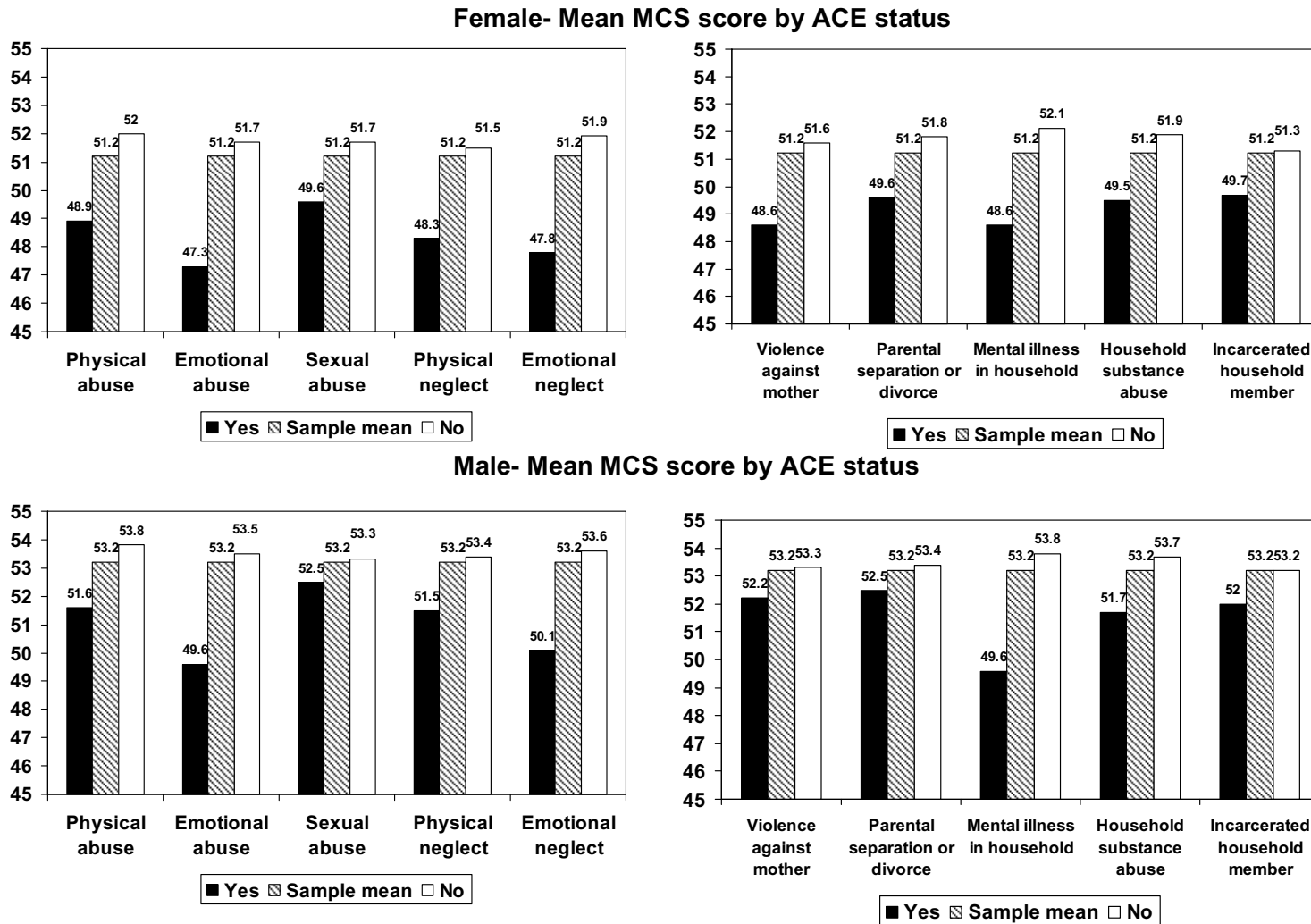


Figure 5. Mean MCS score by ACE status and gender

Table 9

Unadjusted and Adjusted Relationships Between ACEs and Smoking and ACEs and Psychological Distress-Women

ACE	Unadjusted			Adjusted		
	Smoking=ACE	PD=ACE		Smoking=ACE	PD=ACE	
	Odds ratio (95% CI)	Parameter estimate	Pr> t	Odds ratio (95% CI)	Parameter estimate	Pr> t
Abuse						
Emotional						
Yes	1.76 (1.29-2.40)*	-4.388	<0.0001	1.46 (1.05-2.01)*	-3.895	<0.0001
No	Referent			Referent		
Physical						
Yes	1.67 (1.30-2.14)*	-3.051	<0.0001	1.38 (1.07-1.79)*	-2.598	<0.0001
No	Referent			Referent		
Sexual						
Yes	1.44 (1.11-1.86)*	-2.115	<0.0001	1.21 (0.93-1.57)	-1.821	<0.0001
No	Referent			Referent		
Neglect						
Emotional						
Yes	1.44 (1.08-1.93)*	-4.061	<0.0001	1.21 (0.90-1.62)	-3.699	<0.0001
No	Referent			Referent		
Physical						
Yes	1.74 (1.22-2.47)*	-3.185	<0.0001	1.55 (1.08-2.23)*	-2.776	<0.0001
No	Referent			Referent		
Household dysfunction						
Violence against mother						
Yes	1.87 (1.40-2.51)*	-2.981	<0.0001	1.46 (1.08-1.98)*	-2.397	<0.0001
No	Referent			Referent		

