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Interpersonal Victimization as a Risk Factor for ADHD - A Critical Review

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Abstract

In mental health care today, the theory that interpersonal victimization may cause Attention Deficit Hyperactivity Disorder (ADHD) has become progressively influential and is increasingly impacting clinical practice in the field. This is partially the result of a number of studies that have found an association between interpersonal victimization, such as child maltreatment or bullying, and ADHD. However, establishing causality in life-course research is a challenge. Human development is a dynamic and complex process and consequently, abundant with possible confounding factors.

This study attempts to address the research question: Is interpersonal victimization a risk factor for ADHD?

The process began with a systematic search for relevant studies and papers in PubMed and PsychINFO. Longitudinal studies that performed pre- and post-tests of both predictor and outcome variables were included. The initial search results contained hundreds of studies, but only six studies met the criteria and were reviewed. Two studies examined polyvictimization and four studies examined peer victimization.

In reviewing those empirical studies that examined interpersonal victimization as a risk factor for ADHD, results suggest that interpersonal victimization may be a small risk for increased ADHD symptoms. However, this effect seems to be temporary. Moreover, there appears to be substantial confounding affecting the association between victimization and ADHD. When controlling for genetics the association between victimization and ADHD disappears or is substantially reduced. The lack of studies with a minimal control for confounding suggests a need for further research in this area and clinical caution.

Key words: Attention Deficit Hyperactivity Disorder, Attention Deficit Disorder, ADHD, Victimization, Child Abuse, Child Maltreatment, Peer Victimization, Bullying, Polyvictimization, Trauma

Preface

This has been quite the journey.

I would like to thank my advisor, Jennifer Drummond Johansen, for her support and guidance on a long and winding road. I would also like to thank Dawit Abebe for answering my questions on statistics.

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This paper is dedicated to K, B and F; the next generation of neurodivergent rebels.

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1.0 INTRODUCTION

Interpersonal victimization, in its myriad of forms, including child maltreatment, bullying, rape, domestic violence and crime victimization, has been suggested to be causally related to a vast range of both physical and mental health problems. Over the years, a number of studies have reported an association between various forms of victimization and an assortment of mental health diagnoses (Kessler et al., 2010a; Greenfield, 2010; Moore et al., 2017). Nevertheless, when studies attempt to eliminate alternative explanations for these associations, they often reach conflicting results (Schaefer, et al, 2018).

One hypothesis that has gained considerable traction in recent years pertains to the possible causal relationship between victimization and Attention Deficit Hyperactivity Disorder (ADHD). A frequent source of controversy and debate, ADHD affects roughly 5-7% of children and 3-5% of adults (Polanczyk et al., 2015; Thomas et al., 2015; Fayyad et al., 2017). The diagnosis entails patterns of atypical behavior including inattention and/or hyperactivity and impulsivity. Like victimization, it is associated with a multitude of mental health and social issues. Several studies have shown an association between interpersonal victimization and ADHD. Nonetheless, such associations may exist for many reasons.

Establishing causality in life-course research is a challenge. Human development is a dynamic and complex process and consequently, abundant with possible confounding factors. Demonstrating causal relations requires dealing with multiple potential threats to internal validity (Miller et al., 2016; Duncan et al., 2004). When it comes to demonstrating causal connections “few problems in the psychological sciences have been as simultaneously important and intractable as establishing a causal relationship between victimization exposure and psychopathology” (Schaefer et al., 2018, p. 352). Due to obvious ethical reasons, the range of research designs available to study causal effects of victimization are limited. As such, when it pertains to humans; we need to rely on observational studies. These studies need to disentangle causal effects from the countless factors known to be associated with victimization. And while experimental studies using non-humans, such as rodents, are permissible, they are complicated by their own problems of external validity (Schaefer et al., 2018). To what extent can we generalize from rodents to humans?

Rather than confront these challenges, Foster (2010) believes many researchers sidestep them by either giving up causal inference all together or by making causal claims based on unstated assumptions. Although challenging, establishing causal relationships in human development is fundamental to the

field's implicit goal of improving people's lives (Foster, 2010). It is crucial as a basis for implementing policies for promoting mental health and preventing problems.

1.1 Research Question

This paper proposes the following research question:

- *Is interpersonal victimization a risk factor ADHD?*

The term "risk factor" is often used loosely (Bhopal, 2002, pp.163-164). In what is now considered a classic article, Kraemer et al. (1997) defined *risk factor* as:

> a measurable characterization of each subject in a specified population that precedes the outcome of interest and which can be used to divide the population into 2 groups (high risk and low risk). The probability of the outcome in the high-risk group of subjects must be shown to be greater than the probability of the outcome in the low-risk group. (p. 338)

Kraemer et al. proposes specific steps to document and establish a variable as a risk factor. First, the factor must be associated with the outcome. If the factor is correlated with the outcome, the second step is to establish that it precedes the outcome. If this is the case, then the factor may be termed a *risk factor;* if not, it is a *concomitant* or *consequence*. Successive steps are needed to establish a factor as a *causal* risk factor. A risk factor is, in other words, not *necessarily* a causal factor (Bhopal, 2002, p. 164), but analyzing associations and establishing whether something is a risk factor may be a step on the way to uncovering a possible causal relationships (Kraemer et al. 1997, Bhopal, 2002, p.164). Establishing risk factors can indicate which variables we need to account for when teasing apart complex, potential, causal mechanisms, thereby facilitating a deeper understanding of such processes.

1.2 Aim of the Study

This study aims to critically review the empirical research regarding interpersonal victimization as a potential risk factor for ADHD. The belief that victimization may cause similar or identical symptoms to ADHD is gaining considerable traction and is increasingly influencing clinical practice (Nordanger, 2015; Løwgren & Evensen, 2019). To the best of my knowledge, there have not been any systematic reviews that go beyond cross-sectional, association studies regarding this question. Hence the timeliness of this study.

The ultimate purpose of research in the behavioral sciences is to bring about knowledge that benefits humanity. While many people displaying or diagnosed with ADHD traits are highly productive and

fulfilled individuals, a great number suffer. Increased understanding regarding the relationship between victimization and ADHD may increase our ability to provide the right interventions, support and/or services at the appropriate intervals and levels, thereby improving this group's quality of life. Furthermore, the author hopes that this review may give some individuals additional or new insight into their own experiences of victimization and ADHD.

2.0 KEY CONCEPTS AND THEORIES

2.1 Dynamic, Interactional Models of Development

According to Sameroff (2010), theories of development started out with the hope of conforming to the principles of Ockham's razor, with simple linear models determining developmental outcomes. However, as the field developed, it became increasingly clear that beyond short-term predictions, such models were failing to provide meaningful results. Instead, development involved the interplay of complex, multilevel systems, processes and relationships involving "child and parents (…) neurons and neighborhoods, synapses and schools, proteins and peers, and genes and governments" (Sameroff, 2010, p.7).

Lewis (2014), argues that theories of development are generally dominated by two different views of human nature. One perspective sees individuals as the result of mechanistic biological, physical and/or social forces that act upon them. These forces cause the individual to develop and behave a certain way. The other perspective sees individuals as interacting with biological, physical and social environments. The individual both influences and is influenced by such factors in a dynamic process (Lewis, 2014, pp. 3-6). Lerner (1998), similarly, describes contemporary frameworks as models where the individual's development is "embedded within an integrated matrix of variables derived from multiple levels of organization, and development is conceptualized as derived from the dynamic relations among variables in this multi-tiered matrix" (Lerner, 1998, p.1). It is such a complex, dynamic, interactive and relational model of development that is the foundation of the analysis in this paper.

2.2 Causation, Confounding and Reverse Causation

According to Rothman & Greenland (2005) the cause of a specific event can be defined as "an antecedent event, condition or characteristic that was necessary for the occurrence (…) at the moment

it occurred given that other conditions are fixed" (p. 144). Based on this definition it is unlikely that an individual event, condition or characteristic is sufficient cause by itself, therefore this a definition of component causes and not a complete causal mechanism. A complete causal mechanism - or «sufficient cause» - is a set of components that together inevitably lead to the outcome of interest.

A particular outcome may have a range of possible causal mechanisms involving a multitude of causal components, a phenomenon known as multicausality (Rothman & Greenland, 2005, 144-145). Multicausality indicates that many causal mechanisms are sufficient but not necessary—as there are many possible causal mechanisms —and that many causal factors are not necessary components of every sufficient causal mechanism. These causal components are known as *inus conditions*: they are insufficient but necessary in certain sufficient (but not necessary) causal mechanisms and therefore contribute to some cases, but not all cases (Mackie, 1974, in Shadish et al., 2002).

Reciprocal causality may occur when there are bidirectional influences between various phenomena, either in sequence or simultaneously (Zelazo, 2013). This idea is fundamental to the interactional or transactional model of development. For instance, parents and children may influence *each other* in a dynamic relationship (Sameroff, 2020).

Sometimes a "primary" factor leads to an increased risk for a "secondary" factor, that then increases the risk for certain outcomes. In these cases, the secondary factor acts as a *mediator* between the primary factor and the outcome variable (Pearlin and Bierman, 2013, pp. 330). In addition, there may be *moderating* factors, factors that can strengthen or weaken a relationship between two variables (Mirowsky & Ross, 2012, p. 144). For example, social support may moderate the potential negative effects of experiencing a stressful event.

Knowledge about causal mechanisms help us develop effective prevention programs but we need to be cautious about inferring causal effects. Mistaken causal assumptions may have detrimental consequences of a more serious nature than simply «theoretical fallacy». Theories often have practical implications. Simplistic or hasty causal assumptions based on associations can easily lead to mistreatment or worse: transgressions or abuse of power causing serious harm. History has a vast variety of examples. Just a few decades ago, parents of autistic children were treated with "sensitivity classes" because it was believed that autism was caused by "cold mothers". Unfortunately, it is very difficult to undo a belief once it has been established (Watt & Collins, 2019).

2.2.1 Confounding

There are a multitude of pitfalls to making causal inferences based on associations. One potential threat to causal inference is *confounding*. If we compare two groups that differ on a known attribute or predictor variable and discover that one group has a higher occurrence of a certain outcome of interest than the other, it may be easy to assume that it is the known predictor variable that is the cause of the difference in outcome. However, there may be systematic differences between the two groups that are unaccounted for. If these differences are associated with the predictor and outcome variable being studied, they may create a spurious association between the two. This is known as confounding (Shadish et al., 2002, p. 7).

In a perfect experiment, a causal effect of a variable might be inferred if one could compare two groups that were identical in every possible way except for the presence/absence of a single specific variable (Reichardt, 2019). This is what is emulated in experiments through random assignment to treatment and control groups—one hopes to create groups without systematic differences between them. Unfortunately, such perfectly matched groups are almost impossible to achieve in real life as there are a multitude of potential causes of systematic bias (Higgins et al., 2020). Additionally, practical and ethical considerations often limit the possibility of using "true experiments" with random allocation in the social sciences. This tends to leave observational studies as the most viable option. Observational studies regarding causality are especially vulnerable to confounding because the groups being compared are not randomly assigned. Rather, they are naturally formed. All the possible influences on this formation, such as socio-economic factors, level of education, attitudes and so on, may be possible confounding factors when comparing the two groups on some outcome of interest.

In the social sciences, there seems to be an almost unlimited number of variables with possible effects and interactions making it difficult to control for all the variables that might possibly influence the outcome. This is why it is essential to perform pre-test measurements of outcome variables in the groups we are studying so we can identify how groups differ at the outset (Shadish et al., 2002, p.136).

An additional risk of making causal inferences based on associations regards the problem of causal direction. Even if the association of two variables is in fact the result of a causal relationship, there is no way of knowing the direction of this relationship (Shadish et al., 2002, p. 7). In studies showing an association between the variables *ADHD* and *victimization*, it is difficult to determine if *victimization* is causing *ADHD, ADHD* is causing *victimization*, or if there is a reciprocal causal relationship.

Performing pre-test and post-test measurements on both predictive and outcome variables may help with this problem (Shadish et al., 2002, pp. 136-161).

Familial Confounding. Research in developmental psychology in the past few decades has led to increased support for the phenomenon referred to as *gene-environment correlations,* which may lead to familial confounding (Kendler & Baker, 2007; Beauchaine, Gatzke-Kopp & Gizer, 2017; Rutter et al., 2006). Gene-environment correlations, often written as *rGE*, occur because our genes influence our exposure to particular environments (Jaffee & Price, 2007). This may happen through what is known as passive, active and evocative correlations (Plomin et al., 1977). So-called *passive* gene-environment correlations transpire when a parent's genes, which are inherited and therefore shared by the child, influence both parenting behavior and the child's behavior (Jaffee and Strait, 2012). In other words, when a child grows up in a family with parents that exhibit high levels of aggression and the child later exhibits high levels of aggression him/her-self, this might be the result of being in an aggressive environment or it might be because of passive gene-environment correlations where the genes that increased the parent's risk of aggressive behavior were inherited by the child. Furthermore, it might be both.

The second way our genes might influence the environments we are exposed to, is through *active gene-environment correlation*. This occurs when our genes influence our choice of environments. For example, the genes that give rise to lower-than-average mesolimbic dopamine activity, which is thought to influence impulsivity, seems to predispose towards sensation-seeking and high-risk activity. In certain environments, this might lead to substance abuse and associating with delinquent peers (Beauchaine and Neuhaus, 2008), whereas in other environments it might lead to an exciting, high-risk profession, signing up with the resistance army or Doctors Without Borders. In this way, our genes increase our chances of certain types of experiences, but there is always an *interaction* between our genes and our environment in shaping our phenotype.

In addition, a trait may interact with another trait. Impulsivity in combination with anxiety might produce different behavioral tendencies than impulsivity in combination with general fearlessness (Beauchaine and Neuhaus, 2008). Because each individual's genetic make-up is so unique, it is difficult to make simplistic predictions based on any one particular factor.

Lastly, *evocative gene-environment correlations* refers to the way our genetically influenced behavior evokes certain types of reactions in our surroundings. A child with a higher-than-average propensity towards impulsivity or activity might have a harder time following rules and social norms. In cultures

or environments that cherish restraint and order such a child might elicit negative reactions from his or her surroundings.

As we have seen, these processes of gene-environment correlation might lead to *familial confounding* if they are not controlled for in studies. Studies may attempt to control for familial confounding through twin or adoption studies. Twin studies often compare same-sex dizygotic twins with monozygotic (MZ) twins in order to probe for genetic or environmental influences. However, it is important to remember that although MZ twins share the same genes, there may already be differences between them at birth that are caused by different environmental circumstances. For instance, unequal placental sharing frequently occurs with twins. This may lead one child to be small at birth, which may be a risk factor for various health and behavioral problems including ADHD (Fick et al., 2006; Tyson et al., 2008). In addition, monozygotic twins may receive more similar treatment than dizygotic twins.

2.3 Interpersonal Victimization

In this paper, the term *interpersonal victimization* is defined as an occurrence whereby a person is subjected to actions by an individual or group of individuals that break societal norms or are an abuse of power and are likely to cause suffering, physical or emotional harm or substantial impairment of fundamental rights.

This definition is inspired by the UN's Declaration of Basic Principles of Justice for Victims of Crime and Abuse of Power (United Nations, 1985) and Kirchhoff (2010, pp. 112-113). The term includes, but is not limited to child maltreatment (which encompasses both physical and emotional child abuse and neglect), sexual abuse or assault, bullying, intimate partner violence or domestic abuse, violence and criminal victimization.

2.3.1 Risk Factors and Associated Factors

Victimology is the field of research concerned with the process and phenomenon of victimization, its risk factors, context, patterns and consequences. It draws from a vast range of academic disciplines such as sociology, criminology, legal studies and psychology (Fattah, 2010; Kirchhoff, 2010; Zaykowski & Campagna, 2014). Research in the field of victimology has found that although victimization may at times be a random, unpredictable incident, most victimization follows patterns. There are individual and group differences in rates and type of victimization (Fattah, 2010). Whereas some people are never, or rarely, the victims of crimes or violence, others repeatedly experience various forms of victimization—a concept known as polyvictimization (Finkelhor et al., 2009). In

order to understand and explain these individual differences and the dynamic process of victimization, victimology studies individual characteristics, family structure and social relationships, the relationship between victim and perpetrator, socio-demographic factors, environment, history, social roles, routines and lifestyles of victims in addition to cultural and societal factors such as gender ideals or ethnic relations (Walsh & Hemmens, 2019; Fattah, 2010).

The study of victimology has led to the realization that there is a substantial overlap between victims and offenders. Victims and offenders often share the same social and geographical space, so tend to come from the same populations (Fattah, 2010, p. 52). In addition, perpetrators tend to be victims and vice versa (Vaske et al., 2012; Sampson & Lauritsen, 1994). It is possible that victimization and perpetration may have some common causes (Vaske et al., 2012).

While research into risk factors for victimization began with studies looking for singular, direct causal agents, the field has gradually moved towards models of multiple interacting factors (MacKenzie, Kotch & Lee, 2011; Grubb & Posick, 2018). As in interactional, systemic models of development, risk factors may operate on many levels leading to a multilevel perspective (Sampson & Lauritsen, 1994). In other words, though each individual risk factor may not in itself be a sufficient cause, the combined interaction of risk and protective factors drives the etiology of victimization.

While certain factors are implicated in particular forms of interpersonal victimization, others are risk factors for multiple forms of victimization. Alcohol and substance abuse seems to be a risk factor for most forms of victimization. About half of all violent crimes worldwide involve alcohol (Hoaken & Stewart, 2003). Substance use is widely implicated in child maltreatment, homicide, sexual assault and intimate partner violence (Connell-Carrick, 2003; Yang & Maguire-Jack, 2018; Lundholm et al., 2013; Lorenz & Ullman, 2016; Krug et al., 2002; Spencer, et al., 2019; Assink et al., 2019). These associations have been found regarding both the perpetrator and the victim. (Gutwinski, Heinz & Heinz, 2018, pp. 456-457).

Low socioeconomic status (SES) also seems to be a general risk factor for victimization. It is associated with child abuse and neglect (Euser et al., 2013; Sedlak, 2010; Slacket al., 2004; Stith et al., 2009; Connell-Carrick, 2003; Hindley, Ramchandani and Jones, 2006; Liel et al., 2011; Yang et al. 2018; Hunter & Flores, 2021), sexual abuse and assault (Finkelhor et al., 2005; Assink et al., 2019), intimate partner violence (Capaldi et al., 2012, Field & Caetano, 2004; World Health Organization & Pan American Health Organization, 2012; Costa et al., 2015), and crime victimization (Nilsson & Estrada, 2006; Berzofsky et al., 2014). Low SES is also generally considered to be a risk factor for, or

associated with, other risk factors and social issues such as living in an unsafe neighborhood, substance abuse, parental and family stress and mental health issues. For instance, neighborhood characteristics are linked to SES (Mirowsky & Ross, 2012, p.149). Boney-McCoy and Finkelhor (1995) found that living in a dangerous neighborhood was associated with higher levels of sexual victimization in children.

