UNIVERSITY OF CALIFORNIA
RIVERSIDE

The Effects of Community Violence Exposure on Adolescents' Health

A Dissertation submitted in partial satisfaction
of the requirements for the degree of

Doctor of Philosophy

in

Psychology

by

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June 2010

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Acknowledgements

I would like to thank my advisor, Dr. Nancy G. Guerra, for her continual support during my time at UC, Riverside. As my advisor, she has provided me with both the theoretical guidance in discussing research ideas as well as the personal support in encouraging me to pursue my interest in health psychology. She is responsible for supporting the mindfulness and health study I conducted among undergraduate students at UC, Riverside, which has been one of the most fulfilling experiences in my development as a researcher and psychologist. I am also deeply grateful to Dr. M. Robin DiMatteo and Dr. Tuppett M. Yates whose encouragement, guidance, and constructive comments helped me pursue new research challenges and opportunities. Both were excellent mentors whose guidance allowed me to ask better questions and be a better researcher. I also want to thank all the faculty members at UC, Riverside. I feel fortunate to have attended a graduate program with professors and mentors who truly care about their students professionally and personally.

I also wish to extend my warmest thanks to all my ACE-UCR lab colleagues over the past six years, whose support, encouragement, and warmth provided the anchors to continue working on this dissertation. A special thanks goes to Roxie Alcaraz whose concern and care created a warm environment that was both inspiring and professional. I would also like to give a special thanks to my office-mate of four years, Rosa Toro. She always provided an excellent sounding board for theoretical ideas and questions. Without her humor and wit, I may not have made it through those tiring days and nights.

I also want to recognize Faye Harmer in the psychology department whose open door and attentive ear policy made UC, Riverside a home over the past six years. Without her support and continual oversight of us graduate students, my experience would not have been as memorable or as smooth.

Dedication

I dedicate this dissertation to my amazing and unbelievably gracious father, Dr. John Fairborn. He inspired me to pursue my doctoral degree at a very young age, and continues to inspire me with his intellect, kindness, wit, and charm. Without his constant guidance, support, and love, I could not have completed this dissertation. I also dedicate this dissertation to my loving mother who has been a constant figure of support, particularly over the past six years. I could not have asked for a better mother. Last, I dedicate this work to my wonderful husband whose patience and love encouraged me to continue working over these many years as a graduate student.

ABSTRACT OF THE DISSERTATION

The Effects of Community Violence Exposure
on Adolescents' Health

by

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University of California, Riverside, June 2010
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The relations between community violence exposure (CVE) during adolescence and a selection of six co-occurring health-related outcomes were investigated using data from The National Longitudinal Study of Adolescent Health (Add Health). The mechanism by which CVE affected adolescents' health was then examined through depression and self-regulation. The health-related outcomes comprised self-reported assessments of general health, physical problems, BMI (Body Mass Index), sleep problems, physical activity and sedentary behavior from approximately 14,000 adolescents between the ages of 12 and 19. The adolescents were divided into a younger and older group with the break at 16 years and older; analyses were conducted separately for each age group and examined at two time points separated by one year. Hierarchical regression was used to examine the effects of CVE on adolescents' health. The mediation pathways through depression and self-regulation and whether these relations varied by

gender were tested using multiple mediation analyses (Preacher & Hayes, 2008). Gender, socioeconomic status, and childhood maltreatment were entered as control variables. Findings indicated CVE affected all health-related outcomes but were minimal for sedentary behavior and BMI. In general, both depression and self-regulation mediated the effect of CVE on general health, physical problems, sleep, and physical activity. Moderating effects revealed CVE had a more adverse impact on females' sedentary behavior and BMI at the second time point.

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Part One:

The Effects of Community Violence Exposure on Adolescents' Health

Introduction

Adolescents, as a group, are generally healthy. However, as they interact with friends and family and maneuver around challenging obstacles and outside influences, they are confronted with choices, many of which can have long-term health consequences. A very unfortunate fact in this country is that children and adolescents, particularly those living in poor neighborhoods, are exposed to violence within their community, and at their young and vulnerable age they are generally the victims. It seems obvious that witnessing or being a victim of violence would have some negative health consequences, but the process by which one affects the other is complex. It will, for example, depend upon many factors; an incomplete list would include the nature and extent of the violence exposure, the inner resources of the individual, and the support (or lack of support) that is received from family, friends, and community. Because intervention resources are limited and community violence exposure is inevitable for many children, it is important that the relation between violence exposure and health outcomes be understood in all its complexities. The main goal of this study is to examine several health-related choices that adolescents make and how exposure to violence can influence those choices and associated health outcomes.

Community violence exposure has been defined by Krug, Dahlberg, Mercy, Zwi, and Lozano (2002) to be violence between individuals who may or may not know each other and that usually takes place outside of the home. The violence exposure could take the form of either witnessing the violence of being a victim of it. This definition will be adopted in this study. It is in contrast to experiencing violence within the home in the

form of familial physical, sexual, or emotional abuse, which has been shown to have a direct link to poor health. The two types of violence will be treated separately in this study.

First we will discuss the negative impact of violence exposure among adolescents and the importance of its prevention. Violence statistics in the U.S. will be presented followed by a literature review of the negative relation between community violence exposure and health. Next six health-related conditions (general health, physical problems, obesity, sleep, physical activity, and sedentary behavior) will be introduced, which can relate to eventual chronic health problems. A review will then present how problem behavior among adolescents affect their health, revealing that adolescents, even at a young age, are already experiencing serious health problems. Finally, the few studies that discuss how adolescents' own personalities may relate violence to poor health will be cited in order to introduce the concept of mediators.

This literature review will be followed by analyses that expand past research and examine the relation between community violence exposure and six health-related behaviors and outcomes among adolescents. Previous research relating violence and adolescents' health has focused on how violence triggers self-destructive behaviors, such as smoking, alcohol abuse, and drug use. Relatively little work has been devoted to other more passive, but nevertheless, unhealthy behaviors and outcomes such as inactivity, overeating, and obesity. The current study seeks to examine the effects of community violence exposure on some of these more passive conditions, namely the six health-related behaviors and outcomes mentioned in the previous paragraph: general health,

physical problems, obesity, sleep, physical activity, and sedentary behavior. Analyses will further investigate the process in which community violence exposure affects health through two possible mediators, depression and self-regulation. Gender difference as a moderator will be examined. Due to the fact that childhood maltreatment within the home has been related to health outcomes, and must therefore be separated from community violence exposure, it will be included as a control variable in all analyses.

Violence Exposure

The incidents of violence upon and among adolescents unfortunately are relatively common in the U.S., and numerous studies have documented their lasting consequences on adolescent development. This is well documented in The Youth Risk Behavior Surveillance System (YRBSS), which monitors health-risk behaviors and obesity rates among adolescence. This nationwide survey of high school students conducted by the Center for Disease Control and Prevention (CDC, 2008) found 35.5% reported being in one or more physical fights in the 12 months preceding the survey, and 18% reported carrying a weapon on one or more occasions 30 days preceding the survey. In 2004, there were 5,292 young people between the ages of 10 and 24 murdered in which 82% were killed with firearms, and more than 720,000 young people were treated in emergency rooms due to violence in 2005 (CDC, 2007). The financial load on the public from direct and indirect costs of youth violence exceeds 158 billion every year, and underlines the need to address both the causes and the effects of violence among our youth (Children's Safety Network Economics & Data Analysis Resource Center, 2000).

The Effects of Community Violence Exposure

The prevalence of violence exposure in our communities and schools makes us more aware of the need to prevent its adverse consequences. Whitaker and Bastian (1991) reported that one-quarter of the violent crimes experienced by twelve to fifteen year olds and 26% experienced by sixteen to nineteen year olds occur on the street. With regards to adolescent health, the research on community violence exposure that has received the most attention has tended to focus on only a small number of health outcomes, generally limited to mental health, smoking habits, sexual risk behaviors, and alcohol and drug use.

Findings generally support the connection between violence exposure, whether experienced at home, at school, or in the community, and its deleterious effects on the development of adolescents. Research indicated adolescents who have been exposed to community violence are more likely to use drugs, smoke, drink alcohol, and have risky sexual behaviors (Bereson, Wiemann, & McCombs, 2001). A cross-section survey of 517 adolescent girls (mean age = 15.6 years old) was taken at a university-based family clinic where they were asked if they had seen or experienced at any time in their life a robbery, physical attack, rape (or threat of rape), threats against their life, or a murder. Just witnessing violence, but not being a victim of it, increased their risk of using marijuana, alcohol, and tobacco and of having multiple sexual partners by 2 to 3 times compared to the girls who had neither witnessed nor experienced violence. Girls who had actually been a victim of violence had a 2 to 4 times greater risk of using marijuana, alcohol, and tobacco as well as having multiple sexual partners, having sex with strangers, and testing

positive for a sexually transmitted disease. The girls who had been both victimized and had witnessed violence were also 3 to 6 times more likely to have suicidal thoughts.

Vermeiren, Schwab-Stone, Deboutte, Leckman, and Ruchkin (2003) found similar connections between community violence exposure and alcohol and drug use across three adolescent populations in Belgium, Russia, and Connecticut. In a cross-sectional study of 3380 adolescents between the ages of 14 and 17 that controlled for adolescents' own violent behavior, they found that those who reported higher exposure to community violence (witnessing and victimization) used more tobacco, alcohol, marijuana, and hard drugs and concluded that violence exposure can have severe physical and psychosocial health problems. A 2005 study of 409 adolescents who were exposed to higher levels of community violence found that they were 4 times more likely to engage in dangerous sexual behaviors such as unprotected sex, sometimes while intoxicated, and multiple partners (Voisin, 2005). Other research has supported the connection between violence exposure and heightened drug use (Sanders-Phillips, 1997), high-risk sexual behavior (Voisin, 2003; Voisin, Salazar, Crosby, DiClemente, Yarber, & Staples-Horne, 2004) and intention to smoke (Fick & Thomas, 1995).

Exposure to community violence (both witnessing and experiencing) has also been linked to a variety of stress responses and reactions such as crying, withdrawal, depression, anxiety, difficulty in falling asleep and nightmares. Duncan's 1996 review documented substantial evidence that neighborhood violence indeed relates vulnerabilities to Post Traumatic Stress Disorder, depression, and sleep disturbances. In addition, a pilot study conducted at the Hopkins Prevention Research Center examined

the relations among sleep disturbance, community violence, and psychological indices. Self-reported community violence exposure was positively correlated to sleep deprivation among 64 adolescents, ages 16 to 18 years (Cooley-Quille & Lorion, 1999). This study also revealed that adolescents who experienced the highest levels of community violence exposure had the lowest resting pulse, which is unexpected since urban youth tend to have higher blood pressure rates than suburban or rural youth due to crowds and noise (Thomas & Groer, 1986). The study suggests that this may be a protective response so that these adolescents can physiologically adapt to the violent events they experience with frequency in their neighborhoods. They physiologically habituate causing them to become emotionally desensitized to the violence, so that they are not aroused to a "fight or flight" response at every exposure and the violence becomes normalized. Although this biological response has short-term benefits for coping with these stressors, there may be long-term implications on adolescents' health when consistently being forced to suppress a normal emotional response to violence. While not being emotional, adolescents still experience fear in these circumstances which over time can turn into distress and increased physiological reactivity (Turner, Beidel, & Cooley-Quille, 1997).

Although the many studies cited above have implicated violence outside the home to a wide range of health risks, not all health risks are covered in the same detail. There is a gap in the literature addressing whether such violence affects other forms of health-related behaviors and outcomes that may be deemed just as important. One example is obesity, which is one of the most worrying public health concerns for children and adolescents in the United States today. This study intends to close the gap by

investigating the relations between community violence exposure and obesity, as well as other associated, and less studied, health problems comprising general health, physical problems, sleep patterns, physical activity, and sedentary behavior.

Before examining the relation between community violence exposure and these other health problems, it is important to separate out the effects of childhood maltreatment, which generally occurs within the home and has been consistently linked to obesity. In the following paragraphs this link will be elaborated upon by many references in order to show that it is firmly established and must be included in any model involving violence exposure and obesity. As noted above violence exposure inside the home, which will be referred to as childhood maltreatment, will be included in the analyses as a control variable.

Childhood Maltreatment and Health

There is a long list of physical health problems that past abuses, whether physical, sexual, or emotional, can cause. One of the most notable findings when investigating childhood abuse is its clear relation to obesity. A prospective longitudinal study of 84 abused females from ages 6 to 27 investigated the role one type of childhood maltreatment, namely sexual abuse, played in the development of obesity (Noll, Zeller, Trickett, & Putman, 2007). A moderate relation was found while the participants were still adolescents, but by young adulthood (ages 20–27), abused female participants were significantly more at risk for obesity (42.25%) than their non-abused counterparts (28.4%). Noll and colleagues suggested that depression and psychobiological responses might play a mediating role, helping to explain how abuse may lead to obesity.

Another study (D'Argenio, Mazzi, Pecchioli, Di Lorenzo, Siracusano, & Troisi, 2009) of 200 adults also found that early abuse, whether sexual or physical, experienced during the first 15 years of life predicted obesity in adulthood. The study also investigated the role of psychiatric disorders as the mechanism for this relation. Participants were separated into on of the following three group: a control group of non-obese healthy adults, a group of obese adults with no current or past psychiatric disorder, and a third group of obese adults with a current psychiatric disorder. Results indicated that obese individuals were more at risk for depression, anxiety, and overeating, but the psychiatric disorder was not a necessary condition for obesity. An interesting finding from this study was that even minor early life stressors could be linked to later obesity, which is relevant to this study, which is examining the obesity connection to a specific early life stressor, namely community violence exposure.

It is important to add that sexual abuse can lead to other types of eating disorders as evidenced by the statistic that 17% of U.S. women who are bulimic also experienced sexual abuse (Wonderlich, Wilsnack, Wilsnack, & Harris, 1996). It appears that this type of abuse may in some way hinder one's ability to self-control, seen in the overeating associated with both obesity and bulimia.

A study by Noll, Trickett, Susman, and Putman (2006) also implicated early sexual abuse during adolescence to sleep disturbances. Their prospective longitudinal study of 147 females (78 of whom were abused) examined the effect of childhood sexual abuse on later sleep problems in adolescents while controlling for co-occurring psychopathologies. Ten years after disclosure of their abuse, the abused women

experienced significantly more sleep disturbances than those who reported no abuse even after controlling for depression and Post Traumatic Stress Disorder (PTSD). It is relevant to this study since they also found that the sleep problems highly correlated with depression, which will be used as a mediator when developing the relation between community violence exposure and health.

In addition to sexual abuse, physical and verbal abuse have also been implicated in the development of obesity (Williamson, Thompson, Anda, Dietz, & Felitti, 2002; Greenfield & Marks, 2009). Williamson and colleagues' retrospective cohort study assessed self reports of childhood abuse that had taken place before the age of 18 from 13,177 members of California HMO, who at the time of the survey were between the ages of 19 and 92. At least one type of abuse was reported by 66% of participants. Sexual, verbal, physical, and fear of physical abuse were all examined, with results indicating increases in obesity with the number and severity of each type of abuse, but physical and verbal abuse were most strongly correlated with obesity risk.

Previous research has also found a connection between neglect during childhood and obesity as an adult. In a 1974 prospective study the support from family and quality of family structure was assessed, based on teacher perception, of 987 Copenhagen third graders (Lissau & Sorenson, 1994). Ten years later 86% of the now young adult students were reassessed. Those who were perceived to be neglected by parents during childhood were more likely to be obese adults regardless of childhood weight status.

Abuse experienced during childhood appears to be related to one's perception and tolerance to pain as an adult. A study conducted in the early 1990's (Ericsson, Kendall-

Tackett, & Hernandez, 2007) examined the relations between childhood physical and sexual abuse and pain among 1,727 men and women using the National Co-morbidity Survey, a nationally representative sample. Participants were asked if they had suffered from any serious pain-inflicting illnesses in the last twelve months, and if so they were asked to rate their level of pain from a scale of 1 (not at all) to 4 (a lot). Regression and mediation analyses indicated that childhood abuse predicted reports of pain and that this relation was mediated through depression.

In another study, abuse during childhood was linked with diabetes and the presence of diabetic symptoms (Kendall-Tackett & Marshall, 1999). Other diseases linked to abuse include heart disease and cancer and breast and thyroid cancer in women and men respectively (Stein & Barrett-Connor, 2000). Abuse may create an "I don't care about my health attitude," such that abused individuals may be more likely to smoke despite the known health consequences (Edwards, Anda, Gu, & Felitti, 2000), and female adolescents may take higher risks for HIV infection (Johnsen & Harlow, 1996). Moreover, it appears that each additional type of abuse experienced compounds the negative effect on both physical and mental areas of health (Turner, Finkelhor, & Ormrod, 2006).

Researchers have discovered that traumatic events during childhood can lead to actual physiological changes, which may explain the connection between abuse and poor health. There have been numerous studies over the past 15 years showing traumatic events can interrupt the body's ability to maintain hormonal balance (e.g. Bremner, 2005; Kendall-Tackett, 2000). Yates (2007), in particular, gives a detailed overview of how

childhood emotional abuse can lead to negative psychological development through the impairment of measurable biological processes. Citing many published studies, she explains how two stress response systems within the body can suffer from long-term emotional trauma. One system involves the integrated function of the hypothalamus, pituitary, and adrenal glands (known as the limbic-hypothalamic-pituitary-adrenal axis (L-HPA)) which, through the production of the hormone cortisol, acts to increase blood sugar, suppress immune response, inhibit fear and in general prepare the body to deal with stress. Equally important is that cortisol acts to modulate the activity of the L-HPA axis and slow it down when the stressor has passed. The other stress response system, known as NE-SAM (norepinephrine-sympathetic-adrenal-medullary), also involves the hypothalamus and adrenal glands, but this system works through the brain stem and sympathetic nervous system to produce within the adrenal gland a number of hormones that increase heart rate, blood pressure, and in general prepare the body for a "flight-or-fight response" to an acute episode of stress. These two systems must work together to enable the body to create an effective response to outside stress and turn it off when the stress has passed. However, if the body undergoes prolonged exposure to stress and the corresponding stress response does not turn off, it can be damaging. Available resources may be reduced and the two systems can fail to work in balance, creating runaway anxieties, depression and other symptoms of pathology. These symptoms have a biological connection as seen from animal studies where very young animals that were exposed to stress (premature removal from their mothers, for example) were found to have abnormal and prolonged stimulation of the two stress response systems and

decreased receptor density on the cells that have to respond to this stimuli. Yates suggests that disruption in the balance between the L-HPA and NE-SAM stress-response systems can mediate the relation between early childhood abuse and later pathological outcomes which can include major depressive disorder, anxiety disorder, and posttraumatic stress disorder (PTSD).

Another key discovery is that both physical and psychological stress can trigger a inflammatory response from the immune system (Robles, Glaser, & Kiecolt-Glaser, 2005). High stress levels upset the normal checks and balances of the immune system leading to prolonged high levels of inflammatory cytokines (Dhabhar & McEwen, 2001). Such high inflammatory agents are known to increase the risk of a number of serious health problems; among them are heart disease, chronic pain, premature aging, impaired immune function, and Alzheimer's disease (Frasure-Smith & Lesperance, 2005)

All the references cited above present clear evidence for the potentially dangerous effects of childhood abuse; however, the trajectory from childhood abuse to poor health outcomes may be multi-faceted. Most of the abuse discussed in the references refers to abuses suffered within the home. There is another example of abuse, which occurs outside the home, within the community, that usually takes the form of witnessing or being a victim of violence. This type of violence may follow similar patterns and lead to the same or related outcomes, but it has received less attention. For example childhood abuse has been implicated to obesity, and therefore it seems likely that experiencing or witnessing violence outside the home also has a connection to increasing one's likelihood of being overweight or obese. It also seems probable that the effects of violence will have

other health consequences including the ones that are the subject of this study: general health, physical problems, physical activity, sedentary behavior, and sleep disturbances. These consequences, along with the health-related outcome of obesity, and how they are affected by exposure to community violence will be the focus of this study.

The Obesity Problem

Obesity has become one of the most widespread health-endangering physical condition among children and adolescents in the United States (Ogden, Flegal, Carroll, & Johnson, 2002). Data from two National Health and Nutrition Examination Surveys (NHANES) taken during the periods 1976-1980 and 2007-2008 indicate that the prevalence of overweight children and adolescents is increasing. Using 2000 BMI-for-age criteria (Body Mass Index; see Center for Disease Control and Prevention), the percentage of obese children (ages 6 to 11 years) increased from 6.5% to 17%, and the percentage of adolescents (ages 12 to 19 years) who would be considered overweight or obese increased from 5.0% to 17.6% (Hedley, Ogden, Johnson, Carroll, Curtin, & Flegal, 2004; Ogden, Carroll, & Flegal, 2008; Ogden, Carroll, Curtin, Lamb, & Flegal, 2010). Given these current statistics, the nation will not meet the *Healthy People 2010* target of 5%. Inasmuch as obesity is associated with serious health problems, these high obesity rates are a cause of concern, and successful attempts to reduce these rates would have a corresponding benefit to the health of our youth.

Health Risks Associated with Obesity

Obese children and adolescents are at risk for a long list of emotional, social and health problems, including self-esteem, social difficulties, and physical health

(Gortmaker, Must, Perrien, Sobol, & Dietz, 1993). Specific physical health problems linked with obesity are high blood pressure, osteoarthritis, high levels of cholesterol and triglycerides in the blood, type 2 diabetes, heart disease, and stroke (Freedman, Mei, Srinivasan, Berenson, & Dietz, 2007; Must & Anderson, 2003). Some other more uncommon consequences of childhood obesity are sleep apnea and muscular development (Dietz, 2007). Although obesity-associated diseases occur more frequently in adults, they are becoming increasingly more common in children and adolescents. The CDC reports that one in three children born in the U.S. in 2000 will develop type 2 diabetes some time in their life, a byproduct of the rise in childhood obesity.

The determinants of obesity in the U.S. are complex, but one clear picture emerges. Research indicates that childhood obesity is the main antecedent for adult obesity (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997); that is, children and adolescents who are obese are increasingly more likely to be an obese adult. In a review of the literature from 1970 to 1992, one-third of overweight preschool-age children in the study became obese adults and half of school-age children became obese adults (Serdula, Ivery, Coates, Freedman, Williamson, & Byers, 1993). Fat cells develop during childhood and adolescence that will influence physical development in adulthood. Moreover dietary and activity patterns established in childhood and adolescence become habitual overtime and are difficult to change in adulthood. The conclusion here is that it is imperative to better understand the antecedents of obesity and implement obesity prevention efforts before it becomes a reality later in life.

In addition to the individual health burden, obesity is also a significant financial burden on the U.S. healthcare system (Finkelstein, Fiebelkorn, & Wang, 2003). Using the 1998 Medical Expenditure Panel Survey (MEPS) and the 1996-1997 National Health Interview Surveys (NHIS), the national costs for obesity and obesity related-health issues was roughly 78.5 billion dollars or 9.1 of U.S. medical expenditures. This is in comparison to $147 billion per year directed to obesity issues in 2008 (Finkelstein, Trogdon, Cohen, & Dietz, 2009).