Table - (Continued)

Parental separation or divorce						
Yes	1.76 (1.37-2.25)*	-2.173	<0.0001	1.41 (1.09-1.82)*	-1.624	<0.0001
No	Referent			Referent		
Mental illness in the household						
Yes	1.19 (0.92-1.55)	-3.423	<0.0001	1.04 (0.80-1.37)	-3.143	<0.0001
No	Referent			Referent		
Household substance abuse						
Yes	1.41 (1.10-1.81)*	-2.364	<0.0001	1.00 (0.77-1.30)	-1.712	<0.0001
No	Referent			Referent		
Incarcerated household member						
Yes	2.59 (1.83-3.66)*	-1.624	0.0067	2.21 (1.54-3.17)*	-0.925	0.1205
No	Referent			Referent		
Total number of ACEs						
0	Referent	-2.413	<0.0001	Referent	-1.126	<0.0001
1	0.82 (0.56-1.20)			0.71 (0.48-1.04)		
2	1.62 (1.12-2.34)*			1.30 (0.89-1.89)		
3	1.80 (1.21-2.69)*			1.31 (0.86-1.98)		
4+	2.07 (1.49-2.87)*			1.37 (0.97-1.94)		

Note. Adjusted odds ratios adjusted by age group, race, education, parental smoking during childhood, and alcohol use in past month; PD = psychological distress.

* $p < 0.05$

Table 10

Unadjusted and Adjusted Relationships Between ACEs and Smoking and ACEs and Psychological Distress-Men

ACE	Unadjusted			Adjusted		
	Smoking=ACE	PD=ACE		Smoking=ACE	PD=ACE	
	Odds ratio (95% CI)	Parameter estimate	Pr> t	Odds ratio (95% CI)	Parameter estimate	Pr> t
Abuse						
Emotional						
Yes	1.46 (0.99-2.16)	-3.930	<0.0001	1.19 (0.79-1.79)	-3.647	<0.0001
No	Referent			Referent		
Physical						
Yes	1.54 (1.20-1.98)*	-2.273	<0.0001	1.29 (0.99-1.67)	-2.086	<0.0001
No	Referent			Referent		
Sexual						
Yes	0.96 (0.69-1.33)	-0.856	0.0241	0.94 (0.66-1.32)	-0.863	0.0221
No	Referent			Referent		
Neglect						
Emotional						
Yes	1.52 (1.10-2.12)*	-3.561	<0.0001	1.22 (0.87-1.72)	-3.357	<0.0001
No	Referent			Referent		
Physical						
Yes	1.21 (0.84-1.76)	-1.891	<0.0001	1.05 (0.71-1.56)	-1.879	<0.0001
No	Referent			Referent		
Household dysfunction						
Violence against mother						
Yes	1.24 (0.88-1.76)	-1.172	0.0069	0.90 (0.63-1.30)	-0.860	0.0484
No	Referent			Referent		

Table - (Continued)

Parental separation or divorce						
Yes	1.42 (1.09-1.87)*	-0.843	0.0130	1.12 (0.84-1.50)	-0.628	0.0648
No	Referent			Referent		
Mental illness in the household						
Yes	1.20 (0.87-1.67)	-4.220	<0.0001	1.09 (0.78-1.54)	-4.035	<0.0001
No	Referent			Referent		
Household substance abuse						
Yes	1.42 (1.09-1.84)*	-2.015	<0.0001	1.06 (0.80-1.41)	-1.735	<0.0001
No	Referent			Referent		
Incarcerated household member						
Yes	1.67 (1.04-2.68)*	-1.255	0.0583	1.09 (0.66-1.80)	-0.852	0.1994
No	Referent			Referent		
Total number of ACEs						
0	Referent	-1.854	<0.0001	Referent	-1.010	<0.0001
1	1.28 (0.92-1.77)			1.15 (0.82-1.62)		
2	1.58 (1.10-2.26)*			1.31 (0.90-1.90)		
3	1.37 (0.87-2.15)			1.00 (0.62-1.60)		
4+	1.72 (1.18-2.51)*			1.14 (0.76-1.70)		

Note. Adjusted odds ratios adjusted by age group, race, education, parental smoking during childhood, and alcohol use in past month; PD = psychological distress.

*p < 0.05

Hypothesis 5

The final hypothesis predicted that psychological distress would mediate the relationship between ACEs and smoking. Three additional analyses were needed to examine this hypothesis. First, the association between smoking and psychological distress needed to be assessed for women ($p=0.0001$) (Figure 2, line B, p. 45). Given that the association was significant, it was appropriate to conduct mediator modeling. As stated earlier, men were not included in these analyses because ACEs were not associated with smoking in this population. Second, the model containing both ACEs and psychological distress needed to be assessed. In order to determine if psychological distress significantly mediated the relationship between ACE(s) and adult smoking, the Sobel test was used (Sobel, 1982). In addition to the Sobel test, the percent of the relationship between ACEs and smoking that was mediated through psychological distress was also assessed.

