Université de Montréal

# Impact of Maltreatment on Depressive Symptoms in Emerging Male Adults: The Mediating and Moderating Role of Coping Strategies and Cortisol Stress Response

par Yamiley Christina Cantave

École de criminologie
Faculté des études supérieures

Mémoire présenté à la faculté des études supérieures
en vue de l'obtention du grade de maîtrise (M. Sc.)
en criminologie
option mémoire

Août 2017

Université de Montréal

Faculté des Arts et des Sciences

Ce mémoire intitulé :

Impact of Maltreatment on Depressive Symptoms in Emerging Male Adults: The Mediating and Moderating Role of Coping Strategies and Cortisol Stress Response

Présenté Par :

Yamiley Christina Cantave

Évalué par :

Stéphane Guay

Président-rapporteur

Isabelle Ouellet-Morin

Directrice de recherche

Marie-France Marin

Membre du jury

# Résumé

Un nombre substantiel d'études indique que les enfants maltraités seraient plus susceptibles que les autres de vivre un épisode dépressif tôt au cours de la vie ainsi que de voir celui-ci se répéter par la suite. Néanmoins, les facteurs sous-tendant cette relation demeurent nébuleux. La présente étude examine les contributions des stratégies d'ajustement et de la réactivité cortisolaire à l'explication de l'association liant la maltraitance à la dépression. Les données de 156 jeunes hommes âgés de 18 à 35 ans, dont 56 rapportent avoir été maltraités à l'enfance, ont été colligées par le biais de questionnaires auto-rapportés évaluant la maltraitance, les symptômes dépressifs et les stratégies d'ajustement. La sécrétion cortisolaire a été mesurée en réponse au « Trier Social Stress Test » (TSST). Les résultats montrent que les participants maltraités utilisent plus fréquemment les stratégies d'ajustement centrées sur les émotions et celles-ci apparaissent, en retour, plus fortement associées aux symptômes dépressifs. Cependant, les personnes maltraitées utilisant moins fréquemment les stratégies d'ajustement centrées vers les tâches rapportent beaucoup plus de symptômes dépressifs. De surcroît, plus les participants montrent une forte réponse cortisolaire au stress, plus l'association liant la maltraitance aux symptômes dépressifs s'intensifie. En contrepartie, la réponse cortisolaire au stress est liée aux symptômes dépressifs, mais celle-ci n'explique pas la relation notée entre la maltraitance et les symptômes dépressifs. Ces résultats invitent les praticiens à considérer les stratégies d'ajustement comme une cible potentielle afin de prévenir ou réduire les symptômes dépressifs auprès de jeunes adultes maltraités au cours de leur enfance.

**Mots-clés** : Maltraitance, Dépression, Cortisol, Axe HPS, Stratégies d'ajustements, TSST.

# Abstract

An extensive body of work suggest that maltreated individuals are more likely to manifest depression at an early age and are more prone to experience recurrent depressive episodes that persist over long periods of time. However, the mechanism implicated in this association remain elusive. The present study examined the contribution of cortisol response to stress and coping strategies to the association linking childhood maltreatment to depressive symptoms. Data from 156 young men aged 18 to 35, of whom 56 were maltreated, were collected through self-reported questionnaires assessing maltreatment, depressive symptoms, and coping strategies. Cortisol was measured in response to the "Trier Social Stress Test" (TSST). Findings showed that maltreated individuals adopted emotion-oriented coping strategies more frequently and these, in turn, are more strongly associated with depressive symptoms. Conversely, maltreated individuals who were less prone to adopt task-oriented coping strategies reported higher levels of depressive symptoms. In addition, the more participants showed a higher cortisol response to stress, the greater was the association between maltreatment and depressive symptoms. In contrast, while cortisol response to stress is associated with depressive symptoms, it does not explain the association noted between maltreatment and depressive symptoms. These findings invite practitioners to consider coping strategies as a potential target for preventing or reducing depressive symptoms among young adults with a history of childhood maltreatment.



**Keywords**: Maltreatment, Depression, HPA axis, Cortisol, TSST, Coping.

**Table des matières**

# Liste des tableaux

# Liste des figures

# Liste des sigles

ACTH : Adrenocorticotropin hormone

CAR: Cortisol awakening response

CRH : Corticotropin-releasing-hormone

HPA axis: Hypothalamic –pituitary–adrenal axis

LGM : Latent growth model

SEM : Structural equation modeling

SPT : Social provocation task

TSST: Trier social stress test

# Liste des abréviations

e.g.,: For example

i.e.,: That is

*Goals on the road to achievement cannot be achieved without discipline and consistency*
*~Denzel Washington~*

*There is no fashion to be found in playing small and settling for a life that is less that the one you are capable of living ~Denzel Washington~*

# Remerciements

Bien que vous ne liriez probablement jamais ce manuscrit, je vous dédie ces quelques lignes. Je tiens à remercier mes parents pour leur support moral et les sacrifices qu'ils ont dû faire afin que je me rende jusqu'ici. Je remercie énormément ma meilleure amie, Hermeline, pour avoir toujours été là pour moi, pour les nombreuses discussions stimulantes au sujet de ce mémoire et « ta précieuse perspective clinique ». On en reparlera au doctorat.

Je remercie aussi ma directrice, Isabelle, pour sa disponibilité, ses conseils judicieux, sa rigueur et le support moral qu'elle m'a offert lorsque j'en avais besoin. Un grand merci à Amine et Abdelilah pour les discussions enflammées au sujet des statistiques, Excel et tout ce qui se passe dans les nouvelles. Vous allez me manquer !! Enfin, je remercie mon partenaire d'étude, Standley qui a su m'épauler lorsque je doutais de mes capacités.

Si ça prend toute une communauté pour élever un enfant, ça prend toute une communauté pour terminer une maîtrise. Un grand merci à tous !

# Introduction

According to the World Health Organisation, one out of five women and one out of thirteen men have reported being victims of sexual abuse in childhood (World Health Organization, 2016). In addition, one out of four adults have reported having been physically abused in childhood (World Health Organization, 2016), suggesting that a considerable number of individuals are confronted with maltreatment experiences in the general population (Briere & Elliott, 2003; Finkelhor, 1994; Gilbert et al., 2009). Beside its immediate burden, childhood maltreatment is reported to have serious lifelong consequences, including depression (Gilbert et al., 2009; Norman et al., 2012). Specifically, maltreatment is associated with a higher risk of suffering from recurrent and persistent depressive episodes (Harkness, Michael, & Kennedy, 2012; Nanni, Uher, & Danese, 2012). Despite the recurrently noted association between maltreatment and depressive symptoms, the potential mechanisms implicated in this association remain unknown (Liu, 2017).

With this goal in mind, this master-degree thesis sought to evaluate the contribution of cortisol response to stress and coping strategies in the association between maltreatment and depressive symptoms. In the first chapter, a review of the empirical literature will be undertaken. A thorough description of the method used will be detailed in the second chapter, followed by the main results of the study presented in the third chapter. Finally, the limitations as well as the theoretical, empirical and clinical contributions of the thesis will be fully discussed in the fourth chapter.

# Chapter 1: Literature Review

Childhood maltreatment is a major public health, human rights and social concern worldwide. This ill-treatment refers to any acts of commission or omission by a parent or other caregivers that lead to harm, potential for harm, or threat of harm to a child, even if it is not the intended result (Gilbert et al., 2009; Li, D'Arcy, & Meng, 2015). Five categories of maltreatment are broadly recognized: (a) sexual abuse, which includes sexual contact or conduct between a child younger than 18 years of age and an adult or older person; (b) physical abuse, which involves the infliction of bodily assaults on a child by an adult or older person that posed a risk of or led to injury; (c) emotional abuse, which encompasses verbal assaults on a child's sense of worth or well-being or any humiliating or demeaning behaviour directed toward a child by an adult or older person; (d) physical neglect, which extends to the failure of caretakers to provide for a child's basic physical needs, including food, shelter, clothing, safety and health care; and (e) emotional neglect, defined as the failure of guardians to meet children's fundamental emotional and psychological needs, including love, belonging, nurturance, and support (Bernstein et al., 2003). Although each subtype implies a distinctive departure from the normative environment, it is important to keep in mind that most maltreated children have suffered from more than one form of maltreatment (Cicchetti & Valentino, 2006), thus drawing attention to the potential cumulative impact of maltreatment on their functioning.

According to the Canadian Incidence Study of Reported Child Abuse and Neglect, an estimated 235,842 investigations have been initiated in 2008 following allegations of childhood maltreatment (i.e., neglect [34%; 28,939], physical abuse [20%; 17,212], emotional abuse [9%; 7423] and sexual abuse [3%; 2607]), or exposure to violence (i.e., intimate partner violence [34%; 29,259]). Of these cases, approximately 36% (85,440) were corroborated (Trocmé et al.,

2010). These statistics, however, only document cases that have come to the attention of authorities. Thus, the "real" number of Canadians who are victims of childhood maltreatment is thought to be far greater. Indeed, according to the General Social Survey (2014), about 30% of Canadians reported being victims of maltreatment by an adult at least once before the age of 15 (Perreault, 2015). In addition, a recent population survey carried out among 4029 mothers and 1343 fathers in 2012 revealed that four out of five children in Quebec were exposed to behaviours that could be qualified as psychological abuse (i.e., yelling, insults, threats and humiliations) at least once during the last year. Moreover, nearly one out of two of these children (49%) have endured them recurrently (i.e., three or more times) (Clément, Bernèche, Fontaine, & Chamberland, 2013). In addition, more than a third of children in Quebec have suffered from physical abuse (i.e., hitting hands, arms, legs or buttocks, get pinched or shaken) on at least one occasion throughout the year. These events were experienced repeatedly for 11% of the sample (Clément et al., 2013). Given the considerable number of children who are confronted with maltreatment experiences, attention has been devoted to circumscribing the extent to which these experiences affect later functioning.

Aside from the immediate burden related to childhood maltreatment, these experiences have been shown to jeopardize the normative development across many domains of functioning in childhood such as emotional and behavioural regulation, peer relations as well as academic performance (Cicchetti & Toth, 2005). Several prospective studies have also revealed that maltreated children are more likely to suffer from a number of problems once they become adults, including antisocial behaviours, risky sexual behaviours, obesity and physical health problems (Gilbert et al., 2009; Norman et al., 2012). Childhood maltreatment has additionally

been shown to have an impact on later economic productivity in adulthood. That is, maltreated individuals are at a greater risk of being unemployed as well as occupying menial and semi-skilled positions at work (Gilbert et al., 2009). Converging evidence also support maltreatment as an important risk factor for mental health disorders, with depression at the forefront (Gilbert et al., 2009; Shenk, Griffin, & O'Donnell, 2015). For instance, Li and colleagues have revealed in a recent meta-analysis of prospective cohort studies that a history of childhood maltreatment increased the onset of depression in adulthood by a factor of two (Li et al., 2015). Furthermore, maltreated children are more likely to suffer from a first episode of depression at an earlier age (Widom, DuMont, & Czaja, 2007), and are more prone to experience recurrent depressive episodes that persist over long periods (Nanni et al., 2012). Considering the higher probability of suffering from recurrent and persistent depressive symptoms among maltreated individuals, it is of utmost importance that investigators focus on factors that underlie this association. This is the main objective of the present master-degree thesis.

Before identifying the potential mechanisms, a more detailed description of depression symptomatology is in order. Depression refers to a pathological condition marked by a sad, empty or irritable mood, co-occurring with somatic and cognitive changes that significantly disrupt the individual's ability to function (American Psychiatric Association, 2013). Depression is ranked as the second leading cause of years lived with disabilities occurring between 15 and 64 years of age (Ferrari et al., 2013). This condition affects approximately 4% of Canadians each year, with a lifetime prevalence of 24.2% among women and 14.2% among men (Patten, 2009; Patten et al., 2006). Depression is often chronic, recurrent and incrementally harmful over time (Judd, 1997). Accordingly, substantial proportion of remitted depressed

patients report residual symptomatology, suggesting that complete symptomatic recovery following an episode of depression is not the rule (Conradi, Ormel, & De Jonge, 2011). Moreover, depression frequently overlaps with other psychopathologies (e.g., anxiety, substance abuse and impulse control disorder) and seems to interfere with optimal functioning in several domains of life, including social and work-related roles (Kessler et al., 2003). Overall, depression is a chronic and recurrent disorder, the occurrence of which is often associated with a broad range of psychosocial difficulties. Preventing and delaying the emergence of this disorder are thus desirable from a public health perspective.

### 1.1. Maltreatment and Depression: What are the Mechanisms Involved?

While cumulating evidence suggests that childhood maltreatment constitutes a risk factor for depression, the potential mechanisms implicated in this association remain unclear (Liu, 2017). Several factors have been proposed as being involved in risk pathways linking maltreatment with depression, such as a low self-esteem, violent interpersonal tendencies as well as alterations of brain structures and functioning (e.g., hippocampus, amygdala, prefrontal cortex) (Liu, 2017; Twardosz & Lutzker, 2010). For instance, it has been proposed that maltreatment may facilitate the emergence of negative cognitive styles (e.g., rumination, avoidance) which, in turn, may enhance the susceptibility for depression. Similarly, it has been hypothesized that maltreatment may give rise to maladaptive interpersonal styles (e.g., insecure attachment style, conflicts), which, in turn, may lead to a higher vulnerability for depression (Liu, 2017; McCrory, De Brito, & Viding, 2012). Pathways implicated in the physiological and psychological response to stress have received considerable attention in the last decades, given the recurrently noted association

between stressful life events and depression onset and recurrence over time (Monroe & Harkness, 2005). This investigation may best be pursued in a Developmental psychopathology framework, considering the need to assess multiple domains of development (e.g., biological, psychological, social and cultural processes) that are in constant interaction (Cicchetti, 2006). Opting for a multiple level analysis of stress mechanisms thought to be involved in the emergence of depression among individuals who were exposed to childhood maltreatment is essential considering the multifactorial nature of its etiology (Shenk et al., 2015; Toth & Cicchetti, 2013). Increasing knowledge about these risk pathways may promote the development of more effective interventions for these children to support their resilience or promptly redirect the course of the illness once a first episode has been experienced (Liu, 2017; Shenk et al., 2015).