Sleep

We live in a chronically sleep deprived nation; the National Sleep Foundation (NSF) found that adolescents in the U.S. are not getting the sleep they need and that this continues as they move through their teenage years. The NSF recommends 8.5 to 9.25 hours of sleep per night for teenagers between the ages of 11 and 17. Yet the 2006 *Sleep in America* poll taken by NSF of 1,602 adolescents indicated that only 15% of them were meeting these recommendations on school nights. Just under half (45%) reported sleeping less than 8 hours per night on school nights, and high school seniors reported on average only sleeping 6.9 hours. More than half (51%) of adolescents also reported feeling tired or sleep during the day. Among those adolescents who reported feeling sad, depressed, hopeless, nervous, or worrying too much, 73% of them also reported not getting a sufficient amount of sleep, and 59% had feelings of sleepiness during the day. Many of these adolescents (28%) also reported that they felt too tired to exercise over the past two weeks. In contrast those who slept more than 9 hours per night reported positive moods more often.

Sleep is essential for preventing disease and maintaining good health and overall well-being. At the 2007 Society for Research in Child Development, William Dietz proposed sleep as one of the two areas to target for childhood obesity prevention. Chronic insufficient sleep contributes to both sleepiness-related inactivity and potential metabolic changes that can lead to significant weight gain. Spiegel, Leproult, and Van Cauter (1999; 2003) of the University of Chicago, who are leading researchers in sleep loss and circadian rhythms on the endocrine system, found that sleep deprivation, even over a couple of days, can have serious negative effects on the metabolism and hormone levels. In their 1999 study of 11 male participants, they examined how carbohydrate metabolism, thyroid function, and the coordinated activity of the L-HPA axis (as mentioned earlier the L-HPA axis comprises the hypothalamus, pituitary, and adrenal glands that enable the body to respond to stress) were effected after 6 nights of sleep deprivation (4 hours of sleep) and then again after 6 nights of sleep recovery (12 hours of sleep). They found that glucose tolerance, ($p < .02$) and concentrations of thyroid-stimulating hormones ($p < .01$) decreased with sleep deprivation, while evening cortisol (an essential hormone that among other things regulates the body's response to stress) concentrations ($p = .01$) and activity of the sympathetic nervous system (that part of the autonomic nervous system that mobilizes the body for action when one is under stress) increased with sleep deprivation ($p < .02$), all of which are related to weight gain and adiposity.

Knutson, Spiegel, Penev, and Van Cauter's review (2007) on the consequences of sleep deprivation, discussed three primary pathways between insufficient sleep, diabetes, and obesity. These pathways are impairments in the regulation of glucose metabolism,

increase of appetite, and decrease of energy expenditure. Blood sugar levels are regulated by insulin created within the pancreas. The condition where proper blood sugar levels require increasingly higher amounts of insulin is known as insulin resistance. Knutson and colleagues provided a detailed argument that sleep deprivation may impair the ability of the pancreas to regulate glucose levels contributing to the development of insulin resistance, and it is this insulin resistance over time that increases weight. They also pointed out that sleep deprivation disrupts two specific hormones leptin and ghrelin and discussed the contribution that these hormones make to appetite regulation and energy expenditure. Leptin appears to promote satiety and increase energy expenditure while ghrelin, on the other hand, increases appetite and decreases energy expenditure. Data indicate that short sleep duration was associated with a drop in leptin and an increase in ghrelin, leading to feelings of hunger. The two work hand in hand, so disruption of one, or both, will upset the balance between calories in and calories out leading to weight problems.

Other epidemiological studies (e.g., Horne, 2008) indicate that the clinical risks of sleep deprivation (5 hours for adults) may be small but can develop after many years of exposure. Nevertheless these studies suggest that sleepy children tend to exercise less and eat more while other studies (e.g., Lamberg, 2006) propose that sleep, in addition to eating less and exercising more, may by the key to losing weight and preventing obesity especially among our youth. Both cross-sectional and longitudinal studies observed low levels of sleep correlated with BMI across adult and children populations both in the U.S. and other parts of the world, including Canada, France, Germany, Spain, and Japan. In

Spain, Vioque, Torres, and Quiles (2000) found the prevalence of obesity decreased with more sleep, and in Japan, Shigeta, Shigeta, Nakazawa, Nakamura, and Yoshikawa (2001) found those who sleep less than 6 hours were more likely to be obese. U.S. studies also supported that little amounts of sleep increased one's risk of obesity among adults (Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002; Taheri, Lin, Austin, Young & Mignot, 2004; Hasler, 2004) and children (Agras, Hammer, McNicholas, & Kraemer, 2004) and adolescents (Gupta, Mueller, Chan, & Meininger, 2002).

Physical Activity

The physical condition of being overweight or obese results from an energy imbalance: taking in more calories than can be burned off through physical activity. The difference gets stored as fat. Examining factors that determine whether or not a child gets the proper amount of exercise is therefore important, since lack of exercise in children is an antecedent to unhealthy body mass index (BMI) levels in adolescents (Patrick et al., 2004). Increases in physical activity are consistently related to lower BMI (Eisenmann, Bartee, & Wang, 2002). According to the Dietary Guidelines for Americans (U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2005) children and adolescents should participate in at least 60 minutes of moderately intense physical activity most, if not all, days of the week. Caspersen, Powell, and Christenson (1985) defined physical activity as any bodily movement produced by skeletal muscles resulting in energy expenditure. Regular physical activity not only keeps weight down, but it also increases muscular strength, bone mass, and self-esteem while reducing blood pressure, anxiety, and stress. In addition activity level has been indicated in 14% of

deaths such that being active reduces the risk of dying from heart disease, diabetes, cancer, and high blood pressure (McGinnis & Foege, 1993).

Children are less physically active than ever before (CDC, 2008). Recent studies suggest that less than half of our adolescents meet the 2005 Dietary Guidelines for physical activity. In the 1999 national school-based Youth Risk Behavior Survey (YRBS), a component of the Youth Risk Behavior Surveillance System (CDC, 2000), found that of 15,143 students in Grades 9 through 12, only 27% reported that they exceeded the threshold of 30 minutes of moderate activity per day. This may be more of a problem for females since this level of activity among female students was significantly less than among male students (24 % versus 29%), respectively. Vigorous physical activity among high school students is also declining. The more recent YRBS (CDC, 2008) reported that nationwide only 34.7% (male = 43.7%; female = 25.6%) of students were engaging in vigorous activity 5 or more times per week for at least 60 minutes each day, which is far from the physical activity goal of *Healthy People 2010* of 85%. In addition, one in four adolescents (24.9%) did not participate in 60 or more minutes of any kind of physical activity at least one day during the previous seven days.

Despite the clear benefits of physical activity to health (Tremblay, Inman, & Willms, 2000), schools have been reducing or eliminating their physical education programs over the last twenty years (Tremblay, Pella, & Taylor, 1996). With current difficult economic times, physical education opportunities are the first to be impacted. Class sizes are increasing while staff for physical activity programs are decreasing, and

this is most critical for lower income districts in which physical education is the only opportunity in the day for youth to be physically active.

Sedentary Behavior

Sedentary behavior is a quantitative measure of one form of physical inactivity. In the current study it is defined as the amount of time adolescents spend with media, for example television and videogames. It is not the negative of physical exercise; a child could engage in both a high level of physical activity and a high level of sedentary behavior at different times during their day, as it is defined here. Sedentary is becoming an increasingly more critical issue among our youth due to the accessibility of many different forms of technology that traditionally are inactive. One study revealed that 23% of deaths from chronic diseases in the U.S. are due to a sedentary lifestyle (Kujala, Kaprio, Sarna, & Koskenvuo 1998).

In one of the largest studies conducted on the media habits of youth is the Generation M study of the Kaiser Family Foundation (Rideout, Roberts, & Foehr, 2005), which is based on a national sample of 2,032 third to twelve graders (8 to 18 years of age). It examined how long, what types, with whom, and where youth were using media. The study found that our youth live in a media saturated world in which on average they spend nearly 6.5 hours each day with media. This includes television, videos, DVDs, radio, CDs, videogames, computers, and reading (although reading was not included in our definition of sedentary behavior). In addition The Youth Risk Behavior Surveillance Summaries (YRBSS, 2008) report revealed that 35.4% of students watched three or more hours of television (male = 37.5%; female = 33.2%) and 24.9% played video or computer

games or used a computer game (that was not at school) for three of more hours per day (male = 29.1%; female = 20.6%).

In a seminal study examining the relation between television and obesity among children and adolescents (N = 6,965), Dietz and Gortmaker (1985) argue that television viewing predicted weight gain. Among both children (6 to 11) and adolescents (12 to 17), the prevalence of obesity (measured by tricep skinfold) increased with each hour per day of media exposure by 2% even after controlling for baseline weight, season of the year, region, population density, race, socioeconomic status, and other familial variables.

Although a low level of physical activity and a high level of sedentary behavior are not direct serious health problems, there is clear evidence that over time they can be linked to obesity and other health problems (Dietz & Strasburger, 1991). The causes of obesity are complex with a multitude of factors interacting to result in what has come to be regarded by some as a disease. In general 50% of the variation in body weight is inherited (Williamson et al., 2002), so clearly both genetic and environmental factors play a role. Determining a direct link between obesity and all these factors is beyond the scope of this study.

General Health

An important measure in assessing the negative effects of community violence exposure on adolescents' health may also be the adolescents' subjective view of their own health. Community violence exposure may have serious consequences on how adolescents think about their own health. McGee, Liao, Cao, and Coopers' study (1999) provide clear evidence on the strong relation between self-reported health status and

subsequent mortality among both genders and across a wide range of ethnic groups (e.g., Native Americans, Asian/Pacific Islanders, blacks, whites, and Latinos). Their study of over 700,000 adult participants used the one-item questionnaire, "Would you say your health in general is excellent, very good, good, fair, or poor?" Results revealed that a self-report of fair or poor health was associated with increased risk of mortality providing the argument that this one-item measure is a valid indicator of health. Although this study involved adults, it is likely that it also applies to adolescents, and so the subjective judgment of one's own health, using the same one-question criterion, was included in our health-related variables.

Mediators

An important question to address is the underlying mechanism that links community violence exposure to health-related behaviors and outcomes. Are there other variables that are influenced by community violence exposure and in turn affect health outcomes, and if so, what are they and how do they operate? Such other variables include mediators and moderators, which attempt to explain the process in which a predictor causes an outcome. Mediators are commonly used in psychology as well as many other disciplines that require statistical analysis to develop a cause-and-effect relation (e.g., Baron & Kenny, 1986; Preacher & Hayes, 2004; Preacher & Hayes, 2008). Mediation clarifies the nature in which the independent variable indirectly affects the dependent variable through one or more intervening variables; this will be elaborated upon in the analysis section. For now we will describe how and why the mediators used in this study were chosen.

Choosing mediators is not straightforward and is usually based on some conceptual model where one can hypothesize cause and effect relations. Two relevant models for this study have been offered by Kendall-Tackett and Klest (2009) and Guerra and Bradshaw (2008). Kendall-Tackett and Klest have identified five pathways to explain the process from trauma to health. These five pathways comprise physiological, behavioral, social, emotional, and cognitive processes, each of which could lead to poor health, but according to the authors more often combine to create an accumulative deleterious effect.

The Guerra and Bradshaw model attempts to identify factors that both increase the risk of negative health behaviors and factors that decrease the risk. The authors developed a 5-factor model proposing five key attributes of well-adjusted youth. Labeled as *core competencies,* they are: (1) positive sense of self, including self-esteem and positive affect; (2) self-control, including self-regulation and delay of gratification; (3) decision making skills, including social problem-solving skills; (4) moral system of belief, including empathy and moral identity; and (5) social connectedness, including bonding with peers and partners.

These two models (the 5 pathways and the core competencies) offer insight into how community violence exposure affects adolescents' health patterns. This study will examine two potential mediators that include different aspects of both models. The first mediator examined is depression, which falls under the emotional pathway and is the negation of the core competency of positive affect. The second mediator is self-regulation, which falls under the cognitive pathway and the core competency of self-

control. The Diagnostic Statistical Manual of Mental Disorders defines symptoms of depression as including but not limited to feelings of sadness or emptiness, reduced interest in activities, loss of energy, difficulty concentrating, and irritability. Common symptoms in adolescents also include anxiety, anger, and avoidance of social interaction. Individuals suffering from depression may have trouble sleeping or sleep excessively, may lose their appetite or over-eat, or may have higher risk for disease and physical symptoms. Baumeister, Heatherton, and Tice (1994) defined self-regulation as any effort to alter one's own responses. Specifically it is an action, thought, feeling, or desire that overrides the natural or accustomed response for another response (or lack of response). Derryberry and Rothbart (1988) describe regulation as several processes, which include shifting attention, focusing attention, activating control, or inhibiting behavior. Positive affect (the opposite of depression) and self-regulation are basic fundamental skills for healthy living and are often viewed as most critical in the health literature. Specifically community violence exposure may hinder the development of these abilities (i.e. mediators) leading the adolescent to behave in ways that negatively affect such things as their food choices, activity level, mental health, or general health.

One may argue that depression and self-regulation could be use as moderators in the sense that they provide some protection against violence exposure. However, that would assume that violence exposure has no effects on depression levels or the ability to regulate oneself, and as discussed in the next few paragraphs that is not the case. Moreover, there is literature already suggesting that depression may be a key mediator in

explaining the relation between abuse and obesity (Noll, Zeller, Trickett, & Putman, 2007) and abuse and sleep disturbances (Noll, Trickett, Susman, & Putman, 2006).

The Effect of Community Violence Exposure on Mediators

Exposure to community violence has been linked with depression and posttraumatic stress symptoms. A study of 347 adolescents between Grades 7 and 12 examined the relations between violence exposure in home, school, and community and their effects on nine psychological symptoms including interpersonal sensitivity, depression, and anxiety (Lai, 1999). Results indicated that both experiencing and witnessing violence predicted higher levels of depression and self-esteem problems across the three settings, home, school, and community. Many other studies have also shown negative effects on adolescents' mental health due to violence (e.g., Larzman, 2005; Turner, Finkelhor, & Ormrod, 2006; Schwab-Stone, Chen, Greenberger, Silver, Lichtman, & Voyce, 1999).

Cull (1996) found that being a victim of violence affected self-care practices, which she defined as learned purposeful action directed toward maintaining life, health, and well-being. She asserted that experiencing violence decreased the basic skills, motivation and knowledge for self-care, which presumably would make up central components of both our mediators depression and self-regulation. One highlight of her study was that adolescents who scored lowest on self-care also had the most difficulty maintaining a balanced diet and keeping themselves safe.

The Effect of the Mediators on Adolescents' Health

Depression and self-regulation play key roles in how we maintain our health. It may be assumed that if one does not like himself, views himself with little self worth, or in other words exhibits symptoms of depression, he will most likely care little about making efforts to improve his health or may not have the emotional capability to make the effort. In addition, in order to make healthy choices and follow through with associated behaviors the ability to regulate one's behavior has important implications.

Salovey, Rothman, Detweiler, and Steward, (2000) provide an in-depth examination evaluating how emotional states affect physical health. They discussed how positive emotional states could promote healthy perceptions, beliefs, and physical well-being through physiological, cognitive, social and behavioral mechanisms. Positive emotions have been associated with improving immune response and those who are more optimistic may not only cope with health problems more productively and plan for the future, but also their mood may elicit the necessary social support that buffers stressful life experiences. An optimistic mind and exhilarated spirit seem to accompany a health body. Other research has consistently supported the notion that the physiological and psychological symptoms go hand in hand (Kasl & Cobb, 1967). Given the strong connection between physical health and so many psychological and social factors, it seems clear that this connection also applies to the specific mediators discussed here.

When examining the relation between depression and the specific health outcome, obesity, the literature clearly demonstrates that they are related (Mustillo, Worthman, Erkanli, Keeler, Angold, & Costello, 2003; Pine, Goldstein, Wolk, & Weissman, 2001).

Using The National Longitudinal Study of Adolescent Health dataset (Add Health, which was also used for this study), Swallen, Reither, Haas, and Meier (2005) examined whether BMI related to a quality of life measure that consisted of general health, physical health, emotional health, and a school and social functioning assessment among 4743 adolescents, Grades 7 through 12. Using a cross-sectional design, they found poorer emotional and school/social functioning related to higher BMI, but only for those between ages 12 and 14. For this age group, being overweight or obese increased the risk for depression, low self-esteem, and poor school/social functioning. Other cross-sectional studies investigating obesity and emotional health produced similar results across the entire age range of adolescence (e.g., Pinhas-Hamiel, Singer, Pilpel, Fradkin, Modan, & Reichman, 2006); in these studies, they found increases in negative self-image and decreases in self-esteem correlated with increases in BMI levels.

There is some controversy about the direction of the cause and effect relation between depression and obesity. Obese and overweight youth are highly stigmatized and confront discrimination regularly across multiple settings. They are often rated as less likeable, and are the subject of negative social attitudes. Of more concern is that overweight individuals are blamed for their weight problems. They often have to face stereotypes that assert their weight is controllable and caused by their gluttonous or lazy behavior, which can lead to feelings of rejection, shame, or depression. However, what remains unclear is whether obesity leads to depression or depression causes obesity.

A prospective study of 9,374 adolescents in Grades 7 through 12 using the Add Health dataset found that depression at baseline was not associated with obesity at

baseline but was predictive of obesity one year later (Goodman & Whitaker, 2002). Stunkard, Faith, and Allison (2003) also attempted to tease apart the relation between depression and obesity and found that depression in adolescents predicted a greater BMI as an adult. Despite these findings, they concluded that depression and obesity co-occur and that an adverse childhood experience may be triggering the development of both. Stunkard et al. hypothesize that a physiologic mechanism due to stressful experiences may be affecting both depression and obesity. When cortisol levels become elevated, indicating hypothalamic-pituitary-adrenal (HPA) activation, there may be more risk for a build up of abdominal fat along the stomach wall. In fact depression activates the HPA axis; therefore the HPA axis appears to share some responsibility for the relation between obesity and depression (Bjorntorp & Rosmond, 2000).

Infrequent exercise has also been linked with experiencing loneliness, shyness, and feelings of hopelessness (Strauss, Rodzilsky, Burack, & Colin, 2001), which are again closely associated with depression.

Self-regulation has consistently demonstrated its importance in our ability to maintain our health. A study of 539 9th graders in a metropolitan area investigated the relations between self-control and eating and physical activity habits (Wills, Isasi, Mendoza, & Ainette, 2007). Findings supported that good self-control related to more fruit and vegetable intake, more participation in sports, and less sedentary behavior while poor self-control related to more saturated fats intake and less physical activity even after controlling for gender, ethnicity, parental education. Other studies have indicated similar results such that increases in self-regulation correlated with increases in moderate and

vigorous physical activity (Mowad, 2007) and self-regulatory cognitions predicted both healthy and unhealthy eating (Kalavana, Maes, & De Gucht, 2010). It is not surprising self-regulation plays such a central role in our health; these skills require self-discipline to maintain a healthy diet, avoid unhealthy foods and drinks, and spend time regularly exercising.

Alternatively, positive motivation and high skill level, which are related (or opposite to) our mediators, play roles in predicting healthy behavior and have been made part of several conceptual health models (e.g., The Health Belief Model, Rosenstock, 1966; the Theory of Health Preventive Behavior, Kasl & Cobb, 1966).

Moderator

The role of gender as a moderator will be examined since there may be important differences between boys and girls that need to be noted in these relations. For example, violence for boys is more normative compared to girls (CDC, 2008; Department of Health and Human Services (DHHS), 2001), and therefore it is more likely less damaging. Specifically violence exposure may be considered less critical in developmental disruption. For females, the effect might be more marked because it is non-normative, making them more susceptible to consequences of violence. In addition boys may react differently than girls to community violence exposure. Boys may react by losing self-control (assessed by self-regulation) triggered by feelings of retaliation or vulnerability to the violence. On the other hand, it is more socially acceptable for females to express a more affect response to violence, displaying feelings of depression or emotional instability. Swallen and colleagues (2005), in fact, found that girls who were

exposed to community violence were more likely than boys to report poorer general, physical, emotional, and school/social health.

Other Variables

The other independent variables (covariates) in the study will include, in addition to the mediators and gender, childhood maltreatment, and socioeconomic status (SES).

Childhood maltreatment. As previously discussed childhood abuse has been implicated in health-related problems to a considerable degree; therefore, this study will take into account the effects of early maltreatment on the above-mentioned health issues.

Socioeconomic status (SES). Previous research using national data has found that child abuse correlates more highly with income or education level (e.g., socioeconomic status (SES)) than race or ethnicity (Straus & Smith, 1990). It is theorized that low SES families may be more at risk for child abuse due to higher levels of stress that far outweigh coping resources. Along the same lines, low SES families may only be able to afford homes in violent neighborhoods. In addition another study using the Add Health dataset found that both lower household income and lower parental education each were associated with depression and obesity (Goodman, Slap, & Huan, 2003). Only SES will be controlled for as a social indicator and not race or ethnicity.

Age. Analyses were conducted separately for early and late adolescence. Adolescents sixteen years of age and older were categorized in the late adolescence group while those younger than sixteen formed the early adolescence group. Sixteen was determined the cut off age since it is the legal age that adolescents may obtain their driver's license allowing them substantially more independence in the choices they make.

The opportunity to drive is arguably the major contributor to an adolescent's sense of independence. The boundaries of their world expand dramatically as they no longer require their parents to drive them places. In the process older adolescents are freer to choose friends who may be unknown to the parents. Adolescents are very impressionable, and for good or bad, these friends may have a greater influence on an adolescent's decisions around food they eat and activity they undertake than do the parents.

Organizational Framework

The aims of this study fit conceptually within an organizational framework of development. According to this approach, development is viewed as a series of life events and behaviors that organize themselves and integrate in a cause and effect way (Yates, Dodds, Sroufe, & Egeland, 2003). Early experiences establish a foundation from which later experiences are built and shaped. In the context of this paper, the model posits that early exposure to community violence influences development and sets the stage for deleterious events, patterns, and behaviors that lead to poor health-related outcomes. It is not necessary or inevitable that this will occur; it is just that the developmental process tends to self organize around certain profound experiences, and earlier experiences generally are more influential since they impact subsequent adjustment and development.

Here, the organizational process begins with exposure to community violence during childhood or adolescence. Within the organizational framework experiencing violence during childhood is expected to have stronger effects than does similar violence experienced during adolescence. Nevertheless it may be more appropriate when

exploring health outcomes since this period is considered a time of initiating and establishing eating habits, activity choices, and sleeping patterns independent of parents or caregivers.

The course of events that organize themselves from this exposure to violence is the resulting decrease (or enhancement) of the two skills tested as mediators. Depending on how community violence exposure influences self-regulation and emotional health will determine the outcomes of the health-related issues: obesity, general health, physical problems, level of activity, and sleep patterns. It is expected that depression levels will increase while self-regulation skills will diminish under the influence of violence and the corresponding health-related outcomes will be negative.

The Current Study

The current study will expand upon the well-established fact that if an adolescent is exposed to violence in any form it will have a negative impact on his or her health. The purpose of this paper is to first investigate whether exposure to violence in community predicts health-related outcomes and second to understand the organizational pattern in this relation, specifically whether it is mediated by an individual's feelings of depression and poor self-regulation and moderated by gender.

The aspects of health that are impacted will also be specified. Six were singled out, which include both health-influencing behaviors, such as physical activity, and health outcomes, such as obesity. Since obesity is a serious health problem among adolescents and is growing at an alarming rate, it is a primary focus of this study. In fact most of the other health-related variables were chosen because they are commonly

regarded as influencing obesity. There are of course many other aspects of health than the six chosen here. However, the ones chosen represent a good characterization of an adolescent's overall physical health and predisposition toward obesity.