After adjusting for sociodemographic characteristics and other potential covariates, among women, psychological distress mediated 9.6% of the relationship between parental separation and divorce and adult smoking, 12.8% of the relationship between violence against the mother and adult smoking, 13.9% of the relationship between physical neglect and adult smoking, 15.4% of the relationship between physical abuse and adult smoking, and 19.3% of the relationship between emotional abuse and adult smoking (Table 11).

Table 11

Adjusted Mediation Statistics-Women

Type of ACE	Sobel test		% of total mediated
	Test statistic (standard error)	p-value ^b	
Abuse			
Emotional	-3.227 (0.019)	0.00125	19.30
Physical	-3.287 (0.018)	0.00101	15.36
Neglect			
Physical	-2.989 (0.015)	0.00280	13.85
Household dysfunction			
Violence against mother	-3.038 (0.016)	0.00239	12.81
Parental separation or divorce	-2.877 (0.014)	0.00402	9.59

Note. Adjusted odds ratios adjusted by age group, race, education, parental smoking during childhood, and alcohol use in past month; P-values drawn from the normal distribution under the assumption of a two-tailed z-test. The hypothesis is that the mediated effect equals zero.

Summary

The statistical analyses in this study generally supported the association between smoking and ACEs and smoking and psychological distress in the unadjusted models; particularly for women. The only exception among women was the association between mental illness in the household and smoking which was not significant. There was also a graded increase in the unadjusted odds of smoking as the number of ACEs increased. Notably, men exhibited a different profile. There was not a significant association between smoking and emotional abuse, sexual abuse, physical neglect, violence against the mother, or mental illness in the household. Additionally, the association between

incarcerated household member and psychological distress was not significant for men. Moreover, unlike women, there was not a graded increase in the unadjusted odds of smoking as the number of ACEs increased; the association only became significant for two ACEs and four or more ACEs (versus no ACEs).

After further adjusting by select sociodemographic characteristics and other potential covariates, the association between smoking and ACEs among women became attenuated and no longer significant for sexual abuse, emotional neglect, household substance abuse, and total number of ACEs. The association between psychological distress and incarcerated household member also became non-significant. Notably, among men, all the associations between ACEs and smoking became non-significant. With the exception of parental separation or divorce, and incarcerated household member, however, the associations between ACEs and psychological distress remained significant.

While the average age of the study population was 55.9 years, ACEs still appeared to play a significant role in adult smoking, particularly among women. Psychological distress significantly mediated the relationship between adult smoking and emotional abuse (19.3%), physical abuse (15.4%), physical neglect (13.9%), violence against the mother (12.8%), and parental separation or divorce (9.6%) among women.

Chapter 5 includes a brief explanation for why the study was conducted, an interpretation of the findings, recommendations for future research, implications for social change, and limitations of the study.

Chapter 5: Discussion

Introduction

The data for these analyses were collected between April and October 1997 on a sample of adult members of the Kaiser Permanente Medical Care Program in San Diego, California (mean age 55.9). The purpose of this study was to evaluate the potential mediating effect of psychological distress on the relationship between ACEs and smoking by type of ACE, total number of ACEs, and gender.

These analyses address three primary weaknesses in the current literature. First, most child abuse and maltreatment studies have failed to account for a broad range of ACEs and the cumulative effect of multiple ACEs. This has the propensity to underestimate the burden of victimization and possibly lead to inaccurate assumptions about the relationships between specific ACEs and negative outcomes (Anda et al., 1999; Dong, Anda et al., 2003; Felitti et al., 1998; Finkelhor et al., 2005). The present study examined a number of types of abuse, neglect, and household dysfunction in addition to the overall impact of cumulative ACE exposure on the relationships between psychological distress and smoking.

Second, while gender differences in smoking characteristics have been noted, many studies failed to consider the implications of these differences on prevention and intervention programs. For example, as compared to men, women are much more likely to smoke as the result of negative affect (Brandon & Baker, 1991; Husky et al., 2008; McKee et al., 2003); they are less likely to be dependent on nicotine (Bjornson et al., 1995; Gritz et al., 1998; Perkins, et a., 2002; Royce et al., 1997; Ward et al., 1997); they

are less likely to be heavy smokers (Giovino et al., 1994), and they have lower concentrations of cotinine (Bjornson et al., 1995; Etter & Perneger, 2000; Etter, Vu Duc et al., 2000; Glassman et al., 1993; Ward et al., 1997). Notably, however, women have lower quit rates (Perkins & Scott, 2008; Royce et al., 1997; Wetter et al., 1999) and often experience worse withdrawal symptoms during smoking cessation attempts than men (Perkins et al., 1999; Royce et al., 1997). Given the large sample size, analyses examining the relationships among ACEs, psychological distress, and adult smoking were conducted by gender.