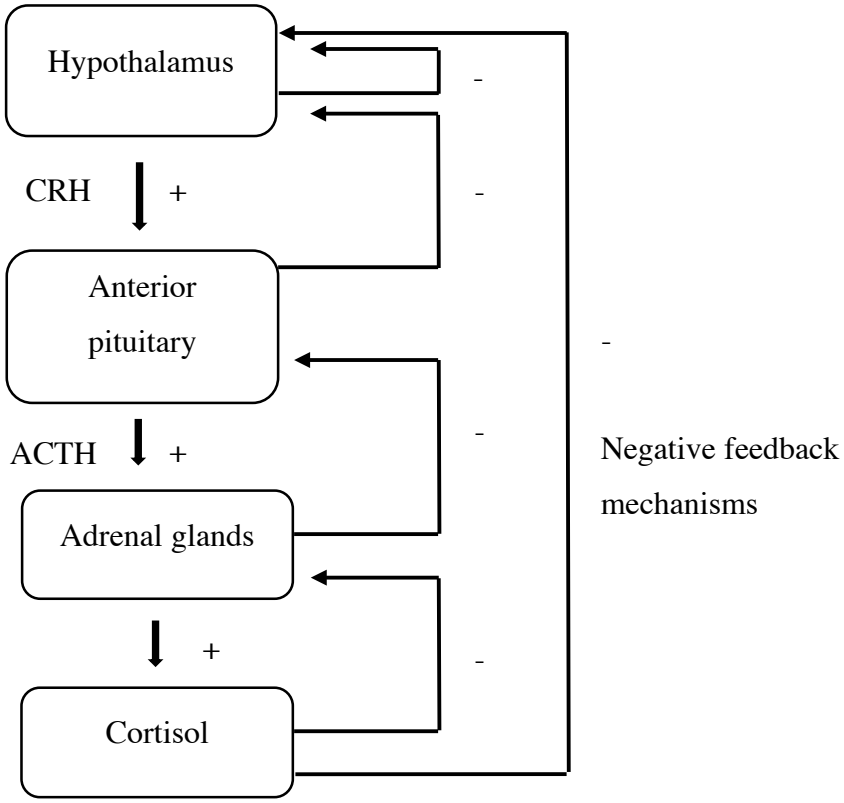
## 1.2 Maltreatment, HPA axis, and Depression

### 1.2.1 The Normative Stress Response: HPA axis and Coping Strategies

Stress is defined as a threat to homeostasis (i.e., the stability of physiological processes that preserve life). That is, situations that are both or either uncontrollable, unpredictable, new and that threaten the ego are expected to trigger a stress response (Lupien, 2010). When a stressful event is experienced over a limited duration, and leaves a sense of exhilaration and accomplishment and the person who is dealing with the situation feels like he or she can master it, it can be referred to as good stress (eustress). Conversely, challenging events such as maltreatment involving a loss of control and mastery as well as experiences that are often prolonged, recurrent, irritating, emotionally draining, physically exhausting or dangerous may result in distress (bad stress) (McEwen, 2007). The concept of allostasis and allostatic load have

been proposed to understand how exposure to chronic and/or severe adverse life events, such as maltreatment, contribute to the onset of mental and physical health problems (Flier, Underhill, & McEwen, 1998; McEwen & Stellar, 1993). Allostasis refers to the process whereby the body regulates vital physiological functions by setting new arousal set points in order to support optimal adaptation to continually changing demands in the environment (McEwen & Stellar, 1993). Successful adjustment to stressful situations requires the short-term activation of the primary physiological stress mediators, including the cardiovascular system and the hypothalamic-pituitary-adrenal (HPA) axis to meet the metabolic needs of the individual (Chrousos & Gold, 1992; Lupien et al., 2006). As can be seen in Figure 1, the perception of stress is expected to trigger the activation of the HPA axis, whereby neurons in the paraventricular nucleus of the hypothalamus release the corticotropin-releasing hormone (CRH), which travels through the hypophysal portal circulation to the anterior pituitary gland. The latter reacts by releasing a pulse of adrenocorticotropin hormone (ACTH), which is carried through the peripheral circulation to the adrenal glands. In response to ACTH, the adrenal glands synthesize and secrete cortisol (Gunnar & Vazquez, 2006; Miller, Chen, & Zhou, 2007). Cortisol is crucial for mobilizing energy during stress (Sapolsky, 2004) and plays a role in returning to the baseline level of HPA axis activity when the stressful situation has ended through the action of negative feedback mechanisms (Olff, Langeland, & Gersons, 2005).

Figure 1. The HPA axis response to stress




Beside the neurophysiological stress response, the Allostasis and Allostatic load model also proposes arguments in favour of the relevance of behavioural stress responses as means to preserve homeostasis, that is, the way individuals cope behaviourally and cognitively with challenging situations (McEwen & Stellar, 1993). Consistent with this theory, Lazarus & Folkman (1984) proposed the Transactional model of stress and coping, in which the psychological stress response is emphasized for its crucial role in interpreting a situation as stressful. The first step of the psychological stress response is the evaluation of cues in the environment, which can be categorized into two basic forms: primary and secondary appraisals. Primary appraisal refers to the assessment of stimuli in the environment that are anticipated as

irrelevant, benign positive and stressful (Lazarus & Folkman, 1984). Environmental signals that do not carry any significance for a person's well-being will be evaluated as irrelevant. Situations that improve an individual's well-being or which hold out a promise of such an outcome will be appraised as benign positive. Encounters that could include harm/loss (situations where damage has already been sustained), threat (situations that are potentially harmful) and challenge (situations that have the potential for gain or growth) will be assessed as stressful. Stressful situations will trigger a secondary appraisal to estimate the consequences of any action that could be taken by the individual to deal with the stressor (Lazarus & Folkman, 1984). Subsequently, individuals are thought to adopt cognitive and behavioural strategies in an attempt to regulate their emotional response (emotion-oriented), to alter or manage the demands of the situation (task-oriented) or to distract themselves from the stressful situation (avoidance-oriented) (Endler & Parker, 1994; Lazarus & Folkman, 1984). In brief, the normative stress response mobilizes individuals cognitively and behaviourally, during which they interpret and react to the perceived stressful situation in addition to the body's neurophysiological stress response (Lupien et al., 2006). Consequently, to evaluate the role of the stress response in the association between maltreatment and depressive symptoms more accurately, both physiological and psychological levels should be analyzed in complementarity.

### 1.2.2 The Pathological Stress Response

Brief activation of physiological stress mediators enable the body to respond adequately to challenging situations (Lupien et al., 2006). However, under conditions of chronic and repeated stress, enduring mobilization of stress mediators through repetitive hits, failure to habituate, and/or prolonged or inadequate response may in the long run, carry substantial cost for the body,

namely a process known as allostastic load (Flier et al., 1998; McEwen & Stellar, 1993). Consistent with the Allostatic load model, exposure to maltreatment early in life may lead to disruptions in the activity of the HPA axis in basal and stressful contexts (McEwen, 1998; McEwen & Stellar, 1993). This hypothesis has gathered the attention of a great number of investigators over the years, for which a brief review is proposed in the following section.

### 1.2.3 Association Between Maltreatment and the HPA Axis Activity

Existing findings suggest an altered HPA axis activity among maltreated individuals (Gonzalez, 2013; Gunnar & Vazquez, 2006; McCrory et al., 2012; Tarullo & Gunnar, 2006). Earlier investigations have mainly focused on basal cortisol levels and diurnal rhythms of which the latter is characterized by a rise in cortisol levels 30 minutes after waking [cortisol awakening response (CAR)] and a subsequent gradual decline throughout the day (Tarullo & Gunnar, 2006). Several studies have reported lower levels of cortisol among maltreated children (Alink, Cicchetti, Kim, & Rogosch, 2012; Bicanic et al., 2013; Kohrt, Worthman, et al., 2015). For example, Dozier and colleagues revealed that children who have been maltreated secreted lower levels of cortisol at awakening compared to nonmaltreated children in a sample of 20- to 60-month-old children (Dozier et al., 2006). Likewise, lower morning cortisol was reported in a sample of 3- to 6-year-old maltreated children residing in foster care in comparison to a group of low-income nonmaltreated children (Bruce, Fisher, Pears, & Levine, 2009). Higher cortisol diurnal levels have also been reported in that context (Doom, Cicchetti, Rogosch, & Dackis, 2013; Gerra et al., 2010; Şimşek, Kaplan, Uysal, Yüksel, & Alaca, 2016; Şimşek, Yüksel, Kaplan, Uysal, & Alaca, 2015). Maltreated 10-year-old children with concurrent PTSD symptoms were shown to have higher levels of cortisol (Carrion et al., 2002), as well as higher

concentrations of 24h urinary free cortisol (De Bellis et al., 1999) in comparison to controls. Altogether, these findings suggest that childhood maltreatment is associated with a disrupted HPA axis activity emerging at an early age and which could take the form of lower or higher cortisol levels among children. However, nonsignificant findings also exist (Bick et al., 2015; Cicchetti & Rogosch, 2001a; Kuhlman, Geiss, Vargas, & Lopez-Duran, 2017).

Similar results have been reported for adults with a history of maltreatment (Power, Thomas, Li, & Hertzman, 2012; Trickett, Noll, Susman, Shenk, & Putnam, 2010; van der Vegt, van der Ende, Huizink, Verhulst, & Tiemeier, 2010; Weissbecker, Floyd, Dedert, Salmon, & Sephton, 2006). Adult men and women who experienced severe childhood maltreatment exhibited a lower cortisol awakening response in comparison with nonmaltreated participants (van der Vegt, van der Ende, Kirschbaum, Verhulst, & Tiemeier, 2009). Furthermore, in a sample of 40-year-old women with chronic pain linked to fibromyalgia or osteoarthritis, individuals who were exposed to severe maltreatment as children showed higher cortisol levels throughout the day during thirty consecutive days (Nicolson, Davis, Kruszewski, & Zautra, 2010). Others have also reported nonsignificant findings (Hulme, McBride, Kupzyk, & French, 2015; Kempke et al., 2015). Overall, childhood maltreatment seems to be associated with a deregulated HPA axis activity that may persist into adulthood, although much work remains to be done to better understand the inconsistency in the reported findings.

In order to gain additional knowledge about the HPA axis (Burke, Davis, Otte, & Mohr, 2005), recent studies have also examined the cortisol response to psychological stress among maltreated individuals. Psychological challenges ideally involved tasks encompassing both

social-evaluative and uncontrollable elements that can naturally provoke a reliable stress response, such as public speaking (Dickerson & Kemeny, 2004; Tarullo & Gunnar, 2006). A trend for lower cortisol response to psychological stress has been observed in maltreated adolescents (MacMillan et al., 2009; Ouellet-Morin et al., 2011; Trickett, Gordis, Peckins, & Susman, 2014). Trickett and colleagues studied boys and girls aged between 9 and 12 years of age who had experienced substantiated (i.e., corroborated) maltreatment during the preceding month. In comparison to the control group, maltreated children showed a lower cortisol response to stress (Trickett et al., 2014). The same pattern of findings was reported in a sample of female adolescents aged between 14 and 19 years of age (Shenk, Putnam, Rausch, Peugh, & Noll, 2014). Higher cortisol response to psychosocial challenges has also been noted among adolescents with a history of childhood maltreatment (Bick et al., 2015; Harkness, Stewart, & Wynne-Edwards, 2011; Rao, Hammen, Ortiz, Chen, & Poland, 2008). For instance, Bick and colleagues reported that adolescents with a history of sexual abuse exhibited a greater cortisol response to stress in comparison to a healthy control group (Bick et al., 2015). Additionally, Harkness and colleagues revealed that maltreated male and female adolescents and young adults aged 12 to 21 years who experienced mild/moderate depression symptoms also showed a higher cortisol response to psychosocial stress (Harkness et al., 2011). Childhood maltreatment may thus contribute to induce a deregulated pattern of cortisol response to psychosocial stress among children and adolescents.

Atypical patterns of cortisol response to stress have also been documented in adulthood (Carpenter et al., 2007; Gonzalez, 2013; McCrory et al., 2012; Voorhees & Scarpa, 2004). In a groundbreaking study, Heim and colleagues reported a greater cortisol response to a

psychosocial stress test among women aged 18 to 45 years, who had been maltreated before puberty (Heim, Newport, et al., 2000; Heim et al., 2002). In contrast, in comparison to controls, lower cortisol response to stress was observed in young adults with a history of traumatic experiences (i.e., maltreatment, parental divorce and loss of significant others) before the age of 18 (Elzinga et al., 2008). Other investigations have, however, not found the expected differences in the pattern of cortisol response to stress between adults who had been maltreated compared to those who were not (Carpenter, Shattuck, Tyrka, Geracioti, & Price, 2010; Rao & Morris, 2015). In summary, empirical evidence cumulated so far suggests that exposure to maltreatment in childhood may lead to disruptions in basal and reactive cortisol activity in childhood and adulthood. Although these disruptions may be adaptive, sustained alterations in HPA axis activity may also precipitate changes in secondary and tertiary outcomes, such as the wear and tear of organs and tissues as well as the diseases that may follow (e.g., cardiovascular diseases and depression) (Danese & McEwen, 2012; Flier et al., 1998; McEwen & Stellar, 1993).

### 1.2.4 Association Between the HPA Axis and Depression

Over the last four decades, an extensive body of work has been generated from investigations assessing the activity of the HPA axis among depressed individuals (Burke et al., 2005; Young, Lopez, Murphy-Weinberg, Watson, & Akil, 2000). This interest is partly motivated by the need to discover key biological mechanisms that may be involved in the recurrently noted association between exposure to stress and depression (Buitelaar, 2013; Burke et al., 2005; Lopez-Duran, Kovacs, & George, 2009). Most of the knowledge regarding the HPA axis functioning in depressed individuals concerns basal cortisol levels and cortisol response to pharmacological challenges following the administration of exogenous substances (i.e., dexamethasone,

metyrapone and CRH) that mimic the effect of the main hormones secreted by the HPA axis (e.g., cortisol, CRH) (Burke et al., 2005). Previous studies have thus revealed higher basal cortisol levels among depressed individuals in comparison to controls (see Knorr, Vinberg, Kessing, & Wetterslev (2010) and Lopez-Duran et al., (2009) for a more in depth review on the subject). In line with these findings, one meta-analysis has also reported that depressed individuals showed a higher cortisol response to pharmacological challenges compared to nondepressed participants (Stetler & Miller, 2011), suggesting deregulated negative feedback mechanisms among this population (Burke et al., 2005).

More recent studies have turned their attention to the cortisol response to psychological probes. Whereas the pharmacological stress response, and to a certain extent basal cortisol indexes, reflect the activity of distinct components of the HPA axis, the cortisol response to psychosocial stress mirrors the endogenous activity of the entire HPA axis system, including higher-level excitatory and inhibitory influences (Burke et al., 2005; Young et al., 2000). This HPA axis index is particularly relevant in the context of depression given that psychosocial stressors activate key emotional circuits (i.e., limbic areas of the brain) that have been reported to be altered in depressed individuals (Dean & Keshavan, 2017; Nestler et al., 2002; Young et al., 2000). Consequently, a better understanding of HPA axis response to social stress may help clarify its contribution to the onset of depression (Ciufolini, Dazzan, Kempton, Pariante, & Mondelli, 2014). While investigations examining the association between HPA axis response to psychological stress and depressive symptoms are limited (Guerry & Hastings, 2011), their results seem to be somewhat consistent with studies using pharmacological challenges. On the one hand, higher cortisol response to stress has been reported among individuals who manifest

depressive symptoms (Dockray, Susman, & Dorn, 2009; Höhne et al., 2014; Lopez-Duran et al., 2009; Luby et al., 2003; Weinstein et al., 2010). For example, higher levels of parent-rated depressive symptoms were related to greater cortisol response to stress in boys and girls aged between 8 and 13 years of age (Dockray et al., 2009). Consistent with these findings, one study revealed that clinically depressed men and women exhibited a higher cortisol response to stress relative to a non-depressed control group (Weinstein et al., 2010). One meta-analysis assessing the association between cortisol response to psychological stress and depression among adult men and women indicated that, in comparison to nondepressed individuals, depressed patients had higher cortisol levels during recovery (i.e., following the offset of the stressor) (Burke et al., 2005). Findings also suggested that a greater cortisol response to stress may be particularly associated with mild/moderate depressive symptoms (Harkness et al., 2011), as well as a short history of the symptomatology (Booij, Bouma, de Jonge, Ormel, & Oldehinkel, 2013). More importantly, adult men with remitted depression showed a higher cortisol response to stress compared to men who never experienced depression (Bagley, Weaver, & Buchanan, 2011). These findings suggest that higher patterns of cortisol secretion in response to stress may be present during depressive symptoms and may even persist after remission.

On the other hand, a handful of studies have also reported a lower cortisol response to stress among depressed individuals (Ahrens et al., 2008; Booij et al., 2013; Burke et al., 2005). For example, one study reported that adolescents with moderate/severe depressive symptoms had a lower cortisol response to stress in comparison to participants with minimal/mild/moderate depressive symptoms (Harkness et al., 2011). Additionally, adolescents and adults with a long history of depressive symptoms showed a lower cortisol response to stress, indicating that

chronicity of depressive problems may be associated with a distinct pattern of cortisol response to stress as well (Ahrens et al., 2008; Booij et al., 2013). Of note, nevertheless, several studies have failed to replicate these findings (Ciufolini et al., 2014; Dienes, Hazel, & Hammen, 2013; Giese-Davis et al., 2006; Young et al., 2000). Overall, these studies suggest that disruptions in the HPA axis activity, including both lower and higher cortisol response to stress, may confer a vulnerability to manifest depression. For the most part, however, these studies failed to consider for past adverse experiences, such as childhood maltreatment, that may have also impacted the HPA axis.