The study can be framed around the following questions about how community violence exposure could affect health and be a lead-up to obesity:

1). What are the concurrent relations between community violence exposure and adolescents' health? What are the effects of community violence exposure on adolescents' health one year later? Are the patterns of these relations different across early and late adolescence? It is expected that adolescents who were exposed to community violence will have worse general health, more physical problems, higher BMI levels, and poorer sleep patterns. They will also be less physically active and more sedentary than those adolescents who were not exposed to community violence. It is also predicted that community violence exposure will continue to have a similar effect on health one year later. Although older adolescents' opportunities for community violence exposure are greater and their parental supervision is less, it is expected that the exposure will have a more deleterious effect on younger adolescents than their older counterpart. Due to their age, they are less resilient to the stress caused by the violence and are less mature and able to cope.

2). Do depression and self-regulation mediate the concurrent relations between community violence exposure and adolescents' health? Do they also mediate the effect of community violence exposure on adolescents' health one year later? Are the patterns of these relations different across early and late adolescence? It is expected that there will

be a mediating effect through both depression and self-regulation between community violence exposure and the specific health-related variables. More specifically, being exposed to community violence will predict increases in depression and decreases in self-regulation which in turn will diminish general health, increase physical problems, raise BMI levels, harm sleep patterns, decrease physical activity, and finally increase sedentary behavior, in concurrent analyses. It is expected that this mediation model will hold one year later. Again, it is also expected that stronger relations will exist among younger adolescents when compared to older adolescents.

3). In what way does the effect of community violence exposure on adolescents' health differ between girls and boys? It is expected that girls will respond to community violence exposure differently than boys. Although boys may be exposed to more community violence, girls may be more sensitive to the violence and become more withdrawn and depressed, and consequently their health may be more negatively impacted.

Part Two:

Method

Method

Participants and Procedures

This research has been drawn from a larger, multi-year, nationally representative research study known as The National Longitudinal Study of Adolescent Health (Add Health), conducted out of the University of North Carolina at Chapel Hill. Four waves of data have been collected that investigate adolescent health-related behaviors from Grades 7 through 12 and their outcomes in young adulthood. The National Institute of Child Health and Human Development (NICHD) in associated with 17 other federal agencies funded the Add Health program. Add Health is one of the most comprehensive surveys of adolescents to examine the influence of the individual, family, school, and community on health behaviors. Two waves of data, separated by one year, were collected between 1994 and 1996. Six years later, in 2001 and 2002, a third wave of data was collected on the same adolescents, who were by this time between the ages of 18 and 26. This was followed by a continuation of data collection in 2007 and 2008 comprising the fourth wave when participants were between the ages of 24 and 32.

For this particular study, however, only data from Wave I and II with a total of 14,723 participants (49% males). Of the participants interviewed at both waves, 51.1% were white, 21.5% were African American, 16.5% were Hispanic, 6.8% were Asian, 2.6% were American Indian, and 1.5% reported no ethnic identity. The economic breakdown of this sample was similar to that of the national income distribution. Household income of participants was assessed at Wave I and used to categorize family income into quintiles as defined by the U.S. Census Bureau. According to their historical

household income tables, upper limits for each quintile of household income were as follows: quintile 1 = $13,426, quintile 2 = $25,200, quintile 3 = $40,100, quintile 4 = $62,841 dollars, and finally quintile 5 consisted of families making over 62,841 dollars. Of families who reported their income 14.2% were in the lowest quintile, 19.1% were in the 2nd quintile, 22.7% were in the 3rd quintile, 24.1% were in the 4th quintile, and 20% were in the 5th quintile. As expected many families did not report their income. Twenty three percent of responses were left blank or were believed to be entered incorrectly, which were then recoded as missing.

The sampling frame consisted of 80 high schools and 52 associated "feeder" middle schools selected systematically to ensure a representative sample with respect to region of country, level of urbanization, school size, school type, and ethnicity. Only schools that included an eleventh grade and had at least 30 students were eligible. Of those eligible, 88% participated in the study. Wave I completed 20,745 adolescent In-Home Interviews in 1995 with Wave II completing just fewer than fifteen thousand. For this study, analyses were based only on participants who gave what were believed to be valid responses.

Established consent procedures defined by the institutional review board of the University of North Carolina at Chapel Hill were used for collecting data. Data were collected with the idea that families, friends, schools, and communities play a role in adolescents' lives, and these influences may encourage healthy or harmful behavior. Waves I and II examined possible influences on adolescents' behavior while Wave III assessed six years later investigated the transition to adulthood and how decisions made

as an adolescent influenced outcomes in emergent adulthood. For this study, only childhood maltreatment, which retrospectively asked adult participants about past abuses, was used from Wave III. Because of the sensitive nature of the subject of childhood maltreatment, it could not be asked in Waves I and II while participants were still adolescents. Although data were collected from students, parents, school administrators, and others, only those collected from the students themselves were used for this study.

A trained researcher conducted the interview at the participant's home. No paper questionnaires were used in order to maintain confidentiality. Instead data were recorded on laptop computers. When the questions were deemed less sensitive, the interviewer read the questions to the participant and entered the appropriate response. For more sensitive material, participants entered their own responses in private to ensure a sense of comfort and confidentiality. On average, interviews were completed in 134 minutes. Approximately 90 minutes consisted of the laptop section while the remaining time was required to for researchers to document a physical description and health assessment of the participant.

Constructs Assessed and Measures

Data collected from the Add Health study were from a wide range of disciplines in the social and behavioral sciences. This particular study examined one predictor (community violence exposure) assessed at Wave I and six outcomes (general health, physical problems, adiposity (BMI), sleep patterns, physical activity, and sedentary behavior) as derived from both the Wave I and Wave II datasets. Based on constructs included in Add Health, two mediating factors (depression and self-regulation) were

formed using Wave I. All covariates included were created using Wave I except for early maltreatment which due to the sensitivity of the question was only assessed at Wave III when participants were adults.

Principal components analysis (PCA) was run to confirm how well items comprising each measure held together to form one or multiple components. PCA was conducted using varimax rotation, and an Eigen value of 1.0 was used to determine the number of components. The scree plot was also performed to confirm the number of components. Results confirmed one component was found for each measure except physical problems, which resulted in two components. However, all thirteen items comprising physical problems were retained as one scale since theoretically all assessed some type of physical health problem.

Wave I

Predictor

Community Violence Exposure (CVE). The predictor variable, community violence exposure, was created from a 5-item measure of the extent to which participants had witnessed or had been a victim of violence within the community ever in their life. The items include (1) witnessing someone being shot or stabbed, (2) being threatened with a knife or gun, (3) being shot at, (4) being stabbed, and (5) being physically assaulted. These items are commonly employed to measure community violence exposure (Guterman, Hahm, & Cameron, 2002; Selner-O'Hagan, Kindlon, Buka, Raudenbush, & Earls, 1998). Using ordinal categories, participants reported 0 (never), 1 (once), or 2 (more than once). Since experiencing violence exposure within one's

community is not common among most adolescents, the five items were dichotomized to zero and one. The sum of the five items made up each participant's final community violence exposure score, which ranged from 0 to 5. The distribution was highly skewed toward zero, with about 75% reporting no community violence exposure; however, it should be noted that this is considered normal for violence exposure in most communities.

Mediators

Depression. A 10-item scale was employed to evaluate how often in the past week participants experienced depressed symptoms. Participants reported their emotional state and feelings using a 4-point scale: 0 (never or rarely), 1 (sometimes), 2 (a lot of the time), and 3 (most or all of the time), which ranged from 0 to 30. The participants were asked questions such as, "were you recently bothered by things that usually don't bother you" and "have you recently felt depressed." Items were coded assigning higher scores to greater levels of depression (reliability measure, $\alpha = .82$).

Self-regulation. This 3-item measure asked participants to rate their ability to regulate their behavior during the last week. Items were coded so that higher scores reflected poor self-regulation (e.g. participants were asked questions suggesting lack of self-regulation such as, "did you have trouble keeping your mind on what you were doing" and "was it hard to get started doing things"). Participants responded using a 4-point scale from 0 (never or rarely) to 3 (most or all of the time) ($\alpha = .62$), which ranged from 0 to 9.

Wave I and II

Health Outcomes

General Health. This one-item measure was self-reported and asked participants one question, "In general, how is your health?" Participants reported their subjective level of health on a 5-point scale (1 = poor; 2 = fair, 3 = good, 4 = very good, and 5 = excellent).

Physical Problems. This was assessed by asking participants whether they had experienced any one or more of 13 different physical problems in the past 12 months (e.g., headache, stomachaches, dizziness, sore throat, or chest pains). For each condition, respondents selected 0 (no) or 1 (yes), creating a scale ranging from 0 to 13. The sum of these responses formed their overall physical-problem score (α = .79 and .78 for Wave I and II, respectively). Similar scales were used in previous studies (e.g., Rhee, 2005).

Adiposity (Body Mass Index (BMI)). Adiposity was measured via body mass index. BMI was calculated using self-reported height and weight data and can serve as a way of measuring adiposity (BMI formula = (weight in pounds/(height in inches)^2)*703). Because participants were adolescents and would continue to grow, their BMI scores depended on both age and gender. Therefore each BMI measurement was calibrated to an age- and gender-dependent table using the 2000 CDC BMI-for-age growth charts. The table consists of a series of curves that indicate where a given BMI measurement for an adolescent of a certain age and gender falls relative to a representative range of BMI scores for all adolescents of the same age and gender. The score is indicated as a percentage of the total range of the representative sample. The BMI-for-age growth

charts were developed from five national datasets, the National Health Examination Surveys (NHES) 2 and 3 and the National Health and Nutrition Examination Survey (NHANES) I, II, and III taken from 1963 to 1994 (Ogden, Flegal, Carroll et al., 2002). The criterion for adolescent obesity was defined as greater than the 95th percentile of the estimated BMI ranges (The Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services). The criterion for overweight status was defined as between the 85th percentile and 95th percentile (U.S. National Center for Health Statistics). A score of 100 in the table, for example, represents the highest BMI measured within CDC's representative sample. Therefore it is possible that someone today could have a score greater than 100, although none were measured in this survey. A score of zero or less was regarded as anomalous and recoded as missing. The total of the calibrated BMI scores was therefore between .10 and 100.

Sleep. Two scales were used in order to assess both the quantity and quality of sleep. Participants were first asked how many hours of sleep per night they usually get and then for a quality measure they were asked whether they usually get enough sleep. Responses for the quality-of-sleep measure were coded as 0 (no, they did not get enough sleep) or 1 (yes, they did). Quantity of sleep was scored by the number of hours of sleep per night. Reported hours of sleep less than 3 or more than 16 hours were considered invalid and recoded as missing. In order to balance the two scores, they were separately standardized and then averaged to make up the participants' final sleep score.

Physical Activity. Physical activity was measured employing methods used in previously published studies (Add Health; Gordon-Larsen, McMurray, & Popkin, 1999;

Knutson, 2005; Nelson, Gordon-Larsen, Adair, & Popkin, 2005). Using a 7-day recall (times/week), participants reported the frequency in which they partook in three different types of activities: (1) bicycling and skating, (2) active sports such as soccer or swimming, etc. (3) exercise such as jogging or dancing. Possible responses included 0 (none), 1 (1 to 2 times per week), 2 (3 to 4 times per week), and 3 (5 or more times per week). Participants' physical activity score was the sum of their responses, which ranged from 0 to 9.

Sedentary Behavior. Far less attention has been focused on quantifying sedentary behaviors than there has been on physical activity; the reliability and validity of sedentary behavior data has been minimally reviewed in the literature. Following guidelines outlined by Nelson, Gordon-Larsen, Adair, and Popkin (2005) whose work also examined sedentary behavior using the Add Health dataset, adolescents' sedentary behavior was measured by the amount of time spent in 3 sedentary activities: (1) watching television, (2) playing video games, and (3) being on the computer. Using a 7-day recall, participants entered the number of hours per week spent on each activity (range = 0 to 168). A final sedentary score was calculated by summing the responses. This wide range of scores was compressed by taking its square root for the regression analyses. Compressing the score prevents a few participants with anomalously high scores from having too large an influence in the regression. It also implies that sedentary behavior is nonlinearly related to CVE in a complicated way that cannot be determined in linear regression. Such a determination is beyond the scope of this study, which seeks only to establish a cause and effect relation.

Covariates

Socioeconomic Status (SES). SES was determined by parents' report of household income for the year 1994. Households were then divided into quintiles according to their gross income in which each quintile represents 20%, or one fifth, of all households. Therefore, family income was categorized into quintiles defined by the U.S. Census Bureau, Housing and Household Economic Statistics Division (U.S. Census Bureau, Current Population Survey, Annual Social and Economic Supplements, Historical Income Tables – Families, 1994). Quintiles were used rather than their specific income in dollars in order to account for inflation and household type. Household type has demonstrated to be strongly correlated with household income. The lowest quintile indicates poverty.

As mentioned previously the income quintiles are as follows: the first quintile consisted of families with an income of 13,426 dollars or less; the 2nd quintile consisted of families who made between 13,426 and 25,200 dollars; the 3rd quintile consisted of families who made between 25,200 and 40,100 dollars; the 4th quintile consisted of families who made between 40,100 and 62,841 dollars; and finally the 5th quintile consisted of families who made more than 62,841 dollars. The scores ranged from 1 to 5 according to the appropriate quintile.

Childhood Maltreatment. This was a 3-item scale measuring the number of times the participant experienced neglect, physical, or sexual abuse as a child (e.g., "By the time you started 6th grade, how often had your parents or other adult care-givers slapped, hit, or kicked you?"). In the Add Health survey the scores were rated as: 0 (never), 1 (1

time), 2 (2 times), 3 (3 to 5 times), 4 (6 to10 times), and 5 (more than 10 times). However because of the relatively low rate of abuse, each of the items were dichotomized to 0 (no) and 1 (yes) and summed to create the final score, which ranged from 0 to 3.

Parental Weight Status. Participants provided self-reports on whether either their biological mother or father was obese, responding 0 (no) or 1 (yes). The sum of these two items formed the parental weight status score, which therefore ranged from 0 to 2. These questions were taken from the public-use dataset from Wave I. Half of the Wave I participants (approximately 6,500) were randomly selected to complete this part of the survey.

Analytic Procedures

Analyses for each specific aim were conducted as explained below using the statistical program SPSS. Descriptive and correlation statistics were first analyzed, and then independent samples t-tests were calculated to determine if early adolescents' experiences might differ substantially from those of older adolescents, who are given more opportunities to make their own decisions, particularly with respect to healthy versus unhealthy choices.

Second a series of hierarchical multiple regression analyses were run to determine the unique effect of community violence exposure (CVE) on each of the six health-related outcomes (see Figure 1). Hierarchical multiple regression analysis, as used in this study, was a two-step process. Step one regressed each health-related outcome on gender, SES, and childhood maltreatment. Step two added community violence exposure to the equation to determine how well the health-related variables were predicted beyond that of

the covariates used in step 1. Parents' weight status was included as an additional covariate for the BMI analysis, and for the longitudinal analyses baseline health values, as measured at Time 1, were included as additional covariates. The statistical measure of improvement will be the change in R-squared, the ratio of variance between the predicted health outcomes and the actual outcomes, or in other words, the amount of variance in the measured outcomes that was accounted for by community violence exposure. The equations in the regression analyses at step 1 were: $H(i) = B_1(i)*(\text{gender}) + B_2(i)*(\text{socioeconomic status}) + B_3(i)*(\text{childhood maltreatment})$, where H(i) represented the i-th health-related outcome, and the B's were the partial regression coefficients or the solutions to the regression equations. In the case of Time 2, the baseline health assessments were also added as independent variables at step 1. The regression equations at step 2 are the same, but with the addition of community violence exposure ($B_4(i)*(\text{community violence exposure})$).

Third the question of multiple mediation was analyzed using methods outlined by Preacher and Hayes (2004, 2008). The two proposed mediators, depression and self-regulation, were introduced into the equations for each health-related outcome and analyzed using multiple mediation regression and bootstrapping, which computes the mediation effects and the confidence intervals of the mediators. The relations among the independent variables, mediators, and dependent variable are shown in the path diagram in Figure 2. The two mediators are located between community violence exposure and the outcomes, first being influenced by CVE and in turn exerting influence on the outcomes. The regression coefficients associated with the paths through the mediators are

known as the indirect effect and defined as the sum of the products a_1*b_1 (depression) and a_2*b_2 (self-regulation). There is also a direct effect of CVE on the outcome through path c'. The sum of the indirect effect and the direct effect equals the total effect, c; that is, $c = c' + (a_1*b_1 + a_2*b_2)$. This method was preferred because unlike previous methods, multiple mediation allowed us to simultaneously test the unique contribution of each mediator in the context of a multiple mediator model. The confidence intervals for the mediating effect were determined by bootstrapping as recommended by Preacher and Hayes (2008).

The path diagram in Figure 3 implies the equations used to investigate the direct (c') and indirect paths (a_1, b_1, a_2, and b_2). They were: $H(i) = b_1*m_1 + b_2*m_2 + c'*x + 3$ covariates + 1 moderator term, where $m_1 = a_1*x$ and $m_2 = a_2*x$. The character H(i) represents one of the six health-related variables, x represents community violence exposure, and the m's are the mediators. The moderator term is explained below.

Mediating variables play an important role in psychological research. They allow us to better understand the mechanisms through which one variable affects another. Previous methods have been used to assess simple mediation hypotheses (i.e., a one-mediator model). The most referenced method is Baron and Kenny's mediation hypothesis (1986) which establishes mediation if three steps are met: (1) the predictor must correlate with the outcome, (2) the predictor must correlate with the mediating variable, and (3) the mediator must correlate with the outcome, while controlling for the predictor. Again this causal steps approach by Baron and Kenny is restricted to testing a single mediator at a time; our current study aims to test two pathways simultaneously. A

common test to determine the significance of the indirect effect has been the Sobel test (1982), but this assumes a normal distribution of the mediator. A more recent and increasingly more popular alternative to test mediation is bootstrapping (Shrout & Bolger, 2002) which makes no assumption as to the distribution of the mediating variables.

The bootstrapping procedure is a re-sampling technique and is accomplished by taking a number of random samples from the original, large sample population (where the number of selected samples is less than the number in the original sample). The indirect effects are computed using these smaller number of samples. The selected samples are then returned to the original population, (this is known as bootstrapping with replacement), a new random selection is made, and another regression is performed. The process is repeated, typically 1000 to 5000 times. The average of these thousands of estimates of indirect effects is the point estimate of a_1, b_1, a_2, and b_2. The distributions of these thousands of estimates determine the confidence interval (CI). For example, if the CI were 95%, the lower bound would be the score that is below 2.5% of all the other numbers, and the upper bound is the score that is above 97.5% of all the other numbers. For a thousand estimates the lower bound of the CI is the 25^{th} estimate, while the upper bound is the 976^{th} estimate when ordered sequentially. The indirect effect is considered statistically significant if the 95%CI does not contain zero. As mentioned above, the sampling distribution can be non-normal and highly asymmetry about the mean.

Preacher and Hayes (2004; Hayes, 2009) make the argument that multiple mediation and bootstrapping add validity and generality to the mediation model. Multiple

mediators are necessary in most cases since the relation between the predictor and dependent variable is typically explained by more than one path, and the distribution of the mediator variables in unlikely to be normal. A comparison study by Williams and MacKinnon (2008) found that confidence intervals obtained by bootstrapping were better than the standard deviations obtained by the Sobel method. The practical implication is that the potential for Type I error (i.e. concluding the null hypothesis is false when in fact it is not) is reduced.

Finally, the interaction between gender and community violence was created and included in the multiple mediation models to test for moderation by gender. Both mediators and moderators are introduced into the analysis as independent variables, but the way they influence the outcome is different. Mediators and moderators are commonly confused, but can be distinguished by the structure of the analysis. As mentioned above, a mediator operates actively and sits between the predictor and the outcome (see Figure 3). A moderator, on the other hand, works passively and specifies when a certain relation will hold more for one group than another (such as only for males, or only for females). It often is used to subdivide the predictor into subgroups in order to determine if certain outcomes are more sensitive to a subgroup (refer to Kraemer, Wilson, Fairburn, & Agras, 2002).

Part Three:

Results

Results

Descriptive Statistics for Total Sample

Community Violence Exposure. At Time 1, 74% reported never having been exposed to violence within their community. Of the 26% who reported some type of exposure, roughly 15% had been exposed to violence outside their home no more than once in their life, 6% reported two times in their life, and 5% reported three or more times. In a breakdown of the types of violence exposures participants experienced, roughly 12% witnessed someone being shot or stabbed, 13% had a knife or gun pulled on them, only 1% were shot, 5% were stabbed, and 12% were assaulted. Since these percentages total more than the 26%, it is apparent that many adolescents experienced more than one type of community violence exposure.

Childhood Maltreatment. Roughly 66% of participants reported never being maltreated (sexual abuse, physically abuse, and/or feeling neglected). Approximately 26% had been abused once in their life while the remaining 8% had been abused two or more times. The most common form of abuse experienced among these adolescents was physical (22%); only 4% reported being sexually abused. Since physical abuse was reported most frequently, if childhood maltreatment did not indicate an effect on any of the six health variables, then physical abuse alone was also tested (only in question 2) to ensure being victimized within the home was captured as a control variable (see Tables 12 and 13). The Pearson r correlation between childhood maltreatment and community violence exposure was significant ($r = .116$, $p < .01$), indicating that those adolescents exposed to community violence may also have experienced some type of maltreatment.

These violence statistics indicate that indeed adolescents from this current sample were exposed to violence within their community and were victim to abuse within the home at an early age. Consequences of violence exposure within the community were explored while controlling for possible violence within the home.

Health. For our sample, roughly 25% of those assessed reported only good general health with about 7% stating their health as fair or poor. Just about half (Time 1 = 45% and Time 2 = 48%) reported experiencing 7 or more physical symptoms in the past year. Somewhat higher than national trends, approximately 15% of participants were overweight and 11% were obese, as defined by the CDC criteria described above. At Time 1, 38% and at Time 2, 45% of participants did not meet national recommendation for adolescents for hours of sleep (seven or less hours of sleep per night as defined by the National Sleep Foundation is inadequate for adolescents). In addition roughly a third reported they regularly do not sleep enough. Over the one-year period, roughly one in five adolescents engaged in no physical activity or only one to two times per week. Approximately a third also spent more than 25 hours per week engaged in sedentary activities.

The means and standard deviations for all study variables used from Time 1 and 2 are summarized in Table 1. The sample means and sample standard deviations are further separated by gender and age and presented in Tables 2 and 3, respectively. A paired samples *t*-test and independent samples *t*-tests were conducted to determine whether the differences in the means among the various subgroups were significant with respect to each other as well as over time. The *t* scores are computed as the difference in the sample

means divided by the standard deviation of the means. The standard deviation of the means is equal to the sample standard deviation divided by the square root of the number of samples, so in effect the *t* score increases with the square root of the number of samples. Since the number of samples in our analysis was large (approximately 14,000 for the total sample and 7,000 for the subgroups), the *t* scores were subsequently large, contributing to the significant differences in means about all the subgroups and over time.