Third, while childhood victimization has repeatedly been shown to increase the risk of adverse health behaviors (Bulik et al., 2001; De Von Figueroa-Moseley et al., 2004; Nelson et al., 2006; Repetti et al., 2002; Rodgers et al., 2004; Simpson & Miller, 2002; White & Widom, 2008; Widom et al., 2007; Widom & Hiller-Sturmhofel, 2001), little research has examined potential mediators in the relationship between ACEs and smoking, the number one cause of preventable mortality in the United States. The few studies that have been conducted examined the potential mediators between ACEs and alcohol and illicit drug use and implicated symptoms of PTSD, depression, antisocial behavior, social phobia, and stressful life events (DeWit et al., 1999; Douglas et al., 2010; Lo & Cheng, 2007; Simpson & Miller, 2002; White & Widom, 2008).

Summary and Interpretation of Findings

Nature and Strength of the Relationships

The first and second research questions were designed to examine the nature and strengths of the relationships between ACEs and smoking and ACEs and psychological

distress. The findings in this dissertation suggest that, overall, even after adjusting for sociodemographic characteristics and other covariates (parental smoking during childhood and alcohol use in the past month), experiencing emotional or physical abuse, parental separation or divorce, or having an incarcerated household member during childhood is associated with adult smoking. Unlike the study conducted by Nelson et al. (2006), which suggested that childhood sexual abuse is associated with risk of subsequent regular smoking—or the study conducted by Al Mamun et al. (2006), which suggested that childhood sexual abuse is associated with young adult nicotine disorder—the analyses in this dissertation did not indicate a significant association between sexual abuse and smoking in either the unadjusted or adjusted model. Notably, however, all associations between ACEs and psychological distress remained significant even after adjusting for covariates.

Effect of Multiple ACEs

The third research question addressed the associations between cumulative number of ACEs and smoking and cumulative number of ACEs and psychological distress. According to Jun et al. (2008), adolescents are at greater risk for smoking if they have experienced multiple forms of abuse. Several studies have also indicated that exposure to a combination of physical and sexual abuse has the most profound impact on smoking risk (Diaz et al., 2002; Jun et al., 2008; Nichols & Harlow, 2004). These findings are in line with the unadjusted analyses conducted for this dissertation. Overall, as the total number of ACEs increased, the odds ratio for smoking increased; ranging from 1.06 among persons with one ACE to 1.90 among persons with four or more ACEs.

This suggests a dose-response relationship between number of ACEs and adult smoking. After adjusting for covariates, however, exposure to multiple ACEs became non-significant, suggesting that variables other than cumulative number of ACEs are actually responsible for the increased risk of smoking. In the models conducted in this dissertation, the strongest predictors of smoking were education level and age. Notably, the relationship between number of ACEs and psychological distress remained significant even after adjusting for covariates.

Gender Differences

The fourth research question was designed to examine potential differences in the relationships between ACEs and smoking and ACEs and psychological distress by gender. A preliminary cross-sectional study of 101 persons conducted by Sacco et al. (2007) suggests that ACEs are associated with serious mental illness or smoking and to a lesser extent with comorbid serious mental illness and smoking. The finding of weak associations between ACEs, serious mental illness, and smoking in this study may be due to the gender difference in the relationship between ACEs and smoking (i.e., the relationship is significant for women but not for men after adjusting for covariates).

Women. In the unadjusted models in this dissertation, with the exception of mental illness in the household, all relationships between ACEs and smoking, including total ACE score, were significant for women (odds ratios ranging from 1.41 for household substance abuse to 2.59 for incarcerated household member). Even after adjusting for sociodemographic characteristics, parental smoking during childhood, and alcohol use in the previous month, many of these associations remained significant (i.e.,

emotional and physical abuse, physical neglect, violence against the mother, parental separation or divorce, and incarcerated household member).

Similar to the non-gender-specific studies conducted by Nelson et al. (2006) and Al Mamun et al. (2006) describing the association between sexual abuse and adult smoking, several studies were specific to women only. These studies also suggest a significant association between sexual abuse and adult smoking in this subpopulation (De Von Figueroa-Moseley et al., 2004; Diaz et al., 2002; Pederson et al., 2008). While a significant association was observed between childhood sexual abuse and adult smoking among women in the unadjusted model in this dissertation, after adjusting for potential covariates, the association was attenuated and no longer significant. With the exception of incarcerated household member, the association between each ACE and psychological distress remained significant even after adjusting for potential covariates.

Men. In the unadjusted models, half of the ACEs (i.e., physical abuse, emotional neglect, parental separation or divorce, household substance abuse, and incarcerated household member) were associated with smoking for men. Unlike the women, after adjusting for sociodemographic characteristics, parental smoking during childhood, and alcohol use in the previous month, all of the relationships became attenuated and were no longer significant. According to the models, the characteristics that were the strongest predictors of smoking among men were low educational attainment and younger age. These findings suggest that, unlike women, ACEs are not associated with smoking among men. Notably, however, with the exception of incarcerated household member

and parental separation or divorce, the relationships between ACEs and psychological distress remained significant, even after adjusting for potential covariates.

Mediating Effect of Psychological Distress

There are several studies that suggest that psychiatric disorders may mediate the relationship between ACEs and adult substance abuse. Studies conducted by Douglas et al. (2010) and Lo and Chen (2007) suggest that the relationship between childhood abuse and substance dependence may be partially mediated by mood and anxiety disorders. DeWit et al. (1999), implicate social phobia as the mediator between adverse life events and chronic stress in childhood and drug dependence in adulthood.