### 1.2.5 The Mediating and Moderating Hypothesis: HPA Axis

Accumulating evidence suggests that childhood maltreatment may be related to disrupted cortisol activity in both basal and reactive contexts. Analogously, disrupted patterns of lower and higher secretion of cortisol has been documented among individuals suffering from depression. These two bodies of research have, nonetheless, often been analyzed separately despite much theorizing postulating that cortisol may be implicated in the association between maltreatment and depressive symptoms; thus, limiting our understanding of the processes that join these three components together (Hagan, Roubinov, Mistler, & Luecken, 2014; Heim, Newport, Mletzko, Miller, & Nemeroff, 2008). Theoretically, cortisol could be implicated in this association in two different ways: either as a mediator (Figure 2, Path A) or a moderator (Figure 3). Several conceptual models have highlighted the mediating role of the HPA axis in the association between maltreatment and depressive symptoms, including the Allostatic load model and the Biodevelopmental framework (McEwen & Stellar, 1993; Shonkoff, 2010). As abovementioned, the Allostatic load model states that exposure to chronic and repeated stress,

such as maltreatment, may lead to disruptions in primary mediators, including the HPA axis. In turn, persistent alterations of these mediators may increase the probability of suffering from a broad range of health problems, including depression (Flier et al., 1998; McEwen & Stellar, 1993). To the best of our knowledge, only one study has tested the mediating role of cortisol response to stress in the association linking maltreatment to internalizing symptoms. Their findings indicated that cortisol response to stress did not account for the association between exposure to violence (i.e., maltreatment and exposure to direct or indirect violence in school, home or neighborhood) and internalizing symptoms among a sample of adolescents (Busso, McLaughlin, & Sheridan, 2017). Additionally, one prospective study revealed that the association between substantiated childhood maltreatment and depressive symptoms in a sample of adolescents was not explained by the participants' total cortisol output (i.e., an estimate that reflects total cortisol levels before and after a psychological stress test), while simultaneously accounting for negative affect, the autonomic response and difficulties in emotion regulation (Shenk et al., 2015). Regarding diurnal cortisol levels, preliminary support for mediation was found in a sample of adult men and women who were exposed to neglect in infancy. These experiences were associated with a flattened diurnal cortisol profile (slower decline during the day), which in turn, was related to higher levels of daily pain, depression and anxiety symptoms (Yeung, Davis, & Ciaramitaro, 2016). In sum, there is a dearth of research on the extent to which cortisol response to stress partly explains the association recurrently noted between maltreatment and depressive symptoms. Consequently, it remains unclear whether cortisol indeed serves as an indirect risk pathway linking the experiences of maltreatment to depression.

Alternatively, cortisol could also act as a moderator in the association between maltreatment and depressive symptoms. Accordingly, not all children will manifest mental health problems following maltreatment (Cicchetti & Rogosch, 2001b). The Biological sensitivity to context model proposes that biological reactivity to stress interact with the context in which an individual is brought up in (Boyce & Ellis, 2005). This conceptual framework somewhat echoes the hypothesis that exposure to maltreatment interacts with cortisol response to stress to enhance the risk of depressive symptoms (moderation model). Preliminary evidence for the modulating role of the HPA axis in the association between maltreatment and depressive symptoms is indirectly found in studies reporting differences in cortisol response to stress between groups who have/have not been exposed to childhood maltreatment and suffered from depression (Heim, Newport, et al., 2000; Heim et al., 2002; Rao & Morris, 2015). For instance, maltreated women with current depression showed a higher cortisol response to stress in comparison to participants with a diagnosis of current major depression, participants with a history of maltreatment and controls (Heim, Newport, et al., 2000). Despite bringing a new light on the possibility that the combination of maltreatment and cortisol should be taken into account to delineate more accurately the cortisol response to stress, these studies do not appropriately test the moderating-based hypothesis. To do so, investigators need to include an interaction term between the two main factors (i.e., maltreatment X cortisol) in their analyses, thus testing whether the impact of the combination of the two factors is greater than the sum of its parts. The few studies that have formally tested the modulating role of cortisol in this association revealed that individuals who were exposed to childhood maltreatment are more likely to manifest higher levels of depressive symptoms if they had a disrupted HPA axis response to stress (Badanes, Watamura, & Hankin, 2011; Hagan et al., 2014; Harkness et al., 2011; Kuhlman et al., 2017;

Rogosch, Dackis, & Cicchetti, 2011; Steeger, Cook, & Connell, 2017; Suzuki, Poon, Papadopoulos, Kumari, & Cleare, 2014). For example, among emerging adults who had a greater cortisol response to stress, participants with higher maltreatment experiences exhibited more internalizing problems in comparison to participants with a lower cortisol response to stress (Hagan et al., 2014). The same findings were reported in another investigation with adolescents who showed a greater cortisol response to stress and who had been exposed to higher levels of family life stress (e.g., financial problems, serious injury or illness of a family member) reporting more internalizing problems compared to those who showed a lower cortisol response to stress (Steeger et al., 2017). Furthermore, one study indicated that the lower cortisol response to stress was, the greater was the impact of physical abuse on parent-rated internalizing symptoms in adolescents (Kuhlman et al., 2017). Conversely, higher cortisol response to stress was found to enhance the impact of emotional abuse on parent-rated internalizing symptoms (Kuhlman et al., 2017), suggesting that lower as well as higher cortisol response to stress may increase the risk of developing depression among maltreated children. In summary, mounting evidence supports the hypothesis that cortisol response to stress either modulate or mediate the association between maltreatment and depressive symptoms. However, studies that have appropriately tested these models are scarce and yield to inconsistent findings. Moreover, to the best of our knowledge, only one study has tested both the mediating and moderating models (Badanes et al., 2011).

### 1.3 Maltreatment, Coping Strategies, and Depression

The study of coping draws from a long history of research in psychology that stems from psychoanalytic and ego theories. Initially, arguments have been presented to suggest that coping

with stress is mainly determined by stable personality attributes and perceptual styles (Livneh & Martz, 2007). Following the pioneering work of Lazarus (1966), researchers shifted their exclusive attention from individual characteristics to focus on a more transactional perspective, whereby coping refers to cognitive and behavioural strategies adopted by a person that are constantly changing, in an attempt to deal with challenging situations (Lazarus & Folkman, 1984; Livneh & Martz, 2007). More specifically, when a situation is perceived as stressful, individuals will adopt strategies in order to manage or alter the demands of the situation (task-oriented), to regulate their emotional response (emotion-oriented) or to distract themselves from the stressful situation (avoidance-oriented) (Endler & Parker, 1994; Lazarus & Folkman, 1984). When the challenging situation is appraised as mildly stressful, individuals will use the three forms of coping strategies at an equivalent rate to solve the problematic situation. Inversely, in situations that are evaluated as moderately stressful, task-oriented coping strategies will be adopted more frequently whereas in highly stressful situations, emotion-oriented and avoidance-oriented coping strategies will predominate (Lazarus & Folkman, 1984). Considering the extreme level of threat that maltreatment poses for adaptation, maltreated children may be more likely to use emotion-oriented and avoidance coping strategies because of the more urgent need to rapidly reduce acute distress, shame and helplessness (Hager & Runtz, 2012; Lazarus & Folkman, 1984; Spaccarelli, 1994). However, in the long run, the use of emotion-oriented coping strategies may become the typical response to deal with stress, irrespective of the perceived stress level of the situation (Lazarus & Folkman, 1984). Emotion-oriented coping strategies may become the predominant response to stressful situations partly because of the maladaptive cognitive schemata (i.e., highly dysfunctional attitudes about the individual that are later embedded into cognitive structures) that maltreatment experiences may trigger, enhancing

the susceptibility that maltreated children appraise challenging situations in a negative and distortional manner (Beck, 1979, 2008; Janoff-Bulman, 1985). Because of the high level of stress that maltreatment experiences may induce, these children may be more prone to adopt emotion-oriented coping strategies (Lazarus & Folkman, 1984). Over time, maltreated children may become accustomed and may adopt emotion-oriented strategies as a typical response to stress even in adulthood, regardless of the level of perceived stress of the situation. In sum, earlier attempts to cope with maltreatment may affect subsequent efforts to cope with stressful situations in adulthood (Hager & Runtz, 2012; Lazarus & Folkman, 1984). Considering the appeal of this hypothesis for intervention, a great body of research have taken interest in examining the proposed association, which will be summarized in the following section.

### 1.3.1 Association Between Childhood Maltreatment and Coping Strategies

An extensive body of research has documented the association between maltreatment and coping strategies (Walsh, Fortier, & DiLillo, 2010). One the one hand, substantial work has been carried out to explore the manner in which maltreated individuals cope with childhood maltreatment experiences in adulthood. Findings from such studies suggest that these individuals often rely on avoidance coping strategies (Brand & Alexander, 2003; Cantón-Cortés & Cantón, 2010; Filipas & Ullman, 2006; Leitenberg, Greenwald, & Cado, 1992; Merrill, Guimond, Thomsen, & Milner, 2003). For instance, adults who were sexually abused in childhood reported that they were more likely to adopt avoidance coping strategies to deal with reminiscences of the memories of maltreatment experiences (Sigmon, Greene, Rohan, & Nichols, 1997). Findings also indicate that individuals who were exposed to childhood maltreatment had a greater inclination to use emotion-oriented coping strategies to deal with the

aftermath of these experiences in adulthood (DiLillo, Long, & Russell, 1994; Long & Jackson, 1993). For example, Long & Jackson (1993) reported that sexually abused adult women had a greater tendency to adopt emotion-oriented coping strategies to deal with childhood maltreatment events in adulthood. However, inconsistent findings have also been noted (Futa, Nash, Hansen, & Garbin, 2003; Runtz & Schallow, 1997). For instance, Runtz and Schallow (1997) reported that individuals who were exposed to physical abuse were more likely to adopt both positive (task-oriented) and negative (avoidance) coping strategies to deal with childhood maltreatment in adulthood. Although findings are not always consistent, maltreated individuals seem to be more inclined to adopt avoidance as well as emotion-oriented coping strategies to deal with the aftermath of childhood maltreatment experiences in adulthood.

Similar findings have been reported when focussing on the way adults maltreated as children cope with stressful situations (Walsh et al., 2010). Leitenberg and colleagues (2004) revealed that adult women who experienced cumulative abuse prior to 16 years of age (i.e., sexual and physical abuse, parental alcohol abuse, domestic violence) employed greater disengagement strategies [similar to avoidance strategies (e.g., problem avoidance, social withdrawal)] when dealing with recent stressful situations (e.g., interpersonal conflicts, negative life events, traumatic events) (Leitenberg, Gibson, & Novy, 2004). Likewise, Hyman and colleagues reported that adult men and women who were severely maltreated relied more on avoidance coping strategies to deal with stress. However, this association could not be detected for task-oriented and emotion-oriented coping strategies (Hyman, Paliwal, & Sinha, 2007). Yet, still other studies reported that maltreated individuals have a greater tendency to adopt emotion-oriented coping strategies in response to stress (Sigmon et al., 1997; Thabet, Tischler, &

Vostanis, 2004). For example, one investigation revealed that sexually abused women had a higher inclination to adopt emotion-oriented coping strategies to manage current stressful life events (Sigmon et al., 1997). Similarly, Thabet and colleagues (2004) reported that adolescent males with a history of physical and emotional abuse were more likely to adopt both avoidance and emotion-oriented coping strategies to deal with stress. Taken together, these results suggest that maltreatment is related to a greater use of avoidance and emotion-oriented coping strategies to manage challenging events, although inconsistent findings have also been noted (Coffey, Leitenberg, Henning, Turner, & Bennett, 1996). More generally, it goes without saying that individuals, and perhaps more so those who were maltreated as children, may prefer to avoid the painful reminiscences and negative emotional states related to challenging events or try to regulate their emotional response to stressful situations (Lazarus & Folkman, 1984; Leitenberg et al., 1992). Accordingly, the use of these strategies are not altogether detrimental, and could be even adaptive in the short-term, providing relief and facilitating the regulation of negative emotions (Lazarus & Folkman, 1984; Sigmon et al., 1997; Wright et al., 2007). However, these strategies may not be adaptive in the long run if they prevent the active cognitive processing needed to integrate and resolve challenging situations (Wright et al., 2007). In sum, a greater tendency to adopt emotion-oriented and avoidance coping strategies may jeopardize the individuals' capacity to deal with stress in their lives and thus they may be more vulnerable to suffer from mental health problems such as depression.

### 1.3.2 Association Between Coping Strategies and Depression

Over the last decades, substantive works have reported a recurrent association between coping strategies and later adjustment (Lazarus & Folkman, 1984; Spaccarelli, 1994; Walsh et al.,

2010). This interest probably stems from the recognition that coping strategies are amenable to change and may thus represent a viable target for intervention (Walsh et al., 2010). Most of these studies have examined the association between coping and general psychological distress (i.e., a composite index of somatization, obsessive-compulsive symptoms, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation and psychoticism), followed closely by psychological outcomes such as PTSD, depression and sexual dysfunction (Walsh et al., 2010). Existing findings suggest that the use of avoidance coping strategies is related to a greater risk of suffering from psychological distress as well as depressive symptoms (Brand & Alexander, 2003; Littleton, Horsley, John, & Nelson, 2007; Wright et al., 2007). For example, one meta-analysis that assessed the association between the use of coping strategies following trauma among a sample of 6,747 individuals suggests that avoidance coping strategies were associated with higher general psychological distress, posttraumatic stress symptoms and depression (Littleton et al., 2007). Congruent with these findings, Griffing and colleagues also reported higher levels of depressive symptoms among adult women who reported a greater use of disengagement coping strategies, a coping resembling avoidance (e.g., problem avoidance, social withdrawal) (Griffing et al., 2006a). A similar pattern of findings was revealed among a sample of adult women who were the victims of incest (Brand & Alexander, 2003). Therefore, converging evidence seems to support avoidance coping strategies as a risk factor for depression.

Existing findings also indicates that emotion-oriented coping strategies may be associated with higher levels of psychological distress and depressive symptoms (Futa et al., 2003; Leitenberg et al., 1992; Long & Jackson, 1993; O'Leary, 2009; Runtz & Schallow, 1997). Leitenberg and

colleagues reported that emotion-oriented and avoidance coping strategies (i.e., denial, emotional suppression, cognitive rumination and avoidance) were related to greater psychological distress among adult women (Leitenberg et al., 1992). Analogously, O'Leary reported that emotion-oriented and avoidance coping strategies (i.e., behavioural disengagement, use of emotional social support, acceptance and suppression of competing activities) predicted increased levels of somatic symptoms, anxiety/insomnia, social dysfunction and severe depression in adult men (O'Leary, 2009). In contrast, Runtz & Schallow (1997) reported that emotion-oriented strategy [i.e., expressing emotion (e.g., talking to family and friends about your feelings, giving yourself permission to feel your feelings and considering any feelings to be ok)] was associated with lower levels of psychological distress among maltreated adult women. These inconsistent findings have led investigators to postulate that emotion-oriented coping may include both positive (e.g., emotional processing and expression) and negative strategies (e.g., self-preoccupation and fantasizing) (Austenfeld & Stanton, 2004; Cantón-Cortés & Cantón, 2010), with different effects on adaptation. An alternative hypothesis is that the timing during which they were used (initially or long after the occurrence of the stressful event) may also be associated with differences in psychological adjustment (Lazarus & Folkman, 1984; Sigmon et al., 1997; Wright et al., 2007). In sum, the adaptiveness of emotion-oriented coping strategies is still open to question.

Higher use of task-oriented coping strategies to deal with stressful events has been long hypothesize to predict better adjustment (Cantón-Cortés & Cantón, 2010; Tremblay, Hébert, & Piché, 1999). For example, Runtz & Schallow (1997) reported that actively seeking change and understanding were associated with less psychological distress among maltreated adult women.