Correlational analyses were conducted to determine whether CVE at Time 1 related first to both mediators at Time 1 and second to each of the six health-related variables concurrently at Time 1 as well as later at Time 2 (Table 4). CVE correlated positively with depression at Time 1 ($r = .188$, $p < .01$) and poor self-regulation **(**$r = .149$, $p < .01$). In addition CVE correlated with all health-related variables at both time periods: 1) general health at Time 1 ($r = -.061$, $p < .01$) and Time 2 ($r = -.068$, $p < .01$); 2) physical problems at Time 1 ($r = .070$, $p < .01$) and Time 2 ($r = .068$, $p < .01$); 3) BMI at Time 1 ($r = .053$, $p < .01$) and Time 2 ($r = .055$, $p < .01$); 4) sleep quantity at Time 1 ($r = -.073$, $p < .01$) and Time 2 ($r = -.066$, $p < .01$) and sleep quality at Time 1 (($r = -.056$, $p < .01$ and Time 2 ($r = -.043$, $p < .01$); 5) physical activity at Time 1 ($r = .062$, $p < .01$) and Time 2 ($r = .043$, $p < .01$); and 6) sedentary behavior at Time 1 ($r = .085$, $p < .01$) and Time 2 ($r = .083$, $p < .01$). Depression and self-regulation also correlated with all health variables at both time periods.

In general the correlation coefficients were small, but as seen from the asterisks most of them were significantly different from zero. Note the very small correlation coefficients between sedentary behavior and physical activity indicating that they can be

mutually exclusive. Note also that the largest correlation coefficients were between a health variable at Time 1 with the corresponding variable at Time 2. Although this is to be expected, it is surprising that the coefficients were not larger. Adolescents' perception of their health had changed over time.

The correlations of gender and age group with the mediators and health variables are reported in Table 5. The gender correlations were computed using gender as a dichotomous variable with males as 0 and females as 1. Negative correlation coefficients indicate higher scores for males while positive coefficients indicate higher scores females. At both time periods, the gender difference in correlations implied that males in general had better general health, higher BMI percentiles, better sleep patterns, more physical activity, and more sedentary behavior. Females, one the other hand, reported more physical problems across time in addition to being more depressed and exhibiting lower levels of self-regulation.

Tables 6 and 7 are correlations between CVE, the mediators, and the health variables at both time points. Table 6 separates the participants by gender, and Table 7 separates them by age. Correlations were computed by separating the adolescents in appropriate groups and computing separate analyses for each group, further quantifying the gender and age group differences seen in Table 5. Results revealed that the differences in correlations between gender and age group were small.

Descriptive Statistics by Gender

The means and standard deviations for the study variables by gender and across time are summarized in Table 2. A paired samples t-test indicated that both males' and

females' health changed over the one-year period with two exceptions: no significant change was found in males' BMI level or females' general health (Table 2). Next independent samples t-tests were conducted to further investigate the differences between males and females across study variables. Results revealed mean gender differences for the study's predictor, mediators, and health-related outcomes.

For community violence exposure, as predicted the mean number of exposures for males (M = .60, SD = 1.04) was reliably greater than for females (M = .27, SD = .66), $t(14605) = 23.48$, $p < .01$. Higher levels of depression and poorer self-regulation skills were more common among females (M = 5.26, SD = 4.58; M = 2.28, SD = 1.77) than males (M = 4.08, SD = 3.64; M = 2.12, SD = 1.64), $t(14661) = -17.23$, $p < .01$ and $t(14693) = -5.61$, $p < .01$, respectively. As seen in Table 2, males reported better general health at both Time 1, $t(14719) = 10.57$, $p < .01$), and Time 2 $t(14718) = 12.37$, $p < .01$, and less physical problems at both Time 1, $t(14699) = -15.89$, $p < .01$, and Time 2, $t(14695) = -19.53$, $p < .01$, than females. Males also presented higher BMI percentiles at both Time 1, $t(14376) = 3.38$, $p < .01$, and Time 2, $t(14179) = 4.87$, $p < .01$. For sleep, males slept more at both Time 1, $t(14682) = 4.11$, $p < .01$, and Time 2, $t(14674) = 5.31$, $p < .01$, and better at Time 1, $t(14712) = 9.59$, $p < .01$, and Time 2, $t(14714) = 10.32$, $p < .01$. Finally, males spent more of their time than females being physically active at Time 1, $t(14721) = 26.27$, $p < .01$, and Time 2, $t(14719) = 24.96$, $p < .01$, and sedentary at Time 1, $t(14657) = 15.05$, $p < .01$, and Time 2, $t(14644) = 15.15$, $p < .01$.

Descriptive Statistics by Age

The means and standard deviations for the study variables by age group and across time are summarized in Table 3. A paired samples t-test revealed that each age group's health significantly changed over the one-year period except for younger adolescents' BMI level (Table 3). Next independent samples t-tests were conducted to further investigate the differences between younger and older adolescents across study variables. Younger adolescents' responses differed from those of older adolescents. The differences were small, but the large sample size enabled the independent samples t-tests to imply that the differences were significant, with the exception of physical problems.

As expected older adolescents (M = .49, SD = .94) were exposed to more community violence than younger adolescents (M = .39, SD = .81), $t(14605) = -8.53$, $p < .01$. Older adolescents (M = 5.20, SD = 4.39; M = 2.37, SD = 1.73) also exhibited more depression and less self-regulation than younger adolescents (M = 4.20, SD = 3.93; M = 2.05, SD = 1.67), $t(14661) = -14.54$, $p < .01$ and $t(14693) = -11.39$, $p < .01$, respectively. As indicated in Table 3, younger adolescents reported better general health at both Time 1, $t(14719) = 3.80$, $p < .01$, and Time 2, $t(14718) = 3.10$, $p < .01$. They also slept more at Time 1, $t(14682) = 27.43$, $p < .01$, and Time 2, $t(14689) = 22.06$, $p < .01$, and slept better at Time 1, $t(14712) = 15.23$, $p < .01$, and Time 2, $t(14714) = 12.50$, $p < .01$. Unexpectedly this younger group also exhibited higher BMI levels at both Time 1, $t(14376) = 4.17$, $p < .01$, and Time 2, $t(14179) = 8.58$, $p < .01$. Although younger adolescents were more physically active at Time 1, $t(14721) = 23.67$, $p < .01$, and Time

2, $t(14719) = 28.23$, $p < .01$, they also exhibited more sedentary behavior than older adolescents at Time 1, $t(14657) = 10.27$, $p < .01$, and Time 2, $t(14644) = 8.79$, $p < .01$.

Because significant differences existed between younger and older adolescents for the majority of health indicators, the analyses were conducted separately for each age group.

Hierarchical Multiple Regression (Question 1)

Hierarchical multiple regression analyses were first conducted to examine whether community violence exposure (CVE) predicted different domains of adolescents' health concurrently and longitudinally after controlling for gender, SES, and childhood maltreatment. As mentioned already the six aspects of health comprise general health, physical problems, BMI, sleep habits, physical activity, and sedentary behavior. Each health-related variables assessed at Time 1 were each regressed separately on community violence exposure reported at Time 1. The same six health-related variables assessed at Time 2 were then regressed on community violence exposure at Time 1. Each health-related variable was analyzed separately for young and old adolescents and at Time 1 and Time 2, giving a total of 24 regressions. As mentioned in the analysis section, gender, SES, and childhood maltreatment were entered sequentially as predictors in the first regression model and followed by the addition of CVE as an independent variable in the second regression model in order to test its contribution to adolescents' health above and beyond the covariates. Baseline health values were entered in model 1 for longitudinal analyses, and parent's weight status was also accounted for in model 1 when examining the effects of CVE on adolescents' BMI. Tables 8, 9, 10, and 11 present the

results from the 2-step hierarchical regression models with structural variables (SES was centered) entered at Step 1 (Model 1) and CVE (square rooted and centered) added at Step 2 (Model 2).

The standardized partial regression coefficients (posted in the "β" column) are computed after all the variables have been reduced to a standard form, which is a mean of zero and a standard deviation of one. That is they have been converted to their Z-scores. It is sometimes convenient to look at the standardized coefficients since they all have the same scale of standard deviations. They indicate how one standard deviation in a particular independent variable affects one standard deviation of the dependent variable.

General Health

Early adolescence. Cross-sectional analyses revealed that the final model accounted for 2.10% of the total variance in younger adolescents' subjective general health at Time 1, $F(4, 4509) = 25.65$, $p < .001$, with CVE explaining zero of the variance but still contributing significantly, (B = -.052, $p < .05$). CVE also predicted poor general health one year later, (B = -.091, $p < .001$), explaining .30% of the variance. The model explained 27.50% of the variance of younger adolescents' general health at Time 2, $F(5, 4508) = 342.95$, $p < .001$.

Late adolescence. Similar to younger adolescents, the CVE-included model at Time 1 accounted for 2.6% of the variance for older adolescents' subjective general health, $F(4, 3798) = 26.53$, $p < .001$, while CVE specifically accounted for an additional .50% of the total variance. The more community violence older adolescents were exposed to, the more likely they reported poor general health at Time 1, (B = -.125, $p < .001$).

Longitudinal analyses revealed that CVE accounted for only an additional .10% of the variance for older adolescents' general health at Time 2 causing the proportion of variance accounted for by the overall (CVE-included) model to increase to 30.10%, $F(5, 3796) = 327.67$, $p < .001$. This compares to an R-squared of only 2.60% at Time 1. Being exposed to community violence at Time 1 increased risk for poor health at Time 2, (B = -.06, $p < .05$).

Physical Problems

Early adolescence. The overall model explained 2.80% of the variance of physical problems at Time 1, $F(4, 4506) = 33.16$, $p < .001$, with CVE accounting for an additional 1.10% of the total variance. Although small, these exposures did increase the likelihood of physical problems (B = .56, $p < .001$). Longitudinal analyses also indicated a strong effect of CVE on physical problems at Time 2 (B = .22, $p = .001$), but the proportion of additional variance accounted for decrease to .10%. Once again both model 1 (without CVE) and model 2 (with CVE) accounted for a much greater percent of the variance at Time 2 (30.5%, $F(5,451)=342.95$, $p < .001$) than they did at Time 1.

Late adolescence. In contrast to younger adolescents, cross-sectional analyses of older adolescents revealed that the overall (step 2) model accounted for more variance in physical problems at Time 1, 3.60%, $F(4, 3794) = 36.93$, $p < .001$, whereas the addition CVE at Time 1 accounted for less variance, only .50%. Nevertheless being exposed to community violence related to more physical health problems (B = .37, $p < .001$). After one year, CVE only accounted for an additional .10% of the variance of physical problems at Time 2. As with general health, the overall model accounted for

considerably more variance in physical problems at Time 2, (33.50% ,$F(5, 3787) = 383.23$, $p < .001$.) than it did at Time 1 (3.60%). While the strength of the effect decreased after one year, findings still revealed that exposure to violence did predict physical health problems at Time 2 (B = .16, $p < .001$).

Adiposity (BMI)

Early adolescence. CVE at Time 1 accounted for an additional .40% of the total variance of younger adolescents' BMI at Time 1 while the overall model (controlling for parental weight status) accounted for only 2.30% of the variance, $F(5, 4091) = 20.67$, $p < .001$. Such experiences, however, still affected BMI at Time 1, (B = 3.72, $p < .001$). After a one-year period, the amount of variance the overall model accounted for increased to 19.30%, $F(6, 3967) = 159.66$, $p < .001$, but with CVE explaining zero of the total variance. At Time 2, it no longer predicted BMI (B = 1.42, *n.s.*).

Late adolescence. For older adolescents, CVE accounted for .20% of the variance of BMI at Time 1 with the overall model explaining 1.70% of the total variance, $F(5, 3433) = 12.61$, $p < .001$. Results also indicated a relation between CVE and BMI at Time 1, (B = 2.23, $p < .05$). One year later, CVE at Time 1 appeared to have no effect on an older adolescent's BMI, (B = .843, *n.s.*), accounting for zero of the variance. The overall model accounted for 23.60% of the total variance, $F(6, 3323) = 172.51$, $p < .001$) as compared to only 1.70% at Time 1.

Sleep

Early adolescence. The overall model among early adolescents accounted for only 2.00% of the variance of sleep at Time 1, $F(4, 4509) = 24.06$, $p < .001$, with CVE,

by itself, accounting for 1.30% of that total. Younger adolescents who were exposed to community violence exhibited poor sleep patterns at Time 1, (B = -.185, $p < .001$). Longitudinal analyses revealed that over time CVE only accounted for .20% of the total variance of sleep at Time 2. Nevertheless a significant effect of CVE on sleep at Time 2 was revealed, (B = -.08, $p < .001$). The final model accounted for 16.00% of the total variance of sleep patterns at Time 2, $F(5, 4507) = 95.54$, $p < .001$, again much greater than the variance explained in Time 1 because of the added factor of baseline sleep.

Late adolescence. As with younger adolescents, the overall model accounted for only a small proportion of variance for older adolescents' sleep at Time 1, which amounted to 1.60%, $F(4, 3798) = 16.81$, $p < .001$. CVE explained an additional .30% of the total variance, and a negative effect was still found (B = -.09, $p < .001$). Adolescents exposed to more community violence exhibited more sleep difficulties. CVE also predicted a small effect after the one-year period (B = -.06, $p < .01$). While the proportion of variance accounted for by the overall model increased to 17.00%, $F(5, 3797) = 156.35$, $p < .001$, most was predicted by earlier sleep patterns. CVE at Time 1 explained only an additional .20% of the variance for sleep at Time 2.

Physical Activity

Early adolescence. After controlling for covariates, CVE accounted for .20% while the overall model explained 4.70% of the variance of early adolescents' physical activity at Time 1, $F(4, 2509) = 56.50$, $p < .001$. Unexpectedly results indicated being exposed to community violence at Time 1 related to being more rather than less physically active at Time 1 (B = .18, $p < .001$). While the overall model explained

19.50% of the variance of physical activity at Time 2, $F(5, 4508) = 750.32$, $p < .001$, CVE at Time 1 did not have a significant effect on physical activity at Time 2 (B = -.05, *n.s.*), explaining zero variance.

Late adolescence. The amount of variance accounted for by the overall model and CVE was slightly more for older adolescents' physical activity level at Time 1, 5.50% and .30%, respectively. The overall model significantly explained this group's physical activity, $F(4, 3797) = 56.14$, $p < .001$, as did CVE (B = .21, $p < .001$). As with younger adolescents, an increase in CVE unexpectedly predicted an increase in physical activity. Older adolescents exposed to community violence at Time 1 were more active at both Time 1 and Time 2 (B = .13, $p < .05$). Longitudinal analyses revealed CVE accounted for .10% of additional variance of physical activity at Time 2 with the overall model accounting for 23.10% of the total variance, $F(5, 3796) = 229.00$, $p < .001$.

Sedentary Behavior

Early adolescence. The overall model explained 3.80% of the total variance of early adolescents' sedentary behavior at Time 1, $F(4, 4489) = 162.69$, $p < .001$, with CVE at Time 1 accounting for an additional .10% of the total variance. Although only to a small degree, CVE affected sedentary behavior at Time 1 (B = .12, $p < .05$). Younger adolescents exhibited more sedentary activities if they had been exposed to violence with the community. Longitudinal analyses revealed similar findings after one year. While the amount of variance accounted for by the overall model increased to 24.80% for sedentary behavior at Time 2, $F(5, 4466) = 296.46$, $p < .001$, most was predicted by baseline

sedentary behavior. CVE predicted sedentary behavior at Time 2 (B = .12, $p < .05$), but accounted for zero of the total variance.

Late adolescence. Analyses among older adolescents indicated similar patterns to their younger counterpart with the overall model accounting for 4.10% of the variance in older adolescents' sedentary behavior at Time 1, $F(4, 3789) = 41.05$, $p < .001$, and CVE accounting for an additional .40% of the total variance. Exposure to community violence was linked to being involved in more sedentary activities (B = .22, $p < .001$). Longitudinal analyses revealed that the proportion of variance accounted for by the overall model was 26.60%, $F(4, 3797) = 56.14$, $p < .001$, with CVE explaining an additional .10%. The partial regression coefficient for CVE was small and not statistically significant (B = .06, *n.s.*).

A Multiple Mediation Model (Question 2)

For the second aim, the Preacher and Hayes (2008) multiple mediation model was used to examine whether depression and self-regulation mediated the concurrent and longitudinal relation between community violence exposure and adolescents' health. As with the hierarchical regressions, the adolescents were divided into two age groups (12-15 years and 16-19 years). For the present study, the Preacher and Hayes model was particularly useful because it allowed for the simultaneous inclusion of two or more mediators while also examining a moderator. Multiple mediation models allow us to obtain the total indirect effect, which consists of depression and self-regulation acting together as a set in mediating the relation between CVE and the health variables and to then examine the specific indirect effects of each mediator. In other words, we can test

and compare the unique ability of each to mediate beyond that of the other mediator (Preacher & Hayes, 2007). The bootstrap estimates presented were based on 5,000 bootstrap samples.

Within the multiple mediation analyses, the following three steps were taken to explore the relations among the predictor, mediators, and outcomes: 1) The total effect (c) and direct effect (c') were examined. 2) The total indirect effect ($a_1*b_1 + a_2*b_2$) was investigated using the confidence intervals as indicators of significance. 3) The specific indirect effects of each mediator (depression = a_1*b_1 and self-regulation = a_2*b_2) for all six outcome variables were then examined. The specific indirect effects tested were as follows: a) the indirect effect of CVE on each health-related variable through depression, controlling for the indirect effect of self-regulation, and b) the indirect effect of CVE on each health-related variable through self-regulation, controlling for the indirect effect of depression. Each a -path was computed using simple linear regression of the mediator regressed on CVE and each b -path was computed using multiple regression of the specific health indicator regressed on the mediator with the inclusion of CVE, the covariates, and the interaction term of gender multiplied by CVE. As mentioned in the analysis section, the equations used were: $H(i) = b_1*m_1 + b_2*m_2 + c'*x$ + 3 covariates + 1 moderator, where $m_1 = a_1*x$ and $m_2 = a_2*x$.

The specific indirect effects tested were as follows: (1) the total effect of CVE on each mediator (i.e. two simple regressions) in order to compute paths a_1 and a_2 through depression, controlling for the indirect effect of self-regulation and (2) computing paths b_1 and b_2 by multiple regressions of each health-related variable on each mediator, while

controlling for the other mediator, the interaction term and the covariates. In total there were three regressions for each health variable: two simple regressions and one multiple regression.

It is important to note that a significant total indirect effect is not a prerequisite for investigating specific indirect effects. It is possible to have significant indirect effects from each mediator in the presence of an insignificant total indirect effect (e.g., one mediator could act as a suppressor, the other as an enabler, so that the sum of their effects could partially cancel each other out).

Gender was tested as a moderator (discussed below in question 3), and gender, SES, and childhood maltreatment, (and parental weight status for outcome BMI) were entered as control variables in all models tested. The partial regression coefficients are given on Tables 12 and 13 for Times 1 and 2, respectively. When examining change over time, previous health indicators were also entered as control variables.

The mediation analyses are summarized on Table 14 where the total, direct and indirect effects are tabulated along with the partial regression coefficients for each mediator and the new R-squared statistic that resulted from adding the mediation. The degree of statistical significance is noted by asterisks, and ~ symbol next to the mediator regression coefficients indicates that the 95% confidence interval (as computed by bootstrapping) did not enclose zero.

As in the hierarchical regressions, the R-squared terms at Time 1 were very small, but nevertheless significant because of the large sample size. The R-squares also

increased appreciably in Time 2 because of the inclusion of the corresponding health variables from Time 1.

The partial regression coefficients for the covariates computed in the mediation regression analyses as presented in Tables 12 and 13 are close to those computed in the hierarchical regression analyses (Tables 8-11). Consequently there is no need to discuss them again in this section, which will instead concentrate on the direct and indirect effects associated with mediation.

General Health

Early adolescence at Time 1. For younger adolescents, a total indirect effect of community violence exposure (CVE) on subjective general health at Time 1 for mediators, depression and self-regulation, was found with a point estimate of -.071 (95%CI between -.089 and -.055). The point estimate is defined by Preacher (2010) as the product of the estimated regression weights linking a*x to m and b*m to y, the latter controlling for x. In early adolescence, those exposed to violence outside the home were depressed and had poor self-regulation, which in turn negatively affected their subjective general health. The specific indirect effects here represent 1) the effect of depression to mediate the relation between CVE and general health, while controlling for self-regulation, and 2) the effect of self-regulation to mediate the relation between CVE and general health, while controlling for depression. Specific indirect effects indicated that both depression (95%CI between -.053 and -.025) and self-regulation (95%CI between -.048 and -.021) mediated the relation beyond that of the other (low self-regulation scores indicated better self-regulation skills). In a pairwise contrast of the specific indirect

effects, neither mediator contributed significantly more than the other (95%CI between -.026 and .017).

The direct effect of CVE on general health was in an unexpected direction (c' = .074, $t(4501) = 2.153$, $p < .05$); that is, younger adolescents exposed to community violence reported better general health. However, the total effect of CVE on general health is the sum of the negative indirect effects and positive direct effect, and consequently it was reduced to a level of insignificance (c = .003, $t(4502) = .089$, *n.s.*).

Early adolescence at Time 2. CVE had no direct or total effect on general health after one year among younger adolescents when controlling for gender, SES, childhood maltreatment, and previous general health (c' = -.013, $t(4500) = -.446$, *n.s.*; c = -.044, $t(3501) = -1.487$, *n.s.*, respectively). However, the difference between the total and direct effects, the total indirect effect through both depression and self-regulation, was different from zero and therefore significant with a point estimate of -.031 (95%CI between -.043 and -.020). The way in which CVE affected early adolescents' general health was through depression and self-regulation. Younger adolescents who were exposed to violence within their community tended to be more depressed and less able to regulate or control their behavior, which in turn over time led to poor general health. Results from the specific indirect effects indicated that depression (95%CI between -.028 and -.006) and self-regulation (95%CI between -.026 and -.005) were both good mediators and that neither mediator contributed significantly more than the other (95%CI between -.020 and .016).

Late adolescence at Time 1. Similar to younger adolescents, depression and self-regulation together as a set mediated the effect of CVE on subjective general health at Time 1 among older adolescents with a total indirect effect of -.044 (95%CI between -.061 and -.028). Exposure to community violence related to higher levels of depression and lower levels of self-regulation while these mediators in turn contributed to poor subjective general health. In addition both depression (95%CI between and -.046 and -.020) and self-regulation (95%CI between -.021 and -.005) contributed to the indirect effect above and beyond the other; yet an examination of the pairwise contrasts of the indirect effects revealed that the specific indirect effect through depression was larger (95%CI between -.035 and -.007). This relation was transmitted largely through the two mediators since no direct effect of CVE on general health was found (c' = -.059, t(3789) = -1.892, *n.s.*). The total effect was significant (c = -.103, t(3790) = -3.196, $p < .01$).

Late adolescence at Time 2. Similar to younger adolescents, a total effect of CVE on older adolescents' subjective general health one year later was found to be significant, but small (c = -.060, t(3789) = -2.2355, $p < .05$). No direct effect was indicated (c' = -.047, t(3788) = -1.766 *n.s.*) suggesting a mediation effect. Indeed, the total indirect effect met significance, with a point estimate of -.013 (95%CI between -.021 and -.006). However, a further examination of specific indirect effects indicated only depression mediated the relation (95%CI between -.019 and -.005). Being exposed to community violence as a child or adolescent led to depression, which in turn negatively affected general health one year later. In addition the latter finding was supported by a pairwise

contrast demonstrating depression to be the superior mediator (95%CI between -.018 and -.001).