Disability seems to be an additional general risk factor for victimization. In their systematic review Jones et al. (2012) found that children with disabilities are more likely to be victims of violence than non-disabled children. A population-based study in the US found that disabled children were 3.4 times as likely to be maltreated as non-disabled children (Sullivan & Knutson, 2007). In addition, studies find that children with disabilities are subjected to more bullying than average (Hong & Espelage, 2012; Maïano et al., 2016; Turner et al., 2011). Adults with disabilities also seem to suffer higher than average levels of violence and victimization (Sin et al., 2009; Smith et al., 2011; Dean et al., 2018).

Lack of social support, large family size, young children, parenting stress, age of parents, intimate partner violence, cramped housing, drug/alcohol abuse, migration history and parental psychopathology are all risk factors for child maltreatment (Euser et al., 2013; Sedlak, 2010; Slack et al., 2004; Stith et al., 2009; Walsh et al., 2003; Connell-Carrick, 2003; Hindley et al., 2006; Liel et al., 2011). Family structure is generally associated with many forms of victimization, including child abuse, sexual abuse and neglect. Not living with both biological parents or living with a step-parent increases risk of victimization in children and youth (Lauritsen, 2003; Turner et al., 2006). In single parent households this seems to be tied to a lack of economic resources and time, while in step-families it might be a direct consequence of the presence of a step-parent in the household, as step-parents might have a higher risk of perpetrating child abuse, especially sexual abuse (Finkelhor & Baron, 1986; Sedlak et al., 2010). Additionally, step-families in general have higher rates of parental unemployment and imprisonment, drug/alcohol problems, homelessness and chronic parental arguing (Turner et al., 2007).

A lack of social support or positive social networks may also be risk factors for interpersonal victimization. Experiencing peer rejection or lack of beneficial friendships have been found to be risk factors for peer victimization (Hong & Espelage, 2012; Herráiz & Gutiérrez, 2016; Klomek, 2020), dating violence (Park & Kim, 2018; Hébert et al., 2019), intimate partner violence (Capaldi et al., 2012; Levendosky et al., 2004), sexual assault (Hawn et al., 2018; Conley et al., 2017) and violence victimization (Schreck & Fisher, 2004). Being part of a constructive social group provides protection.

Several studies also suggest that genes may be a contributing risk factor for such examples of victimization as child maltreatment, peer victimization, adolescent victimization, intimate partner violence and assault (Sartor et al., 2012; South et al., 2015; Bowes et al., 2013; Fisher et al., 2015; Hines & Saudino, 2004; Stein et al., 2002; Afifi et al., 2010). Furthermore, mental health problems have been found to be a risk factor for victimization (Cuevas et al., 2009; Turner et al., 2010; Maniglio, 2009; Bhavsar et al., 2019; Latalova et al., 2014; Kljakovic & Hunt, 2016; Khalifeh et al., 2015). In addition, belonging to various marginalized social groups, such as a sexual minority, may increase the risk of victimization (McGeough & Sterzing, 2018; Button et al., 2012). Many of the risk factors for victimization may be understood in light of the broader concept of *social marginalization,* which can be defined as the position of individuals or groups at the margins of social, political, economic and ecological systems, thereby limiting their access to resources and power (von Braun & Gatzweiler, 2014, p. 3).

These risk factors influence and interact with each other in dynamic and complicated processes. The various attributes of an individual are always embedded in specific contexts which will influence how they play out. Poverty and lack of social support may influence parental stress and mental health, which may influence drug or alcohol consumption, which again may influence employment status. In this way, cumulative risk factors may add up, increasing the total risk. However, it is often difficult to predict how such factors may interact. For instance, Freisthler, Holmes and Wolf (2014) found that parental social network and substance abuse could interact in such a way that large social circles increased the risk of child abuse. This is in contrast to the general finding that access to social networks lower this risk.

2.3.2 Possible Outcomes of Victimization

Most of us have experienced some form of victimization (Darves-Bornoz et al. 2008; Kilpatrick et al., 2013; de Vries & Olff, 2009; Stoltenborgh et al., 2015). Victimization may lead to suffering—physical or emotional pain that may be short or long term. It may change a person's sense of self and sense of safety. Victimization encompasses a vast array of different experiences that vary in both duration, intensity, timing and quality, in addition to individual differences in victims and differing contexts. The manner in which something is experienced by an individual or group is dependent on a multitude of factors. On the whole, the most frequent outcome of victimization seems to be resilience (Bonanno et al., 2011). Nonetheless, victimization is associated with a multitude of negative health outcomes (Kessler et al., 2010a; Greenfield, 2010; Moore et al., 2017). Studies find that victimization is a

possible risk factor for various mental health problems, in particular anxiety and mood disorders, psychosis, suicidality, aggression, PTSD and substance abuse disorders (Moffit et al., 2013; Moore et al., 2017; Macalli et al., 2021; Turner et al., 2006; Lagdon et al., 2014). Cumulative exposure to multiple forms of victimization seems to increase this risk (Turner et al., 2006; Hughes et al., 2017). Nonetheless, it is unclear if there is a direct causal effect, or if the relationship is due to confounding or is mediated through other pathways (South et al., 2015).

Many theories regarding the detrimental effects of victimization on mental health involve possible consequences of the physiological reactions to stress. Stress, however, has no agreed-upon definition. It may be viewed as situations that might be threatening or harmful, that require substantial adaptation, as demands that exceed our resources or as the obstruction of basic needs or goals (Cohen et al., 2019). Such real or potential threats are processed and evaluated by what we often call the stress system, a network of highly complex systems and processes that comprise various brain structures.

Different types of stress involve different networks in the brain (Godoy et al., 2018). For instance, physical stress, such as pain, is predominantly processed in the brain stem and hypothalamic regions. These responses are more or less autonomic. In the first, immediate and short-lasting phase, the sympathetic adrenomedullar system (SAM) gives a rapid physiological response resulting in heightened alertness. This phase is followed by a more long-lasting hormonal process involving the Hypothalamic Pituitary adrenal axis (HPA) and the release of cortisol. Psychological stressors, on the other hand, involve responses that are both physical and cognitive. Elements of the limbic system, such as the prefrontal cortex, hippocampus, amygdala, hypothalamus and nucleus accumbens, have a particularly central role in the appraisal and regulation of the stress response (Ulrich-Lai & Herman, 2009; Godoy et al., 2018). There does not seem to be one stress response. Studies show large inter- and intra-individual responses to stress (Orem et al., 2019; Godoy et al., 2018). In addition, stress reactions, differ based on the timing and duration of exposure (Godoy et al., 2018).

It is obviously difficult to study what happens in the body and mind of individuals during episodes of victimization and so, broadly speaking, we must generalize and speculate based on laboratory studies of a different nature. Several experimental studies on humans find that stress can have a negative effect on attention and executive function, though some studies show improvement under stress and results often vary depending on gender (Shields et al., 2019). Yet, most studies focus on immediate effects in a laboratory. Whether these effects are long-lasting or not is another matter.

A recent meta-analytic review of associations with allostatic load, which reflects the activation of bodily regulatory functions in response to stress, such as the HPA axis, levels of cortisol etc., and cognitive functions found a very small, but significant, association between allostatic load and global cognitive function and executive function (r = 0.08 and r = 0.07 respectively) (D'Amico et al., 2020). Conversely, a longitudinal, twin study of more than 3000 children from two different countries, found no causal link between victimization in childhood and later cognition when controlling for familial confounding (Danese et al., 2017).

Studies of childhood maltreatment and brain structures have often found associations with differences in parts of the brain such as the hippocampus, amygdala, cerebral cortex and corpus callosum (Teicher & Samson, 2016; Hart & Rubia, 2012). However, a systematic review found that most studies up until 2012 were association studies, consisting of small clinical samples, making it difficult to generalize or say anything about cause (Hart & Rubia, 2012).

Better executive function seems to mitigate the effects of stress (Shields et al., 2017; Dileo et al., 2016). Studies on effects of PTSD on cognitive functioning find most deficits were present before trauma and probably make people vulnerable to developing PTSD (Scheeringa, 2021; Bomyea, et al., 2012; Kremen et al., 2012; Gilbertsen et al., 2006; Lebois et al., 2016). Several studies have found ADHD to be a risk factor for developing PTSD (Howlett et al. 2018; Biederman et al., 2014; Lee et al., 2012; Adams et al., 2020). In their systematic review of neurocognitive functioning in PTSD patients, Scott et al. (2015) suggest the possibility that some of the cognitive deficits that have been assumed to be consequences of PTSD might actually be the result of comorbid ADHD.

Animal studies of early life stress show some effects on, what we interpret as, anxious and depressed behavior in rodents (Murthy & Gould, 2018; Derks et al., 2017). Though most studies are time limited and do not give information on the duration of the effects, some studies seem to indicate that the effects are temporary (McEwen & Morrison, 2013) In general, results are somewhat mixed and often vary between different forms of stress, different strains of rodents or different species (rats vs mice etc.) or lack consistency between brain changes and behavioral changes, and finally, tend to suffer from very small samples (Murthy & Gould, 2018; Derks et al., 2017; McEwen & Morrison, 2013). All of the above makes it hard to generalize from findings. As a result, it is difficult to know if we can compare the stressful circumstances of these experiments to the phenomenon of child abuse. Furthermore, most studies show that the majority of rodents are not severely affected by early life stress, so in many ways these studies show how *resilient* rodents are (Murthy & Gould, 2018; McEwan & Morrison, 2013), which might give us cause for thought.

2.4 Attention Deficit Hyperactivity Disorder

2.4.1 General Description

Attention Deficit Hyperactivity Disorder is a classification of a group of persistent atypical behaviors (Kileen, 2019). The cluster of symptoms may be related to inattention and/or impulsivity and hyperactivity. Previously thought to be a childhood disorder, it has been recognized as a possible lifetime condition since the late 90's or early 2000's (Barkley, 2015, p.19) In the International Classification of Diseases (ICD) by the World Health Organization (WHO) and the Diagnostic and Statistical Manual (DSM 5), which is the system used in the US, it is classified under the chapter *Neurodevelopmental Disorders* (Gaebel et al., 2020).

According to the ICD 11, ADHD is "characterized by a persistent pattern (at least 6 months) of inattention and/or hyperactivity-impulsivity that has a direct negative impact on academic, occupational, or social functioning" (icd.who.int). Symptoms must be present prior to the age of 12 and occur across multiple settings, but may vary according to demands and structure of the setting, and must be outside the limits of normal functioning. The ICD-11 describes *inattention* as "significant difficulty in sustaining attention to tasks that do not provide a high level of stimulation or frequent rewards, distractibility and problems with organization". *Hyperactivity* is described as "excessive motor activity and difficulties with remaining still, most evident in structured situations that require behavioral self-control", and *impulsivity* refers to "a tendency to act in response to immediate stimuli, without deliberation or consideration of the risks and consequences". Lastly, symptoms cannot be better explained by other mental, behavioral, neurodevelopmental disorders or effects of substances or medication (Ibid.). The DSM 5 (2013) has similar criteria to the ICD-11.

The symptoms of ADHD seem to be the outer margins of continuous traits (Larsson et al., 2012) and is therefore considered a spectrum disorder. The diagnosis is distinguished from "normal variation" by the degree the aforementioned symptoms interfere with daily functioning across multiple domains. In this regard, ADHD has aspects of both a mental disorder and a disability (Graby, 2015).

Like all mental health conditions, there is some variation in prevalence estimates but generally global estimates fall between about 5-7 % for children (Polanczyk et al., 2014; Thomas et al., 2015, Alhraiwil et al., 2015) and 1.2 - 7.3 % for adults (Fayyad et al., 2007, Fayyad et al., 2017). ADHD seems to be about twice as common in males as in females (roughly about 8% vs. 4%, respectively, in children) but rates of diagnosis exaggerate these differences, which is likely to be partially due to

symptom criteria being based on how the condition typically presents in boys (Derks et al., 2020; Quinn & Madhoo, 2014; Rucklidge, 2010).

ADHD symptom levels are generally quite temporally stable, although they tend to decline over time with the inattentive aspects appearing to be more lasting than the hyperactive/impulsive symptoms (Kessler et al., 2010b). Eilertsen et al. (2019) found a correlation of 0.65 between ADHD symptom levels at age 3 and age 5, while the correlation was 0.44 between age 1,5 and age 5. Law et al. (2014) followed 88 children diagnosed with ADHD between the ages of 3 and 6 over a period of 4-10 years. They found that 70% still met criteria for ADHD at follow-up. High levels of child internalizing and externalizing symptoms in addition to parental psychopathology and low family SES predicted ADHD diagnosis at follow-up. Interestingly, they found that nearly 60% of those who no longer met the criteria for ADHD, met criteria for a different diagnosis, mainly autism.

It is challenging to measure the stability of ADHD because symptoms change as people age and diagnostic criteria have not been sufficiently adapted to adults (Faraone et al., 2010). In addition, several studies indicate that adults with ADHD significantly under report symptoms (Barkley et al., 2002; Sibley et al., 2010). A 10-year follow-up study of boys with ADHD found that 65% no longer met the full criteria after 10 years. However, 78% showed some level of persistence when including those who were currently medicated for ADHD, those with moderately to severely impaired global functioning or those who had sub-threshold symptoms levels (Biederman et al., 2006). In other words, even when individuals no longer meet diagnostic criteria, there may be substantial functional impairment.

2.4.2 Risk Factors and Associated Factors

ADHD is defined by its consequences and observable symptoms and not by its etiology or biological markers (Killeen, 2019). The exact mechanisms and underlying neurological causes of ADHD are unknown. However, studies do indicate that ADHD has a strong genetic component. ADHD has a heritability rate of about 60-90%, meaning genetics explain about 60-90% of the ADHD variation in a population (Burt, 2009; Nikolas & Burt, 2010; Larsson et al., 2014; Faraone & Larsson, 2019; Pettersson et al., 2019). There is no *one* ADHD gene, rather, ADHD seems to be the result of many genes and their interactions (Faraone & Larsson, 2019; Demontis et al., 2019). The high rate of heritability means someone with ADHD has a high risk of having a close family member with the same diagnosis. In other words, children with ADHD tend to have parents with ADHD (Starck et al., 2016; Walker, 1999).

Apart from genetic factors, possible risk factors for ADHD include prenatal factors such as exposure to alcohol (Wetherill et al., 2018; Mattson et al., 2019; Han et al., 2015), malnourishment (Sucksdorff et al., 2021; Black et al., 2008), various medication such as paracetamol and valproate (Ystrom et al., 2017; Christensen et al., 2019), arsenic and manganese (Rodríguez-Barranco et al., 2013), phthalates (Engel et al., 2018), air pollutants (Zhang et al., 2020; Shih et al., 2020; Park et al., 2020) infections/ virus and/or fever (Gustavson et al., 2019), maternal smoking (Huang et al. 2018), asthma (Liu et al. 2019), stress (Manzari et al., 2019), loss (Li et al., 2010), hypertension (Zhu et al., 2016; Pohlabeln et al, 2017; Maher et al., 2018) and diabetes in pregnancy (Ji et al., 2018). In addition, perinatal complications such as cesarean delivery (Zhang et al., 2020), hypoxia and low Apgar score (Zhu et al., 2016) and premature birth or low birth weight, including being small for age at birth (Aarnoudse-Moens et al., 2009) are possible risk factors. Furthermore, malnourishment in childhood (Peter et al., 2016; Galler et al., 2012; Lu et al., 2019), infections/fever (Pohlabeln et al, 2017; Tseng et al., 2020; Köhler-Forsberg et al., 2019), auto-immune diseases (Nielsen et al., 2017) and exposure to lead (Nilsen and Tulve, 2020; Goodlad et al., 2013; Froehlich et al., 2009) all seem to be risk factors. Traumatic brain injury has also been shown to be a risk factor for so-called secondary attention-deficit/ hyperactivity disorder (SADHD) (Narad et al., 2018; Eme, 2012; Schacher et al., 2004; Adayemo et al., 2014). Substantial substance abuse may also lead to symptoms that are similar to ADHD (Sibley et al., 2018).

Some of these associations might be due to gene-environment correlations. For example, one study found that smoking during pregnancy, which has been considered a risk factor for ADHD, might occur because mothers with ADHD are more likely to smoke during pregnancy (Skoglund et al., 2014). In addition, there are association studies indicating possible gene-environment interactions (Nilsen & Tulve, 2020). However, there are so many possible alternative explanations for such associations that it is difficult to draw any conclusions yet (Moore & Thoemmes, 2016). On the whole, there does not seem to be many single, sufficient causes of ADHD. Instead, most cases are likely to be caused by the combined effects of multiple genetic and environmental risk factors that individually have highly limited effects (Faraone et al., 2021, p. 4).

Disabilities are, in general, associated with a range of social problems and health conditions and ADHD is no exception, although there are considerable individual variations within the group. Studies conducted by the WHO have found an odds ratio of 4.4 for having any co-morbid disorder and these odds increase with the number of co-morbid disorders (Fayyad et al., 2017, p.55). ADHD is strongly

associated with Oppositional Defiance Disorder[1] (OR 15.0), occurring in about 60% of children with ADHD (Fayyad et al., 2017; Azeredo et al., 2018), developmental coordination disorder occurs in about 50% (Goulardinsa et al., 2015), while 18-45 % of children with ADHD have some form of reading disability (Germanò & Gagliano 2010, p. 476). In addition, various learning disorders and autism is common (Tistarelli et al., 2020). Having ADHD in combination with a learning disorder or behavioral disorder substantially increases the risk of detrimental outcomes (Cuffe et al. 2020; Sibley et al., 2011; McNamara, 2005).

There is also considerable comorbidity between ADHD and mental health conditions with 65-89% of all adults with ADHD having an additional psychiatric disorder (Sobanski, 2006; Kessler et al., 2006). Many of the detrimental outcomes associated with ADHD may be partly due to its co-morbidities (Kessler et al., 2014). The co-occurrence between ADHD and other mental health conditions seem to be partly due to shared genetic risk factors (Andersson et al., 2020; Brikell et al., 2020). ADHD is also linked to sleep disruptions in both children and adults with ADHD (Lugo et al., 2020) and to higher levels of emotional lability (Sobanski et al., 2010; Beheshti et al., 2020).

People with ADHD seem to have an increased risk of early mortality and a shorter lifespan than average, with some studies finding that individuals with ADHD have an average nine-year shorter lifespan than the general population (Barkley & Fischer, 2019). ADHD has been found to be associated with five to six times higher than average suicide rates (Fitzgerald et al., 2019; Septier et al., 2019; Fuller-Thomson et al. 2020; Balazs & Kereszteny, 2017), self-harm (Allely, 2014), an increase in accidental injuries (Chang et al., 2014; Brunkhorst-Kanaan et al., 2021; Ruiz-Goikoetxea et al., 2018) and higher rates of premature death (Chen et al., 2019b; Sun et al., 2019). Additionally, ADHD is associated with a vast range of medical conditions such as asthma, allergies, eczema, psoriasis, auto-immune diseases (Cortese et al., 2018; van der Schans et al., 2017; Hegvik et al., 2021; Chen et al., 2019a), diabetes (Kapellen et al., 2016; Chen et al., 2018), sleep disordered breathing (Sedky et al., 2014), epilepsy (Brikell et al., 2018; Bertelsen et al., 2016) and obesity (Cortese et al., 2016; Chen et al., 2018).