In addition, Steel and colleagues revealed that seeking social support was linked with less psychological distress in adult men and women (Steel, Sanna, Hammond, Whipple, & Cross, 2004). However, the association between task-oriented and better adjustment is not documented in all contexts. Effectively, others have also revealed that the use of engagement strategies [similar to task-oriented strategies (e.g., problem-solving, cognitive restructuring)] is not associated with psychological adjustment, including depressive symptoms (Cantón-Cortés & Cantón, 2010; Gibson & Leitenberg, 2001; Hébert, Tremblay, Parent, Daignault, & Piché, 2006). Researchers have even reported that task-oriented strategies was associated with greater psychological distress among adult women (Brand & Alexander, 2003; Daigneault, Hébert, & Tourigny, 2006). In regard to these concurrent findings, it has been hypothesized that the adaptiveness of task-oriented coping strategies may depend on an additional factor : coping efficacy (i.e., the perception that the coping strategies adopted by the individuals were effective in achieving the individuals' goals) (Aldwin & Revenson, 1987). Effectively, one study reported that a greater use of negotiation was associated with lower levels of psychological symptoms when these strategies were perceived as effective. Conversely, these strategies were related to higher psychological distress when they were perceived as being ineffective (Aldwin & Revenson, 1987). In summary, accumulating evidence suggests that a greater tendency to rely on avoidance and emotion-oriented coping strategies to deal with stress may enhance the susceptibility for depressive symptoms. However, inconsistent findings have been reported relative to the adaptability of task-oriented and emotion-oriented coping strategies. Considering the hypothesized role of coping strategies in health outcomes, investigators have started to specify their role in the association linking maltreatment to depressive symptoms.

### 1.3.3 The Mediating and Moderating Hypothesis: Coping Strategies

Childhood maltreatment has been proposed to be associated with a greater inclination to adopt avoidance and emotion-oriented coping strategies. In addition, greater use of these coping strategies has been related to higher levels of depressive symptoms. The exact role of coping strategies remains, nevertheless unclear in the association between maltreatment and depressive symptoms. Two dominant models have been put forward overtime; one that involves coping strategies as an indirect pathway linking maltreatment to depressive symptoms (Figure 2, Path B; mediation model) while the other states that emotion-oriented and avoidance coping strategies may enhance the susceptibility to suffer from depressive symptoms among maltreated individuals (Figure 3; moderation model) (Spaccarelli, 1994; Walsh et al., 2010). The mediation model is, for instance, congruent with the Transactional model of stress and coping of Lazarus & Folkman (1984) and the Transactional model of Spaccarelli (1994), which proposed that exposure to maltreatment may inhibit the acquisition of effective coping strategies, thereby leading to an enhance recourse to maladaptive coping strategies to deal with stress. In turn, greater use of these strategies may contribute to a greater vulnerability for a host of health-related problems, including depression (mediation model) (Lazarus & Folkman, 1984; Spaccarelli, 1994). This hypothesis has received considerable empirical support (Fortier et al., 2009; Merrill, Thomsen, Sinclair, Gold, & Milner, 2001; Rosenthal, Hall, Palm, Batten, & Follette, 2005; Steel et al., 2004). For example, one study revealed that more severe experiences of childhood sexual abuse was associated with a greater use of avoidance coping strategies. In turn, the use of these strategies were related to higher levels of psychological distress (i.e., a composite index of somatization, obsessive-compulsive symptoms, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation and psychoticism) among a

sample of female undergraduate students (Fortier et al., 2009; Rosenthal et al., 2005). In addition, Choi and colleagues showed that exposure to childhood maltreatment was linked to avoidance-oriented and emotion-oriented coping strategies, which in turn, predicted depression in a sample of adult women seeking antenatal care (Choi et al., 2015). Merrill and colleagues also reported that the impact of child sexual abuse severity on psychological distress was entirely accounted for by coping strategies among a sample of female navy recruits. More specifically, a history of severe sexual abuse was related with a higher inclination to adopt constructive (alike task-oriented) and avoidance coping strategies to deal with maltreatment experiences. In turn, constructive coping strategies were associated with less psychological distress, whereas avoidance coping strategies were linked to greater psychological distress (Merrill et al., 2001). Altogether, studies conducted so far suggest that coping strategies could partly explain the association between maltreatment and depressive symptoms.

Even though maltreated children have a greater probability to show higher levels of depressive symptoms, several of them report little to no depressive symptomatology in adulthood (Cicchetti & Rogosch, 2001b). Investigators have thus posited that a greater tendency to adopt maladaptive coping strategies may increase the risk to develop depression among maltreated individuals (moderation model) (Gibbs, 1989; Green, Wilson, & Lindy, 1985; Walsh et al., 2010). However, only a few studies have tested this hypothesis. One study indicated that avoidance-oriented coping strategies modulated the impact of maltreatment on psychological distress (Merrill et al., 2001). Although this investigation reported a significant moderation effect, the authors did not specify the direction of this interaction. Thus, it remains unclear whether a greater use of avoidance-oriented coping strategies conferred a vulnerability for depressive symptoms among

maltreated individuals as suggested in bivariate analyses. Another study revealed that coping strategies modulated the association between periods of hemodialysis and depression in a way that patients who used emotion-oriented coping strategies more often reported higher levels of depressive symptoms, whereas those less incline to use these strategies experienced lesser depressive symptoms (Takaki et al., 2005). Finally, Cantón-Cortés & Cantón (2010) assessed the moderating role of maltreatment in the association between coping strategies and PTSD symptoms, which has been reported to often co-occur with depression (Widom et al., 2007). Their findings showed that among sexually abused adult women, the use of avoidance strategies was related to higher levels of PTSD symptoms in comparison to nonabused participants (Cantón-Cortés & Cantón, 2010).

Considerable empirical support exists for the mediating and moderating role of coping strategies in the association between maltreatment and depressive symptoms, although stronger evidence has been noted for the former model to date. The current state of knowledge is, however, constrained by several limitations. First, most studies have mainly focused on sexually abused females, thereby limiting the generalizability of prior results (O'Leary, 2009; Walsh et al., 2010). Considering the fact that maltreated children often suffer from more than one type of maltreatment, these studies may not adequately capture the full extent of maltreatment on well-being or wrongly attribute the consequences of other forms of victimization to sexual abuse (Hager & Runtz, 2012). Second, while it has been hypothesized that coping may affect the association between maltreatment and depressive symptoms, there are relatively few studies that have formally tested this hypothesis, delineating in which contexts coping strategies enhance

the impact of maltreatment on depressive symptoms (Walsh et al., 2010). For these reasons, the present study will contribute to widen the scope of prior investigations.

### 1.4 Problem Statement and Objectives

It is well established that exposure to maltreatment in childhood poses a risk for the experience of depressive symptoms over long periods. However, the mechanisms involved in this association remain elusive (Liu, 2017). Congruent with the Allostatic load model, the physiological and the psychological stress responses may play a role in the association between childhood maltreatment and depressive symptoms. Theoretical and empirical work indeed suggest that cortisol response to stress could partly account for the association between maltreatment and depressive symptoms (McEwen & Stellar, 1993; Shonkoff, 2010; Yeung et al., 2016). Other findings also shed the light on the possibility that a disrupted cortisol activity may enhance the long-term impact of maltreatment on depressive symptoms (Hagan et al., 2014; Steeger et al., 2017). However, very few studies to date have assessed its mediating role in this association and even fewer did so using a rigorous analytical strategy. Finally, only one investigation has assessed both the mediating and the moderating role of cortisol response to stress in the association linking maltreatment to depressive symptoms (see Badanes et al., 2011). Consequently, additional research is needed to examine whether cortisol response to stress is implicated in the association between maltreatment and depressive symptoms.

Studies assessing the psychological stress response also support coping strategies as playing a role in the association between maltreatment and depressive symptoms (Walsh et al., 2010). Empirical and theoretical research suggests that the use of avoidance-oriented and emotion-

oriented coping strategies may partly explain why maltreated individuals have a greater vulnerability for depression (Walsh et al., 2010). A handful of studies have additionally indicated that coping strategies may buffer or exacerbate the impact of maltreatment on depression (Merrill et al., 2001; Takaki et al., 2005). However, most of these researches have predominantly included sexually abused victims and women, limiting the generalization of prior findings to men and to individuals who were exposed to other types of maltreatment experiences. Furthermore, there is a lack of studies examining the hypothesis suggesting that coping strategies may magnify the impact of maltreatment on depressive symptoms (moderation model). To address these limitations, the role of coping strategies in the association between maltreatment and depressive symptoms needs to be examined further.

This study aims to better understand the role played by cortisol stress response and coping strategies in the association between childhood maltreatment and depressive symptoms. Specifically, two objectives were investigated. Firstly, we tested whether cortisol response to stress and/or coping strategies partly explained the impact of maltreatment on depressive symptoms (mediation model). Secondly, we examined if participants with a history of maltreatment were more likely to report higher depressive symptoms when they showed a deregulated pattern of cortisol response to stress or if they use avoidance-oriented and emotion-oriented coping strategies more frequently (moderation model). We refrained from specifying the hypothesized direction of the moderating role of task-oriented coping strategies considering the inconsistent findings regarding its impact on later adjustment.

Figure 2. Model indicating the mediating role of HPA axis response to stress (Path A) and coping strategies (Path B) in the association between maltreatment and depressive symptoms

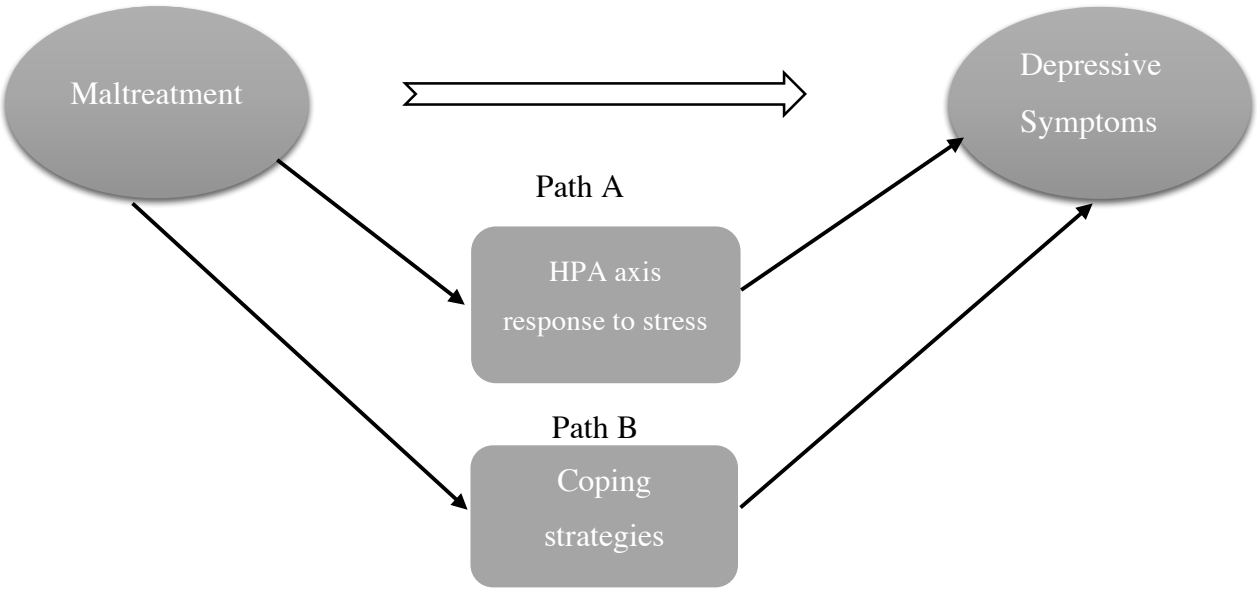
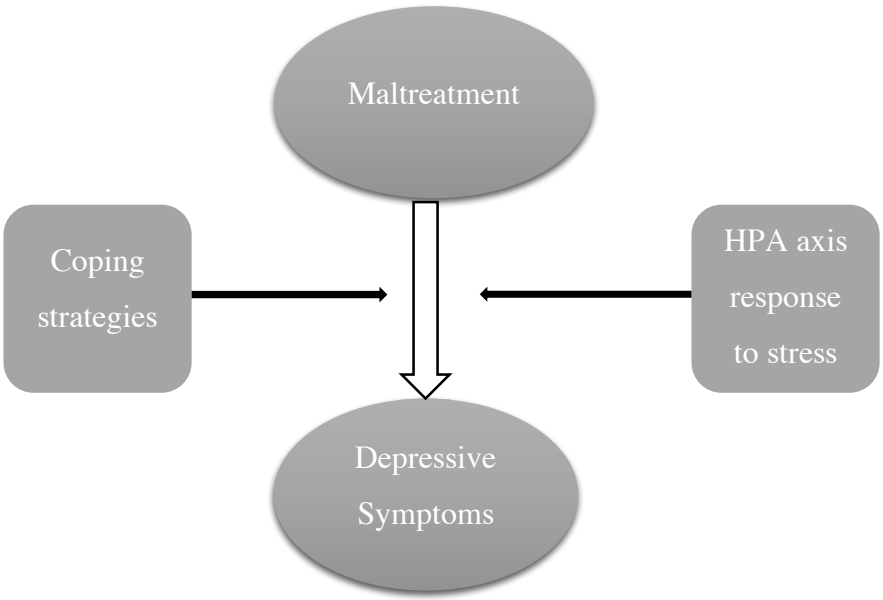



Figure 3. Model indicating the modulating role of HPA axis response to stress and coping strategies in the association between maltreatment and depressive symptoms.



# Chapter 2: Methodology

From a heuristic standpoint, the evidence of an association between a predictor variable and an outcome variable does not translate into a deep understanding of this association (Hayes, 2013). A more profound appreciation of the association between childhood maltreatment and depressive symptoms would theoretically stem from discovering the mechanisms through which maltreatment exerts its long-lasting effect on depressive symptoms (mediation) and in which contexts maltreatment would indeed have an impact on depressive symptoms (moderation) (Hayes, 2013). Therefore, the main objective of the present study is to adopt a mechanistic approach aiming to investigate the moderating and the mediating role of cortisol response to stress and coping strategies in the association linking maltreatment to depressive symptoms. This chapter presents a brief summary of the study from which our data were derived, along with a thorough description of the sample. The procedure chosen as well as a description of the measures used to collect data will be presented. Lastly, the analytical approach selected to test our objectives will be detailed.

### 2.1 Study Sample

Participants were recruited from a larger project entitled: Stress Reactivity and Aggression in Early Adulthood: Do Early Victimization and Regulatory Mechanisms Matter. This study main objective was to identify the mechanisms through which early victimization increases aggression behaviours among a sample of young male adults. The collection took place between July 2013 and December 2014 under the direction of Isabelle Ouellet-Morin.

### 2.2 Participants

Because the study's general objective was to understand the biosocial roots of aggression, and that men are more frequently engaged in these behaviours than women (Archer, 2004), this

investigation only included men. The exclusive selection of men allowed us to avoid the necessity to consider female menstrual cycle phase as well as the use of oral contraception, which have been reported to influence HPA axis activity (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Kudielka & Kirschbaum, 2005). A sample of 160 young men aged from 18 to 31 years ($M_{age}$ = 24.06 years, $SD$ = 3.69) were recruited from the general population. Young adults were targeted for two reasons. First, there is little empirical knowledge about the impact of maltreatment on HPA axis activity and mental health problems during this developmental period (Toth & Cicchetti, 2013). Second, emerging adulthood represents an optimal period to examine mechanisms implicated in the association between maltreatment and depressive symptoms considering that these individuals are at a higher risk for mental health problems (Hagan et al., 2014). Using the manual's recommended classification scores (Bernstein & Fink, 1998), we identified 56 participants (35.9%) who reported experiences suggesting the occurrence of at least one type of maltreatment while the others did not report such experiences (100; 64.1%). Maltreated and nonmaltreated participants were comparable on many sociodemographic factors and lifestyle habits, including their marital status (i.e., single) (87.5% and 85% for maltreated and controls respectively), their reported drug consumption (18.6% and 23% for maltreated and controls respectively) and their cigarette use (25% and 14% for maltreated and controls respectively). Also, the two groups did not differ regarding their age ($t$ (154) = - 0.02, $p$=.34) and their alcohol intake throughout the week ($t$ (154) = -.74, $p$ =.25). However, a greater proportion of maltreated young adults had the flu during the last month in comparison to nonmaltreated young adults ($\chi^2$ (1) = 5.79, $p$ = .02) (see Table 1). Participants ($n$ =1 in the nonmaltreated group, $n$ =3 in the maltreated group) who had previously taken part in research

at the Centre for Studies on Human Stress using the TSST were removed from our analyses because these individuals may have gotten accustomed to the TSST, thus showing a reduced activation of the HPA axis following the stressor. Therefore, our final sample included 156 young men.