Physical Problems

Early adolescence at Time 1. The total effect of CVE on physical problems at Time 1 during early adolescence was significant, (c = .450, t(4500) = 4.365, p < .001), but the direct effect was not (c' = .025, t(4499) = .258, *n.s.*); as in general health the effect of CVE on physical problems was largely explained through mediators. This interpretation is supported by the fact that the total indirect effect of depression and self-regulation between CVE and physical problems was significant, with a point estimate of .425 (95%CI between .340 and .515). Specific indirect effects identified both depression (95%CI between .138 and .233) and self-regulation (95%CI between .183 and .310) as good mediators with about equal effect (95%CI between -.135 and .010).

Early adolescence at Time 2. Mediation analyses revealed significant total indirect effects of depression and self-regulation between CVE and physical problems at Time 2 with a point estimate of .084 (95%CI between .056 and .120). Further analyses on the specific indirect effects, however, indicated that only self-regulation mediating the relation (95%CI between .039 and .096). Depression did not meet significance since the confidence interval included zero (95%CI between -.002 and .044). The effect of CVE on physical problems one year later was through the inability to regulate oneself. This is supported by the fact that a pairwise contrast of the indirect effects indicated that the specific indirect effect through self-regulation was significantly larger than that through depression (95%CI between -.088 and -.006). CVE also had a direct effect on physical

problems at Time 2 (*c'* = .219, *t*(4492) = 2.584 *p* < .01), so mediation in this case was only partial. Adolescents exposed to community violence over time suffered from more physical problems. Thus the total effect was significant (*c* = .303, *t*(4493) = 3.590 *p* < .001) as the sum of the direct and indirect effects.

Late adolescence at Time 1. Findings for the effect of CVE on physical problems at Time 1 among older adolescents were similar to those found among their younger counterparts. The total indirect effect had a point estimate of .179 (95%CI between .108 and .256) and the specific indirect effects indicated that both depression (95%CI between .053 and .120) and self-regulation (95%CI between .046 and .152) mediated this relation while controlling for the other. Both mediators contributed about an equal amount to the total indirect effect (95%CI between -.067 and .036). Therefore, during late adolescence, being exposed to community violence increased depression and decreased self-regulation skills that in turn caused more physical problems. The direct effect of CVE on physical problems just missed significance (*c'* = .164, *t*(3786) = 1.920, *p* = .055), so the total effect was transmitted largely through the mediating paths (*c* = .343, *t*(3787) = 3.692, *p* < .001).

Late adolescence at Time 2. As with general health, the total effect of CVE on later physical problems met significant (*c* = .149, *t*(3781) = 2.00, *p* < .05) whereas the direct effect did not (*c'* = .123, *t*(3780) = 1.655, *n.s.*) indicating a mediation effect. The point estimate of the total indirect effect was .026 (95%CI between .011 and .047) such that being exposed to community violence as a child or adolescent increased depression and decreased self-regulation skills, which then led adolescents to suffer from more

physical problems. An examination of the specific effects indicated that both depression (95%CI between .003 and .03) and self-regulation (95%CI between .002 and .025) mediated the relation, while controlling for the other; the contributions of the two mediators were not significantly different from each other (95%CI between -.016 and .023).

Adiposity (BMI).

Early Adolescence at Time 1. For younger adolescents, the direct effect from being exposed to community violence was significant making it more likely they would be overweight or obese (c' = 3.28, $t(4085) = 2.79$, $p < .01$). Unlike general health and physical problem, however, the mediating effect through depression and self-regulation did not have a significant effect. No total indirect effect (95%CI between -.090 and .636) or specific indirect effects were found (depression had a 95%CI between -.101 and .653; self-regulation had a 95%CI between -.368 and .343). The total effect was significant (c = 3.55, $t(4086) = 3.06$, $p < .01$), which is largely explained by the significant direct effect.

Early adolescence at Time 2. The effect of CVE on BMI at Time 2 indicated different patterns from the concurrent analyses. For younger adolescents no total, direct, total indirect, or specific indirect effects were found for the effects of CVE on BMI one year later (direct effect c' = .321, $t(3961) = .298$, *n.s.*; total effect c = .481, $t(3762) = .452$, *n.s.*). Mediating analyses failed to meet significance (the total indirect effect had a 95%CI between -.145 and .507; the specific indirect effects for depression had a 95%CI between -.274 and .401 and for self-regulation had a 95%CI between -.198 and .450).

Late adolescence at Time 1. During late adolescence, being exposed to community violence just failed to correlate with BMI at Time 1 (direct effect *c'* = 2.046, *t*(3427) = 1.905, *p* = .057; total effect *c* = 2.033, *t*(3428) = 1.900, *p* = .058). Mediating analyses also failed to meet significance (the total indirect effect had a 95%CI between -.207 and .187; the specific indirect effects for depression had a 95%CI between -.230 and .161 and for self-regulation had a 95%CI between -.065 and .240).

Late adolescence at Time 2. As in early adolescence, no significant effects were found for total, direct, indirect, or specific effects of CVE on BMI at Time 2 for older adolescents (direct effect *c'* = -.789, *t*(3318) = -.801, *n.s.*; total effect *c* = -.706, *t*(3319) = -.728, *n.s.*). All mediating analyses failed to meet significance (the total indirect effect had a 95%CI between -.083 and .277; the specific indirect effects for depression had a 95%CI between -.108 and .308 and for self-regulation had a 95%CI between -.125 and .134).

Sleep

Early adolescence at Time 1. The total indirect effect of CVE on sleep at Time 1 was significant with a point estimate of -.094 (95%CI between -.116 and -.074). Younger adolescents who were more exposed to community violence concurrently suffered from more depression and could not effectively self-regulate, which in turn was linked to poor sleep patterns. An examination of specific indirect effects revealed both depression (95%CI between -.054 and -.027) and self-regulation (95%CI between -.072 and -.040) uniquely mediated this relation, and neither appeared to be greater than the other (95%CI between -.006 and .037). CVE also directly disrupted younger adolescents' sleep patterns

(c' = -.065, t(4501) = -2.16, p < .05). Thus the total effect was also significant (c = -.158, t(4502) = -5.12, p < .001).

Early adolescence at Time 2. Taken together the total indirect effect of CVE at Time 1 on sleep at Time 2 through depression and self-regulation indicated a point estimate of -.030 (95%CI between -.043 and -.021). After one year being exposed to community violence also caused younger adolescents to feel more depressed and be less able to self-regulate, which in turn disrupted their patterns of sleep. Specific indirect effects indicated that both mediated the effect of CVE on sleep (95%CI between -.026 and -.006 for depression; 95%CI between -.026 and -.008 for self-regulation). The total effect of CVE on sleep patterns was significant (c = -.062, t(4500) = -2.097, p < .05) while the direct effect was not (c' = -.032, t(4499) = -1.066, *n.s.*), suggesting again this relation is largely explained by the two mediators.

Late adolescence at Time 1. The total indirect effect of being exposed to community violence on sleep patterns at Time 1 among older adolescents, as transmitted through depression and self-regulation, had a point estimate of -.035 (95%CI between -.052 and -.019). The specific indirect effects indicated that both made a contribution (depression had a 95%CI between -.018 and -.004; self-regulation had a 95%CI between -.040 and -.012). In a pairwise contrast between the two mediators, however, self-regulation had the larger effect (95%CI between .001 and .031). The direct effect just missed significance at the .05 level (c' = -.053, t(3789) = -1.918, p = .055), while the total effect remained significant (c = -.087, t(3790) = -3.069, p < .01). Adolescents who were exposed to more community violence indicated worse sleep patterns.

Late adolescence at Time 2. Results indicated both a total effect (c = -.074, t(3789) = -2.845, $p <$ .01) and a direct effect (c' = -.069, t(3788) = -2.642, $p <$ .01) of CVE on sleep at Time 2. Being exposed to community violence disrupted older adolescents' sleep patterns at Time 2. Unlike Time 1, the total indirect effect was not significantly different from zero (95%CI between -.011 and .000). Specific indirect effects were also not significant (depression had a 95%CI between -.010 and .003; self-regulation had a 95%CI between -.007 and .000). The mechanism in which CVE affected sleep one year later was not explained by mediation.

Physical Activity

Early adolescence at Time 1. Depression and self-regulation mediated the effect of CVE on physical activity at Time 1 among younger adolescents with a total indirect effect of -.082 (95%CI between -.116 and -.055). Specific indirect effects demonstrated both depression (95%CI between -.064 and -.012) and self-regulation (95%CI between -.077 and -.022) were effective mediators (95%CI between -.032 and .055). In other words, the effect of CVE on physical activity was transmitted through depression and self-regulation such that those exposed to community violence suffered from more depression and had poorer self-regulation skills, which led to being less physically active. However the direct and total effects, also significant, were in the opposite direction; that is, CVE increased physical activity (c' = .249, t(4501) = 3.03, $p <$ .01) and (c = .167, t(4502) = 2.05, $p <$ .05). However, results were inconsistent with the proposed hypothesis; adolescents who were exposed to violence within their community tended to be more physically active. Although the direction of the relation was unexpected for the

above effects, the mediation hypothesis was supported. Depression and lack of self-regulation acted as depressors on physical activity, but did not overcome the direct positive effect.

Early adolescence at Time 2. The total indirect effect of CVE on physical activity one-year later through depression and self-regulation was significant among younger adolescents with a point estimate of -.078, as the confidence intervals contained a zero (95%CI between -.106 and -.054). Problems with depression and self-regulation may develop among this group due to being exposed to violence within their community. These problems in turn decreased their physical activity level. An examination of the specific indirect effects further supported that both depression (95%CI between -.065 and -.016) and self-regulation (95%CI between -.065 and -.017) mediated the effect of CVE on physical activity over time, while controlling for the other mediator. In addition a pairwise contrast indicated neither was superior (95%CI between -.042 and .041). As in Time 1 the direct effect was in the opposite direction to that of the mediators, but in this case it was too small to create a positive total effect (c' = .114, $t(4500) = 1.551$, *n.s.*; c = .036, $t(4501) = .490$, *n.s*).

Late adolescence at Time 1. For older adolescents, the total indirect effect of CVE on physical activity at Time 1 through the two mediators was small, but significant at -.022 (95%CI between -.041 and -.005). However, by looking at the specific indirect effects it was determined that only self-regulation was significant (95%CI between -.039 and -.008) and not depression (95%CI between -.017 and .015). For older adolescents, being exposed to community violence decreased self-regulation skills, which in turn led

to less physical activity. As with younger adolescents, the direct and total effect of CVE on physical activity was in the opposite direction from the mediating effects (c' = .271, $t(3789) = 3.782$, $p < .001$; c = .249, $t(3790) = 3.483$, $p < .001$, respectively). Being exposed to community violence directly increased physical activity level for older adolescents, despite the suppressing effect of self-regulation.

Late adolescence at Time 2. The total indirect effect of CVE on physical activity at Time 2 through depression and self-regulation was statistically significant with a point estimate of -.013 (95%CI between -.027 and -.001). An examination of the specific indirect effects, however, indicated neither depression (95%CI between -.026 and .003) nor self-regulation (95%CI between -.012 and .007) alone mediated the relation, but rather only the combined effect explained the pathway between CVE and later physical activity. Being exposed to community violence led to problems with depression and self-regulation, which in turn caused older adolescents to be less active. Unlike younger adolescents at Time 2, a total effect (c = .193, $t(3789) = 3.137$, $p < .01$) and direct effect (c' = .206, $t(3788) = 3.334$, $p < .001$) of CVE on physical activity at Time 2 were found among older participants, but again in the unexpected direction.

Sedentary Behavior

Early adolescence at Time 1. No significant effects were found during early adolescence in the mediation pathways for the effect of CVE on sedentary behavior at Time 1. Only covariates, gender and SES had partial regression coefficients that appeared significant. The direct effect (c' = .063, $t(4481) = .825$, *n.s.*), total effect (c = .067, $t(4482) = .894$, *n.s.*), total indirect effect (95%CI between -.020 and .030), and

specific indirect effects for depression (95%CI between -.039 and .011) and self-regulation (95%CI between -.006 and .045) were all below the 95% threshold level for significance.

Early adolescence at Time 2. The longitudinal effects of CVE on sedentary behavior at Time 2 were similar to those determined at Time 1. No significant relations were found for the total, direct, total indirect, or specific indirect effects of CVE on sedentary behavior over time (c = .013, t(4459) = .120, *n.s.*; c' = -.002, t(4458) = -.036, *n.s.;* 95%CI between -.007 and .040; 95%CI between -.017 and .027 for depression; 95%CI between -.009 and .034 for self-regulation, respectively).

Late adolescence at Time 1. Unlike younger adolescents, CVE's direct effect on sedentary behavior at Time 1 (c' = .145, t(3782) = 2.127, $p < .05$) among older adolescents was significant. It is not surprising that older adolescents who were exposed to more community violence tended to stay indoors and be more sedentary by watching more television, being on the computer, or playing videogames. However, as with younger adolescents, no indirect effect or specific indirect effects were found (indirect effect had a 95%CI between -.000 and .030; specific indirect effects for depression had a 95%CI between -.001 and .023 and for self-regulation had a 95%CI between -.000 and .021). Nevertheless the sum of the indirect and direct effects still resulted in a significant total effect of CVE on sedentary behavior at Time 1 (c = .157, t(3783) = 2.322, $p < .05$).

Late adolescence at Time 2. Unlike with younger adolescents, the total indirect effect indicated a weak, but significant mediating effect with a point estimate of .015 (95%CI between .004 and .030). Specific indirect effects revealed that only self-

regulation mediated this relation (95%CI between .0003 and .020); depression was not a significant mediator when self-regulation was taken into account (95%CI between -.004 and .023). Problems with self-regulation triggered by CVE led older adolescents to stay indoors and spend time involved in sedentary activities. Despite the fact that only self-regulation presented a unique effect, a pairwise contrast of the two indirect effects indicated they were not statistically different from each other (95%CI between -.019 and .019). As with early adolescents at both Time 1 and 2, CVE did not demonstrate a total or direct effect on sedentary behavior at Time 2 (c = .069, t(3771) = 1.225, *n.s.*; c' = .054, t(3770) = .952, *n.s.*, respectively).

Moderation (Question 3)

The moderation effect was estimated by the interaction variable added as an independent variable to the multiple mediation regression equations. It took the form of the product of gender and CVE and was meant to capture the interdependence of the two variables. For example, if girls' health was affected differently by CVE than boys', then a significant interaction coefficient would be found. The gender variable was assigned the value of zero for males and one for females. Table 15 lists the partial regression coefficients for the interaction term at both Time 1 and 2 (labeled concurrent and longitudinal) and for both early and late adolescents. As can be seen they are all small, and for the most part not significant.

When investigating the differential pathways between males and females, gender did not moderate any of the concurrent relations between CVE and the health indicators. Two moderation effects were found at Time 2. One was sedentary behavior for early

adolescents (r = .237, $p < .05$); the other was BMI for late adolescents (r = 4.843, $p < .01$). These two gender differences can be seen in Figures 4 and 5, which graph the relations between CVE and sedentary behavior and between CVE and BMI, respectively. Each graph plots males and females separately, and the two plots are clearly different.

Younger males who were exposed to community violence demonstrated decreases in sedentary behavior whereas females who were exposed to community violence exhibited increases in sedentary behaviors after one year. When this difference was investigated through the mediators, being exposed to community violence led to large increases in depression among females and a slight increase among males; however, there did not appear to be a relation between depression and sedentary behavior at Time 2 for either males or females. In line with findings discussed in question 2, depression appeared to not mediate the effect of CVE on sedentary behavior. That is, the first pathway, from CVE to mediator was significant, but not the one between the mediator and sedentary behavior.

One the other hand, males and females differed in the way in which community violence exposure affected sedentary behavior through the other mediator, self-regulation. For females, as community violence exposure increased, self-regulation skills greatly decreased, which unlike depression led to increases in sedentary behavior at Time 2. For males, as community violence exposure increased, only a very slight, if negligible, decrease in self-regulation was found; yet decreases in self-regulation also related to increases in sedentary behavior at Time 2 for males. Self-regulation may help explain how CVE increases sedentary behavior among females but not males. Thus the gender

differences in sedentary behavior appear to be partially due the mediating effects of self-regulation.

During late adolescence, differences existed between males and females in the way in which CVE affected BMI at Time 2. Older females who were exposed to community violence were likely to have increases in BMI levels after one year. Older males' BMI remained roughly the same despite amount of CVE. With regard to the mediators, community violence exposure predicted large increases in depression for females with a moderate increase for males. Observations revealed no relation between depression and BMI at Time 2 for either gender, thus supporting earlier findings that depression did not mediate this relation. For self-regulation, community violence exposure predicted increases in self-regulation for both males and females; however, increases in self-regulation in turn only led to increases in BMI at Time 2 for females while no relation appeared for males. Thus the gender difference again seems to be in the different mediating effects of self-regulation.

Part Four:

Discussion

Discussion

The discussion will be organized into two sections. The first section will discuss our findings organized around the three questions addressed in the paper. We will begin by discussing the effect of community violence exposure on the six health indicators followed by the mechanisms that explain how community violence exposure affects adolescents' health, which has only been addressed minimally in past research. How community violence exposure affects males differently than females will then be reviewed along with a discussion on the effects of childhood maltreatment, gender, and SES on adolescents' health. In addition, how these findings connect with past research and what implications they have on current work with regards to theory and application will also be discussed. The second section will discuss the limitations of the current study and suggest ways in which future research may continue to further our understanding.

Question 1: Does Community Violence Exposure Affect Adolescents' Health?

The main purpose of the study was to investigate the effect of community violence exposure on younger and older adolescents' health, concurrently and over time. In other words, does exposure to community violence have harmful consequences on adolescents' health?

When examining the concurrent effect using Time 1 data, community violence exposure indeed related to all six health outcomes for both younger and older adolescents. All effects were in the expected direction except for physical activity. Adolescents who were exposed to community violence were more physically active, not less, which was the original hypothesis. In light of this unexpected finding, we may

consider the nature of this relation such that it may be physical activity that leads to more community violence exposure rather than the direction hypothesized. Since this finding is based on a cross-sectional analysis, we cannot be certain of how the two affect each other. It is possibly, and even likely, that adolescents who are more physically active spend more time outside, involved in such activities assessed, where the violence may occur. This may be an artifact of a violent neighborhood rather than a cause and effect relation. These adolescents therefore have more opportunities to be a witness or victim to the violence in their community. On a different note, adolescents who are exposed to community violence may use athletic programs or such activities as a protective factor against the fear and stress caused by these exposures. It may also be that active adolescents generally have higher self-esteem levels and therefore are less affected (Tremblay, Inman, & Willms, 2000).

As predicted being exposed to community violence had negative consequences on *all* other areas of health examined for both age groups; adolescents reported poorer general health, more physical problems, higher BMI levels, more disrupted sleep patterns, and more sedentary behaviors at Time 1. These findings are consistent with previous studies investigating the effects community violence exposure (e.g. Duncan, 1996; Cooley-Quille & Lorion, 1996). For the present study, the effects appeared to be especially strong on younger adolescents' physical problems and sleep and older adolescents' physical problems and general health. This tells us that physical problems are one of the main health concerns for younger and older adolescents who are exposed to community violence. Headaches, stomachs, or being sick may possibly be bodily

responses to the violent experiences. In addition medical problems such as stomachs and pain have been symptoms of posttraumatic stress, which may be similar to what is occurring here (e.g, Engelhard, van den Hout, Weerts, Hox, & van Doormen 2009; Dennis et al., 2009; Rauch, Grunfeld, Yadin, Cahill, Hembree, & Foa, 2009).

Research is lacking in this area since much has focused on problem behaviors (e.g., Bereson, Wiemann, & McCombs, 2001; Vermeiren, Schwab-Stone, Deboutte, Leckman, & Ruchkin, 2003) rather than general physical health symptoms that surface from undergoing such stressful experiences. The fear and anxiety that results from the violent exposures may also be what is contributing to younger adolescents' sleep difficulties; older adolescents' sleep was still impacted but to a lesser degree, which may be the result of becoming desensitized to the violence with age (Turner, Beidel, & Cooley-Quille, 1997; Cooley-Quille & Lorion, 1999). It should be pointed out that where older adolescents' general health was strongly affected, younger adolescents' general health was the least affected by CVE at Time 1. This may due to the subjective nature of the general health question such that older adolescents' responses may be more in tune with their physical and emotional health and therefore able to give a more accurate description.

The second part to Question 1 was whether being exposed to community violence still had consequences on adolescents' health after a one-year period. Over time, CVE continued to negatively impact general health, increase physical problems, and disrupt sleep patterns for both age groups, but in general the magnitudes of the effects were diminished when compared to Time 1. By Time 2, CVE had no significant effect on

either BMI or physical activity for younger adolescents, and no significant impact on BMI and sedentary behavior for older adolescents.

Obesity is a condition that is slow to develop, so what effects that CVE may have on BMI may not be apparent for years. Even though no effects on BMI at Time 2 were found in this study, it may be more informative to evaluate their weight status as an adult when years of accumulating weight gain becomes a serious problem. Most studies into the effects of childhood violence exposure and/or abuse on obesity have investigated weight when the child has reached adulthood. This is generally necessary since most studies recognize the potential danger of asking children sensitive questions about abuse, and therefore are retrospective by design (e.g. D'Argenio et. al., 2009; Williamson et. al., 2002; Wonderlich et. al., 1996). The prospective study (Noll, Zeller, Trickett, & Putman, 2007) found a moderate relation between childhood abuse and adolescent obesity, but the abuse was in the form of sexual abuse. Indeed sexual abuse is most likely more traumatic than most of the episodes of community violence exposure reported in this study, and so its effects more dramatic and begin to occur at a younger age. It is not a surprise to see that parental weight had a large effect on adolescents' BMI. The effects of the covariates, specifically childhood maltreatment, were small to negligible with maltreatment dropping into insignificance at Time 2. This was not as expected since a goal of the study was to gain insight into the cause and effect relation between violence and obesity during adolescence.

Physical activity and sedentary behavior also had no effect over time for younger and older adolescents, respectively. As discussed above it may be that being active places

adolescents in areas outside their home where the violence occurs. Parents of younger adolescents have more of an influence than older adolescents' parents to monitor their behavior to avoid dangerous areas. This may be particularly salient at Time 2 for younger adolescents who were initially exposed to community violence because parents may take measures to change their child's activity structure, resulting in less physical activity. Along the same lines, only younger adolescents continue to be more sedentary after their exposure to community violence because parental supervision may limit the younger adolescents' activity to only inside play to avoid unsafe areas in the neighborhood (Groves, 1996). Parents allow for more leniency with older adolescents' activities.

It is not surprising, but still somewhat comforting, that time does diminish the effects of traumatic experiences. The one exception was that younger adolescents' general health was poorer at Time 2. However, when comparing all the indicators over time, it seems more likely that this increase has more to due with the fact that younger adolescents' health was initially low (as mentioned above it had the smallest effect) rather than a true indication of change.