ADHD is also strongly associated with alcohol and substance abuse (Bernardi et al., 2012; Lee et al., 2011; Luderer et al., 2018; Fayyad et al. 2017, p. 47-48). In their meta-analysis, Lee et al. (2011), found that those with ADHD were two to three times as likely to develop substance abuse disorders

[1] Oppositional Defiance Disorder is a behavioral disorder identified by persistent irritability, anger, antagonistic and defiant behavior. Onset is usually early childhood. It is associated with Conduct Disorder in adulthood (Burke, & Romano-Verthelyi, 2018, p. 21).

(SUD). Individuals with SUD and comorbid ADHD tend to use substances at an earlier age, develop an abuse problem faster, and have higher rates of relapses than those without comorbid ADHD (Schellekens, et al., 2020). As previously mentioned, prenatal exposure to alcohol seems to be a risk factor for ADHD. The relationship between ADHD and fetal alcohol spectrum disorder (FASD), which is the neurodevelopmental and physical outcomes of prenatal alcohol exposure, is not well understood (Peadon & Elliott, 2010). FASD includes both fetal alcohol syndrome (FAS) and alcohol-related neurodevelopmental disorder (ARND). Children with FASD may have problems with attention, executive function, impulsivity, emotion regulation, planning, memory, processing speed and disruptive behavior and social difficulties (in addition, children with FAS have specific physical features) (Kodituwakku & Kodituwakku, 2014; Peadon & Elliott, 2010). Up to 94% of those exposed to considerable levels of alcohol prenatally, are diagnosed with ADHD (Peadon & Elliott, 2010). FASD is estimated to affect 0.5-2 in every 1000 births in western countries but prevalence rates vary widely amongst communities (Warren et al., 2001; Lange, 2013; Colom et al., 2021; Chasnoff et al., 2015). FASD seems to be generally under-diagnosed (Morleo et al. 2011; Elliott et al., 2006; Elliott et al., 2007) with some studies finding missed diagnosis rates of over 80% in clinical populations (Chasnoff et al., 2015).

It appears to be difficult to distinguish between children with FASD and children with ADHD alone based on traditional observation scales (Nanson & Hiscock., 1990). However, some studies find differences in cognitive functioning in specific domains and prognosis and treatment response appear to diverge (Peadon and Elliott, 2010). A prospective study comparing children with prenatal alcohol exposure to children with ADHD (without alcohol exposure) found that adaptive behavior skills improved with age in the ADHD group, while the children with a history of alcohol exposure exhibited increased problems with age (Crocker et al., 2009). Individuals with FASD may also respond differently to stimulant medication than other children with ADHD (O'Malley and Nanson, 2002).

A wide variety of studies have shown that children, adolescents, and adults with ADHD are exposed to higher-than-average levels of social rejection (Hoza, 2007). Shea and Wiener (2003) report that children with ADHD are perceived as “different” and subjected to social exile. This rejection seems to be due to both the negative attitudes towards behaviors associated with the diagnosis (such as impulsivity or hyperactivity), as well as towards the diagnosis itself (Lebowitz, 2016; Harris et al., 1992). Studies of adult’s attitudes towards adults with ADHD also find that fictional vignette characters with symptoms of ADHD are more likely to be socially rejected than characters without such symptoms or characters described as having a general medical condition or a «personality flaw»

such as perfectionism (Canu, et al., 2008; Paulson et al., 2005). Unsurprisingly, children, adolescents, and adults with ADHD appear to experience lower levels of social support than average (Emser & Christiansen, 2021; Demaray & Elliott, 2001; Rokeach & Wiener, 2020; Brod et al., 2012).

ADHD is also associated with lower average educational attainment, though the association seems to be moderated by treatment (Zendarski et al., 2020; Kuriyan et al., 2013; DuPaul & Langberg, 2015; Arnold, et al., 2020). In general, disabled people have lower levels of academic achievement (Hammersley et al. 2020, p.53; Cambois et al., 2016; & The World Bank, 2011). It is possible that part of this association is driven by the stigma attached to the ADHD diagnosis (Owens & Jackson, 2017). For instance, studies have found that teachers tend to underestimate the scholastic abilities of children with an ADHD diagnosis regardless of their actual abilities (Metzger & Hamilton, 2020; Eisenberg & Schneider, 2007). Studies show similar results regarding adults (Godfrey et al., 2021).

On average, ADHD is associated with lower socioeconomic status (SES) in both children and adults (Pelham et al., 2020; Russell et al., 2016; Rowland et al., 2018; Sundquist et al., 2015; Choi et al., 2017). The association between adult ADHD and lower socio-economic status is likely to be at least part of the reason why there is also an association between children with ADHD and lower socio-economic status as some studies have found the SES-child-ADHD association to be statistically unreliable when controlling for parental ADHD status (Counts et al., 2005). This link with low SES is not unique to ADHD but seems to be the case for disabilities in general (Kaye, 2009; Palmer, 2011; Cambois et al., 2016; WHO & The World Bank, 2011).

Several studies have found a connection between ADHD and various forms of family structure. ADHD seems to be associated with higher levels of single-parent households, younger parents, divorce, step families and parental conflict (Brown et al., 2017; Kvist et al., 2013; Wymbs et al., 2008; Agha et al., 2013; Kousgaard et al., 2018, pp. 1391; Østergaard et al., 2017; Perales et al., 2017; Fayyad et al., 2017; de Zwaan et al., 2012, p. 79; Pineda, 2020, p. 231). Furthermore, families with children with ADHD seem to experience more parenting stress (Miranda, Tarraga and Ferdandez, 2015; Theule et al., 2013). This relationship is likely moderated by several factors such as symptom severity, additional behavioral issues, parental marital status, lack of social support, family disorganization and parental ADHD symptoms (Theule et al., 2013; Leitch et al., 2019; Mikolajczak et al., 2017).

Moreover, studies find that ADHD is associated with higher rates of criminal behavior (Mohr-Jensen & Steinhausen, 2016). A meta-analysis of 42 studies from 15 countries found the prevalence of ADHD in youth prisons to be 30.1% and 25.5% in adult prisons (Young et al. 2015). Furthermore, ADHD

seems to be associated with gender non-conformity (Bretherton et al., 2021; Warrier et al., 2020; Wang et al., 2014). Finally, ADHD is associated with a substantial amount of stigma, particularly related to taking medication for ADHD (Bussing et al., 2012; Mueller et al., 2012; Masuch et al., 2019; Arcia et al., 2004; Charach et al., 2006; Speerforck et al., 2019) and the public uncertainty regarding the validity of an ADHD diagnosis (Mueller et al., 2012).

As was the case with victimization, many of the factors associated with ADHD can be understood in light of the concept of social marginalization. The marginalization of people with ADHD may be understood as a result of the debilitating effects of ADHD on an individual's functioning. Conversely, it may be understood as a result of structures in society that discriminate against and marginalize people with disabilities.

Notwithstanding the account above, it is worth pointing out that although ADHD seems to be associated with a considerable number of detrimental outcomes, ADHD probably also has many beneficial aspects. Unfortunately, these potential positive associations do not seem to have sparked the interest of the scientific research community.

2.4.3 Perspectives on ADHD

The Individual/Pathological Model. ADHD can be defined and understood in many ways. Traditionally, the individual model of disability (also known as the medical or pathological model) has been the most dominant model. Here, ADHD is understood as something inherently "wrong" with a person's mind, a form of deficit or pathology, and the aim is to prevent, treat or cure such conditions (Oliver, 2013; Ray, 2013). These theories tend to explain ADHD using models of dysfunctional brain networks or psychological dysfunctions or impairments. The pathological models of ADHD include theories of executive dysfunction, deficits in mentalization abilities, delay aversion, working memory deficits, emotional and mental dysregulation, motivational dysfunction, dopamine deficits, suboptimal distribution of energy to cognitive processes, default mode intrusion and inhibitory deficits (Killeen, 2019; Nigg, 2017b; Johnson et al., 2009; Leuzinger-Bohleber et al., 2010; Conway et al., 2011; Posner et al., 2020). The focus is on impairments, deficits and dysfunctions compared to what is considered optimal or normal functioning. There is an implicit evaluation of ADHD as inferior to these normative standards. The theory regarding ADHD as a dysfunction caused by psychological damage in need of psychological interventions or repair, falls under this model.

The Social Model of Disability and the Neurodiversity Movement. Unlike the individual model, the social model differentiates between the impairment itself and the socially constructed disability. The

problems associated with disabilities are due to the fact that society is not adapted to the needs of differently-abled people. Instead, society's organization and structures are based on how the majority function. These structures may take the form of individual prejudice, inflexible educational systems or repressive norms and conventions that operate as social barriers excluding disabled people from participating equally in society (Oliver, 1996). It is not the impairment itself that is the biggest challenge to disabled people, but oppressive social systems. Consequently, it is not mainly the impairment or the disabled individual that needs to be changed or rehabilitated, but society.

The *neurodiversity movement* has its roots in both the disability movement and the survivalist movement and represents a bid for a paradigm shift in psychiatry (Chapman, 2019). According to proponents of neurodiversity, neurological conditions such as autism, dyslexia and ADHD are actual differences in neurological functioning but should not be seen as pathologies needing a cure (Graby, 2015, p. 234; Chapman, 2019, p. 371). Instead, they are seen as natural variations in the human population to be accepted and accommodated (Singer, 1999; Graby, 2015, pp. 232-233). Neurological diversity is just as natural and important as biological diversity. Consequently, the movement aims to gain rights for, and increase recognition and acceptance of, neurodivergent people in society.

The neurodiversity movement challenges the stigma of being neuro-atypical and actively reclaims the diagnoses as something positive. Whereas a diagnosis has traditionally been viewed as something to be avoided, the neurodivergent population often view it as something to be fought for (Singer, 1999, p.65). For many neurodivergent individuals, diagnosis is a relief (Carr-Fanning & McGuckin, 2018). It leads to understanding and acceptance of oneself and to finding one's "tribe": the neurodiverse community. Denial of diagnosis is denial of knowledge about who you are, it is denial of identity and denial of community.

2.5 Theoretical Pathways from Victimization to ADHD

Most theories positing that victimization may cause ADHD are more or less based on psychoanalytic theory and thoughts regarding psychic trauma, attachment theory and self-regulation (Szymanski et al., 2011; Pynoos et al., 1996; Van der Kolk, 2008; Conway et al., 2011; Leuzinger-Bohleber et al., 2012). Self-regulation is a valued quality in many cultures and often equated with moral virtue. The classic example is how Eve's lack of self-control regarding a certain apple led to humanity's fall from grace (Forgas, Baumeister & Tise, 2009, p. 3). Poor self-regulation is related to a vast range of mental health issues such as depression, addiction, bipolar disorder, autism, obsessive compulsive disorders, suicide and, last but not least, ADHD (Nigg, 2017a, p. 2).

Self-regulation is a term encompassing a wide variety of dynamic and complex adaptive systems. There is currently no agreed upon definition of the term but Nigg describes it as the automatic and deliberate regulating processes and systems used to modulate emotion, cognition and behavior (Nigg, 2017a, p. 1-2, 16). Humans self-regulate when we change our behavior in accordance with social expectations and standards or when we override our immediate urges in order to meet long-term goals (Baumeister et al., 2007). Self-regulation may involve a multitude of different systems and processes that may interact and develop across the lifespan in a non-linear fashion (Nigg, 2017). It has been proposed that the conscious act of self-regulation is a limited resource (Baumeister et al., 1998). In other words, the more one actively regulates aspects of the self, for instance during a diet, the less resources one has available for other activities requiring self-regulation. In this way, regulation of the self may be thought of as a muscle, which may be worn out immediately following rigorous exercise, but also strengthened by regular activity.

In contrast, the psychoanalytic view of self-regulation is usually understood as being dependent on the healthy development of fundamental psychic constructs or abilities such as ego-functions, object relations or mentalization (Leuzinger-Bohleber, 2010). The development of these capabilities is largely seen as contingent on a beneficial relationship between infant and caretaker and so is closely tied to attachment theory. Attachment as a theoretical concept is usually attributed to John Bowlby, who was significantly influenced by biology and ethology, and was later expanded on by Mary Ainsworth. Bowlby argued that healthy development in children is dependent on a warm and continuous relationship with a mother figure (Shute & Slee, 2015, p.96). The mother functions as the child's ego and super-ego, regulating the child, while the child gradually acquires these skills itself. According to Bowlby, there were critical periods for attachment, especially the first year. If a child had not experienced such a relationship in this period, the damage would become permanently fixed and very difficult to repair. Bowlby also introduced the idea that attachment relationships lead to working models of the self and others, which then guide an individual's expectations and behavior. According to most attachment theorists there is one optimal type of attachment, called a secure attachment, which is considered to be the result of sensitive parenting (Shute & Slee, 2015, pp. 96- 99).

It is widely theorized by pcychoanalysts that disruptions in attachment may lead to dysregulation (Spinazzola et al., 2018; Fonagy & Bateman, 2016; Leuzinger-Bohleber, 2010). One way this is posited to occur is through impairment of the ability to mentalize. The concept of mentalization is a child of the Theory of Mind and refers to an individual's imaginative capacity to perceive mental states and beahvior as meaningful (Køster, 2017). Mentalization theory as defined by Peter Fonagy and

Anthony Bateman (2016) encompasses both social cognition and self-reflection. According to Fonagy, the ability to mentalize is a developmental accomplishment that is dependent upon attachment and the right interactions between infant and caretaker, in particular mother-infant mirroring. It is speculated that trauma, particularly attachment trauma, leads to loss of this ability to mentalize and often a failure to develop object permanence (Fonagy, 2010, p. 92-107; Fonagy & Target, 1998, p. 95). The traumatized child is unable to distinguish thoughts and feelings, internal and external reality and so «is completely out of touch with his own and others experiences» (Conway et al., 2011, p. 69). It is further claimed that this lack of ability to label and understand their own and others mental and emotional states means the child or adult is unable to regulate emotions. It is this lack of self regulation that causes the symptoms of ADHD (Ibid.).

Although he does note explicitly refer to ADHD, Bessel van der Kolk has proposed a new diagnostic category, Developmental Trauma Disorder, which he believes has many of the same symptoms as ADHD and which he claims are the result of childhood trauma (Van der Kolk & D'Andrea, 2010). In addition to references to attachment, he bases his theory on the assumption that there are certain unique kinds of experiences that constitute "traumatic experiences" and that these events activate a special kind of consciousness or alarm state. During these alarm states people are "cut adrift from the cortex". The parts of the brain responsible for executive functioning "go offline" and verbal abilities are limited (p. 59). These states are then easily triggered and re-activated by often subconscious reminders of the traumatic incident. Van der Kolk uses association studies of child maltreatment and brain dissimilarity to corroborate that trauma damages the brain and leads to structural, biological damage (Van der Kolk & D'Andrea, 2010). In doing so, he crosses over into what may be termed "neuropsychoanalysis" (Blass and Carmeli, 2007) designating the traumatized victim as a neural phenotype.

3.0 BACKGROUND: ADHD AND VICTIMIZATION

3.1 Associations with Various Forms of Victimization Across Ages

Studies have found an association between ADHD and numerous forms of interpersonal victimization across childhood, adolescence and, to a lesser extent, adulthood (Aguado-Gracia et al., 2018; Snyder, 2018; Hellström, 2019; Efron et al., 2018). These associations include child maltreatment (Ouyang et al., 2008; Sugaya, et al., 2012; Boyd et al., 2019), adolescent sexual violence victimization (Ngo et al.,

2018; White et al., 2014), hospitalization of children and adolescents due to interpersonal violence (Lam, 2005) and sexual assault victimization in adolescence and young adulthood (Wymbs & Gidycz, 2021). Several studies have found that children and adults with ADHD seem to have had higher rates of adverse childhood experiences in general, such as a having a parent who served time in jail, parental divorce, death of a parent, witnessing domestic violence, living with someone with a substance abuse problem etc., than typically developing children (Schneider, et al., 2019; Brown et al., 2017; Björkenstam et al., 2017). In addition, a meta study by Hellström (2019) found that children with ADHD had experienced significantly higher rates of polyvictimization compared to typically developing youth.

Although most studies focus on children and youth a growing number of studies are looking at young adults. Studies have found associations between ADHD and assault victimization in men attending college (Snyder, 2019), and sexual victimization in women attending college (Snyder, 2015), intimate partner violence victimization (Wymbs et al., 2019) and dating violence (McCauley et al., 2015).

Studies have found various mediating and moderating factors between ADHD and victimization. For example, White, Buehler and Weymouth (2014) found that race and growing up in a female-headed household moderated the relationship between ADHD and adolescent sexual victimization.

Conversely, there are also studies that do not find associations between victimization and ADHD or that find mixed results regarding specific symptoms, gender or lack of association when controlling for additional factors such as learning disorders or behavior problems (Whitmore et al., 1993; Wozniak et al., 1999; Mukherjee et al., 2019; Fonseca et al., 2019). For instance, in a study of sexual abuse, Merry and Andrews (1994) found no association between the child's age, severity and duration of sexual abuse, previous abuse, relationship between abuser and child or socioeconomic status and psychiatric status of the child (including ADHD). However, they *did* find an association between the child's diagnostic status and the mother's mental health status.

3.2 Early Adversity

Golm et al. (2020) used a quasi-experimental design to compare 72 adoptees from Romanian institutions and 22 adoptees from Korea with no institutional experience. The authors found an association between ADHD and experiencing institutional deprivation though these correlations were no longer significant when controlling for IQ. However, the interpretation of these results is problematic for two reasons. Firstly, the conditions in the Romanian orphanages were potentially detrimental in a multitude of ways. In addition to being socially and emotionally deprived, the majority

of children in these institutions were also malnourished (Stephenson et al., 1994, pp. 82-83), and physical abuse (including blows to the head) was also widespread (Rus et al., 2013). It is not self-evident which of these factors were related to the outcome. Secondly, the two groups compared might differ on factors unrelated to institutional experiences. For example, children in institutions are at an especially high risk of fetal alcohol spectrum disorder (FASD), but the rates vary considerably among different populations (Warren et al., 2001; Lange, 2013; Colom et al., 2021; Chasnoff et al., 2015).

In general, research contamination is a potential complicating factor when studying the effects of child maltreatment or prenatal alcohol exposure as there is considerable overlap between the two groups leading to the risk of confounding (Henry et al., 2007). A systematic review in 2005 found that there were no studies thus far, of maltreated children that examined the incidence of prenatal substance exposure in the samples (De Bellis & Van Dillen, 2005). This has changed in the past few years, though it is still, by far, an understudied field.

Henry et al. (2007) studied the effect of FASD in children who had experienced traumatic stress. The authors found that children who had experienced prenatal alcohol exposure and traumatic stress had lower intelligence scores and more neurodevelopmental deficits such as attention problems—in addition to higher rates of oppositional defiance disorder and social problems—than did abused children without prenatal alcohol exposure. These finding led the authors to conclude that prenatal alcohol exposure has an effect separate from traumatic stress.