Table I. Sociodemographic characteristics of maltreated and nonmaltreated participants

| Characteristics | Nonmaltreated | | | Maltreated | | | | |
|---|---|---|---|---|---|---|---|---|
| | n | % | Mean (SD) | n | % | Mean (SD) | $\chi^2$ | *t* |
| Age | 100 | – | 24.10 (3.78) | 56 | – | 24.09 (3.60) | – | -.02 |
| Single (vs married/common-law partner) | 85 | 85 | – | 49 | 87.5 | – | .18 | – |
| Student (vs employed/unemployed) | 62 | 62 | – | 39 | 69.6 | – | .92 | – |
| College or university degree (vs high school or vocational diploma) | 82 | 82 | – | 40 | 74.1 | – | 1.34 | – |
| Smoker (yes/no) | 14 | 14 | – | 14 | 25 | – | 2.95 | – |
| Alcohol consumption per week | 100 | – | 3.27 (3.80) | 56 | – | 3.75 (4.08) | – | -.74 |
| Drugs consumption (yes/no) | 23 | 23 | – | 16 | 18.6 | – | .59 | – |
| Had flu in the past month (yes/no) | 17 | 17 | – | 19 | 33.9 | – | 5.79* | – |

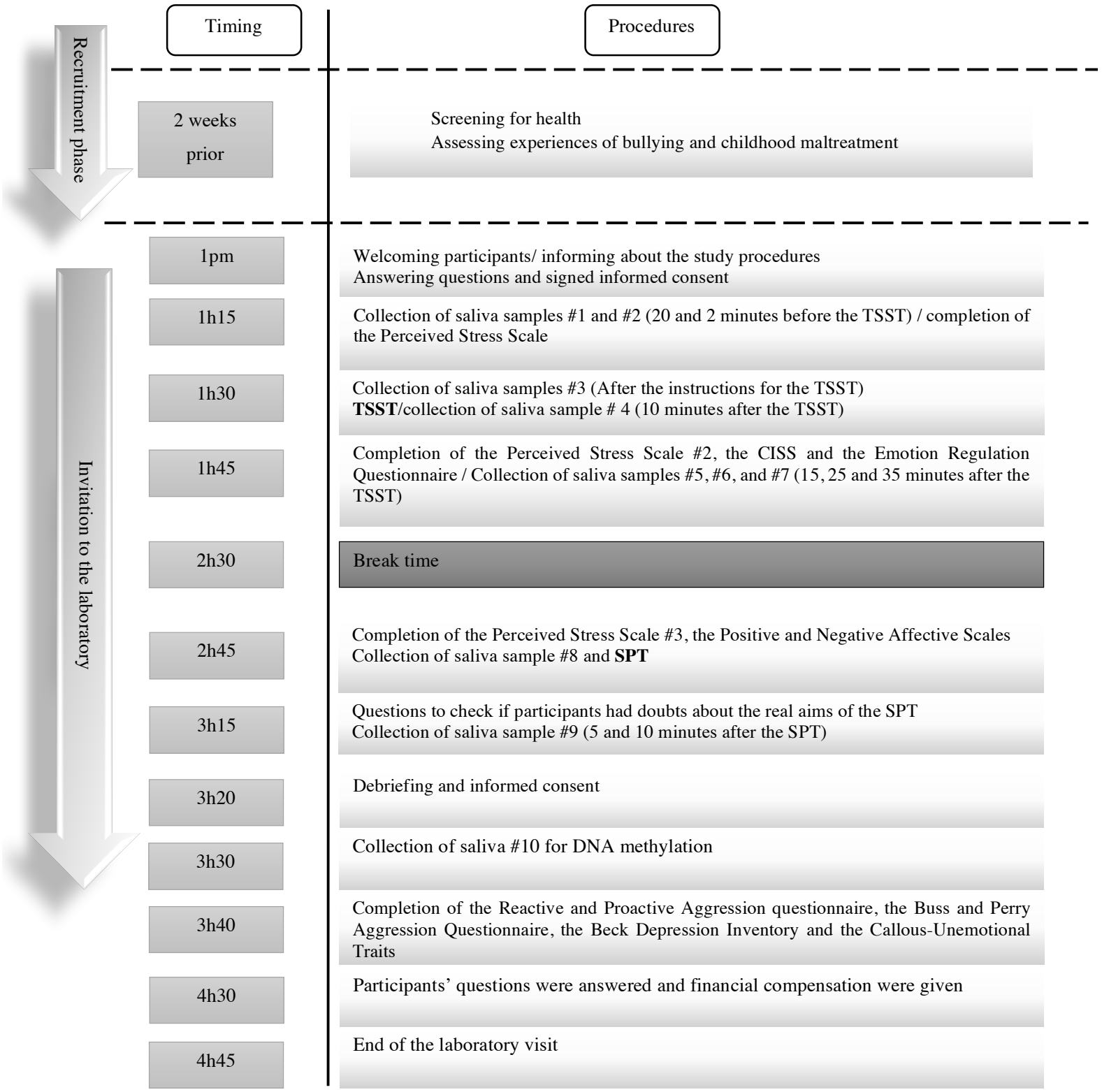
Notes. * = $p < .05$

### 2.3 Procedures

Participants were recruited using ads posted online at the *Centre de recherche de l'Institut universitaire en santé mentale de Montréal's* web site, at the Centre for Studies on Human Stress's web site, on Kijiji (free classified on the Internet) as well as on public billboards. The ads indicated the search for 18 to 35-year-old men living in Montreal and insisted on early life

experiences in order to attract a significant number of participants with early adverse experiences (see Table 2). Trained research assistants conducted a phone interview with interested individuals, screening for health and assessing experiences of bullying (Olweus Bully/victim questionnaire; Olweus, 1996) and childhood maltreatment (CTQ-SF; Bernstein et al., 2003). Eligible participants ($n$ = 160) were invited to pursue to the second part of the study conducted at the Centre for Studies on Human Stress, which lasted approximately 3h30 minutes. At their arrival, participants were once more informed about the study procedures after which they provided signed consent. The visit comprised the completion of two standardized psychosocial tests, the Trier Social Stress Test and the Social Provocation Task, before and following which saliva samples were collected for cortisol, testosterone and DNA methylation of genes associated with stress and emotion regulation. Afterwards, participants responded to self-report questionnaires regarding regulation processes [*Cognitive Emotional Regulation Questionnaire*; (Garnefski & Kraaij, 2007); *Coping Inventory of Stressful Situations*; (Endler & Parker, 1999)], aggression-related phenotypes [*Reactive-Proactive Aggression Questionnaire*; (Raine et al., 2006), *Buss and Perry Aggression Questionnaire*; (Buss & Perry, 1992), *Callous-Unemotional Traits*; (Frick, 2004)], negative affect [*Positive and Negative Affective Scales*; (Watson, Clark, & Tellegen, 1988)], perceived stress [*Perceived Stress Scale*; (Cohen, Kamarck, & Mermelstein, 1983)] and depressive symptoms [*Beck Depression Inventory*; (Beck, Steer, & Brown, 1996)]. The Social Provocation Task (SPT) was used to derive a behavioural-based measure of aggression in a naturalistic context while the Trier Social Stress Test (TSST) was used to induce a reliable physiological stress response. Specifically, the TSST is a well-established, standardized stress paradigm that induces social-evaluative threat (Kirschbaum, Pirke, & Hellhammer, 1993). The protocol included an anticipation phase in

Table II. Illustration of the research procedures



which the participants are given five minutes to prepare for a public speech, followed by the phase during which they had to perform a five-minute speech to convince a "panel of behavioural experts" of their qualification for a chosen job. The participants were later invited to serially substrate as fast and as accurately as possible the number 13 from 2023. Every time they failed, they were asked to start from the beginning by the expert panel. Participants communicated with the panel using an intercom and were filmed with a video camera in a stand-up position in front of a one-way window (Andrews et al., 2007). Both the "panel-in" and "panel-out" methods have been shown to elicit reliable cortisol responses in laboratory settings (Andrews et al., 2007). The TSST took place in the early afternoon for all participants (*M*=13:41, *SD*=0:53). Ethical approval was granted by the Research Center of the Montreal Mental Health University Institute Ethics Committee (Montreal, Canada). Participants received $40 as a compensation for their time. In the present study, the measures of childhood maltreatment, cortisol response to the TSST, coping strategies and depression were considered and thus, described in detail in the following section.

**2.4 Measures**

## 2.4.1. Childhood Maltreatment

Childhood maltreatment was measured with the short form of the *Childhood Trauma Questionnaire* (CTQ-SF), which inquiries about emotional, physical and sexual abuse as well as physical and emotional neglect that have occurred before age 18 (Bernstein et al., 2003). Participants responded to each of the 28 items in the context of "when you were growing up" and answered according to a five-point Likert scale ranging from "never" =1 to "very often" =5, leading to total scores of 5 to 25 on each subscale (plus three items of validity). Acceptable

internal consistency was reported for all subscales (Bernstein et al., 2003). Among this sample, 9.6% ($n$ = 15) experienced physical abuse, 6.4% ($n$ =10) were exposed to sexual abuse, 23.9% ($n$ = 37) experienced emotional abuse, 12.8% ($n$ = 20) had a history of physical neglect and 20.5% ($n$ =32) experienced emotional neglect. According to the questionnaire guidelines (Bernstein et al., 2003), we aggregated participants who had experienced at least one type of childhood maltreatment (maltreated group; $n$ =56, 35.9%) and those who did not (nonmaltreated group; $n$ =100, 64.1%).

### 2.4.2 Cortisol Response to Stress

Cortisol was measured through the collection of five saliva samples via passive drool. The first two samples were collected 20 and 2 minutes before the TSST. The third, fourth and fifth samples were obtained 15, 25 and 35 minutes after the beginning of the TSST. Saliva samples were stored in a -20 °C freezer and analyzed in a single batch at the Centre for Studies on Human Stress with a high sensitivity enzyme immune assay kit (Salimetrics State College, PA, Catalogue No. 1-3102). The range of detection for this assay was between 0.012 and 3 mg/dl and the intra- and inter-assay coefficients of variation were 4.1% and 8.3%, respectively. All samples were assayed in duplicates, winsorized and log-transformed prior to statistical analyses.

### 2.4.3. Coping Strategies

Coping strategies were measured with the *Coping Inventory of Stressful Situations* (CISS), a 48-item self-reported instrument developed to assess individuals' basic coping strategies in the context of stressful situations (Endler & Parker, 1994). The CISS comprises three subscales of

16 items each, measuring task-oriented (e.g., schedule my time better, come up with several different solutions to the problem), emotion-oriented (e.g., blame myself for not knowing what to do, tell myself that it is not really happening to me) and avoidance-oriented coping strategies (e.g., go out for a snack or meal, try to be with other people). Items were rated on a 5-point Likert-type scale with response ranging from "not at all" = 1 to "very much" = 5, higher scores on each subscale indicated greater use of these coping strategies. Highly satisfactory internal consistency has been reported for all subscales (Endler & Parker, 1999). Each subscale was derived by adding up all 16 items. In this sample, the scores varied from 34 to 77 (*M*=58.18, *SD*=10.10) for the task-oriented coping subscale, from 20 to 75 (*M*=42.77, *SD*=9.94) for the emotion-oriented coping subscale and from 18 to 69 (*M*= 39.13, *SD*=11.09) for the avoidance-oriented coping subscale, which parallel the normative range reported for adults by Endler & Parker (1999).

### 2.4.4. Depressive Symptoms

Participants reported their depressive symptoms with the *Beck Depression Inventory-II* (Beck et al., 1996), a widely used 21-item instrument adapted to both clinical and nonclinical populations. Participants had to select the items that best describe the way they felt during the last two weeks. Items were rated on a 4-point Likert-type scale with response ranging from "not present" = 0 to "severe" = 3, with higher scores denoting more severe depressive symptoms. High internal consistency ($\alpha$ = .90) and concurrent validity have been reported for the BDI-II (Storch, Roberti, & Roth, 2004). The total scale was calculated by summing up all the 21 items. In the present sample, participants responses varied from 0 to 44 (*M*=10.48, *SD*=8.79), which is similar to the estimates reported among a normative sample of young adults (Storch et al., 2004).

## 2.5 Statistical Analyses

Preliminary analyses were conducted to evaluate whether participants with a history of maltreatment showed any differences from those who did not report such experiences on their sociodemographic and lifestyle characteristics (i.e., age, marital status, smoking habits, employment status, alcohol consumption per week, drugs consumption and history of flu in the past month) (Table 1). One factor was identified: having had the flu in the past month, which was controlled for in all subsequent analyses. Considering that several factors have been reported to influence HPA axis activity (Nicolson, 2008), additional analyses were carried to identify those uniquely associated with cortisol response to stress (Annexe). Two factors were detected: having had the flu in the past month and being a smoker. Being a smoker was thus also statistically accounted for in all analyses that involved cortisol reactivity. We assessed whether the TSST elicited a physiological stress response by means of a conditional latent growth model (LGM) in Mplus (Version 7.4; Muthén and Muthén, Los Angeles, California). LGM is a special case of structural equation modeling (SEM) which focus on both variable-centered (mean change over time) and person-centered analysis (individual differences in the mean change) (Preacher, Wichman, MacCallum, & Briggs, 2008). This method was selected because it allows to extract, from all five cortisol sample measures, two continuously distributed parameters for subsequent analyses – the cortisol main level (i.e., Intercept) and cortisol response to stress (i.e., Slope). Consistent with a prior examination of the data (Ouellet-Morin et al., under revision), we also confirmed that the TSST indeed triggered an average increment of 0.09 nmol/L (*log transformed base-10*) in this sample (Table 3). The estimated variance component for the slope suggested that sufficient individual differences were present within this sample to test our main hypotheses.

Table III. Fixed, random, and covariance estimates of cortisol change during the baseline and response phases in the total sample of young adults ($n$=156)

| Parameters | Statistics | | |
|---|---|---|---|
| | B | S.E. | Critical ratio |
| *Fixed (means)* | | | |
| Intercept ($y_0$) | 0.66 | 0.03 | 19.21*** |
| Slope ($y_1$) | 0.09 | 0.01 | 8.35*** |
| *Random (variances)* | | | |
| Intercept ($\boldsymbol{\sigma}_0$) | 0.09 | 0.01 | 6.55*** |
| Response slope ($\boldsymbol{\sigma}_1$) | 0.01 | 0.01 | 6.01*** |
| *Covariances* | | | |
| Intercept-response slope ($y_0$, $y_1$) | .01 | .01 | 1.89 |
| Intercept-smoking ($y_0$, $c_1$) | .08 | .07 | .11 |
| Intercept-Flu ($y_0$, $c_2$) | .01 | .07 | .12 |
| Response slope- smoking ($y_1$, $c_1$) | -.07 | .02 | -3.31*** |
| Response slope – Flu ($y_1$, $c_2$) | -.04 | .02 | -2.16* |

Notes. B = unstandardized beta estimates; S.E = Standard error. The critical ratio refers to the ratio of the unstandardized beta estimate over the standard error (B/S.E.). Fit statistics: $\chi^2$ = 350.97, df = 20, CFI = .52, RMSEA = .33, SRMR = .18. * = $p$ <.05; *** = $p$ <.001.