Several studies have specifically examined the relationship between ACEs and drug use and abuse among women. According to a literature review conducted by Simpson and Miller (2002), psychiatric conditions such as depression and anxiety disorders mediate the relationship between child abuse and substance use disorders in women. Moreover, in a study conducted by White and Widom (2008), the authors concluded that PTSD among maltreated girls may increase the risk of subsequent substance use problems.

In this dissertation, while there was not a significant association between ACEs and smoking in men, there was a mediating effect of psychological distress on the relationship between ACEs and adult smoking in women. Among women, over 19% of the relationship between emotional abuse and smoking was mediated by psychological distress, over 15% of the relationship between physical abuse and smoking was mediated by psychological distress, and over 10% of the associations between physical neglect and

smoking and violence against the mother and smoking was mediated through psychological distress. Given this, the identification and effective treatment of psychological distress (e.g., depression, anxiety) among women who experienced various types of ACEs could reduce the risk of adult smoking in this population.

Meaning of Results in Terms of Developmental Psychopathology

As described in chapter 1, the theoretical underpinning for this research was based on developmental psychopathology. According to Cicchetti and Rogosch (1999), excess adverse experiences in combination with few emotional resources, decrease an individual's likelihood of resolving age-appropriate, stage-salient tasks and often leads to modifications in brain anatomy and functioning. This often results in significant deficits in biological, emotional, cognitive, and interpersonal development.

While this dissertation did not find a significant association between ACEs and smoking in men after adjusting for selected covariates, it did for women. This provided an opportunity to probe deeper into the potential mechanisms that link ACEs to smoking in this population. Using a developmental psychopathological approach, it was hypothesized that ACEs, which serve as the adverse experiences, and psychological distress, which occurs as the result of unresolved stage-salient issues and modifications in brain anatomy and functioning, portend the development of the adult smoking; the vulnerability. This was, in fact, partially true for women. Despite decades of time between the actual ACE event and the time of the survey, over 15% of the relationships between physical abuse and smoking and emotional abuse and smoking was mediated through psychological distress and over 10% of the associations between physical neglect

and smoking and violence against the mother and smoking was mediated through psychological distress.

Recommendations for Future Research

Little research has been conducted examining the long-term consequences of ACEs on emotional, behavioral, and social development. Given this, future research recommendations are warranted.

First, while information on the prevalence of child abuse and neglect are available from a variety of data sources, each of these sources has limitations that may bias results. For example, child protective services data may underestimate the true prevalence of maltreatment because the majority of child abuse and neglect goes undetected. In contrast, the use of clinic and hospitalized patients may inflate the prevalence of ACEs. Population-based national and state-based surveillance data are needed in order to more adequately determine the prevalence of abuse in the population as well as the causes, developmental paths, and critical points that link ACEs, psychological distress, and smoking (Widom et al., 2006). Moreover, in order to more accurately interpret findings, eliminate subjectivity, and share information, standard abuse, neglect, and household dysfunction criteria and instruments should be developed (Brodsky & Stanley, 2008).

Second, further research should be conducted on gender differences in the associations between ACEs, psychological distress, and smoking. While psychological distress was significantly related to smoking for both men and women in this dissertation, after adjusting for covariates, the relationship between ACEs and smoking remained significant only for women. This may be due to differences in coping styles and

socialization (Simantov et al., 2000). It is possible that females may develop more passive styles of responding to threats and distressing events as opposed to boys who may engage in a more active coping style (Compas, Malcarne, & Fonacaro, 1988; De Boo & Spiering, in press; Groer, Thomas, & Shoffner, 1992; Nolen-Hoeksema & Girus, 1994; Peterson, Sarigiani, & Kennedy, 1991).

Third, Cicchetti and Toth (1995) suggest that interventions should take into account issues related to the timing of maltreatment and the child's cognitive capabilities to process the experience. According to a study conducted by Jun et al. (2008), there is a dose-response relationship between accumulation and severity of abuse and early onset smoking initiation among adolescent females (Jun et al., 2008). Moreover, according to two studies, one by Thornberry et al. (2001) and one by Kaplow and Widom (2007), adolescent abuse is more strongly associated with drug use in adulthood than abuse experienced in childhood alone. Given this, child abuse and neglect research would benefit from additional questions addressing temporality, intensity, frequency, and duration of maltreatment (Cicchetti & Toth, 1995; Kaplow & Widom, 2007).

Fourth, it is important to consider the victim's past and current environmental circumstances. Many studies only assess stress and trauma within the family environment during childhood and fail to account for broader environmental exposures (e.g., social, economic, neighborhood, social support) that could impact psychological and behavioral development. Moreover, research suggests that persons abused as children are at increased risk of victimization as adults due to environmental factors and poor relationship and coping skills (Coid et al., 2001; McNutt et al., 2002; Messman-Moore, &

Long, 2003; Schaaf & McCanne, 1998), further increasing the risk of psychological distress and licit and illicit drug use.