Mukherjee et al. (2019) studied the effects of neglect on children with FASD. In their study comparing children with FASD with or without postnatal neglect, they found no difference between the groups in developmental outcomes, suggesting that exposure to prenatal alcohol influences these outcomes independent of neglect—and furthermore, that neglect does not worsen the effects of FASD. The authors recommended that professionals who are working with such children be aware that behavioral difficulties in children with both FASD and a history of neglect are "likely to be related to prenatal alcohol exposure and not necessarily reflective of parenting quality" (Mukherjee et al., 2018, p. 27).

Several studies have found an association between ADHD and experiences of child abuse, though results are somewhat mixed regarding types of abuse, ADHD symptom type, gender associations and correlates (Ouyang et al., 2008; Sugaya et al., 2012; Boyd et al., 2019; Sanderud et al., 2016; Gonzalez et al., 2019). A meta-analysis found that people with ADHD were more than twice as likely to have experienced maltreatment as a child, than those without ADHD (Clayton et al., 2018).

Ouyang et al. (2008) conducted a retrospective study of a nationally representative sample of more than 14 000 participants and found a significant association between ADHD inattentive type and the likelihood of supervision neglect (OR 1.6), physical neglect (OR 2.1), physical abuse (OR 1.6) and contact sexual abuse (OR 1.6). They found a smaller association between ADHD hyperactive type and supervision neglect and physical abuse (OR 1.5 and 1.3, respectively), but no significant association with physical neglect or sexual abuse. The study used a propensity score matching model to control for confounding factors such as race and ethnicity, family structure, teenage parents and SES. However, they did not control for substance abuse.

Boyd, Kisely, Najman and Mills (2019) found an association between non-sexual maltreatment in childhood and attention problems at age 14 and 21 when controlling for confounding factors such as age, gender, birth weight, maternal depression, educational level, parental relationship status at birth and family income. In contrast, they did not find a similar association between sexual abuse and attention problems. Again, there was no control for substance abuse.

The association between ADHD and child maltreatment seems to be stronger when ADHD is co-morbid with Oppositional Defiant Disorder or Conduct Disorder. Ford et al. (2000) found that having ODD in addition to ADHD increased the rates of physical abuse from 26% to 73% and the rate of sexual abuse from 11% to 31% in a clinical sample of 165, 6–17 year-olds. In their meta-analysis, Clayton et al. (2018) found that individuals with co-morbid ADHD and ODD/CD had an odds ratio of 2,55 of having been maltreated compared to individuals with only ADHD.

There are several possible reasons for the association between ADHD and child maltreatment. First of all, they share many of the same associations and risk factors such as parental unemployment, substance abuse, poverty and mental health problems (Fuller-Thompson & Lewis., 2015, p. 189; Mulsow et al., 2001; Chronis et al., 2003). Secondly, infants who later develop ADHD and children with ADHD tend to have more challenging temperaments in the form of higher activity, less adaptability, more negative moods, and sleep disturbances which may affect parenting stress and the parent-child relationship negatively (Johnson et al., 2015; Shen et al., 2020; Martin et al., 2019). As previously mentioned, families with children with ADHD seem to experience more parenting stress than average (Miranda, et al., 2015; Theule et al., 2013; Yousefia et al., 2011). Schneider et al. (2019) found that the rates of resilience for families (such as the ability to talk about problems, work together to solve problems, perception of strengths to draw on and hope for future) were similar for families with children diagnosed with ADHD and families with typically developing children when controlling

for ACEs and demographic variables. Nonetheless, this resilience was reduced in families with a child with more than one diagnosis (Schneider et al., 2019).

Thirdly, genetic factors may come into play as parents of children with ADHD are more likely to have ADHD themselves (Thapar, et al., 2007). This might increase the risk of maltreatment further. Some studies seem to find that child abuse is associated with parental ADHD. For instance, in a sample of children with ADHD, Becker-Blease & Freyd (2008) found that 77% of abused children had a parent with ADHD while only 33% of non-abused children did. A systematic review found a small association between parental ADHD and harsh parenting (Park et al., 2017, p. 30). A Turkish study of children with ADHD, ages 6-11, found that maternal hyperactivity/impulsivity was associated with emotional abuse of boys and emotional neglect in both boys and girls, while paternal attention deficit was associated with sexual abuse (Gul and Gurkan, 2018). Similarly, Tachibana et al. (2016) found a small association between mothers' ADHD symptoms and child maltreatment with impulsivity having an independent association with maltreatment. Rodriguez, Gonzalez and Foiles (2018) found that maternal ADHD symptoms predicted potential risk of child abuse through their children's symptoms of ADHD but only weakly through the mother's parenting style. Fujiwara et al. (2014) found that maternal ADHD symptoms in pregnancy were associated with child mistreatment in the first three years after birth but that the association became non-significant when adjusting for maternal autism.

All the same, these finding should not necessarily be interpreted to mean that ADHD is a direct cause of child abuse. For instance, these studies often have a limited degree of control for confounding factors. As previously mentioned, the association might be caused by confounding or mediating factors such as low SES, substance abuse, low parental age, parental stress, parental conflict, lack of social support, single headed households, stepparents, mental health and psychosocial problems, which are associated with both parental ADHD and risk of child maltreatment.

3.2.1 Genetic Influences

One way to study the potential effects of familial confounding is through twin studies. In a children-of-twins study, Silber, Maes & Eaves (2012), looked at 2 674 twins and their 2 454 children to determine if parental antisocial behavior is an actual environmental risk factor for depression and/or hyperactivity (and conduct disturbances) in children. They found that the impact of parental antisocial behavior on children's hyperactivity was entirely genetic but that the impact on depression was entirely environmental (Ibid, p.668).

A study from Sweden examined the relationship between child maltreatment and ADHD in a sample comprising 8 192 nine-year-old MZ and DZ twins (Dinkler et al., 2017). In their co-twin control analysis, they found that most of the association between child maltreatment and ADHD was explained by common familial factors, but that maltreated boys had a slightly higher level of ADHD symptoms compared to their non-maltreated MZ twins (the mean difference was 1.47 points on the A-TAC scale from 0-19). Conversely, they did not find any difference in symptom levels in twin girls.

Similarly, a retrospective Swedish study of 18 168 adult twins found that genetics accounted for part of the association between childhood maltreatment and adult symptoms of ADHD, but not all (Capusan et al., 2016). In contrast to the Dinkler study, they did not find different associations based on gender. When limiting the study to abuse prior to the age of seven, they found similar effect sizes as when they included maltreatment up to the age of 18, indicating the associations were independent of age of maltreatment. The study found the strongest association between ADHD and physical neglect, while the association with physical abuse was unsubstantial and non-significant.

Although these studies suggest that the association between ADHD and child maltreatment is not entirely explained by familial confounding, neither of them measured ADHD symptoms at the outset, so they were unable to estimate causal direction. However, they do indicate how important it is to control for familial confounding when studying possible causal relationships between ADHD and child maltreatment, as familial confounding apparently accounts for a substantial part of the association.

3.3 Peer Victimization

Several large studies have also found an increased rate of peer victimization in children with ADHD symptoms (Holmberg & Hjern, 2008; Bacchini et al., 2008; Unnever & Cornell, 2003; Yang et al., 2013). This risk is higher for many groups of children with disabilities, but some studies find that children with ADHD have an especially high risk, even compared to other disabled groups (Blake et al., 2016). Various factors have been found to mediate or moderate the association between ADHD and peer victimization, such as friendships, Oppositional Defiant Disorder, Developmental Coordination Disorder, self-control, gender and physical size (Jia & Mikami, 2014; Cardoos & Hinshaw, 2011; McQuaade et al., 2018; Unnever & Cornell, 2003; Fite et al., 2014; Dewey & Volkovinskaia, 2018) There are also findings that imply that environmental differences play a role in these associations, such as whether or not children attend mainstream schools or specialized schools, with children in mainstream schools having a higher risk of victimization (Chan et al., 2018). On the other hand, some

studies have been unable to find any significant correlations between ADHD and peer victimization when controlling for other learning disorders (Klomek et al., 2016) or have found mixed results or different results for boys and girls (Wiener & Mak, 2009).

3.4 ADHD as Risk Factor for Victimization

The correlation between ADHD and interpersonal victimization may also be explained by the fact that ADHD seems to be a risk factor for victimization. Studies have found ADHD and/or genetic liability for ADHD to be a risk factor for victimization in the form of child abuse (Leppert et al., 2020; Zwicker et al., 2020; Stern et al., 2018), peer victimization (Törn et al., 2015; Blake et al., 2016; Cardoos & Hinshaw, 2011; Schoeler et al., 2019), sexual abuse in childhood, adolescence and young adulthood (White et al., 2014; Christoffersen, 2020; Ohlson Gotby et al., 2018; Wymbs & Gydycz, 2021) dating and intimate partner violence (McCauley et al., 2015; Guendelman et al., 2016; Wymbs et al., 2019; Wymbs et al., 2017), violent crime in childhood and adolescence (Christoffersen, 2019) and adverse childhood or life experiences in general (Lugo-Candelas et al., 2020; Li et al., 2021).

Any claimed observation of causation must therefore establish *temporality* - in other words it is necessary to demonstrate that *the cause precedes the effect* (Rothman & Greenland, 2005, p. 148). Establishing temporality does not prove causation - as few things do - but it might point us in the right direction. As studies have found that ADHD may be a risk factor for victimization, any study regarding victimization as a risk factor for ADHD has to control for the possibility of reverse causation.

3.5 Complex Interactions

The complexity in interactions between variables influencing ADHD and victimization is illustrated in the figure below using SES and substance use/abuse as example variables. Because all the variables

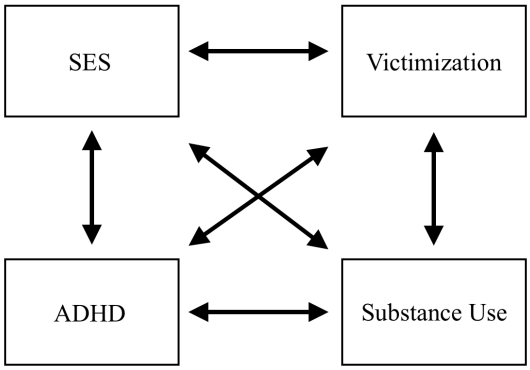


Figure 1. Possible pathways between ADHD, victimization, SES and substance use.

are interconnected, changes in any one variable will potentially result in changes in the other variables which might feed back into the original variable. In addition, all of these variables are influenced by other factors outside of the diagram so the entire system is always in a potential state of change.

4.0 METHOD

4.1 Design

According to Booth et al. (2016) a systematic review involves a methodical approach to the search for relevant literature or research, its appraisal, and synthesis and analysis of findings. These methods should be explicit and reproduceable (Booth et al. 2016, pp. 9-28). This review has relied mainly on the recommendations of Boothe et al. (2016) and Siddaway et al. (2019) when making decisions regarding method and design. Nevertheless, some recomendations have not been possible due to lack of resources and other practical restrictions. For example, only studies published in peer reviewed journals have been included, although this increases the risk of publication bias. In addition, although most reference lists were reviewed to uncover other studies, this was not done sytematically. Furthermore, although it was attempted to document all aspects of searches, some features of the databases, which would have simplified this, were not discovered until after the final search. Therefore some processes, such as saving searches, were done manually, which may have increased the risk of accidental mishaps.

Although the research design should ideally be planned in advance and followed, this review has changed course during the process. At the outset, I had hoped to research the broader relationship between victimisation and ADHD and therefore my research question consisted of two parts: Is interpersonal victimization a risk factor for ADHD and is ADHD a risk factor for victimization? However, it became apparent that such a broad topic was beyond the scope of this thesis. Due to time, resources and space constraints, my research therefore focused on the first question alone. Nonetheless, this initial broad literature review was useful in gaining an overview of the topic and in writing the background section. For example, most of the references in Chapter 2.4 are a result of this initial review.

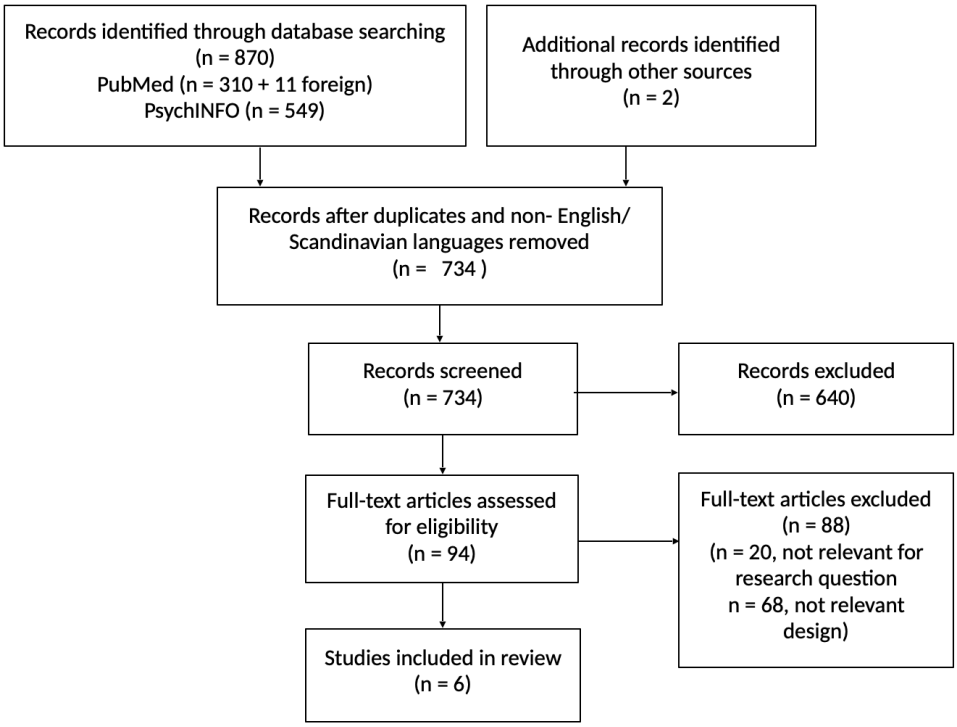
4.2 Search Strategy

PubMed and PsychINFO were searched between the 20th of June 2020 and the 12th of October 2020 for English or Scandinavian-language articles published in peer reviewed journals. PsychINFO is a database produced by the American Psychological Association focusing on psychology and related fields, spanning the timeline from 1806 forward and containing over two million references from all over the world (Apa.org). PubMed is the free version of Medline. It is the world's largest database of biomedical and life science literature and contains over 30 million citations from 30 000 publications (Pubmed.gov).

The keywords and MESH terms (Medical Subject Headings) used were: *Attention Deficit Disorder* / *Attention Deficit Disorder with Hyperactivity* / *Attention-Deficit/Hyperactivity Disorder* / *ADHD* in combination with the terms *Psychological Trauma* / *Emotional Trauma* / *Child Neglect* / *Child Abuse* / *Sexual Abuse* / *Maltreatment* / *Adverse Childhood Event* / *Childhood Adversity* / *Physical Abuse* / *Emotional Abuse* / *Bullying* / *Peer Victimization* / *Intimate Partner Violence* / *Domestic Violence* / *Rape* / *Assault* / *Crime Victim / Victimization.* A search was also conducted in both databases for all articles containing the terms *ADHD* / *Attention-Deficit Hyperactivity Disorder* / *Attention Deficit Disorder* / *Attention Deficit Disorder with Hyperactivity* / *Attention-Deficit/Hyperactivity Disorder* and *victimization* in the title or abstract. The term *violence* was not used as trial searches indicated that it did not give new or different results relevant to the study, but increased the number of irrelevant results pertaining mostly to the perpetration of violence. This was also the case for the single term *abuse* as results then largely dealt with substance abuse and did not add more relevant studies. In addition, *early life stress* gave about 150 results in PsychINFO, but no new studies and mostly animal studies. The term *trauma* alone gave mostly medical/physical trauma—particularly head trauma, so these terms were not used. Trial searches were done in Google Scholar, in addition to PsychINFO and PubMed, but this did not yield additional results.

Total search results yielded 321 articles in PubMed and 549 articles in PsychINFO. After removing the duplicates and articles that were not in English or a Scandinavian language, 732 articles remained. Two more articles were identified through other sources (PubMed suggestion and article reference list). In accordance with the initial broad search that included studies looking at ADHD as a risk factor for victimization, a total of 640 articles were excluded based on the title or abstract. The remaining 94 full text articles were reviewed. Based on the narrowed research question, six studies were chosen (see flow chart).

Figure 2. Flow Diagram



4.3 Criteria for Inclusion and Exclusion

Criteria for inclusion in the review were original empirical studies with human subjects, published in peer-reviewed journals, with relevance to the research question. As the research question regards measurements of risk, only quantitative studies were included. Only studies that strictly pertained to ADHD were included. Studies that did not directly pertain to ADHD but assessed risk regarding factors or constructs connected with or encompassing a limited part of ADHD, such as externalizing behavior, executive function or impulsivity, were excluded as it would be difficult to clearly define the line for inclusion/exclusion. Studies were included that used both dimensional and categorical measurements of ADHD and interpersonal victimization.

Some interpersonal experiences that might be damaging, but not necessarily norm breaking, were excluded. The social rejection of, especially, children with ADHD has been reasonably well established and extensively researched and is beyond the scope of this review. It is worthwhile though, to note that recent research points to possible reciprocal, cascade effects between peer rejection and ADHD symptoms (Tseng et al., 2014). When victimization has been joined into larger conglomerate variables (such as peer rejection), the study has been included although this means it is more difficult to draw conclusion regarding victimization alone.

Because this review is concerned with interpersonal victimization as a possible risk factor, research design has been an important criteria for inclusion or exclusion. As the classic RCT study is not a viable option to explore the research question, observational studies must be used (Shadish, et al. 2002). Making causal inference based on observational studies is, as mentioned earlier, fraught with risk, especially due to threats to internal validity. Therefore, this review has made some assessments regarding study design. This is not in any way meant to imply that threats to validity have been eliminated in the chosen studies, but that a certain level of adjustments to method and design have been made in order to establish temporality. The focus has been on design *features* rather than design *labels* as the latter are often ambiguous (Reeves et al., 2017).

According to Kraemer et al (1997) demonstrating a risk factor:

> requires obtaining its measure on the subjects in a sample free of the outcome of interest, then distinguishing those who do from those who do not subsequently have the outcome of interest (...) (and establishing) a statistically significant association between (risk) factor and outcome. (p. 338)

This forms the basis for the inclusion criteria in this review. As the focus is on change over time, longitudinal studies that attempt to establish temporality, and control for reverse causation, by measuring both the predictor and the outcome variable at base have been included. Studies that measure the same variable in the same individual over time provide information on both intra-individual (within person) and inter-individual (between person) change over time (Duncan, T. & Duncan, S., 2009; Shadish et al., 2002, p. 136). All studies that have used a form of pre-test, post-test design have been included, as long as the pre-test/post-test variables are the same. This means that studies have been included that use proxy measurement. Proxy measurement has certain fundamental flaws, which will be discussed later. However, these choices have been made in order to be flexible enough to include as wide a variety of studies as possible and facilitate transparency about the research material.