Our main research objectives were tested in two steps. First, we evaluated whether cortisol response to stress (i.e., slope) and/or coping strategies could partly explain the association between maltreatment and depressive symptoms (mediation model). Mediation analysis is a statistical method used to assess whether the association between a predictor variable (X) and an outcome variable (Y) can be explained by their association to a third variable (M) (Field, 2013). Tests of mediation have traditionally adopted the causal steps approach (Baron & Kenny, 1986; Hayes, 2013). According to this statistical framework, in order to claim a significant mediation effect, the investigator needs to establish an association between the predictor (X) and the outcome (Y), between the predictor (X) and the mediator (M) and between the mediator (M) and the outcome (Y) in addition to showing that the association between the predictor (X)

and the outcome (Y) is partially or totally reduced (partial or total mediation) after the inclusion of the mediator (M) in the model (Baron & Kenny, 1986; Hayes, 2013). However, this method does not quantify the magnitude of the indirect effect and does not require any inferential test about this effect (Hayes, 2013). To avoid these limitations, we tested the mediation models with the SPSS macro Process v2.16 (Hayes, 2012), which formally tested the significance of the indirect effect of cortisol response to stress and coping strategies, taken separately, in the association between maltreatment and depressive symptoms. Accordingly, our tests of mediation included a bias-corrected bootstrap confidence interval for the indirect effect based on 10,000 bootstrap samples. Bootstrapping generates an empirically derived representation of the sampling distribution of the indirect effect, which is utilized for the construction of the confidence intervals of its estimate. This approach makes no assumptions about the shape of the distribution of the indirect effect, thus yielding inferences that tend to be more accurate than the normal theory approach (i.e., the Sobel Test) (Hayes, 2013). In order for the mediation test to be considered as significant, the lower and the upper bounds of the confidence interval for the indirect effect need to be entirely above zero (Hayes, 2013). Second, we assessed whether cortisol response to stress and coping strategies, taken separately, had a conditional effect on the association between maltreatment and depressive symptoms using the SPSS macro Process v2.16 (Hayes, 2012). Moderation analysis evaluates the combined effects of two or more predictor variables on an outcome (Field, 2013). Post-hoc analyses of significant interaction effects were probed using the Johnson-Neyman technique, which identifies regions of significance of an interaction effect when the moderator is continuously distributed. Thus, this technique contributes to avoiding the pick-a-point approach traditionally employed to define low, average and high values of the modulator (Hayes, 2013). The Johnson-Neyman technique

computes the ratio of the conditional effect of X (the predictor) on Y (the outcome) given M (the mediator) to its standard error. A *p*-value for the obtained ratio is calculated using the *t* distribution, after which an inference is made. This approach calculates the values of the mediator in a way that the ratio of the conditional effect to its standard error is equivalent to the critical *t* value associated with the level of significance chosen for the inference (Hayes, 2013).

# The Article: Impact of Childhood Maltreatment on Depressive Symptoms in Young Male Adults: The Role of the HPA Axis and Coping Strategies

## Introduction

Childhood maltreatment is a major public health and social concern worldwide (Li et al., 2015). Converging evidence suggests that maltreatment is an important contributor of mental health disorders, with depression at the forefront (Gilbert et al., 2009; Shenk et al., 2015). Depression is ranked as the second leading cause of years lived with disabilities occurring between 15 and 64 years of age, affecting approximately 4% of Canadians each year for a lifetime prevalence of 24.2% among women and 14.2% among men (Ferrari et al., 2013; Patten et al., 2006; Patten, 2009). Childhood maltreatment is also related to a higher risk of suffering from recurrent and persistent depressive episodes (Harkness et al., 2012; Nanni et al., 2012). Despite the consensus regarding the impact of maltreatment on depression, the mechanisms underlying this association remains unclear (Liu, 2017). This is problematic as a better understanding of these processes may, eventually help to improve early detection of risk and promote more effective interventions to support resilience (Liu, 2017; Shenk et al., 2015).

The concept of allostasis and allostatic load have been proposed to better understand how exposure to chronic and/or severe adverse life circumstances, such as childhood maltreatment, contributes to the onset of physical and mental health problems (Danese et al., 2009). Allostasis refers to the process whereby the body regulates vital physiological functions by setting new thresholds for arousal to support optimal adaptation to continually changing demands in the environment (McEwen & Stellar, 1993). In response to stress, allostasis may be indicated by the short-term activation of primary physiological stress mediators [e.g., the cardiovascular system and the hypothalamic-pituitary-adrenal (HPA) axis] (Chrousos & Gold, 1992; Lupien et al., 2006). Under conditions of chronic and repeated stress, however, the enduring mobilization of

primary stress mediators, either through repetitive hits, failure to habituate, prolonged response or inadequate response, is hypothesized to have substantial cost for the body and lead to a cascade of changes in physiological systems, a phenomenon referred to as allostastic load (McEwen & Stellar, 1993). Consistent with the Allostatic load model, exposure to maltreatment early in life is hypothesized to disrupt the activity of primary stress mediators, such as the HPA axis (Danese et al., 2009). Prior research has indeed revealed that maltreatment is associated with a deregulated HPA axis activity taking the form of lower or higher cortisol basal levels and diurnal patterns among children (Bruce et al., 2009; De Bellis et al., 1999), and of which the effects are still perceptible in adulthood (Nicolson et al., 2010; van der Vegt et al., 2009). An altered pattern of cortisol response to psychological stress challenges has also been noted according to past or concurrent experiences of childhood maltreatment (Tarullo & Gunnar, 2006). Most of these studies have reported a lower cortisol response to psychosocial stress among maltreated individuals (Carpenter et al., 2007; Elzinga et al., 2008; MacMillan et al., 2009; Ouellet-Morin et al., 2011; Trickett et al., 2014). Nonetheless, higher cortisol response to psychosocial challenges has also been documented in adolescents and adults exposed to maltreatment (Harkness et al., 2011; Heim et al., 2002; Heim, Ehlert, & Hellhammer, 2000; Rao et al., 2008), in addition to nonsignificant findings (Rao & Morris, 2015). Taken together, these results suggest that childhood maltreatment may place a strain on physiological systems designed to support adaptation to the environment, indicated by an altered pattern of cortisol secretion in both basal, diurnal rhythms and reactive contexts. Although these changes may theoretically be adaptive, protecting the body from the detrimental effects of stress, they may also enhance the risk to develop a host of health-related problems, including depression (Flier et al., 1998; McEwen & Stellar, 1993).

Existing findings also suggest a disrupted HPA axis activity among depressed individuals (Buitelaar, 2013; Heim et al., 2008). Higher basal cortisol levels have been reported among depressed individuals in comparison to control participants (Knorr et al., 2010; Lopez-Duran et al., 2009). Congruent with these findings, one meta-analysis also revealed a higher cortisol response to pharmacological challenges in depressed participants in comparison to controls (Stetler & Miller, 2011). More recent investigations have turned their attention to the cortisol response to psychosocial stress. Whereas the pharmacological stress response, and to a certain extent, basal cortisol indexes reflect the activity of distinct components of the HPA axis, cortisol response to stress mirrors the endogenous activity of the entire HPA axis system (Burke et al., 2005; Young et al., 2000). This HPA index is particular relevant in the context of depression considering that psychosocial stressors activate key emotional circuits that have been reported to be altered in depressed individuals (Dean & Keshavan, 2017; Young et al., 2000). These studies have also reported a deregulated HPA axis response to psychological stress in depressed individuals. Specifically, higher cortisol response to psychological stress (Dockray et al., 2009; Lopez-Duran et al., 2015) and during recovery (Burke et al., 2005) have been found in depressed adolescents and adults, most notably among those with mild/moderate depressive symptoms (Harkness et al., 2011). Lower cortisol response to stress has alternatively been reported in adolescents with a long history of depressive problems (Booij et al., 2013), adolescents with moderate to severe depressive symptoms (Harkness et al., 2011), as well as in adult women with remitted major depression disorder (Ahrens et al., 2008). Again, however, nonsignificant findings have been reported (Ciufolini et al., 2014; Young et al., 2000). Overall, available findings point to possible disruptions in HPA axis activity, which have been hypothesized to mark a higher susceptibility to experience depressive symptoms. However, the association

between HPA axis activity and depressive symptoms have often been investigated without considering the participants' early life experiences, including childhood maltreatment (Hagan et al., 2014; Heim et al., 2008).

By and large, the HPA axis may increase the risk of depression among individuals who have been maltreated as children in two different ways. On the one hand, cortisol response to stress may serve as a mediator, linking maltreatment to later depression such that greater disruptions in HPA axis activity would, in turn, increase the risk for depressive symptoms on the long run (Danese & McEwen, 2012). To the best of our knowledge, only one study has tested the mediating role of cortisol response to stress in this association, indicating that this biological marker did not account for the observed association between maltreatment and internalizing symptoms in a sample of adolescents (Busso et al., 2017). In regard to basal levels and diurnal cortisol patterns, a handful of investigations has formally tested this hypothesis, of which some did not find evidence of a mediating effect of cortisol in this association (Badanes et al., 2011), whereas others reported that a flattened diurnal cortisol profile partly explained the association between the experience of neglect in infancy and higher levels of daily pain, depression and anxiety symptoms in adult men and women (Yeung et al., 2016). In sum, the extent to which cortisol response to stress partly mediate the association recurrently noted between maltreatment and depressive symptoms is still open to question.

On the other hand, the HPA axis activity may also play a moderating role in the association between maltreatment and depressive symptoms. In a nutshell, not all children will suffer from mental health problems following childhood maltreatment (Cicchetti & Rogosch, 2001b).

Increased risk to develop psychopathology following exposure to maltreatment may thus vary according to a host of factors, including biologically-based individual differences, as proposed in the Biological sensitivity to adverse contexts (BSC) model (Boyce & Ellis, 2005). Differences in HPA axis activity of depressed participants who were maltreated and those who were not offer preliminary evidence that the HPA axis may act as a moderator of that association (Heim, Newport, et al., 2000; Heim et al., 2002; Rao & Morris, 2015). However, this possibility needs to be formally tested within a statistical analytic framework that includes an interaction term between the two investigated factors (i.e., maltreatment x cortisol). Only a few studies have offered such a test, reporting, for the most part, evidence for a moderating impact of cortisol in the association linking maltreatment to internalizing symptoms, in which higher cortisol response to stress enhances the risk for higher levels of internalizing symptoms following maltreatment (Badanes et al., 2011; Hagan et al., 2014; Kuhlman et al., 2017; Steeger et al., 2017; Suzuki et al., 2014). For instance, Hagan and colleagues reported that the impact of maltreatment on internalizing symptoms was higher among young adults who also exhibited a greater cortisol response to stress in comparison to those who did not (Hagan et al., 2014). A same pattern of findings was reported in the context of family stress (e.g., financial problems, serious injury or illness of a family member) (Steeger et al., 2017). Taken together, cortisol response to stress has been hypothesized to act as a mediator and a moderator in the association between maltreatment and depressive symptoms. To this day, however, the findings remain inconsistent and to the best of our knowledge, only one study to date have tested both models (see Badanes et al., 2011).

In addition to the neurophysiological mediators, additional mechanisms present at other levels of analysis have been proposed, including the way individuals cope behaviourally and cognitively with challenging situations (McEwen & Stellar, 1993; Walsh et al., 2010). Interestingly, this idea echoes a long history of research in psychology that stems from psychoanalytic and ego theory (Livneh & Martz, 2007; Zeidner & Endler, 1996). Following Lazarus (1966) pioneering work, researchers shifted their focus from the exclusive attention to individual characteristics (i.e., personality attributes and perceptual styles) to adopt a more transactional perspective, whereby psychological stress refers to a particular relationship between the environment and the individual that is evaluated as taxing or exceeding the person resources and endangering his well-being (Lazarus & Folkman, 1984). Accordingly, individuals are proposed to adopt cognitive and behavioural strategies in an attempt to alter or manage the demands of the situation (task-oriented), to regulate their emotional response (emotion-oriented) or to distract themselves from the stressful encounter (avoidance-oriented) (Endler & Parker, 1994; Lazarus & Folkman, 1984). However, when extreme threats, such as maltreatment experiences, occur early in life, at a period when children are expected to have fewer abilities and resources to adapt to their environment, the acquisition of effective coping strategies may be constrained (Lazarus, 1966; Spaccarelli, 1994). Consistent with these theorizations, several studies have reported a greater use of avoidance-oriented and emotion-oriented coping strategies among adults exposed to childhood maltreatment, as a way to either deal with the aftermath of these experiences or in response to other stressful situations (Brand & Alexander, 2003; Hyman et al., 2007; Long & Jackson, 1993; Sigmon et al., 1997; Thabet et al., 2004). Inconsistent findings have also been documented (Coffey et al., 1996; Runtz & Schallow, 1997). It is understandable that maltreated individuals are more likely to adopt avoidance and emotion-

oriented coping strategies considering that they may acutely provide relief and may help to regulate negative emotions. However, these strategies are generally assumed to place individuals at a greater risk to suffer from depression in prolonged stressful situations such as it may be the case for maltreatment (Filipas & Ullman, 2006; Johnson, Sheahan, & Chard, 2004).

An extensive body of work indeed suggests that a greater inclination to adopt emotion-oriented and avoidance-oriented coping strategies is related to higher levels of psychological distress and mental health problems (Filipas & Ullman, 2006; Leitenberg et al., 1992; Runtz & Schallow, 1997), including depressive symptoms (Griffing et al., 2006b; O'Leary, 2009; Wright et al., 2007). Conversely, task-oriented coping strategies is not associated with well-being in all contexts. Indeed, while some studies have reported that inclination to adopt task-oriented coping strategies is associated with better psychological functioning (Runtz & Schallow, 1997), others have either indicated that the use of these strategies is related to psychological distress (Brand & Alexander, 2003; Daigneault et al., 2006), or is without consequences to psychological functioning (Cantón-Cortés & Cantón, 2010; Gibson & Leitenberg, 2001). As a potential explanation for these inconsistent findings, it has been hypothesized that task-oriented coping strategies may jeopardize well-being in situations where these strategies are perceived to be ineffective in achieving the individual's goals, but may have the opposite effect in a context where task-oriented coping strategies resolve the stressful situation (Lazarus & Folkman, 1984).

Building from isolated evidence indicating that coping strategies is related to childhood maltreatment and psychological adjustment, investigators aimed to better understand their role in the association between maltreatment and depression. Coping strategies may serve as both a

mediator or a moderator of later adjustment following maltreatment (Walsh et al., 2010). Theoretical and empirical work indeed suggests that maltreatment may enhance recourse to maladaptive coping strategies to deal with stress which, in turn, contribute to increasing the susceptibility to suffer from depression (mediation model) (Fortier et al., 2009; Merrill et al., 2001; Rosenthal et al., 2005; Spaccarelli, 1994; Steel et al., 2004). For instance, adult women seeking antenatal care with a history of childhood maltreatment were more likely to adopt avoidance and emotion-oriented coping strategies which, in turn, predicted depression (Choi et al., 2015).

An alternative hypothesis is that the impact of maltreatment on depressive symptoms should be magnified among participants who are more prone to use avoidance and emotion-oriented coping strategies (moderation model) (Walsh et al., 2010). However, very few researchers have formally tested coping strategies as a moderator of this association. One study revealed that avoidance coping strategies modulated the association between maltreatment and psychological adjustment, including depressive symptoms (Merrill et al., 2001). However, the authors did not specify the direction of this conditional effect, so it remains unclear if the greater use of avoidance-oriented coping strategies enhanced (or reduced) the risk for depressive symptoms among maltreated individuals. Coping strategies were also found to affect the association between periods of hemodialysis and depression, showing that emotion-oriented coping strategies was related to higher levels of depressive symptoms in patients undergoing long periods of hemodialysis (Takaki et al., 2005). Taken together, coping strategies may either mediate or modulate the association between maltreatment and depressive symptoms, although, to this day, stronger evidence has been noted for the former model. These contributions are,

however, constrained by two main limitations. First, most studies have focus on sexually abused females, thereby limiting the generalizability of prior result to men and individuals who have been exposed to cumulative maltreatment experiences (O'Leary, 2009; Walsh et al., 2010). Second, while coping strategies may enhance or buffer later risk for depressive symptoms among maltreated individuals, few studies have formally tested this hypothesis (Walsh et al., 2010).