Fifth, between 12% to 22% of persons who were abused as children exhibit competency and adjustment across multiple life domains, otherwise known as resiliency (Jaffee, Caspi, Moffitt, Polo-Tomas, & Taylor, 2007). While research in this area warrants further evaluation, studies to date indicate that potential factors that lead to resiliency include guidance and supervision by parents or other family members, positive relationship with a nonabusive family member, high-functioning families, stable living conditions, positive peer relationships, structured school environment, supportive relationships with teachers or other adults in the community, higher education aspirations, reflectiveness in meeting new situations, responsiveness to others, and high IQ (DuMont, Widom, & Czaja, 2007; Garnezy, 1991; Haskett, Nears, Ward, & McPherson, 2006; Jaffee et al., 2007; Tiet et al., 1998). More specific to the dissertation hypotheses, according to the stress-coping theory (Thoits, 1986), parental emotional or instrumental support is posited to buffer the relationships between negative life events, mental distress (e.g., anxiety, depression), and substance use and abuse (Galaif, Stein, Newcomb, & Bernstein, 2001; Greenberg, Siegel, & Leitch, 1983; Jun et al., 2008; Mermelstein, Cohen, Lichtenstein, Baer, & Kamarck, 1986; Umberson, 1987; Wills, 1990; Wills & Cleary, 1996; Wills, Vaccaro, & McNamara, 1992). Factors that hamper resiliency include parental substance abuse or mental health problems and living in high crime neighborhoods with low social cohesion (Jaffee et al., 2007). Given these findings,

future research should consider potential resiliency factors which may interrupt the ACE, psychological distress, and smoking pathway.

Finally, genetic influences on smoking behavior, nicotine dependence, and mental illness warrant further consideration. Dopamine receptor genes, transporter genes (i.e., serotonin and dopamine), and other genes related to the metabolism of nicotine are currently being examined in molecular epidemiology studies (Batra, Patkar, Berrettini, Weinstein, & Leone, 2003; Yoshimasu & Kiyohara, 2003). Notably, there is a high comorbidity between mental illness and nicotine use and many of the suspect genes are common to both (Yoshimasu & Kiyohara, 2003). Determining potential genetic vulnerabilities to psychological distress and subsequent smoking can aid in identifying individuals at increased risk and set the groundwork for the development of personalized treatment approaches (Tyndale, 2003).

Implications for Social Change

From a broad perspective, comprehensive strategies such as increased communication between family practice, internal medicine, preventive medicine, mental health professionals, public health, policy makers, and law enforcement are needed to identify children at risk for ACEs (Felliti et al., 1998). According to Anda et al. (2006), medicine and public health are “fragmented by categorical funding, organizational boundaries, and a symptom-based system of medical care” (Anda et al., 2006). Due to this, health care professionals often do not receive the training necessary to identify and treat families in which children are exposed to ACEs or adults who have experienced ACEs (Anda et al., 2002; Dubowitz, 1988). Moreover, fragmentation in the institutions

and funding streams that deal with child maltreatment must be eliminated. For example, child protective services deal primarily with abuse by caretakers and the criminal justice system excludes victimization not generally dealt with by police. In addition, while important child maltreatment research is being conducted in public and mental health agencies as well as universities, this research is not finding its way into the hands of policy makers (Cicchetti & Toth, 1995).

More specific to the findings in this dissertation, gender differences were found in the relationships between ACEs, psychological distress, and smoking. While psychological distress was found to be associated with smoking among both men and women, ACEs were only associated with smoking among women. In addition, psychological distress partially mediated the relationship between ACEs and smoking among women. Given these gender differences, gender-based intervention and prevention strategies are warranted; particularly for adolescent females and women who have experienced ACEs. Given that, as compared to men, women who have experienced ACEs have higher annual healthcare costs due to lower perceived general health and greater emotional and physical disability (Arias, 2004; Chartier, Walker, & Naimark, 2007; Davis, Luecken, & Zautra, 2005; Felitti et al., 1998; Finestone, Stenn, Davies, Stalker, Fry, & Koumanis, 2000; Molnar et al., 2001; Thompson, Aria, Basile, & Desai, 2002; Walker, Gelfand et al., 1999; Walker, Unutzer, et al., 1999), the finding of a gender difference in the relationship between ACEs and smoking has several social implications that warrant further consideration.

First, it is important to identify potential modifiable risk factors for smoking onset in adolescents (e.g., ACEs), as well as build resiliency and positive social support networks for abused children to potentially decrease the prevalence of smoking among children and adolescents exposed to maltreatment. Second, given the mediating effect of psychological distress on the relationship between ACEs and smoking in women, it is not surprising that women often have worse smoking cessation rates than men (Bjornson et al., 1995). Programs that combine skills training and long-term follow-up may be particularly important for this population. Third, given that psychological distress is associated with smoking among both men and women, and that negative affect is highly associated with smoking, pharmacological treatment (Carmody, 1992), and tools for developing alternative coping strategies, and enhancing social support structures (Simantov et al., 2000) may be important components of smoking cessation strategies in this population.

Limitations

There are a number of limitations that should be considered when interpreting this analysis. As described in chapter 1, many of these limitations (e.g., different characteristics between participants and non-participants, test-retest reliability, self-reporting status of ACEs and smoking, generalizability), have been previously examined in the ACE Study (Anda et al., 1999; Dong, Anda et al., 2003; Dube et al., 2004; Edwards, Anda et al. 2001; Felitti et al., 1998). Three limitations, in particular, however warrant further examination.