4.4 Quality Assessment of Studies

There is no “gold standard” for evaluating the quality of observational studies. In their systematic review of tools used to assess the quality of observational studies examining risk factors, Shamliyan, Kane and Dickinson (2010) observed that there was a relatively low consensus concerning quality components for specific types of research, but that there was general agreement on what constitutes major “universal» flaws, such as selection bias, confounding, or misclassification (also known as information bias or measurement bias) (Shamliyan et al., 2010, p. 1067). This view is shared by the

Cochrane Handbook for Systematic Reviews of Interventions, which states that non-randomized studies are particularly vulnerable to confounding, selection bias and information bias (Higgins et al. 2020).

Selection bias. Confounding has been discussed earlier, so will not be discussed again, but a few words on information bias and selection bias are in order. Selection bias refers to bias in the sample selection. When a sample is not based on a random selection from the population it risks being skewed and therefore not representative of the population. This may affect the study's external validity. Selection bias may also occur, as previously mentioned, when allocation to comparison groups is not random. This may lead to a confounding of the effects of risk factors with population differences between the groups, which poses a risk to the study's internal validity. In addition, longitudinal studies have to contend with what are often substantial attrition rates. Measures should be made to evaluate whether or not attrition is related to any of the study variables and might threaten the validity of the study (Shadish et al., 2002, pp.334-337).

Information bias. Information bias refers to biased or inaccurate measurement, recording or interpretation of information leading to erroneous estimations. Any study of variables, such as ADHD, that cannot be measured directly has to contend with the issue of *construct validity*. Construct validity refers to the extent that the concrete measurements used in a study actually represent the phenomenon we are attempting to capture (Shadish et al., 2002, pp. 64-82). In general, there is no measure of ADHD that is not based on some form of standardized interview or checklist. The ADHD diagnose, as most psychiatric diagnoses, is based on observable symptoms, which is inherently difficult to measure objectively. There is no objective test which other instruments can be measured against. The appraisal of validity of measuring instruments for psychiatric diagnoses are often based on how such instruments relate to other instruments thought to measure the same thing or how internal constructs relate to each other (Jozefiak & Berg-Nielsen, 2016). Measuring instruments should ideally be validated. They should also be reliable and show measurement equivalence by measuring the same thing across repeated measurements both across time, individuals and contexts (Casper et al., 2015).

When we study change over time, we often use longitudinal designs where data is collected from individuals on repeated occasions. For instance, ADHD symptoms may be measured in children on a yearly basis over the course of several years. If we are to accurately interpret any change in our data regarding these constructs or latent variables over time, it is important that we are actually measuring the same thing at the various waves (Millsap and Cham, 2012, p. 109). The measures and subsequent data should relate to the same factors and constructs when the child is 5 years old, as when the child is

7 years old. Even if the same measurement instrument is used at all waves, they may not be capturing the same thing because humans develop and change with time (Millsap and Cham, 2012, p. 109; Casper et al., 2015). Therefore, lack of measurement equivalence over time poses a possible risk for information bias in longitudinal studies.

Another potential threat regarding measurement data is rater bias. Subjectivity in rating can be particularly detrimental if ratings of outcome are given "unblinded". If the rater has information about the exposure status of the individuals they are rating, the rating might be influenced by the rater's own hypotheses about causal connections (Minder et al., 2018). This is why it is best to use blinded raters and to avoid using the same rater across several variables, especially across predictor and outcome variables. Rating scores also vary according to who is rating. For example, there is low inter-rater reliability between teachers and student when it comes to bullying and between adolescents, parents and teachers when it comes to ADHD symptoms (Rønning et al., 2009; Valo & Tannock, 2010). In other words, *who* is rating *what* influences results. Using several modes of rating of the same variable, diminishes the risk of information bias (Shadish et al., 2002, p. 75).

4.5 Risk of Bias in Reviews

The review itself also faces risk of bias. Particularly, selection bias by the reviewer and publication bias (Booth et al., 2016). The way we conduct research and interpret what are usually ambiguous scientific findings, is influenced by beliefs, values and discourses. These assumptions and mindsets shape the very questions we ask, the way we attempt to answer them, evaluate results and present our findings. In this particular case, the reviewer's background—both clinical experiences working with victims of violence and abuse and personal experience with the disability movement—has probably influenced the choice of topic, viewpoint and interpretations and therefore poses a risk for bias. Search results should ideally be gone over by several reviewers in order to avoid reviewer bias but this was not possible. However, results were reviewed twice a few months apart to make sure that no relevant studies were missed.

In addition, excluding so-called *grey literature*, is apt to increase risk of publication bias as published literature does not represent a "random sample" of research results (Booth et al., 2016, p.120). For instance, statistically significant outcomes are more likely to be reported in published studies (Chan & Altman, 2005) and the results of many studies are never published at all.

4.6 Ethical Considerations

I have chosen to write about groups that are often marginalized and stigmatized. In so doing, it has been a priority not to add to such stigma. For eample, it has been important that this paper not reproduce ableist attitudes. Ableism refers to the discrimination on the basis of perceived or actual ability due to a prejudiced attitude which prefers or values the “normal”, species typical, over the abnormal (Campbell, 2009, p. 5). The unilateral focus on what disabled people are lacking when compared to the typical population, and the value given to certain skills and behaviors in accordance with what is “normal”, inevitably leads to the subordination of disabled people. "Disability then is cast as a diminished state of being human”. (Campbell, 2001, p. 44). In line with such thoughts, I have attempted to present the topics and findings of this study in a way that is in keeping with respect and dignity. For example, I have endeavored to avoid value-laden words, such as “deficit” and “dysfunction” whenever possible. However, this was not always feasible when describing certain findings, theories and models.

5.0 RESULTS

5.1 General Overview of Included Studies

Six longitudinal studies examine prospective associations of interpersonal victimization and ADHD symptoms in children, adolescents and young adults. Three of the studies look exclusively at the potential effects of peer victimization (Ji et al., 2019; Murray et al., 2020; Singham et al., 2017), while one study (Stenseng et al., 2016) combines peer victimization with two other variables (peer dislike and peer discord) into one variable of peer rejection. Two studies examine child maltreatment. One of the studies (Jimenez et al., 2017) combines child maltreatment with household dysfunction into one variable of adverse childhood experiences, while the other study examines child maltreatment alone and in combination with other victimization experiences (Stern et al., 2018). Two of the studies are twin studies. None of the studies were published prior to 2016.

5.1.1 Samples

The study samples have been drawn from the UK (Stern et al., 2018; Singham et al., 2017), Norway (Stenseng et al., 2016), Switzerland (Murray et al., 2020), China (Ji et al., 2019) and the US (Jimenez et al., 2017). The subjects are children and adolescents up to young adulthood and range in age from

Table 1 Overview of included studies

Author (year)	Aim	Sample	Designs and models	Measures	Co-variables	Results
Ji et al. (2019)	Test possible bidirectional effects between attention problems and peer aceptance, rejection and victimization. And explore potential sex group differences.	N = 2157 pupils ages 9-16 , from 14 elementary schools in urban city in eastern China, participating in the Longitudinal Study of Chinese Children and Adolescents. (China).	Longitudinal study. Peer relations were assessed annually from age 9-16. Attention problems were assessed at age 9 and 15. Latent growth model & autoregressive cross-lagged panel model to explore bidirectional effects. Multi-group models to test gender differences.	BIDIRECTIONAL VARIABLES: ***Peer relations*** (measured at age 9, 10, 11, 12, 13, 14, 15 and 16.): • *Peer victimization*: The Multidimensional Peer Victimization Scale, (self-report, ordinal, valid and reliable). • *Peer Acceptance and rejection:* Standard sociometric procedures (Peers, Interval, valid, results standardized.) ***Attention problems*** (measured at age 9 and 15.): Child Behavior Checklist (CBCL. (Mother report, ordinal, valid and reliable).	Child gender and age, sibling status, parental education level and family income.	<u>Latent Growth Model:</u> • Found all aspects of peer relation (acceptance, rejection and victimization) trajectories predicted attention problems at age 15. Higher intercept and less of a decline in slope of victimization between age 9 and 15, was associated with attention problems at age 15. • The latent intercepts and slopes of peer relation trajectories (except slope of peer acceptance) were found to explain 22% of the variance in attention problems at age 15. <u>Cross-Lagged Panel Model:</u> • Found victimization at age 12 did not predict attention problems at age 15. Only peer rejection at age 12 predicted attention problems at age 15 (r = 0.09). Peer rejection at age 12 was found to mediate the stability of attention problems between age 9 and age 15.
Jimenez et al. (2017)	Assess the extent to which ACEs (adverse childhood experiences) are independently associated with ADHD at age 9.	N = 1572 children age 5-9, from single parent households in 20 cities, in Fragile Families and Child Well Being cohort. (USA).	Secondary analysis using publicly available data from Fragile Families and Child Wellbeing Cohort Study. Used data from year 5 and year 9 follow-up. Logistic regression model.	PREDICTOR VARIABLE: ***Adverse childhood experiences*** (measured age 5 and 9): • Maltreatment: Psychological aggression: CTS-PC Neglect: CTS-PC and mother confirmed contact w/ Child Protective Services because of neglect Physical abuse: CTS-PC and mother reported contact w/ Child Protective Services because of physical abuse Sexual abuse: mother reported contact w/ Child Protective Services because of sexual abuse • Household dysfunction: Maternal depression, substance use, incarceration of parent, witnessing domestic abuse OUTCOME VARIABLE: ***Received ADHD diagnosis*** (measured age 5 and 9)***:*** Mothers were asked the question: «Has a doctor or health care professional ever told you that the child has attention deficit disorder or ADHD?» Categorical.	Child gender, mother's race, ethnicity, impulsivity and education level, parental relationship status at time of birth, family poverty status.	• Did not find statistically significant association between number of ACE (child maltreatment and household dysfunction) before age 5 and receiving ADHD diagnosis by age 9. • Found association between ACE occurring between age 5 and 9 and receiving ADHD diagnosis by age 9 when controlling for ACEs and ADHD diagnosis occurring before age 5. Increased number of ACE increased AOR for receiving diagnosis. 1 ACE = AOR: 1.9 (95% CI 1.2-3.0); 2 ACEs: AOR = 2.1 (95% CI 1.2-3.8); 3 or more ACEs: AOR: 2.2 (95% CI 1.1-4.3). Most common ACEs: substance abuse, neglect, maternal depression
Murray et al. (2020)	Provide evidence on whether ADHD symptoms impact bullying victimization/ perpetration levels and vice versa».	N = 1526, children ,age 11-17 from 56 schools in Zurich. (Switzerland)	Longitudinal study. Measured bullying and ADHD at ages 11, 13, 15 and 17. Autoregressive latent trajectory model with structural residuals to explore bidirectional effects.	BIDIRECTIONAL VARIABLES ***Peer victimization:*** Zurich Brief Bullying Scales, (ZBBS). (Self-report, ordinal, measured age: 11, 13, 15, 17,) ***ADHD symptoms*** **:** • 4 questions from the the Social Behavior Questionnaire (SBQ). (Self-report, measured age: 13,15,17) • 8 items from the SBQ (Teacher report, ordinal, measured age: 11, 13, 15). Unknown if instrument is validated for ADHD.	Child gender.	• Found prospective association between victimization at 11 and inattention at 13 (β = 0.16) according to teacher report and H/I according to teacher report (β = 0.13). • Found association for girls but not boys when association of victimization age 11 and attention age 13 was studied separately for boys/girls. • Did not find association between victimization at age 13 and inattention at age 15. • Found <u>negative</u> association between victimization at age 13 and H/I at age 15 according to teacher report (β= - 0.10). • No association between victimization and inattention according to self report.
Singham et al. (2017)	«Characterize the concurrent and longitudinal contribution of exposure to bullying to mental health in childhood and adolescence using a twin differences design to strengthen causal inference.»	N = 11,108 twins, age 11-16 drawn from the Twins Early Development Study, a population-based cohort recruited from population records of births in England and Wales between January 1, 1994, and December 31, 1996. (UK)	Twin, cohort study. Exposure (victimization) measured at age 11 and 14. Outcome (mental health) measured at age 11 and 16. Structural equation model (phenotypic estimates) and ordinary least square through origin regression (DZ and MZ estimates). Three sets of analysis: concurrent, 2 yrs and 5 yrs after exposure. Three types of analysis: unadjusted phenotypic estimate, DZ same sex twin differences and MZ twin differences.	PREDICTOR VARIABLE: ***Peer victimization:*** The Multidimensional Peer Victimization Scale (self-report, measured age: 11 and 14, ordinal, valid and reliable). OUTCOME VARIABLE: ***ADHD symptoms:*** • *Hyperactivity*: subscale of the SDQ (5 items), (parent-report, measured age: 11 and 16 and child-report, measured age: 11). • *Inattention*: subscale of the Conners Parent Rating Scales - Revised (9 items), (Measured age: 11 and 16). • *Hyperactivity-impulsivity*: Conners parent (9 items), (Measured age: 11 and 16). A total score for Connors parent was also computed based on the 9 items for each dimension (18 items in total). Nominal, valid and reliable.	Child gender, genetics.	• Found small, temporary effects of victimization on inattention (β=0.037) and hyperactivity (β=0.094) when controlling for genetics. • No significant association between peer victimization and parent-rated hyperactivity, hyperactivity/impulsivity or total ADHD score. • Phenotypic associations decreased over time but all phenotypic associations remained significant over 5 year time frame. • Prospective associations did not survive twin control analysis.
Stenseng et al. (2016)	Test «the hypothesis that symptoms of ADHD predict increased peer rejection and, reciprocally, that peer rejection predicts increases in ADHD symptoms.»	N = 995 children, age 4-8, taking part in two birth cohorts of children born between 2003-2004 of parents living in Trondheim. (Norway).	Cohort study. ADHD and peer relations measured at age 4, 6 and 8. Autoregressive cross-lagged panel model to explore bidirectional effects.	<u>BIDIRECTIONAL VARIABLES</u> ***Peer rejection:*** Teacher Report Form (Achenbach), 3 questions (child not liked, gets teased, doesn't get along with other children), (Teacher-report, ordinal, measured age: 4,6 and 8). ***ADHD symptoms:*** Preeschool Age Psychiatric Assessment/ Child and Adolescent Psychiatric Assessment (PAPA/CAPA), (Parent-report, measured age: 4, 6 and 8)	Child gender.	• Found more peer rejection at age 4 predicted more ADHD symptoms at age 6 (β = 0.20). • Did not find similar associations were between age 6 and age 8. • If ADHD symptoms were separated according to subtype, study found that more peer rejection at age 4 predicted more hyperactivity/impulsiveness (β = 0.20) and inattentiveness (β = 0.12) at age 6 and more peer rejection at age 6 predicted more H/I and IA at age 8 (β = 0.14 for both).
Stern et al. (2018)	Investigate the associations in childhood and young adulthood between exposure to abuse/ neglect/ polyvictimization and ADHD, when controlling for confounders.	N = 2232 twins from birth cohort born 1994-1995 in England and Wales, Environmental Risk Longitudinal Twin Study. Teenage mothers over selected to replace selective drop out of high risk families. (UK)	Twin, cohort study. ADHD and victimization measured at age 5, 7, 10, 12 and 18. Logistic regression model to examine associations between abuse/neglect and ADHD diagnoses in childhood and young adulthood. Linear regressions to examine group differences. Autoregressive cross-lagged panel model to explore bidirectional effects. Control for familial confounding by examining twins difference scores using continuous variables to maximize variation in measures.	<u>BIDIRECTIONAL VARIABLES</u> **Victimization:** ***Victimization in childhood*** *(physical and sexual abuse by an adult, emotional abuse and neglect, physical neglect, bullying by peers, domestic violence)*: Clinical protocol from MultiSite Child Development Project (Mother-report, child age 5, 7, 10 and 12). Expanded at age 7,10 and 12 to include questions if child had ever been intentionally harmed physically or sexually by an adult or been in contact with welfare agency. Inter-rater agreement. ***Childhood polyvictimization:*** *Summed experiences of victimization* (3 categories: exposure to zero, one, two or more types of victimization). ***Victimization in adolescence*** *(Abuse/neglect, peer/sibling victimization and family violence):* JVQ, clinical interview, 45 questions (measured age 18) ***Adolescence polyvictimization:*** Summarizing all seven forms of victimization **ADHD:** ***Childhood ADHD*** (measured at age 5, 7, 10 and 12): Rutter Child Scales (mother and teacher reports, 18 symptoms), cut off more than 6 symptoms (2 symptoms co-endorsed). Positive childhood ADHD diagnosis if positive at age 5, 7, 10 and 12. **Young Adult ADHD:** DSM IV, Diagnostic Interview Schedule). Private structured interviews for diagnosis according to DSM-5. (Measured age: 18).	Child gender, parent SES, child IQ at age 5, youth IQ at age 18, Conduct Disorder (parent and teacher report), mother's depression, youth mental health at age 18 (DSM IV, Diagnostic Interview Schedule), genetics.	• Found association between child maltreatment and ADHD in childhood (β=0.23), association was reduced when controlling for sex, age 5 IQ and SES (β=0.13) and became non-significant when controlling for Conduct Disorder. • Did not find support for child maltreatment being a risk factor for ADHD in young adulthood. • ADHD was associated with poly-victimization in childhood and adolescence. Association between childhood ADHD scores and poly-victimization scores was explained by genetic factors. But association between adolescent ADHD scores and poly-victimization scores was partially environmental.

four to eighteen years old. All the studies, with one exception (Stenseng et al., 2016), have samples larger than 1000. Four of the samples are from urban populations (Ji et al., 2019; Jimenez et al., 2017; Murray et al., 2020; Stenseng et al., 2016). Two of the samples recruited their subjects via schools (Ji et al., 2019 and Murray et al., 2020). One was recruited through a city-based public health service (Stenseng et al., 2016), and one was a stratified sample, oversampled for unmarried mothers (Jimenez et al., 2017). Two studies were drawn from the same population cohort of twins in England and Wales, the Twins Early Development study (Stern et al., 2018; Singham et al., 2017). One of these studies oversampled for teenage mothers (Stern et al., 2018). The two studies that used oversampling for single and young mothers examined the prospective associations of child maltreatment and childhood adverse experiences (Stern et al., 2018; Jimenez et al., 2017).

5.1.2 Measures of Interpersonal Victimization

The assessment and measurement of victimization vary widely amongst the studies. Some studies use multiple informants and clinical interviews, observations and surveys (Stern et al., 2018) while other studies used three questions to the pupil's teachers (Stenseng et al., 2016). The two studies regarding child abuse and adverse childhood experiences seem to have more extensive evaluations of victimization than the studies regarding peer victimization. One study used mothers as their informant for adverse childhood experiences (Jimenez et al., 2017). The other study used parents, self-reports and observation by interviewer (Stern et al., 2018).

The studies regarding peer victimization mostly used surveys based on self-report. Two of the studies on peer victimization (Ji et al., 2019 and Singham et al., 2017) used the Multidimensional Peer Victimization Scale (MPVS) (Mynard & Joseph, 2000) that uses self-reports on 16 items to assess various forms of peer victimization, such as physical, verbal and relational victimization and property destruction, although one study apparently excluded the two questions on property destruction (Ji et al., 2019). One study used the Zurich Brief Bullying Scale which was developed for a larger research project the study was a part of (Murray et al., 2021). This is a similar self-report survey that measures the same four types of peer victimization as mentioned above in the MPVS. The fourth study on peer victimization (Stenseng et al. 2016) used teacher reports on three items from the Teacher Report Form (Achenbach & Rescorla, 2000).