This study aims to better understand the role played by cortisol response to stress and coping strategies in the association between childhood maltreatment and depression. Specifically, two objectives were investigated. Firstly, we tested whether cortisol response to stress and/or coping strategies partly explained the impact of maltreatment on depressive symptoms. Secondly, we examined if participants with a history of maltreatment were more likely to report higher levels of depressive symptoms when they showed a deregulated pattern of cortisol response to stress or if avoidance-oriented and emotion-oriented coping strategies were used more often. Considering the inconsistent findings regarding the moderating impact of task-oriented coping strategies on later adjustment, we will refrain from specifying the hypothesized direction of its effect in the association between maltreatment and depressive symptoms.

## Methodology

### Participants

Because the study's general objective was to understand the biosocial roots of aggression, and that men are more frequently engaged in these behaviours than women (Archer, 2004), the sample only comprised men. Participants were recruited using ads posted online and on public

billboards inviting them to participate in a study about early-life experiences. Trained research assistants conducted a phone interview with interested individuals, screening for health and experiences of childhood maltreatment. The short form of the *Childhood Trauma Questionnaire*, which inquiries about emotional, physical and sexual abuse and neglect that have occurred before age 18, was used because of its demonstrated validity in community samples (CTQ-SF; Bernstein et al., 2003). Participants responded to each of the 28 items in the context of "when you were growing up" and answered according to a five-point Likert scale ranging from "never" =1 to "very often" =5, leading to total scores of 5 to 25 on each subscale (plus three items of validity). Acceptable internal consistency has been reported for all subscales (Bernstein et al., 2003). Using the manual's recommended classification scores (Bernstein & Fink, 1998), we identified 56 men (35.9%) who reported experiences suggesting the occurrence of at least one type of maltreatment (i.e., victims), whereas the remaining ($n$=100; controls) were not. The sample thus included 156 participants aged from 18 to 35 years ($M$=24.06, $SD$=3.69).

**Procedures**

We invited the participants to take part in our study, which lasted about three hours 30 minutes. During that time, they participated in the Trier Social Stress Test (TSST), a well-established, standardized stress paradigm that induces social-evaluative threat by subjecting participants to a five-minute mock job interview in front of a "panel of behavioural experts," followed by five minutes of mental arithmetic. Participants were asked to serially substrate, as fast and as accurately as possible, the number 13 from 2023. Every time they made a mistake, they were asked to resume from the beginning by the expert panel. Participants communicated with the panel using an intercom and were filmed with a video camera in a stand-up position in

front of a one-way window (Andrews et al., 2007). Both the "panel-in" and "panel-out" methods have been shown to elicit reliable cortisol responses in laboratory settings (Andrews et al., 2007). The TSST took place in the early afternoon for all participants (*M*=13:41, *SD*=0:53). Ethical approval was granted by the Research Center of the Montreal Mental Health University Institute Ethics Committee (Montreal, Canada).

**Measures**

*Cortisol response to stress* was measured through the collection of five saliva samples via passive drool. The first two samples were collected 20 and 2 minutes before the TSST. The third, fourth and fifth samples were obtained 15, 25 and 35 minutes after the beginning of the TSST. Saliva samples were stored in a -20 °C freezer and analyzed in a single batch at the Centre for Studies on Human Stress with a high sensitivity enzyme immune assay kit (Salimetrics State College, PA, Catalogue No. 1-3102). The range of detection for this assay was between 0.012 and 3 mg/dl and the intra- and inter-assay coefficients of variation were 4.1% and 8.3%, respectively. All samples were assayed in duplicates, winsorized and log-transformed prior to statistical analyses.

*Coping strategies* were measured with the *Coping Inventory of Stressful Situations* (CISS), a 48-item self-reported instrument developed to assess individuals' basic coping strategies in the context of stressful situations (Endler & Parker, 1994). The CISS comprises three subscales of 16 items each measuring task-oriented (e.g., schedule my time better, come up with several different solutions to the problem), emotion-oriented (e.g., blame myself for not knowing what to do, tell myself that it is not really happening to me) and avoidance-oriented coping strategies

(e.g., go out for a snack or meal, try to be with other people). Items were rated on a 5-point Likert-type scale with response ranging from "not at all" = 1 to "very much" = 5, higher scores on each subscale indicated greater use of these coping strategies. Highly satisfactory internal consistency has been reported for all subscales (Endler & Parker, 1999). Each subscale was derived by adding up all 16 items. In this sample, the scores varied from 34 to 77 (*M*=58.18, *SD*=10.10) for the task-oriented coping subscale, from 20 to 75 (*M*=42.77, *SD*=9.94) for the emotion-oriented coping subscale and from 18 to 69 (*M*= 39.13, *SD*=11.09) for the avoidance-oriented coping subscale, which parallel the normative range reported in a sample of adults (Endler & Parker, 1999).

Participants reported their depressive symptoms with the *Beck Depression Inventory-II* (BDI-II) (Beck et al., 1996), a widely used 21-item instrument adapted to both clinical and nonclinical populations. Participants had to select the items that best describe the way they felt during the last two weeks. Items were rated on a 4-point Likert-type scale with response ranging from "not present" = 0 to "severe" = 3, with higher scores denoting more severe depressive symptoms. High internal consistency ($\alpha$ =.90) and concurrent validity have been reported for the BDI-II (Storch et al., 2004). The total scale was calculated by summing up all 21 items. Participants responses varied from 0 to 44 (*M*=10.48, *SD*=8.79), which is similar to the estimate reported among a normative sample of young adults (Storch et al., 2004).

**Statistical Analyses**

Preliminary analyses were conducted to examine whether participants with a history of maltreatment differ in their sociodemographic and lifestyle characteristics (i.e., age, marital status, smokers, employment status, alcohol consumption per week, drugs consumption and

history of flu in the past month) from those who had not (Table 4). One factor was identified: having had the flu in the past month, which was controlled for in all subsequent analyses. Given that many factors have been reported to affect HPA axis activity (Nicolson, 2008), additional analyses were carried to identify those uniquely associated with cortisol response to stress. Two factors were detected: having had the flu in the past month and being a smoker. Being a smoker was thus also statistically accounted for in all analyses that involved cortisol reactivity. Consistent with a prior examination of the data (Ouellet-Morin et al., under revision), we also confirmed that the TSST induced a reliable, significant increase of cortisol in response to the TSST (see Table 5), as tested with a conditional latent growth model (LGM) in Mplus (Version 7.4; Muthén and Muthén, Los Angeles, California). The LGM was selected because it allows to extract two parameters for subsequent analyses – the cortisol main level (i.e., intercept) and cortisol changes over time in response to the TSST (i.e., slope), while controlling for the confounders. Table 5 also revealed that sufficient individual differences were present within this sample to test our main hypotheses, as indicated by the estimated variance component for the slope.

Table IV. Sociodemographic characteristics of maltreated and nonmaltreated participants

| Characteristics | Nonmaltreated | | | Maltreated | | | | |
|---|---|---|---|---|---|---|---|---|
| | n | % | Mean (SD) | n | % | Mean (SD) | $\chi^2$ | *t* |
| Age | 100 | – | 24.10 (3.78) | 56 | – | 24.09 (3.60) | – | -.02 |
| Single (vs married/common-law partner) | 85 | 85 | – | 49 | 87.5 | – | .18 | – |
| Student (vs employed/unemployed) | 62 | 62 | – | 39 | 69.6 | – | .92 | – |
| College and university degree (vs high school and vocational diploma) | 82 | 82 | – | 40 | 74.1 | – | 1.34 | – |
| Smoker (yes/no) | 14 | 14 | – | 14 | 25 | – | 2.95 | – |
| Alcohol consumption per week | 100 | – | 3.27 (3.80) | 56 | – | 3.75 (4.08) | – | -.74 |
| Drugs consumption (yes/no) | 23 | 23 | – | 16 | 18.6 | – | .59 | – |
| Had flu in the past month (yes/no) | 17 | 17 | – | 19 | 33.9 | – | 5.79* | – |

Notes. * = $p < .05$

Main analyses were conducted in two steps. First, we evaluated whether cortisol response to stress (i.e., slope) and/or coping strategies could partly explain the association between maltreatment and depressive symptoms. Tests of mediation have traditionally adopted the causal steps approach (Baron & Kenny, 1986; Hayes, 2013). However, this method does not quantify the magnitude of the indirect effect and does not require any inferential test about this effect. To avoid these limitations, we tested the mediation models with the SPSS macro Process v2.16 (Hayes, 2013), which formally tested the magnitude of the indirect effect of cortisol levels and coping strategies in the association linking maltreatment to depressive symptoms. Accordingly, our tests of mediation included a bias-corrected bootstrap confidence intervals for the indirect effect based on 10,000 bootstrap samples. Bootstrapping generates an empirically derived representation of the sampling distribution of the indirect effect, which is utilized for the construction of the confidence interval of its estimate. In order for the mediation test to be considered as significant, the lower and the upper bounds of the confidence interval for the

indirect effect need to be entirely above zero (Hayes, 2013). Second, we assessed whether cortisol response to stress and coping strategies, taken separately, had a conditional effect on the association linking maltreatment to depressive symptoms, again using the SPSS macro Process v2.16. Post-hoc analyses of significant interaction effects were probed using the Johnson-Neyman technique, which identifies regions of significance of an interaction effect when the moderator is continuously distributed (Hayes, 2013).

Table V. Fixed, random, and covariance estimates of cortisol change during the baseline and response phases in the total sample of young adults ($n$=156)

| Parameters | Statistics | | |
|---|---|---|---|
| | B | S.E. | Critical ratio |
| *Fixed (means)* | | | |
| Intercept ($y_0$) | 0.66 | 0.03 | 19.21*** |
| Slope ($y_1$) | 0.09 | 0.01 | 8.35*** |
| *Random (variances)* | | | |
| Intercept ($\boldsymbol{\sigma}_0$) | 0.09 | 0.01 | 6.55*** |
| Response slope ($\boldsymbol{\sigma}_1$) | 0.01 | 0.01 | 6.01*** |
| *Covariances* | | | |
| Intercept-response slope ($y_0$, $y_1$) | .01 | .01 | 1.89 |
| Intercept-smoking ($y_0$, $c_1$) | .08 | .07 | .11 |
| Intercept-Flu ($y_0$, $c_2$) | .01 | .07 | .12 |
| Response slope- smoking ($y_1$, $c_1$) | -.07 | .02 | -3.31*** |
| Response slope – Flu ($y_1$, $c_2$) | -.04 | .02 | -2.16* |

Notes. B = unstandardized beta estimates; S.E = Standard error. The critical ratio refers to the ratio of the unstandardized beta estimate over the standard error (B/S.E.). Fit statistics: $\chi^2$ = 350.97, df = 20, CFI = .52, RMSEA = .33, SRMR = .18. * = $p$ <.05; *** = $p$ <.001.

## Results

*Did cortisol response to stress explain the association between maltreatment and depressive symptoms?*

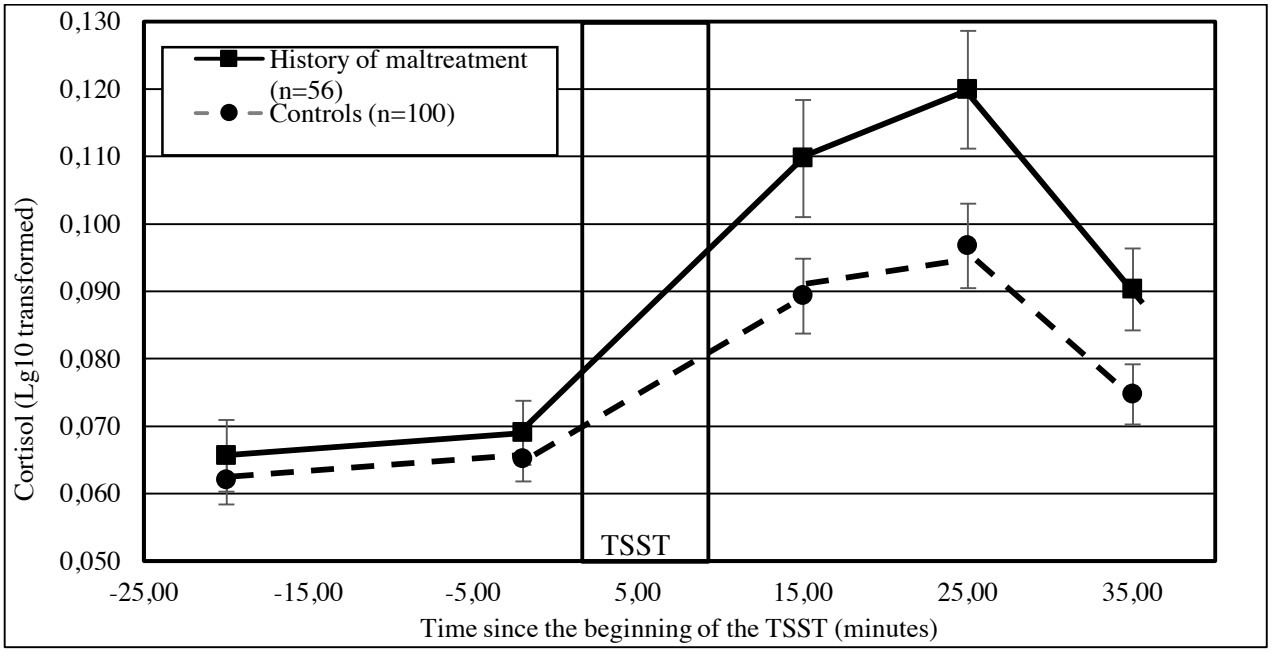
Maltreated participants reported higher levels of depressive symptoms [Mean (SD) = 13.45 (10.39) and Mean (SD) = 8.82 (7.30) for maltreated and controls respectively; $t$ (154) = 3.25, $p$ <.001]. Additionally, as reported previously (Ouellet-Morin et al., under revision), they also exhibited greater cortisol changes over time in response to the TSST compared to the controls (Figure 4) [$t$ (152) = 2.17, $p$ =.03] which, in turn, was associated with lower levels of depressive symptoms [$t$ (151) = -2.32, $p$ =.02]. Of note, the association between maltreatment and depressive symptoms remained significant following the inclusion of cortisol in the predictive model [$t$ (151) = 3.47, $p$ <.001]. We tested whether cortisol response to stress had a significant indirect effect in the association linking maltreatment to depressive symptoms using a bias-corrected bootstrap confidence interval. Our analyses indicated that the bootstrap confidence interval was not above zero (point estimate = -.59, BC 95% CI = -1.75 to -.06), suggesting that cortisol response to the TSST did not mediate the association between maltreatment and depressive symptoms.

*Did coping strategies explain the association between maltreatment and depressive symptoms?*

Maltreated participants adopted emotion-oriented coping strategies more frequently to deal with stressful situations compared to the control group [Mean (SD) = 44.78 (9.50) and Mean (SD) = 41.65 (10.05) for maltreated and controls respectively; $t$ (154) = 1.90, $p$ =.05] which, in turn, was related to higher levels of depressive symptoms. Again, the impact of maltreatment on depressive symptoms remained significant following the inclusion of emotion-oriented coping strategies to the predictive model [$t$ (152) = 6.29, $p$ <.001]. Conversely, emotion-oriented coping strategies had a significant indirect effect, partly explaining the impact of maltreatment on depressive symptoms, as indicated by a bias-corrected bootstrap confidence interval that was

entirely above zero (point estimate = 1.28, BC 95% CI = .03 to 2.85). Maltreatment status was not, however, associated with task-oriented coping strategies [Mean (SD) = 58.21 (11.41) and Mean (SD) = 39.85 (11.45) for maltreated and controls respectively; $t$ (154) = .17, $p$ = .97], although participants who adopted task-oriented strategies more frequently reported lesser depressive symptoms [$t$ (152) = -7.27, $p$ <.001]. Moreover, task-oriented coping strategies did not explain the link between maltreatment and depressive symptoms (point estimate = -.14, BC 95% CI= -1.56 to 1.44). Maltreated participants did not differ either from the controls relative to their use of avoidance-oriented coping strategies [Mean (SD) = 37.84 (10.37) and Mean (SD) = 39.85 (11.45) for maltreated and controls respectively; $t$ (154) = 1.09, $p$ = .28], nor these strategies were associated with depressive symptoms [$t$ (152) = 1.01, $p$ = .31]. Additionally, avoidance-oriented coping strategies did not mediate the association between maltreatment and depressive symptoms (point estimate= -.14, BC 95% CI = -.91 to .10).