After a one-year period, the effects of community violence had the most serious impact on both younger and older adolescents' sleep and younger adolescents' general health. This may be similar to symptoms experienced from Posttraumatic Stress Disorder (PTSD), in which the distress and fear caused by the traumatic experience re-surface in nightmares and other sleep problems (e.g., Babson & Feldner, 2010; Krakow et al., 2001; Groves, 1996). As with the analyses at Time 1, the effect of CVE on adolescents' sleep was greater for younger than older adolescents one year later. These findings inform us

that sleep problems may be one of the most serious consequences of violence on adolescents' long-term health.

In a comparison of the effects between age groups, younger adolescents' physical problems, BMI, and sleep were more negatively affected by CVE while older adolescents' general health, physical activity level, and sedentary behavior were more affected at Time 1. However after one year, CVE in general had a larger effect on younger adolescents' than older adolescents' health. Although older adolescents were exposed to more community violence, it appears that younger adolescents are more emotionally and physically vulnerable to the consequences of violence exposure, which is in line with being less immature and more vulnerable at a younger age.

It is important to note, however, that the effect of CVE on all the health outcomes is quite small, and it may be the large sample size that allows them to be statistically significant. In light of the small effects, we do not regard them as negligible since the accumulative negative effect, which may reinforce one another, can stress adolescents' overall health.

A comment about the general fit of the regression equations is necessary. In general the proportion of variance explained by the addition of CVE was less than two percent across age groups and time periods. The proportion of variance accounted for by the final model at Time 1, which includes the covariates and CVE (model 2), were low, ranging between roughly 2% and 5%. While the proportion of variance explained increased to 16% to 34% at Time 2, this was largely due to the inclusion of the participants' corresponding health status at Time 1. The independent variables chosen for

this study do not explain the whole story. Instead other influences outside of those chosen here appear to have greater effects on explaining adolescents' health behaviors and outcomes. In fact in the introduction we pointed out the many serious consequences of violence exposure on adolescents' health are drug abuse, smoking and unsafe sexual practices. However, the purpose of this study was to examine the less studied consequences of CVE, and one reason that they are less studied is that they are understood to be small. Yet we argue that they remain important since their accumulative effect may be potentially detrimental.

Summary. There are three findings to highlight for the study's first question: community violence exposure had one of the strongest adverse effects on physical problems at Time 1 and sleep at Time 2 for both age groups. Second, in general the effects diminished over time. Third the effects were more severe among younger adolescents, suggesting the younger one is exposed to community violence, the more negative effects it will have on health in the long run, supporting the organizational framework discussed above. There were a few additional interesting findings. One being that physical activity increased with CVE rather than decreased as expected, possibly suggesting that those who are exposed may become involved in athletic programs or other sport activities to avoid further exposures. An alternative explanation may be that the activities kept adolescents outdoors more often, and therefore they had more opportunities for witnessing or being a victim. We also found that being exposed to more community violence related to higher BMI levels; however there was no lasting effect

over time. Last, CVE and the covariates chosen here had a significant impact on the health-related outcomes even though they were small.

Question 2: Do Depression and Self-regulation Mediate the Effect of Community Violence Exposure on Adolescents' Health?

The second question of the study aimed to determine the underlying mechanisms by which community violence exposure negatively impacts adolescents' health. More specifically *how* does CVE harm one's health and *what* are the processes at work? This study investigated two pathways through mediation analysis.

The mediators, depression and self-regulation, were chosen because they are known to be important determinants in adolescent health, and there is a plethora of evidence indicating they are affected by violence exposure. They have been incorporated into many models that were developed to understand the factors underlying adolescents' health; among them are the models of Guerra & Bradshaw (2008) and Kendall-Tacket & Klest (2009).

One of the main findings of the study was that both depression and self-regulation did in fact mediate the effect of CVE on adolescents' general health, physical problems, and sleep at Time 1. In other words, adolescents in both groups who are exposed to community violence tend to be more depressed and less able to regulate their behavior and thoughts, which in turn can lead them to feel worse about their general health, have more physical problems, and have more disruptions in their sleep. CVE also negatively affected younger adolescents' physical activity by increasing depression and decreasing self-regulation skills, but only self-regulation explained the decreases in older

adolescents' physical activity. Researchers have suggested that the stress from experiencing a traumatic event can have detrimental effects, particularly on their sleep habits (Cooley-Quille, & Lorion, 1999; Krakow et al., 2001; Noll et al., 2006) and physical health (e.g, Dennis et al., 2009), which is in line with our findings. It is interesting that the adverse effects on physical health are apparent in adolescents' own subjective ratings of their health and through the more objective list of physical symptoms. It is also worthy to note that the way in which CVE adversely affected these health outcomes was primarily through the mediating process, with the exception of younger adolescents' sleep. That is, there was little *detrimental* direct effect on general health and physical problems at Time 1.

What was unexpected at Time 1 was that the direct effect of CVE improved younger adolescents' general health. The indirect effect, however, is in the more expected negative direction so that the sum of the indirect and direct effects for younger adolescents' general health produced an insignificant total effect. This is in contrast to the hierarchical regression results where CVE's negative effect on general health was significant, although still small. Both the indirect effect and the hierarchical regression result were in the expected negative direction in which CVE exposure potentially harms general health. The positive direct effect is interesting, but it may only be a spurious effect of the sampling statistics.

Although small, the indirect effects for physical activity are interesting in the sense that they are negative in sign as expected, whereas the direct and total effects are positive. The negative indirect effect is more in line with the initial expectation that CVE

would drive adolescents inside and thereby decrease their amount of physical activity. The initial expectation would apply to those adolescents who have poor self-regulation skills or who are more emotionally vulnerable to traumatic events, and the indirect effect supports this expectation. Such adolescents may have been in the minority, however, since the total effect of CVE was positive, increasing physical activity. In an earlier discussion it was suggested that the positive effect on physical activity may be adolescents coping with CVE through high levels of physical activity. Such a pathway is more along the lines of research (Biddle, 1993; Wills et al., 2007; Strauss et al., 2001) suggesting there is protective effect of exercise and participation in sports against a number of unhealthy conditions such as poor self-esteem, loneliness, depression, and sedentary behavior.

There was no indirect effect on BMI and sedentary behavior at Time 1. Yet what was interesting was that CVE had a direct effect on younger adolescents' BMI, increasing it as expected and just missed significance for older adolescents. Also CVE had a direct effect on older adolescents' sedentary behavior, increasing it as expected. This suggests that being exposed to community violence, especially during early adolescence when one is more vulnerable, can lead to weight gain but not through feeling depressed or lacking self-regulation skills. Younger adolescents may find refuge from CVE in food while older adolescents escape by playing video games and watching television.

An explanation for the weak mediating link between CVE and BMI is that the pathways between BMI and depression are reversed. As discussed early, it is unclear whether being depressed leads to weight gain or whether weight gain causes depression.

That is, BMI may be the cause for depression, and possibly self-regulation, not the other way around as assumed in this study. Such a pathway is more consistent with some research findings (Swallen, Reither, Haas, & Meier, 2005; Mustillo et al., 2003). If this reversed pathway were more correct, then depression would have nothing to do with a causal relation between CVE and BMI, and there would be no proposed mediating effect. Whether BMI mediates the effect of CVE on depression should be explored in future studies.

As with the effects of CVE on health in the hierarchical analysis, in general the indirect effects in the mediator analysis decreased with time suggesting a healing effect. However, this must be considered in light of the many studies that relate childhood violence exposure to adult disorders, clearly indicating time does not heal all adverse effects; it may only help. Many of the studies refer to adolescents who had repeatedly been exposed to violence over a long period of time, usually in the form of parental maltreatment. Only 11% and 8% of the adolescents in this study were exposed to community violence or maltreatment more than once, respectively, so it is unlikely that many of them experienced the severe level of violence exposure referred to in the literature.

Although the effects were smaller at Time 2, depression and self-regulation continued to mediate the effect of CVE on adolescents' health. Mediation was again found for general health, physical problems, sleep, and physical activity in the expected direction. However, a few mediation paths differed from Time 1. That is, only self-regulation mediated the effect of CVE on younger adolescents' physical problems and

only depression mediated the effect of CVE on older adolescents' general health. The depression due to CVE seems to cause older adolescents to feel worse about their general health one year later, which makes sense since general health is a subjective measure and thus often determined by emotional health. The indirect effect on older adolescents' sleep just missed significance, and interestingly while there was no longer a direct effect on older adolescents' sedentary behavior, an indirect effect was still found through self-regulation. Older adolescents, who may not be able to regulate their behavior, may become easily enticed by videogames and television.

A direct effect of CVE was found on younger adolescents' physical problems and older adolescents' sleep. It must be noted that there was also a direct effect on older adolescents' physical activity but again increasing activity level rather than decreasing it as expected.

Again the effects were small and generally insignificant for BMI and sedentary behavior at Time 2. Given all the studies linking CVE to depression and depression to BMI, the insignificant indirect link was unexpected. However, weight gain could be slow to develop, and as mentioned earlier most studies relate violence exposure in adolescents with obesity as an adult. Whatever effects CVE might have had on weight gain may not yet be seen.

The lack of effect of CVE on younger adolescents' sedentary behavior across time was somewhat unexpected given that CVE did have a small but significant effect in the hierarchical analysis. Apparently the younger age group in this study did not turn to video games or television as a refuge. The explosive popularity of video games, in particular,

indicates that they are an activity of choice rather than a possible response to an outside influence. Modern video games involve interactivity and a certain amount of manual dexterity that may make them more enjoyable to older adolescents, possibly explaining the effects seen on this group. It is even possible that the pathway between sedentary behavior and CVE is reversed, and that sedentary behavior keeps adolescents inside and protects them from community violence exposure. This conjecture is not supported in the hierarchical analysis, however, but it might be an interesting future study. Video games continue to get more sophisticated, which in turn makes them more appealing, so their effect on adolescents, whatever it might be, will also increase.

Summary. In general adolescents are healthy so when they show signs of either emotional or physical problems, it is concerning. This study has demonstrated that those adolescents who are exposed to violent events in their neighborhood have an increased risk for health problems. As hypothesized, findings have explained the process by which the violence exposure can be detrimental on health, namely through increases in depressed symptoms and decreases in the ability to self-regulate behaviors and thoughts. Specifically exposure to community violence negatively affects adolescents emotionally and cognitively which in turn leads to poorer general health, more physical problems, more sleep disruptions, and less physical activity. Despite a few differences, mediation through depression and self-regulation continued to explain the effect of CVE on health one year later but to a less degree, in line with the diminished effects of CVE over time. Most of the time, depression and self-regulation did not explain how community violence exposure affects weight gain (BMI) and sedentary behavior. However, it should be noted

that CVE did have a direct effect on BMI and sedentary behavior at Time 1 but effects diminished to negligible after one year.

It should be noted that choosing mediators and mediation paths is not straightforward, which is indicated in this study by the lack of indirect effects on BMI unexpected directs effects on physical activity and that they possibility could be explained more fully by a different pathway. In one study (Grant et al., 2005) CVE was found to be a mediator between poverty and psychological symptoms, which is quite different from our use of SES as a covariate. The choice of mediators and pathways will probably always be difficult and subject to controversy, and it will have an effect on any conclusions reached in studies such as this.

Question 3: Does Gender Moderate the Effect of Community Violence Exposure on Adolescents' Health?

The third goal of the study was to investigate whether the effect of community violence exposure affects males differently than females. The process by which being exposed to community violence affects health may be better explained through depression and self-regulation if one gender were more susceptible to them than the other. It was expected that females would exhibit more symptoms of depression due to the violence exposure and therefore their health would be more adversely impacted. As pointed out by the partial regression coefficients in Table 15, males and females were relatively similar in the way in which CVE affected them. The same pathways existed across gender at Time 1; only two health indicators were affected differently one year later, sedentary behavior and BMI. For younger adolescents, females who were more

exposed to CVE became more sedentary one year later whereas the reverse effects were seen in males who became slightly less sedentary one year later. For older adolescents, females exposed to CVE had higher BMI levels one year later whereas males who were exposed had relatively no change in their BMI levels. Figures 4 and 5 illustrate how the mediating paths differed between males and females for sedentary behavior and BMI, respectively. Not as expected, self-regulation mediated the path from CVE to sedentary behavior as well as from CVE to BMI for females such that being exposed to community violence decreased self-regulation which then led to increases in sedentary behavior for younger females and increases in BMI for older females. It is possible that it is this sedentary behavior that occurs in early adolescence that leads to BMI increases seen in later adolescence. A clear pathway through self-regulation was not found for males. While symptoms of depression increased with exposure to community violence, they did not predict increases in sedentary behavior and BMI levels.

Summary. Community violence exposure had similar effects on males and females' health. The one exception to this was that at Time 2 CVE affected sedentary behavior and BMI differently for females than it did for males. The effect of CVE led to decreases in self-regulation, which then lead to increases in sedentary behavior for younger females and BMI for older females. Males became less sedentary with increased exposure but not through changes in depression or self-regulation. This might offer insight into effective strategies that work best for each gender in future health-improving interventions. Based on these results, efforts might be best applied by focusing on improving young girls' self-regulation skills, particularly around eating and weight

control. Future research may want to explore this path for males in order to better understand the process in which CVE affects these two outcomes, which may be of importance when trying to explain why boys are less susceptible.

Effects of Covariates

Childhood maltreatment. What was unexpected was the small effect that childhood maltreatment had on the health outcomes, which contradicts a large body of evidence. For both age groups, childhood maltreatment had a small, negative effect on general health and physical problems but had no relation with BMI, physical activity, or sedentary behavior. There are a number of possible explanations for this that relate to how childhood maltreatment was assessed in the study and not to a lack of a cause-and-effect relation. Specifically, the three items comprising maltreatment were posed at Wave III and thus were retrospectively examined. This brings bias into the measure since the now-early-adults may have suppressed maltreatment experiences that occurred to them at such a young age, relegating them to less importance, or simply wanting to forget them. It is also possible that participants felt uncomfortable about answering such personal and confidential questions and consequently chose to ignore them or provide incorrect information. Moreover most of the childhood maltreatment was reported to be in the form of physical abuse, such as slapping. Possibly a certain percentage of early adults, subject as a child to that kind of physical abuse from their parents, would in retrospect regard it as normal disciplinary behavior, not abuse.

Weight problems have been specifically linked to sexual abuse (e.g., Noll, Zeller, Trickett, & Putman, 2007; D'Argenio, Mazzi, Pecchioli, Di Lorenzo, Siracusano, &

Troisi, 2009; Williamson, Thompson, Anda, Dietz, & Felitti, 2002; Greenfield & Marks, 2009), so it is somewhat surprising that for both age groups, childhood maltreatment did not account for higher BMI levels. The lack of evidence here may be due to the low percentage of participants in the study who reported sexual abuse (6%), so this very severe form of maltreatment may not have had enough power to influence the maltreatment effect. As mentioned earlier high BMI levels may be slow to develop, so the effects of childhood maltreatment may still be observed sometime in the future.

Gender. Across all analyses, four of the health variables that implied the potential for better overall health favored males while two favored females. Males reported better general health, fewer physical problems, better sleep patterns, and more physical activity. Females, on the other hand reported lower BMI levels and less sedentary behavior. The higher physical activity is not surprising for males since they have been traditionally more involved in athletics and other physical activities (Trost et al., 2002; Sallis, Prochaska, & Wendell, 2000). The lower sedentary behavior for females is not surprising since the types of sedentary behaviors examined (video and computer games) tend to be more male-oriented activities.

Males' more positive responses about their general health and physical problems implied that they were more optimistic about their health status. The fact that boys reported higher BMI levels is inconsistent with the National Health and Nutritional Examination Survey (NHANES), which reported that girls between the ages of 7 and 16 have slightly higher BMI scores. The large sample numbers in this study make it

improbable that the higher male BMI score is a sampling error, so the inconsistency remains unexplained.

Socioeconomic status (SES). Adolescents from higher SES families reported better subjective general health, but they also reported more physical problems at both time periods. This may not be surprising since a higher income may have allowed participants more opportunities to obtain medical care, and therefore they could have been more cognizant of any health problems. Findings also supported the expectations that higher SES is linked to more physical activity and less sedentary behavior. Those from higher SES families most likely lived in safer neighborhoods with more opportunities for physical play outside the home Moreover they can afford to partake in activities associated with a cost, such as tennis instructions or soccer leagues.

In general, SES was a good predictor of health status. Socioeconomic disadvantage is one of the most prevalent factors underlying poor health. In describing the devastating effects of poverty among our youth, Schorr argued "poverty is the greatest risk factor of all. Family poverty is relentlessly correlated with …violent crime" (p. xxii, 1988). The findings here support previous work that there is an interactive relation among poverty, violence, and health (e.g., Groves, 1996; Goodman, Slap, & Huang, 2003). In drawing a connection between CVE and health it was necessary to consider SES. The relation between CVE and SES in this study was significant and negative ($r = -.13$, $p < .001$). Low SES forces children to live in poor neighborhoods, which are breeding grounds for violent crime and often do not have the proper health resources to care for them. In addition poor neighborhoods are less likely to have

playgrounds and parks that are safe, forcing adolescents to either play in unsafe areas or stay inside out of harm's way.

Limitations and Future Research

The current study had several limitations that need to be addressed. First, the questions used to measure the variables were chosen before this study was anticipated; there was no control over the data gathering process. Although there are many advantages to using on a large, nationally representative dataset, the one main disadvantage is that the basis upon which the constructs are created is limited to the predetermined questions. Additional questions relating to community violence exposure that might have been pertinent to the present study may be how brutal the event, how regular was the event, or how familiar was the perpetrator or victim. Such questions would have improved the ability to understand and accurately evaluate the violent experience. Whether or not the perpetrator or victim is known can play an important role in the severity of the effect, and there is no indication if multiple community violence exposure occurred regularly or many years apart.

The CVE assessment consisted of four victimization questions and one witnessing of violence question. There is ongoing debate over whether the two should be conceptualized as separate dimensions, since it seems likely that the negative effect of witnessing violence in the community would not be as serious as being the victim. Margolin and Gordis (2004) argue that it is not witnessing and victimization that should be distinguished, but rather researchers need to be concerned with how to evaluate severe from mild exposures. However, this measure attempts to address this issue by making the

witnessed violence very severe, namely witnessing someone being shot or stabbed. Witnessing a violent act, particularly if it is a friend or relative, is clearly a severe traumatic experience and can be viewed as indirect victimization (Finkelhor, Omrod, Turner, & Hamby, 2005). That is, the witness would perceive the event as life threatening, whether it one's own life or the life of someone else.

In addition the two forms of violence, witnessing and victimization, were highly correlated ($r = .94$, $p < .001$), suggesting they typically co-occur. Moreover of the 26% of participants exposed to community violence, only 6% were just a witness. Adding the two types of community violence exposure also reduced the skew in the CVE distribution and improved the model fits. This was further supported by a separate mediation analysis conducted using just those adolescents who were victims of violence. The pattern of effects remained roughly the same. We therefore followed the strategy of many previous studies (e.g., Schwab-Stone et al., 1999; Brady, 2008) and did not separate adolescents exposed to community violence into witnesses and victims.

It would be still interesting, however, for future research to determine if certain types of violence, depending on their severity, have more impact than others. It may also be of interest to compare how the frequency of the exposures impacts health; that is, how do the effects of a one-time extreme exposure to community violence differ from frequent, low-level exposures? If possible such studies should incorporate a prospective childhood maltreatment covariate to improve the accuracy of the maltreatment score and better separate the effects of community violence exposure and childhood maltreatment.

This study also did not take into account adolescents' own violent behavior. A well known outcome of experiencing violence is to become violent (e.g., Dodge, Bates, & Pettit, 1990; Resnick, Ireland, & Borowsky, 2004). Future studies may want to control for adolescents' violent behavior, in order to determine whether it is linked to poor health.

As seen in this study, socioeconomic status (SES) plays a role in all the health outcomes, and it may be just as important a predictor of health outcomes as community violence exposure. Low SES status may force families to live in unsafe neighborhoods, and future studies should follow up on work of Grant et. al. (2005) to determine if unsafe neighborhoods affect adolescents' health through the mediation of community violence exposure. The existence of such a link would suggest that providing safe neighborhoods (e.g., safe parks and centers) would be an effective way to improve adolescents' health. That may be a more reachable objective than preventing neighborhood violence.

Physical activity and sedentary behavior are also limited in their characterization because of type of questions that were asked. There is a broad spectrum to these behaviors that is difficult to capture by asking adolescents a small set of specific questions as done in this large national study. Formulating questions for a study like this is important, and there is no doubt that the designers of the Add Health survey were careful to design the questions in such a way that specific behaviors and social conditions would be accurately recognized. However, users of the Add Health data may have other conditions in mind for which the questions are less relevant or incomplete. While we benefited from a very large, nationally represented sample, there are certain additional

items that would possibly have better assessed our set of targeted questions. For example, physical activity in this study, as in many others (e.g., Gordon-Larsen, McMurray, & Popkin, 1999; Knutson, 2005; Nelson, Gordon-Larsen, Adair, & Popkin, 2005) was assessed only by the frequency of exercise or participation in a number of specific sports. Physical activity, in general, is difficult to assess because there are other aspects that need to be taken into account to reliably capture how physically active one is. Future studies should include the intensity, the duration, the frequency, and the type of activity, which were not addressed in this measurement. A better understanding of the term "physical activity" may help us better understand how community violence exposure is positively linked to increased physical activity.

Sedentary behavior was defined as the amount of time adolescents spend with media, for example television, videogames, and computers. There is some concern with this measure because it may in fact be capturing a type of personality that enjoys gaming rather than identifying someone who has withdrawn from active play because of violence exposure. Traditionally screen time has been exclusively passive; however, that is changing as video games are now interactive and require skill, quick reflexes, and manual dexterity. The questions assessing sedentary behavior should be brought up to the date. Nevertheless in order to obtain a general understanding of what sedentary behavior means across different studies, our criteria for sedentary behavior are similar to those used elsewhere (e.g., Gordon-Larsen, McMurray, & Popkin, 2000; Knutson, 2005; Nelson, Gordon-Larsen, Adair, & Popkin, 2005) even though they may no longer capture

the original concept of the behavior. Future research should include other types of sedentary behavior, particularly those that may be more female-oriented.

For both physical activity and sedentary behavior the questions used to characterize the condition are the same in many different studies. This is necessary so that characterizations will be on the same scale and be understood by all researchers. In many cases therefore it could be a disadvantage to change the questions, or introduce new ones even if possible.

All scales in this study were based on self-reports and were consequently subject to personal biases. Adolescents' responses may reflect health behaviors and experiences they would like to possess, rather than what they do possess. This may have been particularly true for females' self-report of weight (BMI), since weight can be a very sensitive topic for adolescent girls. This is a possible explanation to the higher male BMI scores in this study, which is contrary to national trends. In the case for boys, they may have exaggerated their reporting of violence exposure in order to seem more worldly and experienced. It may be important for future research to also include observational and/or third party data to verify the accuracy of adolescents' responses.

As mentioned throughout the paper, the direct and indirect effects of CVE on the health-related variables chosen for this study were small, and the same is true for the other independent variables. The large sample size allowed most of these small effects to be regarded as statistically significant, with the surprising exception of BMI, where the effect of CVE was not significant at Time 2 for both age groups. Weight problems have been specifically linked to abuse in many previous studies, but most of them report on

weight problems acquired in adulthood, giving the accumulative effect of weight gain from violence exposure plenty of time to be noticed. It may be of interest to re-evaluate the adolescents in early adulthood and mid-adulthood in order to investigate any lasting effects of CVE. Although the Add Health dataset, which was used in this study, includes additional waves, many of the variables used here were not assessed at these later times.