5.1.3 Measures of ADHD

ADHD was assessed both as a dimensional construct (Stenseng et al., 2016; Murray et al., 2020; Ji et al., 2019; Singham et al., 2017) and as a categorical construct (Jimenez et al., 2017) with one study utilizing both (Stern et al., 2018). Most studies used various diagnostic instruments based on DSM IV to measure ADHD symptoms, such as the Children's Behavior Checklist (Ji et al., 2019), the Preschool Age Psychiatric Assessment/Child and Adolescent Psychiatric Assessment (Stenseng et al., 2016), the Connors scales and the Strengths and Difficulties Questionnaire (Singham et al., 2017), and the Rutter Child Scales (Stern et al., 2018). However, one study used eight items regarding attention and hyperactivity/impulsivity from the Social Behavior Questionnaire for the teacher report and four items, from the same survey, for self-report (Murray et al., 2020). This questionnaire is, seemingly, not based on diagnostic criteria. One study measured ADHD based on a question to the mother regarding whether or not she had ever been told by a health care professional that her child had ADHD (Jimenez et al., 2017). Three of the studies relied solely on parents for information regarding ADHD symptoms/ diagnoses (Ji et al., 2019; Jimenez et al., 2017; Stenseng et al., 2016); one study used teachers and self-report (Murray et al., 2020); one used parent and self-report (Singham et al., 2017); and one used parent, teacher, self-report and co-informant (Stern et al., 2018).

5.1.4 Co-variables

The only co-variable that was consistently controlled for across all the studies was gender. In fact, in two of the studies' this was the only co-variable (Stenseng et al., 2016; Murray et al., 2020). In addition to gender, two studies controlled for genetic factors (Singham et al., 2017; Stern et al., 2018), while three studies included such co-variables as family sociodemographic factors and other factors related to the child or parent (Jimenez et al., 2017; Ji et al., 2019; Stern et al., 2018). All three of these studies included the families'd financial situation as a co-variable and two included parental educational level (Jimenez et al., 2017; Ji et al., 2019). Otherwise, the variables controlled for varied markedly.

5.1.5 Designs and Statistical Models

All the studies were longitudinal, although they varied in timeframe from 4 to 13 years. They also varied in their number of measurement waves, with some studies utilizing the minimum of two waves (Jimenez et al., 2017) and others including as many as six waves (Ji et al., 2019). The interval between waves also differed, although most fell between 1-2 years. Some studies measured all variables at every wave (Jimenez et al, 2017; Stenseng et al., 2016), while most of the studies varied the number

and time of measurements to a greater or lesser extent, with the result that the number of associations are not necessarily strictly related to the number of waves and do not necessarily cover the whole timeframe.

The studies chosen have all used statistical models, though they vary in type and complexity. Three of the studies explored association using several different types of models (Ji et al., 2019; Stern et al., 2018; Singham et al., 2017) while three studies relied on one type of model. Logistic regression models, which are usually used when the outcome variable is dichotomous, were used by two studies (Jimenez et al., 2017; Stern et al., 2018). Otherwise, various types of models were used, such as autoregressive, cross-lagged panel models (Stenseng et al., 2016; Stern et al., 2018; Ji et al., 2019), an autoregressive, latent trajectory models with structural residuals (Murray et al., 2020), a growth curve model (Ji et al., 2019), unspecified structural equation model and an ordinary least square through origin regression model, which is a linear regression model without the intercept (Singham et al., 2017).

5.2 Polyvictimization and Adverse Experiences Involving Family

Jimenez et al. (2017) examined the association between adverse childhood experiences (ACEs) and receiving an ADHD diagnosis in an urban, birth cohort of 1,572 children in the US, oversampled for unwed mothers. ACEs and received ADHD diagnosis was assessed in two waves, at age 5 and 9. At age 5 data was collected from approximately 84% of the baseline sample while at age 9 about 74% of the original sample remained. The study does not contain further information regarding attrition analysis. ACEs included psychological aggression, physical abuse, neglect, sexual abuse, maternal depressive symptoms, maternal substance use, violent treatment by a caregiver, and incarceration of the father.

Information about child maltreatment was gathered during in-home interviews when the child was 5 and 9 years old, using both a question about Child Protective Services (CPS) involvement for neglect, physical abuse and/or sexual abuse and through the Conflict Tactics Scale: Parent Child Version (CTS-PC) (Straus et al., 1998). Psychological aggression was based solely on the CTS-PC and sexual abuse was based solely on CPS involvement for sexual abuse. In addition, mothers were asked about their own depressive symptoms, substance abuse and domestic abuse in the past year and whether the child's father was currently in jail. The ACE variable was grouped into the categories 0, 1, 2 and 3 ≥ ACEs. ADHD diagnosis was coded as yes/no based on asking the mother if a health care professional had ever told them that the child had «attention deficit disorder or ADHD».

In the study sample, 41% of the children had experienced at least one ACE prior to age 5, and 42% had experienced some form of ACE between ages 5 and 9. The most commonly occurring ACEs were substance abuse (11% before age 5 and 16% between age 5 and 9) and neglect (11% before age 5 and 11% between age 5 and 9), closely followed by maternal depressive symptoms (11% and 10%) and physical abuse (9%). Sexual abuse was the most infrequently experienced form of ACE (0.6 - 0.9%). An estimated 10.6% of the children had received an ADHD diagnosis by the age of 9 years.

The authors estimated the odds for the outcome variable (mother-reported ADHD diagnosis) based on the two predictor variables (ACEs before age 5 and ACEs between the age 5 and 9) using both adjusted and unadjusted logistic regression models. In the unadjusted model, they found a statistically significant association between 2 ACEs and 3 ≥ ACEs before age 5 and 1, 2 and 3 ≥ACEs between ages 5-9, and mother-reported ADHD diagnosis at age 9.

The authors subsequently adjusted for potential confounders, including child gender, race and ethnicity, maternal education and parent relationship at baseline, maternal impulsivity and reported ADHD diagnosis at age 5 as well as family income at age 9. The adjusted ORs (AOR) between ACEs before age 5 and a parent-reported ADHD diagnosis were then considerably reduced and the results were no longer statistically significant (AOR, 1; 95% CI, 0.6-1.6; AOR, 1.5; 95% CI, 0.9-2.6 and AOR, 1.6; 95% CI, 0.8-3.2). However, though reduced, the association between ACEs between ages 5 and 9 and mother-reported ADHD diagnosis by age 9, remained statistically significant (AOR, 1.8; 95% CI, 1.2-2.9; AOR, 2.1; 95% CI, 1.3-3.6 and AOR, 2.2; 95% CI, 1.1-4-1).

In other words, children who had more than one adverse experience between the ages of 5 and 9 had a higher risk of having received an ADHD diagnosis by the age of 9 than children without such experiences. The risk increased with the number of adverse experiences, while adverse experiences prior to the age of 5 did not increase the risk of diagnosis when controlling for potential confounders.

Stern et al. (2018) explored the association between ADHD in childhood and young adulthood and experiences of abuse/neglect using a cohort study of 2,232 twins. Home visits were conducted when the children were 5, 7, 10, 12 and 18. In addition, questionnaires were sent to the childrens' teachers and participants selected two co-informants when they were 18 years old.

Exposure to various forms of victimization was measured at all waves. At ages 5, 7, 10 and 12 years, a standardized clinical interview of primary caregivers and observation assessed participants' exposure to sexual or physical abuse by an adult; emotional abuse and neglect, domestic violence and bullying by peers. Physical and sexual abuse of the child by an adult was estimated using the standardized clinical protocol from the MultiSite Child Development Project (Dodge, Bates, & Pettit, 1990;

Lansford et al., 2002) at age 5. At consecutive interviews (7, 10, 12) this was expanded to include additional contexts for child harm. Physical neglect, emotional abuse and neglect was based on interiews and observations during home visits (age 5, 7, 10 and 12). At age 7, 98% participated, at age 10, 96% participated, at age 12, 96% participated and at age 18, 93% partcipated. There was no difference in regards to SES between those partcipating at age 18 and those at baseline. Analysis was restricted to those 2040 individuals who had submitted data on ADHD in childhood and adulthood.

Peer victimization was assessed using both mother and child reports at ages 7 and 10. Mothers were also asked about experiences of domestic violence when the child was 5, 7 and 10 using the Conflict Tactics Scale. When the twins were 18, they were interviewed separately using an adapted version of the Juvenile Victimization Questionnaire 2nd revision (JVQ-R2) about adverse experiences from the age of 12 (Hamby et al., 2011). The interview covered peer/sibling victimization, internet/mobile phone victimization, crime victimization, sexual victimization, maltreatment, neglect and family violence. All information regarding victimization was gathered into a dossier for the individual participants. Experiences of victimization were rated by severity using a six-point scale and then grouped into three classes: no exposure, some exposure and severe exposure.

A childhood diagnosis of ADHD was assessed using parent and teacher ratings of 18 symptoms of inattention and hyperactivity-impulsivity according to the DSM IV and the Rutter Child Scale (Rutter et al., 1970). For criteria to be met, six or more symptoms in the previous six months had to be reported by a parent or teacher with at least two symptoms endorsed by the co-informant. Participants were considered to have childhood ADHD if they met the criteria for ADHD at age 5, 7, 10 or 12. At 18, co-informants and private structured interviews with participants regarding 18 symptoms of inattention and hyperactivity-impulsivity, according to the DSM V, were used to evaluate an ADHD diagnosis. In total, 12% met the criteria for childhood ADHD (71% male), while 8.1% met the criteria for ADHD at age 18 (52% male). Amongst children with ADHD, 47.8% had an additional comorbid conduct disorder. The correlation between ADHD in childhood and young adulthood was $\beta = 0.41$.

A majority of the children (73%) had not experienced severe victimization by the age of 12, while 20.7% had only one experience, with 6.3% having two or more experiences of severe victimization. In adolescence, 64.8% had not experienced severe victimization, while 19.2% had had one experience and 15.9% two or more experiences (of severe victimization).

The authors found an association between abuse/neglect in childhood—which included physical, sexual and emotional abuse and physical and emotional neglect—and ADHD in childhood. This

association extended to other forms of victimization. Findings were similar when they separated ADHD symptoms into the inattention and hyperactivity/impulsivity subscales. The following findings were without co-twin analysis, which was only included when examining polyvictimization and ADHD, which will be described in a later section.

On average, children with ADHD had experienced more abuse/neglect than children without ADHD. The same was true for adolescents. Amongst children with ADHD, 13.8% had experienced severe abuse/neglect, and 27.7% moderate abuse/neglect, while in the group without ADHD, 6.4% had experienced severe abuse/neglect, with 17.6% experiencing moderate abuse/neglect.

Children that had been exposed to any abuse/neglect had a higher risk of meeting diagnostic criteria of ADHD than children who had not been exposed to abuse (OR 1.75, 95% CI 1.42-2.16) and this risk increased with severity of abuse/neglect (moderate abuse/neglect: OR 2.02, 95% CI 1.43-2.86, $p < 0.001$, severe abuse neglect: OR 2.78, 95% CI 1.72-4.48, $p < 0.001$).

When controlling for sex, IQ at age 5 and SES, the risk between any abuse/neglect and ADHD in childhood was reduced (AOR 1.37, $p < 0.05$) and when maternal depression was added as a co-variable, it was further reduced (AOR 1.31, $p < 0.05$). Similarly, when controlling for SES and age 5 IQ, in addition to sex, the overall association between childhood abuse/neglect and childhood ADHD was reduced from $\beta = 0.23$ ($p < 0.001$) to $\beta = 0.13$ ($p < 0.01$).

The authors found that abuse/neglect was concentrated among those with comorbid ADHD and conduct disorder. Amongst the children with ADHD alone, 7% had experienced severe abuse/neglect (which is a similar number to the non-ADHD group where 6.4% had experienced severe abuse/neglect). Amongst children with comorbid ADHD and conduct disorders, 21.1% had experienced severe abuse. Interestingly, this number was equally high (20.9%) in children with conduct disorder issues but no ADHD diagnosis. When controlling for sex, age 5 IQ, SES and conduct disorder, the association between abuse/neglect and ADHD in childhood became non-significant ($\beta = 0.02$, $p > 0.05$).

When examining prospective associations between childhood abuse/neglect and ADHD at age 18, using a cross-lagged panel model and controlling for sex, there were no significant results ($\beta = 0.09$, $p = 0.115$). In other words, abuse/neglect in childhood did not increase the risk of ADHD symptoms as a young adult.

Stern et al. (2018) also explored the relationship between ADHD and polyvictimization. In childhood, they found a modest association between levels of polyvictimization and ADHD symptom scores (r =

0.13 $p < 0.001$). However, this association disappeared when comparing MZ twins ($r = 0.07$, $p > 0.101$) indicating that the association was accounted for by familial confounding. In contrast, the association between polyvictimization and ADHD symptom scores in adolescence/young adulthood remained significant after controlling for genetics ($r = 0.17$, $p < 0.001$), indicating that this association is partly environmentally-driven.

Table 2. Findings regarding family- and polyvictimization.

Study	Victimization Measurement	Victimization time frame	ADHD measurement	Concurrent or Prospective Association	Unadjusted Association	Adjusted Associations	Variables adjusted for
Jimenez et al. (2017)	Age 5, ACE = 1 (parent-report)	Prior 1-4 yrs. (Varied for different ACEs.)	ADHD diagnosis age 5-9 (parent-report)	Prospective	No significant association.	No significant association.	Child gender, mother's race, ethnicity, impulsivity and education level, parental relationship status at time of birth, family poverty status.
	Age 5, ACE = 2 (parent-report)		ADHD diagnosis age 5-9 (parent-report)	Prospective	OR 2.1 (95% CI,1.3-3.3)	No significant association.	Child gender, mother's race, ethnicity, impulsivity and education level, parental relationship status at time of birth, family poverty status.
	Age 5, ACE ≥ 3 (parent-report)		ADHD diagnosis age 5-9 (parent-report)	Prospective	OR 2.6 (95% CI,1.5-4.7)	No significant association.	Child gender, mother's race, ethnicity, impulsivity and education level, parental relationship status at time of birth, family poverty status.
	Age 9, ACE =1 (parent-report)	Prior 1-4 yrs. (Varied for different ACEs.)	ADHD diagnosis age 5-9 (parent-report)	Concurrent/ Prospective	OR 1.9 (95% CI,1.3-2.9)	AOR 1.9 (95% CI, 1.2-3.0)	Child gender, mother's race, ethnicity, impulsivity and education level, parental relationship status at time of birth, family poverty status. Diagnosis received by age 5. ACe prior to age 5.
	Age 9, ACE = 2 (parent-report)		ADHD diagnosis age 5-9 (parent-report)	Concurrent/ Prospective	OR 2.4 (95% CI,1.5-3.8)	AOR 2.1 (95% CI, 1.2-3.8)	Child gender, mother's race, ethnicity, impulsivity and education level, parental relationship status at time of birth, family poverty status. Diagnosis received by age 5. ACe prior to age 5.
	Age 9, ACE ≥ 3 (parent-report)		ADHD diagnosis age 5-9 (parent-report)	Concurrent/ Prospective	OR 3.1 (95% CI,1.8-5.4)	AOR 2.2 (95% CI, 1.1-4.3)	Child gender, mother's race, ethnicity, impulsivity and education level, parental relationship status at time of birth, family poverty status. Diagnosis received by age 5. ACe prior to age 5.
Stern et al. (2018)	Age 5, 7, 10, 12 Severe child abuse/ neglect (parent-report)	Age 0-12	Childhood ADHD diagnosis (Age 5, 7, 10 and 12) (parent- and teacher report)	Concurrent	OR 2.78 (95% CI, 1.72-4.48)	AOR 1.37 (95% CI, 0.97-2.64)	Sex, IQ, SES
	Age 18 Severe child abuse/ neglect (self-report)	Age 12-18	Age 18 ADHD diagnosis (self-report and co-informant)	Concurrent/ Prospective	OR 3.86 (95% CI, 2.33-6.39)	AOR 3.59 (95% CI, 2.10-6.14)	Sex, IQ, SES
	Age 5, 7, 10, 12 Child abuse/neglect (parent-report)	Age 0-12	Age 5, 7,10, 12 ADHD symptoms (parent- and teacher-report)	Concurrent/ Prospective	$\beta = 0.23$ ($p<0.001$)	No significant association. ($\beta=0.02$, $p>0.05$)	Sex, IQ, SES, childhood conduct disorder
	Age 5, 7, 10, 12 Child abuse/neglect (parent-report)	Age 0-12	Age 18 ADHD symptoms (self-report and co-informant)	Prospective	No significant association.* ($\beta=0.09$ $p>0.05$)	No significant association. ($\beta=0.06$, $p>0.05$)	Sex, IQ, SES, childhood conduct disorder (in addition to cross-sectional overlap and stability of variables)
	Age, 5, 7, 10, 12 Polyvictimization (parent-report)	Age 0-12	Age 5, 7,10, 12 ADHD symptoms (parent- and teacher-report)	Concurrent/ Prospective	$r = 0.13$ ($p < 0.001$)	No significant association. ($r = 0.07$, $p > 0.101$)	Genetics.
	Age 18 Polyvictimization (self-report)	Age 12-18	Age 18 ADHD symptoms (self-report and co-informant)	Concurrent/ Prospective	$r = 0.18$ ($p < 0.001$)	$r = 0.17$ ($p < 0.001$)	Genetics.

* Controlling for cross-sectional overlap and stability of variables.

In summary, both studies found short-term associations between adverse childhood experiences pertaining to family and ADHD diagnosis in childhood. These associations were reduced when controlling for potential confounders such as SES. The association between polyvictimization and ADHD in childhood disappeared when controlling for genetics. However, the association remained in adolescents. When followed into early adulthood, Stern et al. (2018) no longer found an association between childhood abuse/neglect and ADHD.

5.3 Peer Victimization

5.3.1 Young Children

Stenseng et al. (2016) followed a Norwegian community sample of 995 children at baseline from the ages of 4 to 8 years old. ADHD symptoms were measured via parents' reports, and information on peer rejection was provided by their teachers when the children were 4, 6 and 8. Total attrition rates were 33%, but drop-out was unrelated to child mental health.

The variable of *peer rejection* was measured through three questions pertaining to the degree to which a child was not liked by other children, did not get along with other children and/or got teased a lot (using a three-point scale). The scale correlated moderately (r = 0.58) at age six with the Revised Olweus Victimization Scale, which is a self-report survey that measures physical, verbal and indirect bullying experiences (Kyriakides et al., 2006).

ADHD was assessed via parents with the Preschool Age Psychiatric Assessment when the child was 4 and 6, and with the Child and Adolescent Psychiatric Assessment when the child was 8 years old. Results were converted into quantitative scores. Overall, the study found the level of peer rejection increased from T1 to T3, while inattentiveness and hyperactivity/impulsivity increased from T1 to T2. From T2 to T3 inattentiveness remained relatively stable while hyperactivity/impulsivity decreased. Boys were more likely than girls to be rejected and to exhibit ADHD symptoms at age 4. The authors used an autoregressive, cross-lagged panel model to test reciprocal effects, but only prospective associations from peer rejection to ADHD are reported here. The full model fit was evaluated as excellent (*X^2= 118.66, (df = 73, p 0.001); RMSEA = 0.025; CFI = 0.962; TLI = 0.946; SRMR = 0.056*).