First, the ACE Study data are cross-sectional and do not collect specific information on temporality. While the temporal relationship between ACEs and adult smoking is apparent in this study, the temporal relationship between psychological distress and ACEs and psychological distress and smoking could not be determined and was inferred solely from the literature. While most current literature suggests the majority of psychiatric disorders associated with smoking occur prior to smoking initiation, other pathways (e.g., bi-directional association, both result from common environmental and genetic factors, smoking precedes psychological distress) have been posited (Costello et al., 1999; Moolchan et al., 2000). Longitudinal studies would further clarify the relationships between ACEs, psychological distress, and adult smoking.

Second, there may be gender differences in the recall or admittance of ACEs. Notably, in a study conducted by Widom & Morris (1997), the authors found that men with a history of documented sexual abuse in childhood were less likely than women to retrospectively report that the incidence occurred.

Third, the age of the data might affect its relevance. Notably, a study conducted by Dube, Felitti, Dong, Giles, and Anda (2003) examining the relationship between ACE score and six health problems, including smoking, across four successive birth cohorts (1900-1931, 1932-1946, 1947-1961, and 1962-1978), suggests that the effects of ACEs on the risk of various health problems are unaffected by social or secular changes.

Summary

In conclusion, this study suggests that there is an association between ACEs and smoking among women; an association between ACEs and psychological distress in both

men and women; and that psychological distress partially mediates the relationship between ACEs and smoking among women. Given the gender differences in these relationships, gender-based intervention and prevention strategies are warranted. Further elucidating potential mediators in the relationships among ACEs, psychological distress, and smoking may lead to more effective targeted intervention and prevention programs. Consideration should be given to factors such as resiliency, social support, genetic and environmental characteristics, and temporality, intensity, frequency, and duration of maltreatment in future research.

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Curriculum Vitae

ACADEMIC EXPERIENCE

- 2007-Present Candidate for Doctor of Philosophy – Epidemiology,
Walden University, Minneapolis, Minnesota
- May 14, 2001 Masters of Public Health – Biostatistics,
Emory University, Rollins School of Public Health,
Atlanta, Georgia
- 1989 Bachelor of Arts – Psychology
State University of West Georgia, Carrollton, Georgia

ACADEMIC HONORS and ORGANIZATIONS

Emory University, Rollins School of Public Health, Atlanta
Delta Omega Phi Honorary Society
Nominated for the 2001 Shepard Science Award

State University of West Georgia, Carrollton, GA
Phi Kappa Phi Honor Society
Pi Gamma Mu -National Honor Society for the Social
Sciences
Graduated Cum Laude

RELEVANT PROFESSIONAL EXPERIENCE

- 2002-Present Epidemiologist (full-time)
Centers for Disease Control and Prevention
National Center for Chronic Disease Prevention and Health
Promotion
- Originated and designed the “Anxiety and Depression Module”
which was implemented in the 2006 Behavioral Risk Factor
Surveillance System (BRFSS). The BRFSS is the largest
continuously conducted telephone survey in the world and

is critical for monitoring health risk behaviors, clinical preventive practices, and health care access related to chronic diseases and injury. The “Anxiety and Depression Module” is the first of its kind to examine the relationships between anxiety, depression, health behaviors, quality of life, and chronic diseases at the national, state, and local level.

Author on a number of peer-review manuscripts using surveillance data (e.g., Behavioral Risk Factor Surveillance System, National Health Interview Survey). Topics include the relationship between adverse health behaviors, impaired quality of life, and mental illness; and chronic diseases, impaired quality of life, and mental illness.

1995-2000

Computer Specialist (full-time)

2000-2001

Health Scientist (full-time)

2001-2002

Epidemiologist (full-time)

Centers for Disease Control and Prevention

National Immunization Program

Responsible for creating the Influenza, Measles, Rubella, Haemophilus Influenzae Type b, Pertussis, and Mumps Surveillance Systems. Responsibilities included developing systems to collect and update data from 50 states-based data sets, producing data entry and analysis screens, and conducting data analysis for manuscripts.

Lead data manager for the Vaccine Adverse Events Reporting System (VAERS) from 1996-1999. Responsibilities included the development, management, and execution of the VAERS data management contract to assure reliability, readability, and usefulness of the data as well as analyzing, evaluating, and recommending approaches and techniques for operating across multiple platforms.

Lead data manager for the Edmonston-Zagreb Measles study (1997). This included supervision of several programming and statistical contract staff. Responsibilities included developing mechanisms for storing, retrieving, and securing data; converting data from various software packages and data record layouts; combining data; and assisting contract and CDC staff with analytic issues.

Responsible for assuring the development of the National Immunization Program's (NIP) National Electronic Disease Surveillance System (NEDSS). This included working with branch staff to develop questionnaires, coordinating development efforts with other CIOs, and attending NEDSS Operational Working Group meetings, contract meetings, NIP meetings, and national stakeholders meetings. Served as a member of NEDSS Reporting Integration and Core Development teams responsible for developing reporting standards for NEDSS.

Originate, design, analyze, and wrote peer-reviewed articles regarding immunization coverage among children.