In the present study, only two mediating pathways between community violence exposure and health-related outcomes were explored, yet other important mediation processes may exist. Future research could investigate how these other physiological, behavioral, and social mechanisms might be mediating or directly affecting the CVE-health relation. This study also did not investigate possible factors that might buffer or negate the effects of community violence exposure on adolescents' health. Future studies could address the question on why some adolescents appear to be more resilient to the effects of community violence exposure. One obvious factor to be included is parental monitoring and support. Social support by peers may also protect adolescents from adverse effects of violence. Personal characteristics of the adolescent, such as coping skills, athletic ability, and popularity among peers, would be other factors that explain resilience that future research may consider.

It must also be noted that the depression mediator may reflect the child's emotional state at the time, not whether or not he or she was truly depressed. Easily triggered reactions to community violence exposure could enhance the depression score and increase the apparent mediating effect. Nevertheless such enhanced emotional

vulnerability appears to have a mediating effect, even if it may not be depression as defined clinically.

Other mediators could have been chosen, and some of the health related outcomes in this study could be potential mediators for other outcomes. It would be reasonable, for example, to think of sleep as a mediator for general health, physical problems, or BMI.

Implications and Conclusions

Community violence exposure had its largest effect on physical problems, sleep, and general health. It had its smallest effect on BMI and sedentary behavior. Physical activity fell somewhere in between. In general this was true for both age groups and across time points.

The total effects on general health, physical problems, and sleep were seen in the hierarchical regression analysis, which was conducted without mediation. A second analysis that included the mediators of depression and self-regulation revealed that much of the effect of CVE on these outcomes was indeed mediated in the sense that the computed indirect effects were statistically significant, with a few exceptions for older adolescents at Time 2. These results were consistent with the pre-study expectations. However, despite being statistically significant all the effects were small. In general a one unit increase in community violence exposure produced less than a 10% change in the corresponding unit of the health variable. Moreover the small R-squared statistic indicated that the independent variables chosen in this study, including community violence exposure, gender, socioeconomic status, and childhood maltreatment, did not fully explain the health outcomes, and that other factors must have been involved.

Among them probably were health-endangering behaviors such as alcohol and drug abuse, smoking, and unsafe sexual practices, which are known to be consequences of community violence exposure.

With only a few exceptions the magnitude of the CVE effects was less at Time 2 than it was at Time 1, indicating a possible healing effect of time. This is comforting, although it is not always the case given that so many studies relate childhood violence exposure to obesity and psychological disorders in later life. It is possible that harmful effects of many severe, traumatic events are suppressed or slow to develop and do not show up for many years after the event(s).

The community violence exposure effect on physical activity was also small and generally significant. What was interesting with this health outcome was the indirect mediating effects were in an opposite direction to that of the direct and total effects. The indirect effects of community violence exposure were negative and indicated that depression and lack of self-regulation can cause physical activity to decrease as a consequence of community violence exposure. This was the expected effect. On the other hand the direct and total effects were positive, giving rise to a counter-intuitive notion that community violence exposure actually increased physical activity. It was more likely that physical activity acted as a buffer to the adverse effects of violence exposure, such that many of the adolescents used physical activity as an escape from the violence, and consequently were more resilient. This suggests that increasing the level of physical activity among adolescents, in particularly coaching them so they become more proficient in the activities they engage in, may make them more resistant to the effects of violence

exposure. The increased resistance could come in the form of increased self-confidence, a wider circle of friends, or even from the physiological benefits that are known to accompany vigorous exercise. Such a suggestion could be regarded as conventional wisdom and obvious, but empirical evidence as seen here provides a solid argument for the design of such programs.

Community violence exposure had the least effect on BMI and sedentary behavior. The small effect on BMI was particularly unexpected given the many studies relating obesity to both violence exposure and the two chosen mediators. Obesity, however, is slow to develop, and many studies relate childhood violence exposure to adult obesity. The deleterious effects of community violence exposure on obesity may not have shown up yet. Moreover the model proposed for this effect may not have been framed properly. There is plenty of evidence to suggest that obesity may cause depression rather than the other way around as this study has it.

With regard to the effect of community violence exposure on sedentary behavior, it was originally expected that the violence would drive adolescents indoors forcing them to turn to television, video games, or computers for entertainment. Such an effect was not seen, and upon reflection it is not surprising. It is likely that adolescents are turning to video games by choice, not by outside influences. Video games are increasingly more popular and enjoyable as they become more interactive and require skill, manual dexterity and fast reflexes to master. Indeed, video games are also becoming more popular among adults. It may be that community violence exposure is increasing sedentary behavior but not specifically through increases in media. Future research may

want to address this limitation and examine the effects using different criteria for sedentary behavior.

Moderating effects of gender showed up for younger adolescents' sedentary behavior and older adolescents' BMI at Time 2. Females exposed to community violence had increases in sedentary behavior whereas males had a slight decrease one year later; females exposed to community violence had increases in BMI whereas males' BMI showed no change one year later. In all other circumstances the interaction variable measuring the moderation effect never rose to significance. There were, however, gender differences in the sample population with respect to almost all the health-related variables, community violence exposure, and the mediators.

With respect to the covariates, it was unexpected that childhood maltreatment would have such a minor effect on the health-related outcomes. The maltreatment assessment was made retrospectively, about six years after the health and community violence assessments were made, and so it is possible that memories of maltreatment were suppressed or forgotten. Moreover the number of adolescents who reported any kind of maltreatment was small relative to the total number of participants in the survey. This was particularly true for repeated episodes of maltreatment, which were reported by only 8% of participants. Possibly this number is too small to allow maltreatment to be captured as a significant influence on health. There are of course many studies that show quite clearly that maltreatment has a significant impact on health. However, many of those studies involve a large population of individuals who were maltreated. Their health

outcomes were then compared with a control group. It is quite different from this study where a sample of the general population is used, not a sample of maltreated adolescents.

Unlike maltreatment socioeconomic status was an important factor in all the health-related outcomes, with the not-surprising exception of sleep. Poor families are often limited to the neighborhood they can afford and are forced to live in more dangerous neighborhoods where chances for violence exposure increase. Affluent families, on the other hand, live in safer neighborhoods and can provide their children better medical care and more opportunities for safe physical activity. Using socioeconomic status and community violence exposure as separate independent variables assumes that they are independent, but of course they are not. A better model may have been to put one or the other into the model as a mediator. Results from such a study may point the way toward more effective strategies to reduce the impact of community violence. For example, it may have been found that poor neighborhoods become dangerous and thus become a causative factor for community violence. A better strategy might be to focus proactively on making the neighborhoods safe rather than spending most of the resources trying to solve violent crimes after they have been committed.

The current study highlights two main issues. One, that is obvious, is the importance of violence prevention efforts among adolescents. Second and less obvious is that it may be important for such efforts to address the depression and low self-regulation that result from being exposed to community violence. This study provides evidence that these two pathways may be key in preventing poor health that result from community

violence exposure. Although some of the health variables used in this study may not be in and of themselves dangerous or unhealthy, if continued over long periods of time, they will have substantial consequences on long-term health. Adolescents are generally healthy, but if they maintain poor health behaviors, the consequences will catch up to them.

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Table 1

Means, Standard Deviations, and Paired t-Tests of Study Variables by Time

	Time 1		Time 2		Paired
	M	*SD*	*M*	*SD*	*t*-tests
Predictors					
Community Violence Exposure	.43	.88			
Mediators					
Depression	4.68	4.19			
Self-Regulation	2.20	1.71			
Outcomes					
General Health	3.88	.92	3.91	.90	$t(14717) = -4.05$**
Physical Problems	7.17	2.73	7.32	2.64	$t(14648) = -7.48$**
Adiposity (BMI percentile)	59.46	28.52	58.67	28.72	$t(13909) = -3.03$*
Sleep Quantity	7.78	1.39	7.58	1.39	$t(14647) = 15.15$**
Sleep Quality	.72	.45	.70	.46	$t(14706) = 5.32$**
Physical Activity	3.74	2.14	3.52	2.09	$t(14720) = 12.26$**
Sedentary Behavior	23.38	21.83	21.85	21.09	$t(14589) = 7.97$**

Note. *M* = mean. *SD* = standard deviation. * $p < .01$. ** $p < .001$.

Table 2

Means, Standard Deviations, and Paired t-Tests of Study Variables by Gender and Time

	Time 1		Time 2		Paired
	M	*SD*	*M*	*SD*	*t*-tests
Males					
Predictors					
Community Violence Exposure	.60	1.04			
Mediators					
Depression	4.08	3.64			
Self-Regulation	2.12	1.64			
Outcomes					
General Health	3.96	.89	4.01	.89	$t(7168) = -4.14$**
Physical Problems	6.80	2.73	6.89	2.62	$t(7147) = 2.95$**
Adiposity (BMI percentile)	60.43	28.73	59.96	28.79	$t(6822) = 1.25$
Sleep Quantity	7.83	1.37	7.65	1.38	$t(7133) = 9.81$**
Sleep Quality	.76	.43	.74	.44	$t(7161) = 3.20$**
Physical Activity	4.21	2.16	3.95	2.11	$t(7168) = -9.55$**
Sedentary Behavior	26.14	23.36	24.54	22.83	$t(7105) = 5.37$**
Females					
Predictors					
Community Violence Exposure	.265	.66			
Mediators					
Depression	5.26	4.58			
Self-Regulation	2.28	1.77			
Outcomes					
General Health	3.80	.92	3.82	.91	$t(7547) = -1.68$
Physical Problems	7.73	2.60	7.51	2.73	$t(7526) = 7.63$**
Adiposity (BMI percentile)	58.54	28.28	57.44	28.60	$t(7085) = 3.03$*
Sleep Quantity	7.73	1.43	7.53	1.40	$t(7512) = 11.59$**
Sleep Quality	.70	.46	.66	.47	$t(7543) = 4.29$**
Physical Activity	3.30	2.02	3.11	1.98	$t(7550) = 7.77$**
Sedentary Behavior	20.76	19.92	19.30	18.96	$t(7482) = 5.98$**

Note. *M* = mean. *SD* = standard deviation. * $p < .01$.** $p < .001$.

Table 3

Means, Standard Deviations, and Paired t-Tests of Study Variables by Age and Time

	Time 1		Time 2		Paired
	M	*SD*	*M*	*SD*	*t*-tests
Younger Adolescents					
Predictors					
Community Violence Exposure	.36	.81			
Mediators					
Depression	4.20	3.93			
Self-Regulation	2.05	1.67			
Outcomes					
General Health	3.91	.89	3.93	.89	$t(7557) = -2.39$*
Physical Problems	7.15	2.70	7.31	2.62	$t(7535) = 5.28$**
Adiposity (BMI)	60.51	28.35	60.86	28.11	$t(7135) = -.79$
Sleep Quantity	8.08	1.35	7.84	1.39	$t(7532) = 13.72$**
Sleep Quality	.78	.42	.75	.43	$t(7550) = 5.55$**
Physical Activity	4.14	2.12	3.98	2.08	$t(7556) = 6.17$**
Sedentary Behavior	25.19	22.54	23.34	21.95	$t(7470) = 6.62$**
Older Adolescents					
Predictors					
Community Violence Exposure	.49	.95			
Mediators					
Depression	5.20	4.39			
Self-Regulation	2.37	1.73			
Outcomes					
General Health	3.85	.93	3.89	.92	$t(7175) = -3.35$*
Physical Problems	7.18	2.76	7.34	2.67	$t(7138) = 5.29$**
Adiposity (BMI)	58.52	28.72	56.73	29.24	$t(6772) = 5.21$*
Sleep Quantity	7.46	1.38	7.32	1.41	$t(7113) = 7.55$**
Sleep Quality	.67	.47	.65	.48	$t(7154) = 2.08$**
Physical Activity	3.32	2.08	3.04	1.98	$t(7162) = 11.41$**
Sedentary Behavior	21.48	20.89	20.29	20.05	$t(7117) = 4.55$**

Note. *M* = mean. *SD* = standard deviation. * $p < .01$. ** $p < .001$.

Table 4

Intercorrelations Among Predictor, Mediators, and Health-related Outcomes

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. CVE W 1	1.00															
2. Depres-sion W 1	.19*	1.00														
3. SR W 1	.15*	.55*	1.00													
4. GH W 1	-.06*	-.25*	-.21*	1.00												
5. GH W 2	-.07*	-.22*	-.18*	.52*	1.00											
6. PP W 1	.07*	.34*	.38*	-.23*	-.18*	1.00										
7. PP W 2	.07*	.26*	.29*	-.14*	-.20*	.55*	1.00									
8. BMI W 1	.05*	.01	.02+	-.07*	-.07*	-.01	-.02+	1.00								
9. BMI W 2	.06*	.02+	.02*	-.14*	-.14*	-.02+	-.04*	.42*	1.00							
10. Sleep Quantity W 1	-.07*	-.16*	-.16*	.06*	.07*	-.09*	-.08*	-.01	-.03*	1.00						
11. Sleep Quantity W 2	-.07*	-.11*	-.09*	.05*	.07*	-.07*	-.11*	-.02	-.02*	.37*	1.00					
12. Sleep Quality W 1	-.06*	-.25*	-.31*	.12*	.11*	-.20*	-.17	-.01	.01	.32*	.18*	1.00				
13. Sleep Quality W 2	-.04*	-.20*	-.22*	.09*	.13*	-.17*	-.22*	.00	-.01	.18*	.34*	.38*	1.00			
14. PA W 1	.06*	-.12*	-.10*	.19*	.16*	-.03*	-.04*	.01	.02*	.08*	.06*	.09*	.07*	1.00		
15. PA W 2	.04*	-.13*	-.11*	.16*	.19*	-.04*	-.03*	.02+	.03*	.11*	.09*	.11*	.10*	.47*	1.00	
16. SB W 1	.08*	.02*	.04*	-.05*	-.04*	.01	-.01	.04*	.07*	.05*	.03*	.03*	.02*	.02 +	.03*	1.00
17. SB W 2	.08*	.04*	.05*	-.05*	-.05*	-.01	.00	.03*	.06*	.03*	.06*	.02 +	.03*	.01	.02*	.42*

Note. CVE = community violence exposure. SR = self-regulation. GH = general health. PP = physical problems. PA = physical activity. SB = sedentary behavior. W1 = wave 1. W2 = wave 2. * $p < .01$. + $p < .001$.

Table 5

Correlations of Gender and Age with Mediators and Health-related Outcomes

	Gender	Age
Mediators		
Depression	.14**	.15**
Self-regulation	.05**	.11**
Time 1 Outcomes		
General Health	-.087**	-.03**
Physical Problems	.13**	.01
Adiposity (BMI percentile)	-.03**	-.04**
Sleep Quantity	-.03**	-.25**
Sleep Quality	-.08**	-.14**
Physical Activity	-.21**	-.20**
Sedentary Behavior	-.12**	-.10**
Time 2 Outcomes		
General Health	-.10**	-.03**
Physical Problems	.16**	.00
Adiposity (BMI percentile)	-.04**	-.07**
Sleep Quantity	-.04**	-.21**
Sleep Quality	-.09**	-.12**
Physical Activity	-.20**	-.26**
Sedentary Behavior	-.12**	-.08**

Note. ** $p < .01$.

Table 6

Correlations Among Predictor, Mediators, and Health-related Outcomes for Males and Females

	Males			Females		
	Predictor	Mediators		Predictor	Mediators	
	CVE	Depression	Self-regulation	CVE	Depression	Self-regulation
Mediators						
Depression	.23**			.23**		
Self-regulation	.16**			.17**		
Time 1 Outcomes						
General Health	-.08**	-.21**	-.17**	-.09**	-.26**	-.23**
Physical Problems	.10**	.32**	.37**	.10**	.34**	.39**
BMI percentile	.05**	.00	.02	.05**	.03*	.02
Sleep Quantity	-.11**	-.18**	-.16**	-.05**	-.15**	-.15**
Sleep Quality	-.07**	-.23**	-.28**	-.07*	-.25**	-.33**
Physical Activity	.03*	.12**	-.11**	.02	-.07**	-.08**
Sedentary Behavior	.05**	.03*	.05**	.08**	.05**	-.05**
Time 2 Outcomes						
General Health	-.09**	-.20**	-.16**	-.09**	-.21**	-.18**
Physical Problems	.11**	.23**	.28**	.09**	.26**	.29**
BMI percentile	.03*	-.02	-.01	.08**	.06**	.06**
Sleep Quantity	-.10**	-.11**	-.09**	-.05**	-.10**	-.09**
Sleep Quality	-.08**	-.18**	-.21**	-.04**	-.19**	-.23**
Physical Activity	.01	-.11**	-.10**	.01	-.11**	-.10**
Sedentary Behavior	.04**	.04**	.06**	.10**	.08**	.05**

Note. CVE = community violence exposure. * $p < .05$. ** $p < .01$.

Table 7

Correlations Among Predictor, Mediators, and Health-related Outcomes for Younger and Older Adolescents

	Younger			Older		
	Predictor	Mediators		Predictor	Mediators	
	CVE	Depression	Self-regulation	CVE	Depression	Self-regulation
Mediators						
Depression	.23**			.14**		
Self-regulation	.18**			.12**		
Time 1 Outcomes						
General Health	-.05**	-.22**	-.19**	-.07**	-.26**	-.21**
Physical Problems	.09**	.35**	.38**	.05**	.34**	.38**
BMI percentile	.06**	.02	.03*	.06**	.01	.02
Sleep Quantity	-.07**	-.25**	-.14**	-.05**	-.13**	-.14**
Sleep Quality	-.08**	-.14**	-.29**	-.02*	-.22**	-.31**
Physical Activity	.05**	-.12**	-.08**	.10**	-.09**	-.08**
Sedentary Behavior	.08**	.03**	.05**	.10**	.04**	-.05**
Time 2 Outcomes						
General Health	-.07**	-.20**	-.17**	-.06**	-.23**	-.18**
Physical Problems	.08**	.26**	.29**	.06**	.27**	.29**
BMI percentile	.07**	.04**	.04**	.05**	.01	.03*
Sleep Quantity	-.06**	-.12**	-.10**	-.05**	-.06**	-.06**
Sleep Quality	-.05**	-.19**	-.21**	-.03*	-.18**	-.22**
Physical Activity	.02	-.14**	-.11**	.10**	-.08**	-.08**
Sedentary Behavior	.08**	.03**	.05**	.10**	.06**	.06**

Note. CVE = community violence exposure. * $p < .05$. ** $p < .01$.

Table 8

Summary of Hierarchical Regression Analysis for Younger Adolescents at Time 1 (N=7558)

Variable	B	Model 1 SE(B)	ß	B	Model 2 SE(B)	ß
General health						
Gender	-.134	.026	-.76***	-.141	.026	-.079***
SES	.071	.010	.106***	.069	.010	.102***
Maltreatment	-.074	.018	-.061***	-.071	.018	-.058***
CVE				-.052	.026	-.030*
Adjusted R^2		.021***			.021***	
F for model		32.823			25.651	
Physical Problems						
Gender	.622	.079	.117***	.695	.079	.131***
SES	.077	.030	.038*	.101	.030	.050**
Maltreatment	.219	.055	.060***	.184	.055	.050**
CVE				.563	.078	.109**
Adjusted R^2		.017***			.028***	
F for model		26.410			33.160	
BMI						
Gender	-2.782	.870	-.050**	-2.309	.876	-.041**
SES	-1.779	.335	-.083***	-1.614	.336	-.075***
Maltreatment	-.463	.610	-.012	-.691	.611	-.018
Parents' weight	5.155	.736	.105***	5.167	.671	.105***
CVE				3.719	.869	.067***
Adjusted R^2		.019***			.023***	
F for model		21.173			20.671	
Sleep						
Gender	-.126	.024	-.079***	-.150	.024	-.094***
SES	-.010	.009	-.017	-.018	.009	-.030*
Maltreatment	-.038	.016	-.034*	-.026	.016	-.024
CVE				-.185	.023	-.119***
Adjusted R^2		.007***			.020***	
F for model		10.995			24.056	
Physical Activity						
Gender	-.825	.062	-.194***	-.801	.062	-.189***
SES	.144	.024	.090***	.152	.024	.094***
Maltreatment	.066	.043	.023	.055	.043	.019
CVE				.180	.061	.043***
Adjusted R^2		.045***			.047***	
F for model		72.356			56.495	
Sedentary Behavior						
Gender	-.582	.057	-.149***	-.567	.058	-.145***
SES	-.167	.022	-.113***	-.162	.022	-.109***
Maltreatment	.077	.040	.028	.069	.040	.026
CVE				.121	.057	.032*
Adjusted R^2		.037***			.038***	
F for model		58.189			44.800	

Note. SE = standard error. * $p < .05$. ** $p < .01$. ***. $p < .001$.