Findings indicated that peer rejection at age 4 predicted increased ADHD symptoms at age 6 ($\beta = 0.17$, $p < .01$), but no significant association was found between peer rejection at age 6 and ADHD symptom score at age 8.

Symptoms of ADHD were then separated to test for differences in patterns relating to the subtypes inattentive and hyperactive/impulsive. The findings suggest that the effect on symptoms of inattention and hyperactivity/impulsivity vary according to age. The prospective association of peer rejection on hyperactivity/impulsivity was stronger from age 4 to 6 ($\beta = 0.20$, $p < .01$) than from age 6 to 8 ($\beta = 0.14$, $p < .05$). The association between peer rejection and inattention remained relatively stable across ages ($\beta = 0.12$, $p < .05$ and $\beta = 0.14$, $p < .05$). This pattern fits the overall development of inattention and hyperactivity/impulsivity symptoms in the sample. The study did not find a significant difference when separating results by gender. There were no other co-variables in the study.

5.3.2 Middle Childhood and Adolescence

Ji et al. (2019) used data from the Longitudinal Study of Chinese Children and Adolescents (LSCCA) that was carried out from 2006 to 2015, to test for longitudinal, bidirectional associations between attention problems and peer acceptance, rejection and victimization during the transition from childhood to adolescence (ages 9 to 16). The sample consisted of 2,157 students (51.9% male) from the city of Jinan in eastern China and was representative of Chinese urban school children with respect to ethnicity, parental education and income.

Peer relations were measured every year from the age of 9 to the age of 16 using self-reports and peer nomination. Physical and relational forms of peer victimization were assessed through self-report, using 14 items adapted from the Multidimensional Peer Victimizations Scale (Mynard and Joseph, 2000). The mean scores for peer victimization at different ages ranged from 0.27 (SD 0.39) to 0.59 (SD 0.51) on a scale from 0-3. Attention problems were measured twice, at ages 9 and 15, using the Children's Behavior Checklist (Achenbach, 1991). The children's behavior was rated by mothers on a 3-point Likert scale from 0 = *not true* to 2 = *very true or often true*. The mean score for attention problems was 0.33 (SD 0.29, range 0.00 to 1.6) at age 9, and 0.42 (SD 0.36, range 0.00 to 1.71) at age 15. There was moderate stability for attention problems between 9 and 15 ($r = 0.41$). Co-variables included child gender, age and sibling status in addition to parental education level and family income.

Percentage of missing data on the different variables ranged from 1.3% to 38.4% with peer relations at 16 years having the highest rate of missing data. Missing data was related to parental education and correlated with observed and unobserved variables but correlations were all smaller than 0.38.

A latent growth model was used to estimate the contribution of the level (intercept) and changing rate (slope) of peer relations on attention problems at age 15. First, an unconditional model was used to appraise the intercepts and slopes of peer victimization, acceptance and rejection. For peer

victimization, the estimated intercept was 0.51 and the slope was - 0.03 which indicated a small, overall decline in victimization from 9 to 15.

After controlling for attention problems at age 9, attention problems at age 15 were regressed on the intercept and slopes simultaneously in order to gauge the unique contributions of peer victimization, acceptance and rejection trajectories on age 15 inattention. Model fit was evaluated as good (CFI = 0.92; X (234) = 1735.34, $p < 0.001$; RMSEA = 0.055 90% CI 0.052,0.057; TLI = 0.91).

In the latent growth model, the trajectories of peer relations (acceptance, rejection and victimization) were all found to predict attention problems at age 15. The latent intercepts and slopes of peer rejection and peer victimization and the latent intercept of peer acceptance (but not the slope) were found to explain 22% of the variance in attention problems at age 15. Attention problems at 15 were associated with the intercept and slope of peer victimization (intercept $\beta = 0.12$, $p = 0.002$ and slope $\beta = 0.13$, $p = 0.002$), indicating that a higher initial level of peer victimization and less of a decrease over time, was associated with more inattention at age 15.

A cross-lagged panel model was used to examine prospective, bidirectional associations between peer relations and attention problems. After estimating paths from attention difficulties to peer relations, significant paths from peer relations to attention problems at age 15 were considered. The former results are not reported here. In the crossed lagged panel model, peer victimization at age 12 did not predict attention problems at age 15. Only peer rejection at age 12 predicted attention problems at age 15 ($r = 0.09$). Peer rejection at age 12 was found to mediate the stability of attention problems between age 9 and 15. The study did not find any gender differences in their results.

A Swiss study (Murray et al., 2020) used data from the Zurich Project on Social Development from Childhood to Adulthood (z-proso) to explore whether bullying, victimization and/or perpetration affect ADHD symptoms and vice versa in young adolescents ages 11-17. The z-proso study is a longitudinal cohort from a socioeconomically and ethnically diverse sample of school children in Zurich, Switzerland. The total number of participants was 1483 for the self-report sample and 1526 for the teacher-rated sample at baseline. Total drop-out was 14% and analyses of attrition indicated that drop-out was related to ADHD status (OR 1.30).

The study used self-reported data on ADHD symptoms at age 13, 15 and 17 and teacher-reported symptoms of ADHD at age 11, 13 and 15 (Social Behavior Questionnaire, Tremblay et al., 1991) in combination with self-reports of bullying victimization and perpetration at age 11, 13, 15 and 17 (the

Zurich Brief Bullying Scale, Murray, Eisner et al., 2021). There was low to moderate correlation between teacher- and self-reports on ADHD symptoms.

Autoregressive latent trajectory models were fit with structured residuals to examine whether victimization levels at age 11, 13 and 15 impacted ADHD symptom levels at age 13, 15 and 17. A prospective association was found for victimization levels at age 11 and teacher reported inattention and hyperactivity/impulsivity symptoms at age 13 ($\beta = 0.16$, $p < 0.05$ and $\beta = 0.13$, $p < 0.05$). When examining this association by gender, they found the association increased for girls ($\beta = 0.20$, $p < 0.015$) while it decreased and became non-significant for boys ($\beta = 0.14$, $p = 0.062$). However, no significant prospective associations were found between victimization at age 13 and teacher-reported inattention at age 15 ($\beta = 0.02$, $p = 0.668$) and there were no significant prospective associations between victimization and self-reported ADHD symptoms at any age (13-15: $\beta = 0.03$ $p = 0.577$ and 15-17: $\beta= 0.07$ $p = 0.160$).

Singham et al. (2017) used a twin difference design to examine concurrent and longitudinal effects of exposure to bullying on the mental health of children and adolescents. The sample consisted of 11,108 twins (5,894 girls and 5,214 boys) drawn from the Twins Early Development Study, a population-based cohort of births in the UK between January 1994 and December 1996. Data collection took place at ages 11, 14 and 16 years. At age 11 and 14, children completed the multidimensional Peer Victimization Scale that consists of the subscales of Physical Bullying, Verbal Bullying, Property Attacks and Social Manipulation. Outcomes, which included various aspects of mental health such as depression, anxiety and ADHD symptoms, were measured at age 11 and 16. ADHD symptoms were measured using several instruments including the Inattention-Hyperactivity subscale of the Strengths and Difficulties Questionnaire (SDQ) (parent-reported version, and child-reported at age 11), and the Conners Parent Rating Scale-Revised with the subscales Inattention and Hyperactivity/Impulsivity. The number of twins with data for the various outcomes ranged from 11 108 to 4 706 (subsample at 14 yrs). No further analysis of attrition is given in the article.

The study estimated the relationships between childhood exposure to bullying and each outcome. Structural equation modelling was used for an unadjusted phenotypic estimate, while an ordinary least square through origin regression (RTO) was used for DZ and MZ twin estimates. Opposite sex DZ twins were excluded from DZ analysis. Within twin differences in outcome were regressed onto within twin differences in exposure to bullying. In addition, analyses controlling for prior twin differences were conducted. Analyses were done for concurrent associations at age 11, for 2-year longitudinal associations (between bullying exposure at age 14 and age 16 outcomes) and 5-year longitudinal

association (between bullying exposure at age 11 and outcomes at age 16). Moderation by sex and non-linear associations were also explored.

In general, all the phenotypic estimates, which looked at the whole group without controlling for twin status, found concurrent and prospective association between exposure to bullying and total mental health difficulties scores. The strength of these associations decreased when estimating DZ estimates and were even further reduced in MZ estimates, indicating the effects of shared environmental and/or genetic confounding on the associations.

Concurrent phenotypic associations between exposure to bullying and total parent-rated score on the Connors was β = 0.231 (95% CI, 0.210 - 0.253). The MZ estimates remained significant for the concurrent associations between exposure to bullying and child rated hyperactivity and inattention symptoms of the SDQ (β = 0.094, 95% CI, 0.042 - 0.147), but not parent-rated. In addition, inattention, but not hyperactivity/impulsivity based on parent-ratings on the Connors scale, remained significant in the MZ estimate (β = 0.037, 95% CI, 0.0003 - 0.072). These findings imply that exposure to bullying contributes to concurrent symptoms of ADHD (though the effects seem to be quite small).

Prospective associations depended on whether genetic effects were factored in. Phenotypic associations remained significant for the entire five-year period while MZ associations did not. Longitudinal phenotypic associations between exposure to bullying and total parent-rating on the Connors scale was β = 0.185 (95% CI, 0.151 to 0.221) after two years, and β = 0.189 (95% CI, 0.163 - 0.216) after five years. The MZ estimates of associations between bullying exposure and ADHD symptoms were all non-significant after two years (with 95% CI ranges for standardized betas of all measures encompassing zero). Some of the associations of the MZ estimates were significant at five years but did not survive additional control for prior twin differences. The findings that phenotypic estimates of associations were more long lasting than MZ estimated associations, suggests that causal effects of exposure to bullying on ADHD symptoms are more temporary than the confounded associations.

In summary, findings are somewhat mixed, but studies found some small associations between peer victimization and increased ADHD symptoms. However, there were also some findings of non-significant associations. When controlling for familial confounding concurrent associations were considerably reduced and prospective associations became non-significant.

6.0 DISCUSSION

6.1 Potential Bias in Studies

6.1.1 Measurement Bias

There is some variability in the use of instruments and sources of measurement int the included studies. Almost all of them use modified versions of instruments or use certain instruments to measure slightly different concepts or populations than what they have been developed for or in slightly different ways than they were intended. This presents some challenges to the evaluation of their psychometric properties. To what degree can we generalize from studies regarding reliability and validity of particular instruments on particular populations to the results obtained from modified versions on different populations?

Some studies used reliable instruments but with unknown validity. For instance, one study used a questionnaire (SBQ) with unknown validity for the measurement of ADHD (Murray et al., 2020). As the authors state in another article regarding the same cohort: «SBQ is not used for diagnostic purposes but to track developmental changes in normative samples. The focus has been reliability not validity» (Murray et al., 2019, p. 676). Although, reliability is an important aspect of measurement, if we don't know what it is we are measuring, it may be difficult to generalize from the study results to the common construct of ADHD. In general, the studies seem to place more emphasis on reliability, particularly internal consistency which tends to be exemplified by referring to Cronbach's α, than validity. Casper et al. (2015) argue that most measures of bullying have sacrificed validity in order to achieve reliability.

One study (Jimenez et al., 2017) does not actually measure ADHD symptoms but rather uses diagnosis received as the outcome variable. Measuring a received diagnosis is not the same as measuring actual symptoms. A received diagnosis may potentially be affected by multiple factors that are unrelated to symptom levels. Although the study attempted to control for one such factor (access to health care) by incorporated a question regarding health check-up in prior year, there are many other factors that may influence the receival of a diagnosis, such as additional behavior issues and involvement of school or social services. In addition, this measurement does not relate equally to the construct of ADHD over time, as rates of children receiving a diagnosis naturally increases with time and gradually encompass a greater proportion of children with ADHD. It is possible that the children who received a diagnosis between age 5 and 9, had ADHD before the age of 5 but had simply not yet received a diagnosis. Furthermore, it is conceivable that this is what led to both the increased rate of victimization and the

diagnosis between age 5 and 9. In general, clinical psychiatric diagnosis are perceived as unreliable measures (Josefiak & Berg-Nielsen, 2016). All in all, the use of such a proxy measurement might not be a valid and reliable measurement of actual ADHD symptoms and the study results are therefore vulnerable to measurement bias.

In their systematic review of child maltreatment measurment instruments, Saini et al. (2018), point out that there is a low level of evidence for most instruments and that no instrument seems to be superior across settings and population. In addition to the specific instrument, there is the issue of who is doing the reporting. Again, there is no gold standard and there is substantial debate about who gives the most accurate estimate. Parent-, child- and professional-reports often differ and all are prone to bias, so it is considered beneficial to use a multi-informant approach (Compier-de Block et al., 2017). The two studies regarding child maltreatment both used parent-report, though one of them supplemented this with child report at age 18. Heavy reliance on parent-report of child maltreatment may be a source of measurement bias.

Self-reports regarding peer victimization have many advantages, such as victims being able to privately report incidents that are not readily observable but they are also prone to bias (Casper et al., 2015). Most of the studies regarding peer victimization have used self-reports. One of the studies on polyvictimization used both self-report and parent-report but otherwise the studies have only used one informant which may make them vulnerable to measurement bias. In addition, one of the studies relied solely on teacher-report on peer-rejection. A substantial amount of negative social activity amongst children occurs when they are unsupervised and teachers tend to underestimate levels of victimization so this may also be a source of bias. Furthermore, several of the studies used the same rater on both predictor and outcome variables which, as previously described, may pose an additional risk for information bias.

In addition, a potential problem regarding the studies on polyvictimization is that they do not discriminate between various forms of victimization/ACEs. This leads to problems relating both to the difficulty in seperating potentially different effects of different forms of victimization and to whether particular aspects of certain forms of victimization may have distinct effects. For instance, child maltreatment may encompass physical abuse which may include head trauma or it may involve neglect which may include malnourishment, all or any of these aspects might be potential risk factors for ADHD. This contributes to confusion regarding what it is, exactly, that we are measuring the effect of?

6.1.2 Confounding

Overall, most of the studies have little control for confounding variables. In view of the amount of possible confounders in the relationship between victimization and ADHD, this is somewhat surprising. Although the two studies that examine child maltreatment have the most control for confounders, they have both omitted to include some arguably relevant factors. For example, there is no control for prenatal factors. One study includes substance abuse in parents as a predictive variable but does not control for FASD. In addition, one of the most common forms of ACEs in this study was maternal depression. In view of the potential genetic association between ADHD and depression/mood disorders (Brikell et al., 2020; Weber et al., 2011; Noorozi et al., 2019) this represents a potential for familial confounding in results. Furthermore, none of the studies have used parental ADHD as co-variables. Considering the apparent level of heritability in ADHD and the possible association between parental ADHD and maltreatment, one would think this would have been an interesting aspect to explore.

In general, there is little control for other diagnoses or issues such as mental health, learning difficulties, disabilities or behavioral problems in the studies. This is especially true of some of the studies regarding peer victimization where two of the studies had no other co-variables than child gender. Of all the studies, only one study had conduct problems as a co-variable. In line with previous findings, they found that the association between victimization and ADHD was concentrated among those children with comorbid behavioral disorders and that behavioral problems predicted later victimization (Stern et al. 2018). This suggests that such issues are important to control for.

Although controlling for confounders may be statistically difficult, it would benefit studies if they explicitly account for the existence of such factors and their possible effects on results.

6.1.3 Other Issues

Overall, very few studies met inclusion criteria and so the number of studies in this review is small. In addition, studies encompass diverse age groups, examine different forms of victimization and use different measurements, which makes comparisons challenging. Different types of victimization may have different effects at different ages. Four of the studies only measure one form of victimization. As several studies indicate there may be a dose response effect to victimization, restricting the study to only one type of victimization may obfuscate the effects of polyvictimization.

The limited number of studies makes the total picture vulnerable to the bias of individual studies, as each part has a larger impact on the whole. For instance, only one study examines peer rejection in the

age group 4-8. This makes it difficult to draw conclusions until findings have been replicated. Out of two of the studies examining child maltreatment and polyvictimization, one of the studies, as mentioned, used received diagnosis and not a measure of ADHD symptoms as outcome and so it is difficult to generalize to actual ADHD symptoms. If one excludes this study, there is only one remaining study examining child maltreatment and polyvictimization, so again, findings should be replicated before drawing conclusions.

Several of the studies had substantial levels of attrition and missing data. Two of the studies noted that attrition was related to predictor or outcome variables. However, several articles did not give information on attrition analysis or the numbers given regarding rates of attrition were difficult to interpret. In general, the articles often lacked explicit information, such as model fit values or design details, that would have been helpful in regards to evaluating studies and interpreting the findings. Furthermore, most studies have used standardized effect sizes, which, as Flora (2018) points out, are difficult to interpret and compare across studies (Flora, 2018, p.61). Standardized coefficients are based on sample estimates, such as standard deviations. These vary from study to study and therefore this complicates the process of comparing results.

6.2 Interpreting Results

As discussed in the previous section, results need to be interpreted with caution as there is considerable risk of bias. Moreover, the use of standardized coefficients complicates the interpretation of results. Whereas unstandardized coefficients relate directly to the variable in an intuitive manner, standardized coefficients are based on the rather technical and obscure concept of units of standard deviations (Flora, 2018, p.61). In addition, the studies are often missing important information, such as the potential score range, standard deviation or group means, which may help in the interpretation of effect sizes. Furthermore, effect sizes for non-significant results are sometimes not give.

Despite these limitations, a tentative analysis follows.

Only two studies look at victimization as risk factor for ADHD diagnosis as a categorical outcome and one of the studies does not actually measure ADHD symptoms (Jimenez et al., 2017). When looking at concurrent associations one study found that ACEs occurring between age 5-9 was associated with the receival of ADHD diagnosis age 5-9 (Jimenez et al., 2017). When looking at prospective associations of ACEs prior to age 5 and diagnosis received between ages 5-9, Jimenez et al. (2017), first found an

increased risk, but after adjusting for possible confounders there was no longer a significant association.

Stern et al. (2018) found that childhood maltreatment (age 0-12) increased the risk for ADHD both in childhood and at age 18 (Stern et al., 2018). However this was without adjusting for genetics or prior ADHD diagnosis in the prospective association. When adjusting for prior ADHD symptom levels using a cross-lagged panel with autoregressive effects they did not find a prospective association between child maltreatment and ADHD symptoms at age 18. This may indicate that child maltreatment is not a long-term risk factor for ADHD. On the other hand, this prospective association was estimated taking childhood ADHD symptoms into account. These childhood symptom levels may have been influenced by victimization experiences in childhood. However, the study found that the association between ADHD symptoms and polyvictimization in childhood was accounted for by genetics, so it is difficult to draw any firm conclusions.