1989-1995

Computer specialist (full-time)
Centers for Disease Control and Prevention
National Center for Infectious Disease

Responsible for analyzing data for a number of mortality projects including Hodgkin's disease, Creutzfeldt-Jakob disease, hemophilia, and chronic liver disease. Lead data manager for the initial Persian Gulf War Syndrome Epi-Aid and the initial Hantavirus outbreak investigation. Responsibilities included creating data entry systems for deployment in the field; developing systems to store, retrieve, and secure data; converting data from various formats; combining data with different data layouts; and producing data quality and analysis reports. Responsible for creating the Rabies Surveillance System which included developing a system to collect and update data from 50 state-based data sets, creating data entry and analysis screens; and conducting analysis for the yearly rabies surveillance report.

PROFESSIONAL AWARDS

National Center for Infectious Disease

Nominee in the Information Category (GS -11 and below),
Outstanding CDC Employee of the Year – 1991.

Recognition Award, Hepatitis/Retrovirus False Reactivity in Blood Donors (Unit Commendation) – 1993.

Recognition Award, Hantavirus (Unit Citation) – 1994.

Recognition Award, Persian Gulf Syndrome (Research-Operational) – 1996.

National Immunization Program

Special Act or Service Award, Edmonston-Zagreb Measles Project – 1997.

Statistical Research and Services Award, Vaccine Adverse Reporting System – 1997.

Secretary's Award for Distinguished Service – 1998.

Special Act Group Award, Smallpox Response Plan – 2002.

Special Act Group Award, National Electronic Disease Surveillance System – 2002.

National Center for Chronic Disease Prevention and Health Promotion

Special Act or Service Award, Scientific Support, Arthritis Program -2007.

PUBLICATIONS

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 doi:10.1159/000109793
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POSTER PRESENTATIONS

Strine TW, Chapman PD, Kobau R, Balluz L. Depression and anxiety as self-reported health impairments: Correlates and implications. Presented at the 56th Institute on Psychiatric Services. American Psychiatric Association. October 9, 2004. Atlanta, Georgia

Strine TW. Depression and anxiety: a new public health surveillance strategy. Presented at 22nd Annual BRFSS conference. March 5-9, 2005, Atlanta, Georgia.

Strine TW, Chapman DP. Associations between anxiety, health-related quality of life and health behaviors. In: Proceedings of the 2006 American Psychiatric Association Institute for Psychiatric Services; 2006 October 5-8; New York, NY. Arlington, VA: American Psychiatric Association, 2006.

Strine TW, Balluz LS, Mokdad AK, Ribble JL. Provisional Depression and Anxiety Module analysis, 2006. Presented at the 24th Annual Behavioral Risk Factor Surveillance meeting, Atlanta GA, March 24-28, 2007.

Strine TW, Chapman DP, Balluz L, Moriarty D, Mokdad AH. Life satisfaction, health-related quality of life, and health behaviors among U.S. adults. Presented at the 2007 American Psychiatric Association Conference, San Diego, California, May 19-24, 2007.

Strine TW, Mokdad AH, Balluz LS. Depression and Anxiety in the United States: Findings from the 2006 Behavioral Risk Factor Surveillance Survey. Presented at the

59th Institute on Psychiatric Services, New Orleans LA, October 11-14, 2007.

Strine TW, Dhingra S. Provisional Mental Illness and Stigma Module analysis, 2007. Presented at the 25th Annual Behavioral Risk Factor Surveillance meeting. Orlando, Florida, March 15-19, 2008.

Strine, T.W., Dhingra. S., Okoro, C., Zack, M., Balluz, L.S., Berry, J.T., Mokdad, A.H. State-based differences in the prevalence and characteristics of untreated persons with serious psychological distress. Presented at the 26th Annual Behavioral Risk Factor Surveillance meeting. Atlanta, Georgia, March 14-18, 2009.

ORAL PRESENTATIONS

Analyzing and interpreting data from the Depression and Anxiety Module with an emphasis on the Patient Health Questionnaire 8 (PHQ-8). Presented at the 24th Annual Behavioral Risk Factor Surveillance meeting, Atlanta GA, March 24-28, 2007.

Potential data analysis using the Anxiety and Depression Module: Behavioral Risk Factor Surveillance Survey, 2006. Presented at the 2007 Joint National Conference on Mental Health Block Grant and National Conference on Mental Health Statistics, Washington DC, May 29-June 1, 2007.

Depression and anxiety in the U.S.: Findings from the 2006 Behavioral Risk Factor Surveillance System. CDC Mental Health Workgroup. Mental Health Surveillance Conference. Atlanta, GA, September 6, 2007.

Depression and anxiety in the United States: Findings from the 2006 Behavioral Risk Factor Surveillance Survey. Presented at the 59th Institute on Psychiatric Services, New Orleans LA, October 11-14, 2007.

Provisional Mental Illness and Stigma Module analysis, 2007. Presented at the 25th Annual Behavioral Risk Factor Surveillance meeting. Orlando FL, March 15-19, 2008.

2007 BRFSS Mental Illness and Stigma Module. Presented at the 2009 National Grantee Conference on Mental Health Block Grant and Data. Washington DC, June 17-19, 2009.