Table 9

Summary of Hierarchical Regression Analysis for Younger Adolescents at Time 2 (N = 7558)

Variable	B	Model 1 SE(B)	ß	B	Model 2 SE(B)	ß
General health						
Gender	-.097	.023	-.055***	-.109	.023	-.061***
SES	.035	.009	.051***	.031	.009	.046***
Maltreatment	-.023	.016	-.018	-.017	.016	-.014
General health T1	.509	.013	.506***	.507	.013	.504***
CVE				-.091	.022	-.052***
Adjusted R^2		.272***			.275***	
F for model		423.147			342.954	
Physical Problems						
Gender	.463	.064	.090***	.494	.065	.096***
SES	.083	.024	.042**	.093	.025	.047***
Maltreatment	.137	.045	.039**	.125	.045	.035**
Physical problems T1	.510	.012	.529***	.506	.012	.524***
CVE				.221	.046	.044**
Adjusted R^2		.304***			.305***	
F for model		491.973			396.942	
BMI						
Gender	-2.501	.804	-.045**	-2.234	.810	-.041**
SES	-1.371	.311	-.064***	-1.310	.313	-.061***
Maltreatment	.909	.562	.023	.821	.564	.021
Parents' weight	8.347	.708	.169***	8.357	.708	.169***
BMI T1	.374	.014	.373***	.373	.014	.371***
CVE				1.420	.805	.026
Adjusted R^2		.193***			.193***	
F for model		190.864			159.657	
Sleep						
Gender	-.116	.022	-.071***	-.127	.023	-.078***
SES	-.006	.009	-.010	-.010	.009	-.016
Maltreatment	-.060	.016	-.053***	.055	.016	.049***
Sleep T1	.391	.014	.381***	.385	.014	.376***
CVE				-.082	.002	-.051***
Adjusted R^2		.158***			.160***	
F for model		211.901			172.656	
Physical Activity						
Gender	-.424	.057	-.102***	-.430	.057	-.104***
SES	.088	.021	.056***	.086	.021	.055***
Maltreatment	.031	.039	.011	.034	.039	.012
Physical activity T1	.393	.013	.403***	.393	.013	.403***
CVE				-.049	.055	-.012
Adjusted R^2		.195***			.195***	
F for model		274.584			219.811	
Sedentary Behavior						
Gender	-.276	.051	-.071***	-.261	.051	-.067***
SES	-.118	.019	-.080***	-.113	.019	-.077***
Maltreatment	-.070	.035	-.026*	-.077	.035	-.029*
Sedentary behavior T1	.454	.013	.468***	.463	.013	.467***
CVE				.115	.050	.031*
Adjusted R^2		.248***			.248***	
F for model		368.888			296.464	

Note. SE = standard error. T1 = Time 1. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 10

Summary of Hierarchical Regression Analysis for Older Adolescents at Time 1 (N =7166)

Variable	B	Model 1 SE(B)	ß	B	Model 2 SE(B)	ß
General health						
Gender	-.188	.030	-.102***	-.222	.031	-.120***
SES	.062	.011	.089***	.055	.011	.079***
Maltreatment	-.066	.021	-.050**	-.055	.021	-.042**
CVE				-.125	.027	-.078***
Adjusted R^2		.021***			.026***	
F for model		27.958			25.534	
Physical Problems						
Gender	.795	.086	.148***	.897	.088	.167***
SES	.179	.032	.088***	.200	.033	.099***
Maltreatment	.245	.061	.065***	.212	.061	.056**
CVE				.372	.077	.080***
Adjusted R^2		.031***			.036***	
F for model		41.251			36.925	
BMI						
Gender	-2.835	.975	-.049**	-2.245	1.002	-.039*
SES	-.323	.371	-.015	-.186	.374	-.009
Maltreatment	.586	.701	.014	.409	.704	.010
Parents' weight	5.679	.844	.114***	5.644	.844	.113***
CVE				2.232	.888	.044*
Adjusted R^2		.015***			.017***	
F for model		14.155			12.607	
Sleep						
Gender	-.156	.026	-.096***	-.180	.027	-.111***
SES	-.036	.010	-.059***	-.041	.010	-.067***
Maltreatment	-.055	.019	-.048**	-.047	.019	-.041*
CVE				-.086	.024	-.061***
Adjusted R^2		.013***			.016***	
F for model		17.939			16.812	
Physical Activity						
Gender	-.913	.066	-.219***	-.855	.068	-.205***
SES	.097	.025	.062***	.109	.025	.069***
Maltreatment	-.031	.047	-.011	-.050	.047	-.017
CVE				.212	.059	.059***
Adjusted R^2		.052***			.055***	
F for model		70.377			56.139	
Sedentary Behavior						
Gender	-.585	.063	-.149***	-.525	.064	-.134***
SES	-.188	.042	-.128***	-.176	.042	-.119***
Maltreatment	.020	.044	.007	.000	.044	.000
CVE				.221	.056	.065***
Adjusted R^2		.037***			.041***	
F for model		49.391			41.046	

Note. SE = standard error. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 11

Summary of Hierarchical Regression Analysis for Older Adolescents at Time 2 (N=7558)

Variable	B	Model 1 SE(B)	ß	B	Model 2 SE(B)	ß
General health						
Gender	-.062	.025	-.034*	-.077	.025	-.043**
SES	.029	.009	.043**	.026	.009	.038**
Maltreatment	-.051	.017	-.040**	-.046	.017	-.036**
General health T1	.520	.013	.534***	.517	.013	.532***
CVE				-.055	.022	-.035**
Adjusted R^2		.300***			.301***	
F for model		407.479			327.669	
Physical Problems						
Gender	.429	.069	.083***	.474	.072	.092***
SES	.059	.026	.030*	.069	.026	.035**
Maltreatment	.248	.049	.068***	.235	.049	.064***
Physical problems T1	.532	.013	.551***	.529	.013	.549***
CVE				.158	.062	.035*
Adjusted R^2		.334***			.335***	
F for model		476.722			383.227	
BMI						
Gender	-2.278	.881	-.039**	-2.055	.906	-.035*
SES	-1.000	.335	-.045**	-.952	.338	-.043**
Maltreatment	.876	.634	.021	.807	.638	.019
Parents' weight	8.420	.764	.168***	8.414	.764	.168***
BMI T1	.434	.015	.430***	.434	.015	.429***
CVE				.843	.806	.017
Adjusted R^2		.236***			.236***	
F for model		206.791			172.513	
Sleep						
Gender	-.073	.024	-.045**	-.090	.025	-.055***
SES	-.020	.009	-.033*	-.024	.009	-.039**
Maltreatment	.010	.017	.009	.016	.017	.014
Sleep T1	.401	.015	.402***	.399	.015	.399***
CVE				-.060	.022	-.043**
Adjusted R^2		.168***			.170***	
F for model		193.160			156.354	
Physical Activity						
Gender	-.486	.058	-.123***	-.452	.059	-.114***
SES	.033	.021	.022	.041	.022	.027
Maltreatment	.025	.040	.045**	. 113	.040	.041**
Physical activity T1	.411	.014	.433***	.409	.014	.431***
CVE				.129	.051	.038*
Adjusted R^2		.230***			.231***	
F for model		284.245			229.003	
Sedentary Behavior						
Gender	-.373	.053	-.100***	-.357	.054	-.096***
SES	-.083	.020	-.059***	-.080	.020	-.057***
Maltreatment	.069	.037	.026	.064	.037	.024
Sedentary behavior T1	.458	.014	.481***	.457	.014	.480***
CVE				.062	.047	.019
Adjusted R^2		.262***			.266***	
F for model		344.730			276.187	

Note. SE = standard error. T1 = Time 1. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 12

Effects of Control Variables in Multiple Mediation Models at Time 1

Covariate	Gender	SES	Childhood Maltreatment (PA)	Parental Weight
Dependent				
Early Adolescence				
General Health	-.091***	.056***	-.044*	
Physical Problems	.410***	.172***	.032(.214**)	
Adiposity (BMI)	-2.647**	-1.539***	-.797(.172)	5.180***
Sleep	-.087***	-.034***	.006(-.030)	
Physical Activity	-.744***	.139***	.084*	
Sedentary Behavior	-.553***	-.161***	.070(.1259*)	
Late Adolescence				
General Health	-.152***	-.047***	-.014(-.040)	
Physical Problems	.590***	.210***	.036(.255**)	
Adiposity (BMI)	-2.100*	-.201	.402(1.522)	5.636***
Sleep	-.119***	-.041***	-.013(-.040)	
Physical Activity	-.829***	.109***	-.030(-.066)	
Sedentary Behavior	-.533***	-.174***	-.012(-.063)	

Note. PA = physical abuse. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 13

Effects of Control Variables in Multiple Mediation Models at Time 2

Covariate	Gender	SES	Childhood Maltreatment (PA)	Health at T1	Parental Weight
Dependent					
Early Adolescence					
General Health	-.091***	.026**	-.007(-.022)		
Physical Problems	.465***	.109***	.100*	.467***	
Adiposity (BMI)	-2.332**	-1.281***	.737(1.344)	.373***	8.325***
Sleep	-.107***	-.017*	-.043**	.353***	
Physical Activity	-.380***	.070***	.065(.046)	.385***	
Sedentary Behavior	-.263***	-.109***	-.085*	.463***	
Late Adolescence					
General Health	-.056*	.025**	-.033(-.061*)	.502***	
Physical Problems	.444***	.077**	.204***	.497***	
Adiposity (BMI)	-1.764	-.893**	.714(.547)	.434***	8.414***
Sleep	-.078**	-.024**	.547(-.021)	.387***	
Physical Activity	-.449***	.037	.125**	.407***	
Sedentary Behavior	-.388***	-.078***	.050(-.008)	.455***	

Note. T1 = Time 1. PA = physical abuse. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 14

Summary of Multiple Mediation Analysis for Adolescents' Health

Variable	Young T1	Young T2	Old T1	Old T2
General health				
C	.003	-.044	-.103**	-.060*
c'	.074*	-.013	-.059	-.047
C – c'	-.071~	-.031~	-.044~	-.013~
Depression	-.038~	-.017~	-.032~	-.010~
Self-regulation	-.033~	-.014~	-.012~	-.002
R^2	.061***	.283***	.088***	.308***
Physical Problems				
C	.450***	.303***	.343***	.149*
c'	.025	.219**	.164	.123
C – c'	.425~	.084~	.179~	.026~
Depression	.183~	.019	.083~	.015~
Self-regulation	.242~	.065~	.096~	.011~
R^2	.180***	.315***	.190***	.342***
BMI				
C	3.545**	.481	2.033	-.706
c'	3.276**	.921	2.046	-.789
C – c'	.269	.160	-.014	.081
Depression	.267	.057	-.069	.079
Self-regulation	.002	.103	.055	.003
R^2	.025***	.196***	.018***	.240***
Sleep				
C	-.158***	-.062*	-.087**	-.074**
c'	-.065*	-.032	-.053	-.069**
C – c'	-.094~	-.030~	-.035~	-.005
Depression	-.039~	-.015~	-.010~	-.003
Self-regulation	-.054~	-.016~	-.025~	-.002
R^2	.104***	.171***	.069***	.172***
Physical Activity				
C	.167*	.036	.249***	.193**
c'	.249**	.114	.271***	.206***
C – c'	-.082~	-.078~	-.022~	-.013~
Depression	-.036~	-.040~	.001	-.011
Self-regulation	-.047~	-.039~	.021~	-.002
R^2	.057***	.204***	.062***	.236***
Sedentary Behavior				
C	.067	.013	.157*	.068
c'	.063	-.002	.145*	.052
C – c'	.004	.016	.013	.017~
Depression	-.013	.004	.005	.008
Self-regulation	.018	.012	.008	.009~
R^2	.039***	.251***	.044***	.266***

Note. C = total effect. c' = direct effect. C – c' = total indirect effect. T1 = Time 1. T2 = Time 2. ~ Indicates the confidence interval does not include zero. * $p < .05$.** $p < .01$. *** $p < .001$.

Table 15

Summary of Moderation Effects of Gender Between CVE and Adolescents' Health

Variable	Early Adolescence	Late Adolescence
Concurrent		
General Health	-0.073	-0.022
Physical Problems	0.000	-0.185
Adiposity (BMI)	0.135	0.591
Sleep	-0.002	0.063
Physical Activity	0.061	-0.084
Sedentary Behavior	0.134	0.183
Longitudinal		
General Health	-0.084	0.030
Physical Problems	-0.219	-0.025
Adiposity (BMI)	2.182	4.843**
Sleep	-0.025	0.053
Physical Activity	-0.148	-0.191
Sedentary Behavior	0.237*	-0.042

Note. CVE = community violence exposure. * $p < .05$.** $p < .01$.

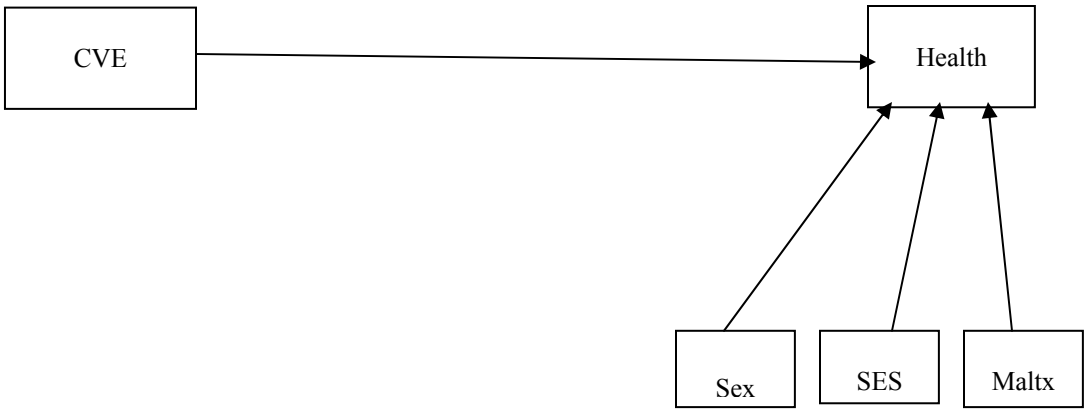

Figure 1. The above model is the path diagram for the effect of community violence exposure on adolescents' health. It illustrates the path from community violence exposure (CVE) to the six health-related while controlling for the gender, SES, and childhood maltreatment (SES = socioeconomic status; Maltx = childhood maltreatment; parent's weight status was also entered as a control variable for outcome, BMI).

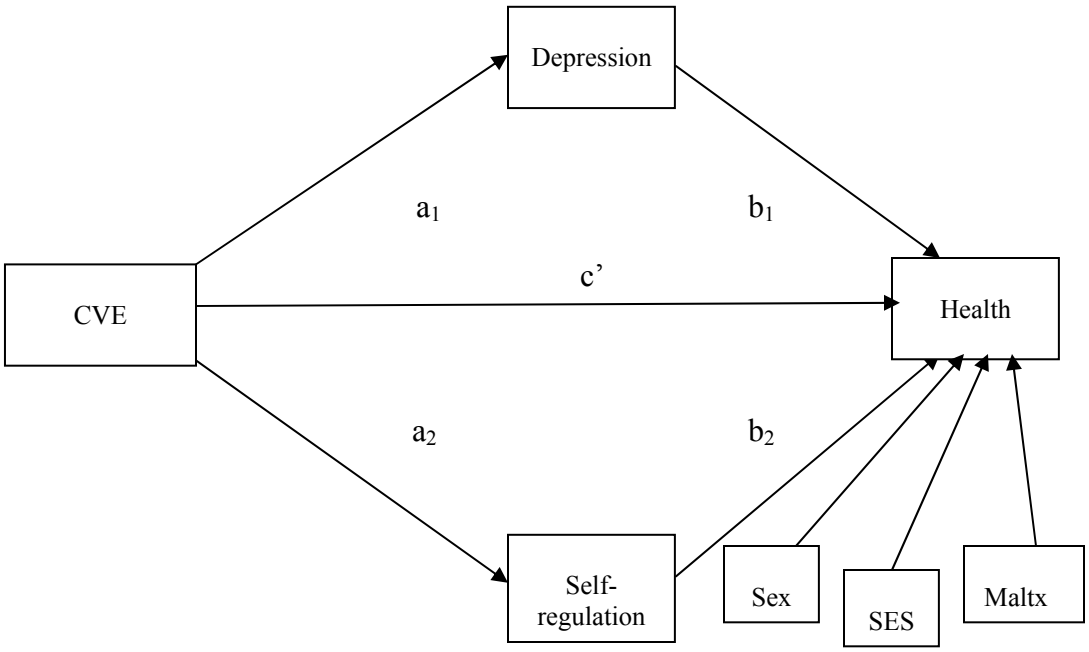

Figure 2. The above model is the path diagram for mediation. It illustrates the two mediating pathways from community violence exposure (CVE) to the six health-related outcomes (SES = socioeconomic status; Maltx = childhood maltreatment; parent's weight status was also entered as a control variable for outcome, BMI). The total effect of CVE on health can be determined by the equation, $c = c' + (a_1*b_1 + a_2*b_2)$. A complete model used in the analyses can be seen in Figure 3.

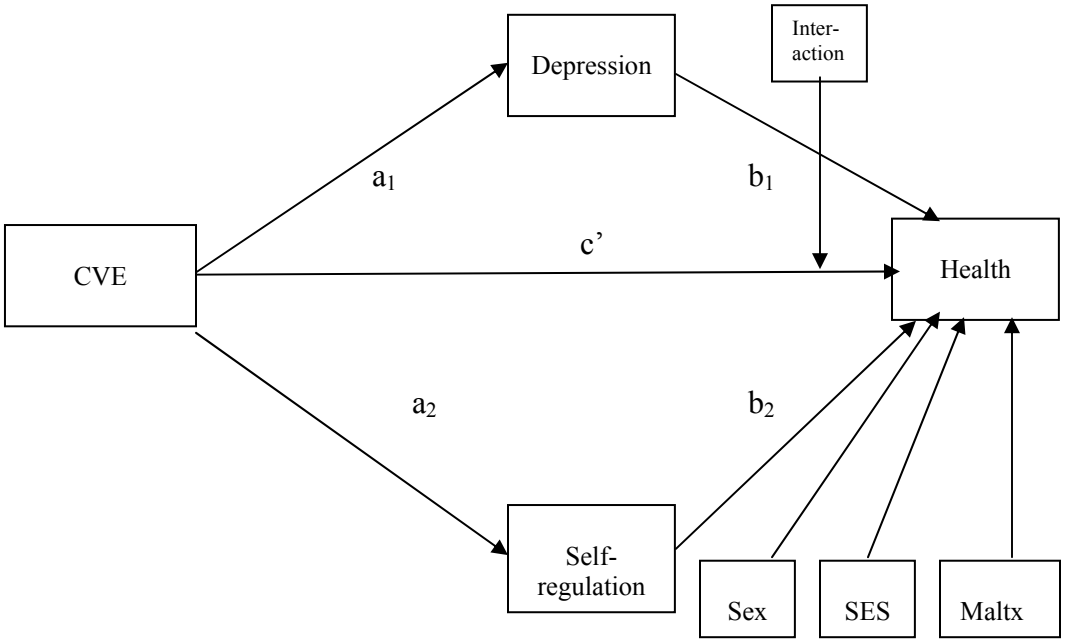

Figure 3. The above model is the path diagram for mediation including the interaction term and covariates. It illustrates the two mediating and one direct pathways from community violence exposure (CVE) to the six health-related outcomes with additional pathways from the interaction term (gender) and the three covariates entered as control variables (SES = socioeconomic status; Maltx = childhood maltreatment; parent's weight status was also entered as a control variable for outcome, BMI). The health variable can be determined by the following equations, $y = b_1*M_1 + b_2*M_2 + c'*x + 3$ covariates + (Gender*CVE), where $M_1 = a_1*x$ and $M_2 = a_2*x$.

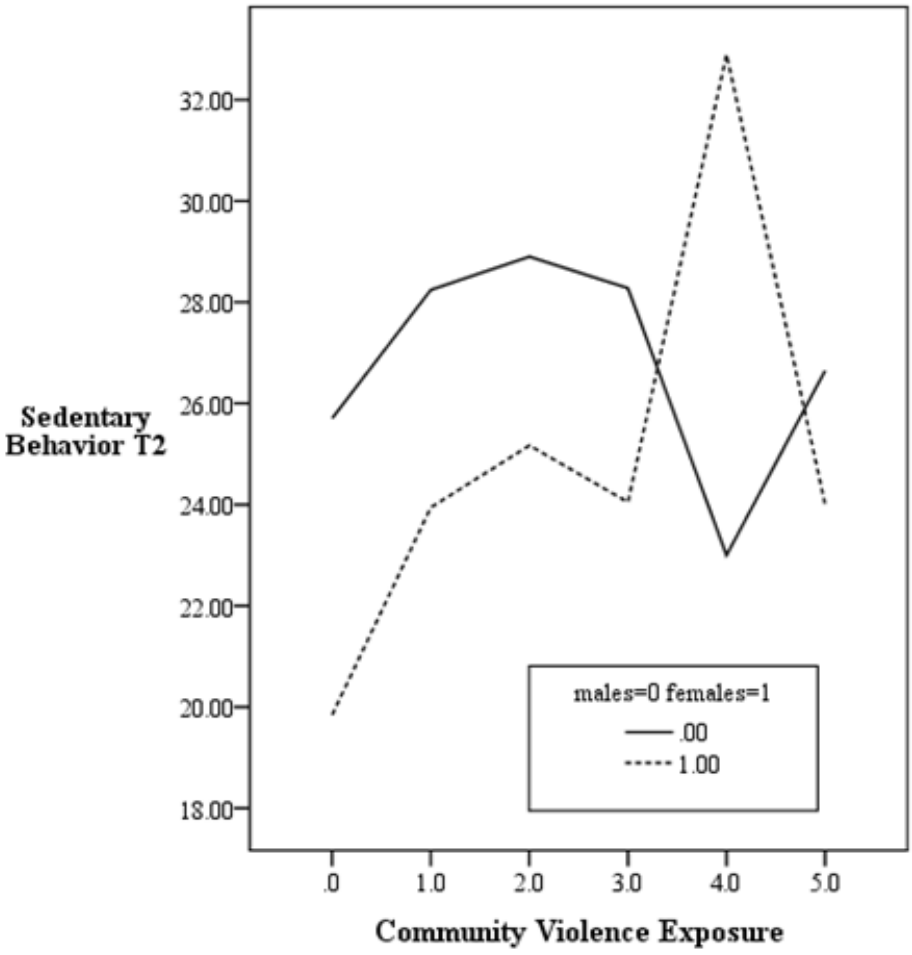

Figure 4. The effects of community violence exposure (CVE) on younger adolescents' sedentary behavior at time point 2 differed by gender. Females' scores, depicted by the green line, increased with more exposure. A slight decrease in sedentary behavior was found for males with more exposure, indicated by the blue line.

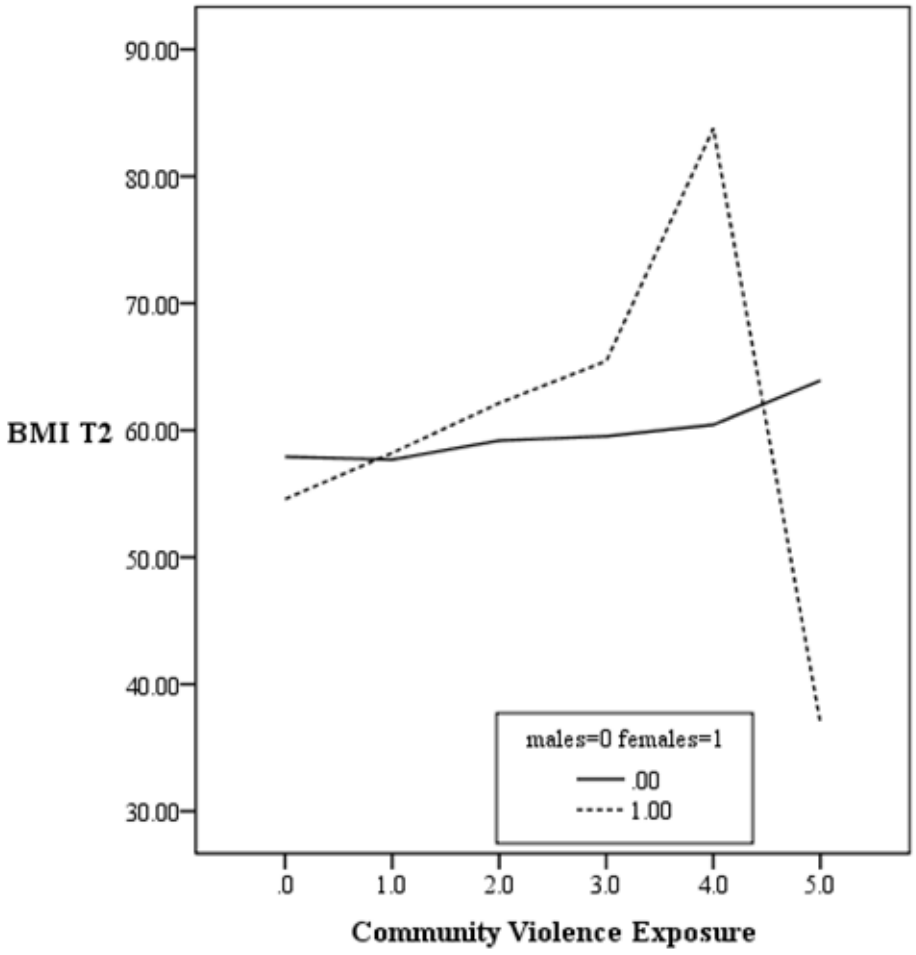

Figure 5. The effects of community violence exposure (CVE) on older adolescents' BMI at time point 2 differed by gender. Females' scores, depicted by the green line, increased with more exposure, except with extreme levels of CVE. A slight increase in males' BMI was demonstrated with more exposure, indicated by the blue line.