When examining concurrent associations between polyvictimization and ADHD in childhood (age 0-12) Stern et al. (2018) found a modest association but this disappeared when they controlled for genetics. Similarly, Singham et al. (2017) did not find significant concurrent associations between peer victimization and total ADHD symptoms at age 11 when controlling for genetics. This indicates that the concurrent association between victimization and ADHD in childhood may be driven by genetic factors. However, Singham et al. (2017) did find a significant, though small, concurrent association between victimization and increased self-reported symptoms of hyperactivity and parent-reported symptoms of inattention. These findings imply that there may be small, concurrent effects on specific symptoms. Both the concurrent and the prospective associations between victimization and ADHD seem to be affected by familial confounding.

Stern et al. (2018) found that child maltreatment was particularly associated with conduct disorder. In fact, the association between maltreatment and ADHD in childhood disappeared when controlling for conduct disorder. This is in accordance with previous results (Ford et al., 2000; Clayton et al., 2018). In contrast, the association between victimization and conduct problems was unaffected by ADHD status. This may indicate that the association between victimization and ADHD is confounded by conduct problems, which is highly associated with ADHD. In addition, Stern et al. (2018) found that conduct disorders in childhood predicted later abuse/neglect.

Stern et al. (2018) also found find that polyvictimization did not increase the risk of ADHD diagnosis in childhood when controlling for genetics. If these findings are replicated it raises the question of why

Jimenez et al. (2017) found that the number of adverse childhood events predicted increased risk of receiving an ADHD diagnosis. One potential explanation regards the association between child maltreatment and behavioral issues. It would have been interesting to explore whether the association between maltreatment and receival of ADHD diagnosis is mediated by conduct problems.

The remaining four studies (Ji et al., 2019; Murray et al., 2020; Stenseng et al., 2016; Singham et al., 2017) examined peer victimization and ADHD on a dimensional scale. Again, only one study examined young children, and the focus was not exclusively victimization but rather peer rejection, so it is difficult to generalize to victimization alone. The study found a small prospective association between peer rejection and total ADHD symptoms between ages 4-6 but not between ages 6-8. However, when breaking down symptoms into two groups (inattention and hyperactivity/impulsivity) they found a small effect both between ages 4-6 and 6-8. There are several potential explanations for this. First of all, there was a much higher stability in ADHD symptoms between age 6-8 ($\beta = 0.79$) than 4-6 ($\beta = 0.59$) which may indicate that symptoms stabilize with age leaving less room for change. In addition, it may be that effects are related to specific symptoms and so do not have enough power to show up on combined symptom scales.

Several studies found different strengths of associations between victimization and inattention compared to hyperactivity/impulsivity. In early childhood Stenseng et al. (2016) found that peer rejection was more closely associated with impulsivity/hyperactivity than inattention. This difference disappeared between ages 6-8. The differences may be explained by developmental changes in children as they age. Alternatively, they may be due to the fact that attention problems could be difficult to observe in children before they start school where they are given tasks that require more attentional control.

Murray found a negative association between peer victimization and hyperactivity/impulsivity in adolescents. Considering that peer victimization seems to be associated with depression (Simmons et al, 2021; Stefanny et al., 2021) it would be interesting to examine if a negative association between peer victimization and hyperactivity/impulsivity is mediated or moderated by depression.

Although all of the studies tested for different patterns for gender, only one study found a difference in effects. Murray et al. (2020) found that the association between peer victimization at age 11 and teacher reported inattention at 13 was substantially larger for girls than for boys, in fact, it was non-significant for boys. This is in contrast to both Singham et al. (2017) and Ji et al. (2019) who studied similar age-groups. Hopefully, future studies will examine this further.

Table 3. Findings regarding prospective associations.

Note: Findings that mix concurrent and prospective associations are not included.

	Victimization Measurement	Victimization timeframe	Measure-ment Interval	ADHD	Association
Jimenez et al. (2017)	Age 5, ACE = 1 (parent-report)	Prior 1-4 yrs. (Varied for different ACEs.)	4 yrs	Age 5- 9 received diagnosis (parent-report)	No significant association.
	Age 5, ACE = 2 (parent-report)		4 yrs	Age 5- 9 received diagnosis (parent-report)	OR: 2.1 (95% CI,1.3-3.3) AOR: No significant association.
	Age 5, ACE ≥ 3 (parent-report)		4 yrs	Age 5- 9 received diagnosis (parent-report)	OR 2.6 (95% CI,1.5-4.7) AOR: No significant association
Stern et al. (2018)	Age 5, 7, 10, 12 Child abuse/neglect (parent-report)	0-12 yrs	6-13 yrs	Age 18 ADHD dimensional (self-report and co-informant)	No significant association ($\beta = 0.09$, $p > 0.05$)
Stenseng et al. (2016)	Age 4 peer rejection (teacher-report)	Unknown. (Concurrent?)	2 yrs	Age 6 ADHD dimensional (parent-report)	$\beta = 0.20$ ($p < 0.01$)
				Age 6 IN dimensional (parent-report)	$\beta = 0.12$ ($p < 0.05$)
				Age 6 H/I dimensional (parent-report)	$\beta = 0.20$ ($p < 0.01$)
	Age 6 peer rejection (teacher-report)		2 yrs	Age 8 ADHD dimensional (parent-report)	No significant association
				Age 8 IN dimensional (parent-report)	$\beta = 0.14$ ($p < 0.05$)
				Age 8 H/I dimensional (parent-report)	$\beta = 0.14$ ($p < 0.05$)
Murray et al. 2020	Age 11 peer victimization (self-report)	Unknown. (Concurrent?)	2 yrs	Age 13 ADHD dimensional (self-report)	No significant association ($\beta = 0.02$, $p > 0.05$)
				Age 13 IN dimensional (teacher-report)	$\beta = 0.16$ ($p = 0.002$) (girls: 0.20 $p = 0.015$, boys: non-significant)
				Age 13 H/I dimensional (teacher report)	$\beta = 0.13$ ($p = 0.039$) (Boys: 0.16. Girls: 0.07):
	Age 13 peer victimization (self-report)		2 yrs	Age 15 ADHD dimensional (self-report)	No significant association ($\beta = 0.02$, $p > 0.05$)
				Age 15 IN dimensional (teacher-report)	No significant association ($\beta = 0.02$, $p = 0.668$)
				Age 15 H/I dimensional (teacher report)	$\beta = -0.10$ ($p = 0.039$) (NB! Negative association.) (Boys: - 0.12. Girls: - 0.03.)
	Age 15 peer victimization (self-report)		2 yrs	Age 17 ADHD dimensional (self-report)	No significant association ($\beta = 0.07$, $p > 0.05$)
Ji et al. (2019) Cross-lagged panel model	Age 12 peer victimization (self-report)	Current semester.	3 yrs	Age 15 IN dimensional (parent-report)	No significant association
	Age 9 peer victimization (self-report)		6 yrs	Age 15 IN dimensional (parent-report)	No significant association
Singham et al. (2017)	Age 14 peer victimization (self-report)	Prior (1) year.	2 yrs	Age 16 ADHD dimensional (parent-report)	Unadjusted: $\beta = 0.185$ (95%CI, 0.14-0.21) Adjusted: No significant association
				Age 16 IN dimensional (parent-report)	Unadjusted: $\beta = 0.185$ (95%CI, 0.15-0.22) Adjusted: No significant association
				Age 16 H/I dimensional (parent-report)	Unadjusted: $\beta = 0.134$ (95%CI 0.10-0.17) Adjusted: No significant association
	Age 11 peer victimization (self-report)		5 yrs	Age 16 ADHD dimensional (parent-report)	Unadjusted: $\beta = 0.189$ (95%CI 0.16-0.22) Adjusted: No significant association
				Age 16 IN dimensional (parent-report)	Unadjusted: $\beta = 0.184$ (95%CI 0.16-0.21) Adjusted: No significant association
				Age 16 H/I dimensional (parent-report)	Unadjusted: $\beta = 0.149$ (95%CI 0.12-0.18) Adjusted: No significant association

Of the included studies examining peer victimization and ADHD symptoms in middle childhood/ adolescence only two explore concurrent associations. Ji et al. (2019) found that victimization trajectories between age 9-15 predicted age 15 inattention and Singham et al. (2017) find some small associations between peer victimization and ADHD symptoms at age 11, but, as previously mentioned, the strengths of the associations were considerably reduced when adjusting for genetics.

Overall, there is relatively little evidence to indicate that there are long-term prospective associations between peer victimization and ADHD symptoms in middle childhood and adolescence. Although, both Murray et al. (2020) and Singham et al. (2017) found some evidence for a small prospective association, Singham et al. (2017) found that it disappeared when they controlled for genetics and prior twin differences. This indicates that confounded effects may be longer lasting than potential causal effects.

6.3 Implications for Theory and Practice

So far, there does not appear to be substantial evidence for the threory that victimization leads to ADHD. What few sound studies we have that examine victimization as a risk factor for ADHD find relatively small associations between victimization and ADHD symptoms or no significant associations. When effects are measured over sufficient time, they seem to be temporary. There appears to be confounding affecting the association between victimization and ADHD. Familial confounding seems especially influential. When controlling for familial effects the association between victimization and ADHD symptoms disappears or is considerably reduced. This does not mean that a causal connection does not exist between victimization and ADHD but theories regarding a notable causal effect have not been confirmed by the few studies that have been done. Considering this, we should probably be careful about basing clinical practice on what is, so far, more of a hypothesis than an established fact.

Results seem to be more in line with the theory regarding self-regulation as a limited resource which is drained under challenging circumstances (Baumeister et al., 2007), than with theories that posit longterm, structural damage. In light of these findings, it might be fruitful to consider a model where victimization functions as a moderator that may temporarily increase the strength of ADHD symptoms, rather than as a causal factor.

Substantial symptoms of ADHD in someone who has experienced interpersonal victimization, that persevere after circumstances have changed, should probably not automatically be considered

symptoms of trauma. Considering the heightened frequency of victimization amongst those with ADHD, victimization experiences should not exclude an ADHD diagnosis. It is potentially problematic if mental health professionals refrain from giving an ADHD diagnosis to someone who fulfills criteria for ADHD simply because they have experiences of interpersonal victimization. It may keep people from receiving the correct accommodations. Berg et al. (2018) found that children with autism who experienced adverse childhood events received a later diagnosis than others. The possible detrimental effects of not getting the right help and support in a timely manner may be considerable.

Problems seem to arise when the presence of one condition excludes the presence of the other in the eyes of service providers. Just because you have ADHD that doesn't mean you are not also abused and just because you are abused that does not mean you do not also have ADHD. Due to the higher level of victimization experiences in individuals with ADHD, there is a higher-than-average chance that patients presenting with trauma related disorders have (undiagnosed) ADHD. A full diagnostic evaluation should therefore include an assessment for ADHD and other neurodevelopmental disorders (Dinkler et al., 2017).

Service providers should also be aware that children with ADHD, particularly those with behavioral problems, are at an increased risk of experiencing victimization and should screen for this possibility. Presenting such screening as routine based on statistical probability, rather than suspicion based on the idea that ADHD symptoms may be symptoms of abuse, may help to alleviate feelings of mistrust and misunderstanding on the part of parents.

If abuse and victimization does not lead to ADHD then claiming it might, has the potential to cause substantial damage. The idea that childhood trauma is being mistaken for ADHD adds to the stigmatization of parents of children with ADHD. Many of these parents have ADHD themselves and might already be marginalized and subjected to ableist attitudes and prejudice because they do not conform to «normal» behavior. By creating a culture of suspicion directed at parents of children with ADHD, society simultaneously denies children and adults proper, adequate help and support and places families under increased stress and duress.

It can be argued that the focus on the individual and trauma as the cause of ADHD symptoms subterfuges the social and cultural processes that stigmatize and marginalize individuals with these characteristics and place them at increased risk of victimization, thereby taking the effects of society and culture out of the equation (Hollomotz, 2012). It is no longer necessary to change the way society treats people with ADHD because it is not ADHD that is a risk factor for victimization, rather it is

victimization that is a risk factor for ADHD. There is no need for the majority to confront its attitudes towards, and treatment of, differently abled minorities because the majority's attitudes and treatment are not in any way implicated in the explanatory model. The association between ADHD and victimization is understood as an individual problem - thereby obscuring the role of society.

In general, there seems to be a lack of understanding in mental health care of how disability interacts with social and cultural processes. Donna Reeve (2000) has stated that this lack of understanding of the prejudices faced by disabled people may lead to oppressive rather than empowering experiences in mental health care. She suggests a form om «disability counceling» that is not limited to the personal and individual but is also fundamentally social and political. Such an approach would be based on a social model of disability, rather than a medical or pathological model, and aimed at empowerment.

6.4 Strengths, Limitations and Future Directions

6.4.1 Strengths and Limitations

To the best of the author's knowledge, this is the only review of studies that attempt to establish temporal order in the association between victimization and ADHD. As such, it offers unique insights into the relationship between interpersonal victimization and ADHD. Furthermore, although the number of studies is limited, the studies generally had reasonably large samples which gives a certain amount of validity to findings.

There are also some limitations. It is possible that relevant studies were missed. The reviewer spent a sconsiderable amount of time doing trial searches and looking over studies and their reference lists before the final search. However, final searches were only done in two databases and so relevant studies that were not contained in these database may have been missed. The review may have benefited from a more extensive search that including other databases such as ASSIA or Scopus. In addition, certain search terms were not used as they led to so many irrelevant results. The review was also limited to studies published in English or Scandinavian languages. These factors may further have increased the risk of missing some potentially relevant studies.

What is more, only studies published in peer-reviewed journal were included. Consequently, relevant but unpublished studies may have been missed. Moreover, the tendency to publish studies with positive results increases the risk of results being skewed. The small number of studies and lack of replicated findings suggests caution in interpreting results.

In addition, this is the author's first literature review and lack of prior experience may have resulted in mistakes. Some of these have already been mentioned but there may be others the author is not aware of. Furthermore, the reviewer has a limited background in statistics and cannot guarantee that important aspects have not been missed or misunderstood.

6.4.2 Future Direction

There is still a lot to learn about the relationship between interpersonal victimization and ADHD. There is a need for further research in order to examine if interpersonal victimization is a risk factor for ADHD or increased ADHD symptoms and if so, to what degree and for how long. Questions remain regarding what form of victimization may be a risk and at what age. If victimization is a risk factor, future studies need to untangle what aspects of victimization may be resposible for such an increased risk. The relationship between victimization and ADHD conceivably involves complex interactions among multiple factors. Future research should take this into consideration and avoid study designs based on simplistic causal assumptions. New statistical models such as growth curve models have the potential to offer more accurate insights into intra- and inter-individual change over time based on multiple variables (Duncan, T. & Duncan, S., 2009).

7.0 CONCLUSION

This study has examined if interpersonal victimization is a risk factor for ADHD. To answer this question empirical studies that attempted to establish temporality regarding the association between victimization and ADHD have been reviewed. Overall, there seems to be a paucity of studies that go beyond cross-sectional designs as only six studies met inclusion criteria.

Results based on these studies indicate that interpersonal victimization may be a small risk factor for a temporary increase in ADHD symptoms. However, there does not appear to be a strong relationship between victimization and ADHD symptoms. When studies control for familial confounding the association disappears or is substantially reduced. In addition, there seem to be several other potential confounding factors affecting the association between victimization and ADHD.

The small number of studies to date and the lack of control for possible confounding factors implies caution regarding causal inference. Subsequently, a similar caution is warranted in clinical practice.

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APPENDIX

A. Search Strings

PsychINFO search string:

1 exp Attention Deficit Disorder with Hyperactivity/ or exp Attention Deficit Disorder/
2 ADHD.mp.
3 exp Emotional Trauma/ or psychological trauma.mp.
4 child abuse.mp. or exp Child Abuse/
5 sexual abuse.mp. or exp Sexual Abuse/
6 physical abuse.mp. or exp Physical Abuse/
7 intimate partner violence.mp. or exp Intimate Partner Violence/
8 domestic violence.mp. or exp Domestic Violence/
9 exp Rape/ or rape.mp.
10 assault.mp. or exp Crime Victims/
11 exp Bullying/ or peer victimization.mp.
12 victimization.mp. or exp Victimization/
13 maltreatment.mp
14 exp Childhood Adversity/ or adverse childhood events.mp.
15 child neglect.mp. or exp Child Neglect/
16 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14 or 15
17 1 or 2
18 16 and 17
19 victimization.ab. or victimization.ti.
20 ADHD.ab. or ADHD.ti. or Attention-Deficit/Hyperactivity Disorder.ab. or Attention-Deficit/Hyperactivity Disorder.ti. or Attention-Deficit Hyperactivity Disorder.ab. or Attention-Deficit Hyperactivity Disorder.ti. or Attention Deficit with Hyperactivity Disorder.ab. or Attention Deficit with Hyperactivity Disorder.ti. or Attention Deficit Disorder.ab. or Attention Deficit Disorder.ti.
21 19 and 20
21 18 or 21

PubMed Search string:

Search: (((ADHD[Title/Abstract]) OR (Attention Deficit Hyperactivity Disorder[Title/Abstract])) OR (Attention-Deficit Hyperactivity Disorder[Title/Abstract])) OR (Attention Deficit Disorder[Title/Abstract]) OR (Attention Deficit Disorder with Hyperactivity[Title/Abstract]) OR (Attention-Deficit/Hyperactivity Disorder[Title/Abstract]) AND (Victimization[Title/Abstract]) OR ((victimization[Other Term]) OR ("victimization"[Other Term])))) OR("emotional trauma"[Other Term]) OR ("sexual abuse"[Other Term])) OR ("maltreatment"[Other Term])) OR ("emotional abuse"[MeSH Terms])) OR ("emotional abuse"[Other Term])) OR ("childhood adversity"[Other Term])) OR ("physical abuse"[Other Term])) OR ("physical abuse"[MeSH Terms])) OR (victimization[MeSH Terms])) OR (victimization[Other Term])) OR ("bullying"[MeSH Terms])) OR ("bullying"[Other Term])) OR ("adverse childhood experiences"[MeSH Terms])) OR ("adverse childhood experiences"[Other Term])) OR ("child abuse"[Other Term])) OR (child abuse[MeSH Terms])) OR (child abuse, sexual[MeSH Terms])) OR ("child abuse, sexual"[Other Term])) OR ("peer victimization"[Other Term])) OR (peer victimization[MeSH Terms])) OR (child neglect[MeSH Terms])) OR ("child neglect"[Other Term])) OR ("psychological trauma"[MeSH Terms])) OR ("psychological trauma"[Other Term])) OR (domestic violence[MeSH Terms])) OR ("domestic violence"[Other Term])) OR ("intimate partner violence"[MeSH Terms])) OR ("intimate partner violence"[Other Term])) OR ("crime victims"[MeSH Terms])) OR ("crime victim"[Other Term])) OR ("rape"[MeSH Terms])) OR ("rape"[Other Term])) OR ("assault"[Other Term])))) AND (((((((("ADHD"[Other Term]) OR (attention deficit disorder[MeSH Terms])) OR (attention deficit disorder with hyperactivity[MeSH Terms])) OR ("Attention-Deficit/Hyperactivity Disorder"[Other Term])) OR ("Attention-Deficit Hyperactivity Disorder"[Other Term])) OR ("Attention Deficit Hyperactivity Disorder"[Other Term])) OR ("Attention Deficit Disorder"[Other Term])))