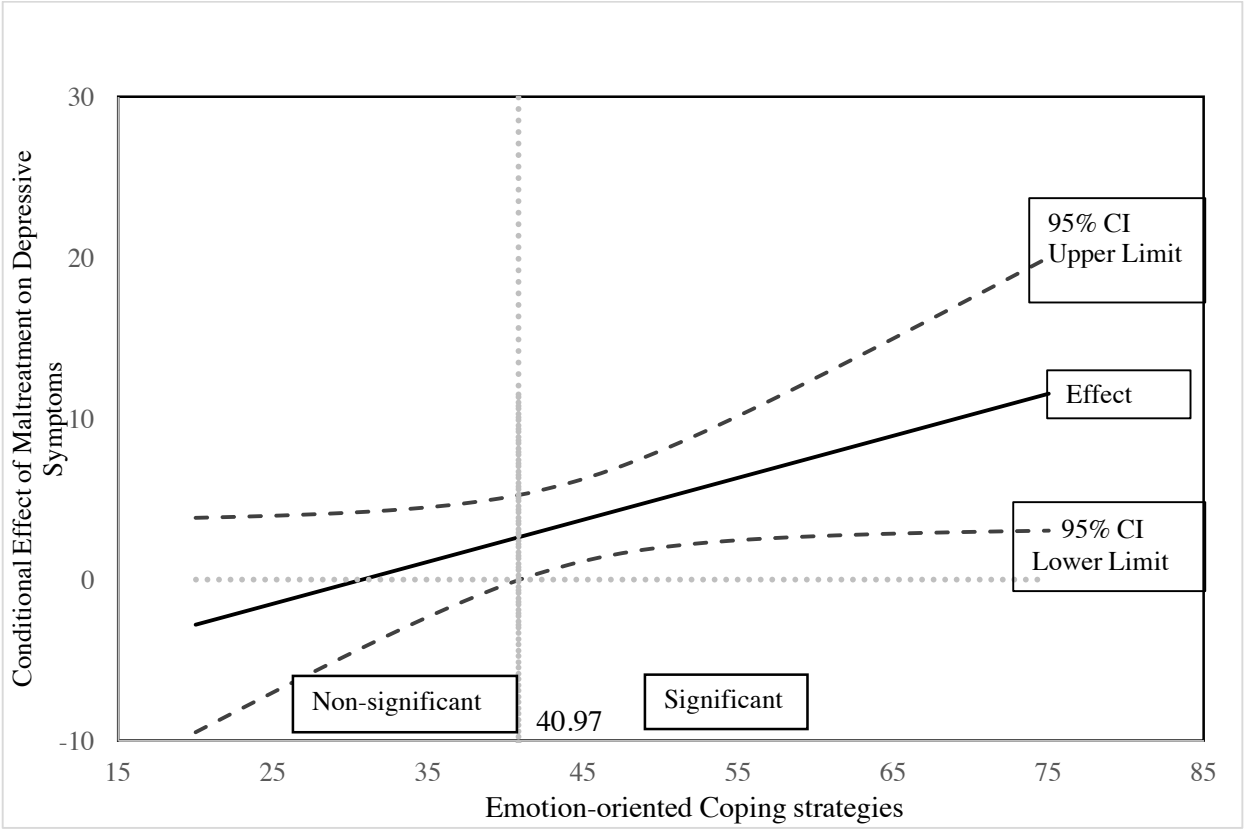
Figure 4. Cortisol response (± SEM) to the TSST according to childhood maltreatment



*Did the association between maltreatment and depressive symptoms vary according to the cortisol response to stress and the use of coping strategies?*

The cortisol response to the TSST did not moderate the association between maltreatment and depressive symptoms [$F$ (3,152) = 5.82, $p$ = .24]. Conversely, the association between maltreatment and depressive symptoms varied according to the frequency participants reported using emotion-oriented coping strategies [$F$ (4,151) = 14.40, $p$ =.05]. Considering the arbitrariness of the pick-a-point approach (i.e., splitting values of the moderator into groups defined as low, average and high) commonly used to illustrate the direction of an interaction, post-hoc analyses were conducted using the Johnson-Neyman technique to see if the effect of maltreatment on depressive symptoms fluctuated alongside the continuum of emotion-oriented coping strategies, thus highlighting where the moderating effect of cortisol is statistically significant and nonsignificant. As can be seen in Figure 5, the two blacks dashed lines represent the 95% confidence interval limits and the solid black line correspond to the conditional effect of maltreatment on depressive symptoms. When the horizontal dotted line touches the confidence interval limits, the conditional effect of maltreatment on depressive symptoms is nonsignificant. The vertical dotted line reveals the position at which the confidence interval limits cross the horizontal dotted line, indicating a significant effect. Figure 5 thus shows that maltreated participants reporting a moderate to high use of emotion-oriented coping strategies to deal with stress (i.e., scored greater or equal to 40.97 on the subscale) had higher depressive symptoms in comparison to the controls. Conversely, the impact of maltreatment on depressive symptoms could not be detected among those who reported using emotion-oriented coping strategies unfrequently (i.e., scored lesser than 40.97 on the subscale).

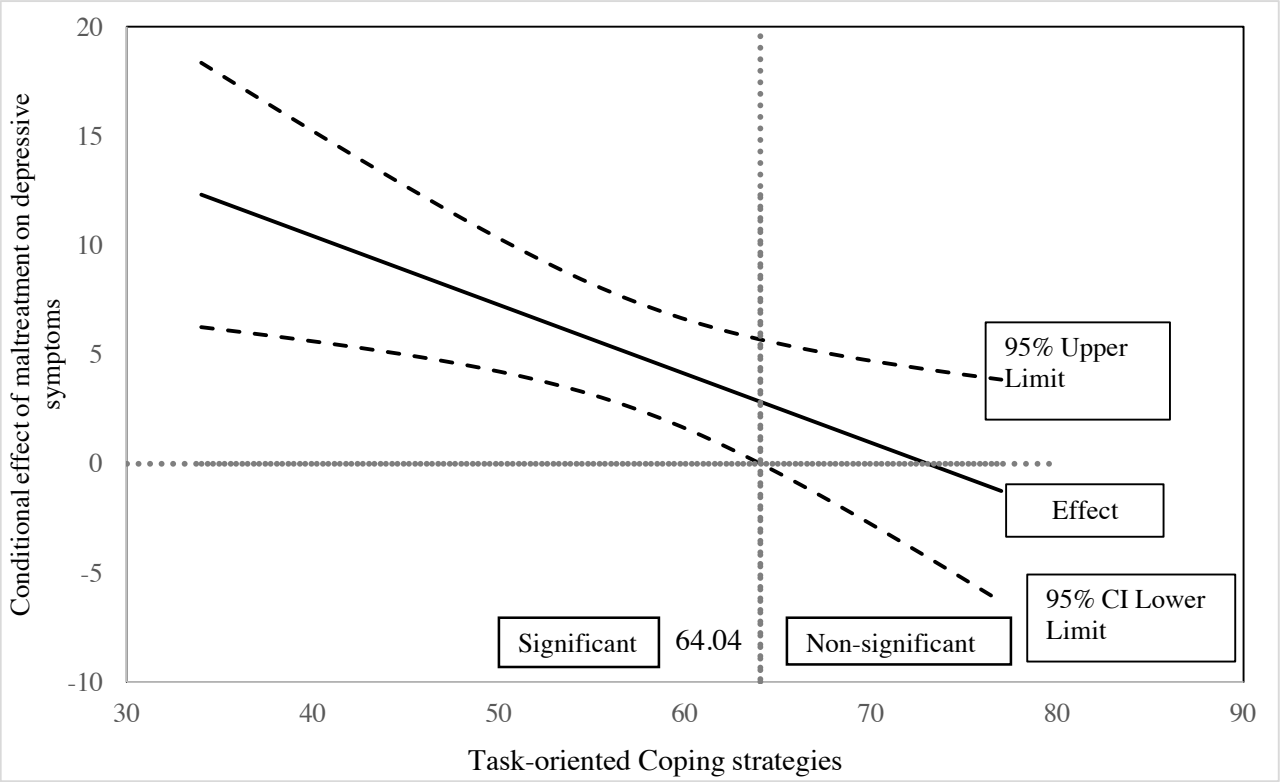
Figure 5. A visual representation of the impact of maltreatment on depressive symptoms according to the use of emotion-oriented coping strategies




Similarly, the association between childhood maltreatment and depressive symptoms varied according to the use of task-oriented coping strategies [$F$ (4,151) = 19.19, $p$ =.008). Post-hoc analyses were once again conducted to identify the boundary conditions of the moderating effect. As shown in Figure 6, maltreated participants evidenced greater levels of depressive symptoms compared to controls among participants reporting lower to moderate use of task-oriented coping strategies (i.e., scored lower and equal to 64.04 on the subscale). Inversely, childhood maltreatment was not associated with depressive symptoms among participants who reported higher use of task-oriented coping strategies (i.e., scored higher than 64.04 on the

subscale). Avoidance-oriented coping strategies did not affect the impact of childhood maltreatment on depressive symptoms [$F$ (4,151) = 2.87, $p$ = .87].

Figure 6. A visual representation of the impact of maltreatment on depressive symptoms according to the use of task-oriented coping strategies



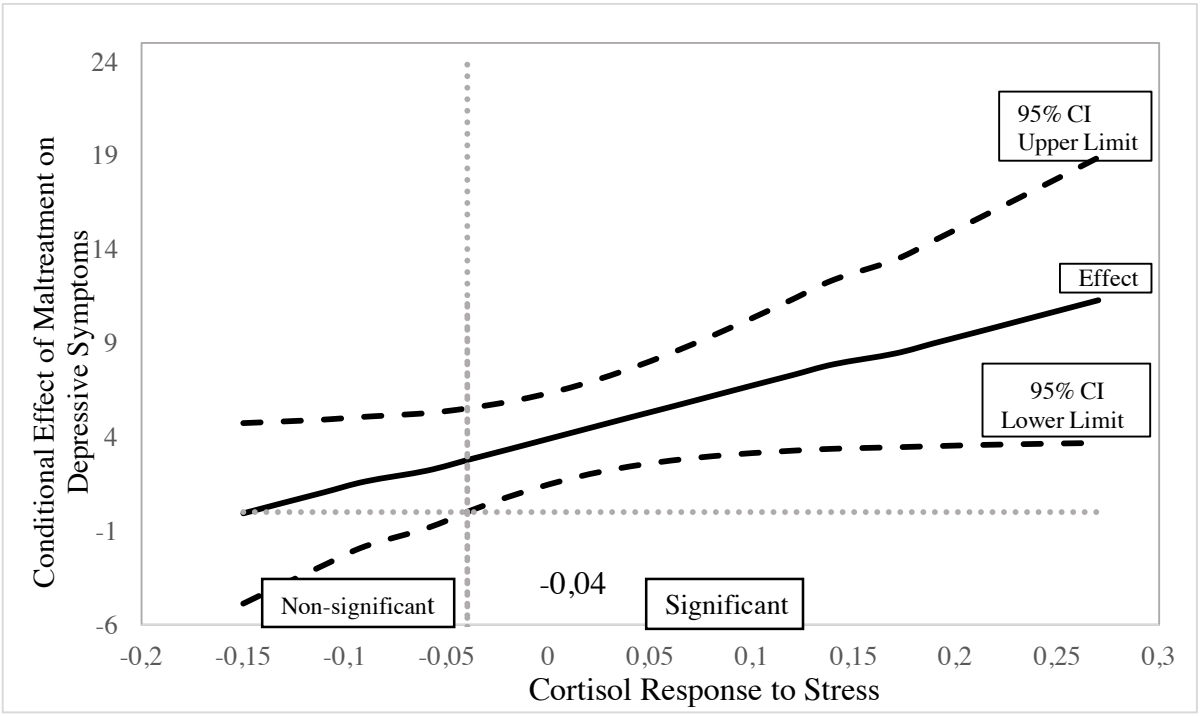

Of note, task-oriented coping strategies were significantly associated with emotion-oriented coping strategies ($r$ =.32, $p$ = .001). Conversely, task-oriented and avoidance coping strategies ($r$ =.14, $p$ = .08) as well as emotion-oriented and avoidance coping strategies ($r$ =.14, $p$ = .09)

only showed a trend for significance. Consequently, we reexamined our mediation and moderation analyses for each coping strategies while controlling for the others to determine whether our findings were specific to each coping strategy. The same pattern of findings was detected for the mediation analyses. Similarly, the results for the moderating role of task-oriented and avoidance-oriented coping strategies in the association linking maltreatment to depressive symptoms were unchanged. However, emotion-oriented coping strategies no longer affected the association between maltreatment and depressive symptoms when task-oriented was accounted for [$F$ (4, 151) = 24.78, $p$ = .36]. Additionally, our mediation and moderation findings remained the same when the area under the curve with respect to increase ($AUC_I$) was used as the physiologic indicator of the responsiveness of the HPA axis instead of the slope, when simultaneously controlling for the first measure of cortisol collected 20 minutes before the TSST. More specifically, maltreated participants showed a greater cortisol response to the TSST in comparison to nonmaltreated individuals [$t$ (150) = 2.01, $p$ =.05], which in turn, was associated with lower levels of depressive symptoms [$t$ (152) = -3.12, $p$ <.001]. However, cortisol response to stress did not affect [$F$ (3, 151) = 7.07, p=.39] nor did it accounts for the association linking maltreatment to depressive symptoms (point estimate = -.67, BC 95% CI = -1.79 to -.08).

Considering that cortisol response to stress was marginally associated with emotion-oriented ($r$ = -.14, $p$ = .07) and significantly associated with task-oriented coping strategies ($r$ = .17, $p$ = .03), we rerun our mediation and moderation analyses for these coping strategies while statistically controlling for the impact of cortisol response to stress. The same pattern of findings emerged, indicating that cortisol response to stress did not explain our results. For the same

reasons, we reanalyzed the mediation and moderation models for cortisol response to stress while simultaneously accounting for emotion-oriented and task-oriented coping strategies. While our findings concerning the mediation role of cortisol response to stress were replicated, we found that the association between maltreatment and depressive symptoms varied according to the magnitude of participants' cortisol response to stress [$F$ (8, 147) = 14,91, $p$ = .05]. As can be seen in Figure 7, maltreated individuals who displayed moderate to high levels of cortisol response to stress reported higher levels of depressive symptoms (i.e., showed a response greater or equal to -.04 on the continuum). Inversely, childhood maltreatment did not influence depressive symptoms among participants who evidenced a lower cortisol response to stress (i.e., showed a response lower than -.04 on the continuum).

Figure 7. A visual representation of the impact of maltreatment on depressive symptoms according to the cortisol response to stress



## Discussion

The present study examined the role of cortisol response to stress and coping strategies in the association between maltreatment and depressive symptoms. Our results extended current knowledge, some of which were in line with earlier theorizations and empirical evidence. Three findings have retained our attention. First, this study showed that lower cortisol response to stress is related to higher levels of depressive symptoms. On the one hand, this finding is consistent with previous investigations suggesting a lower cortisol response to stress in depressed individuals (Ahrens et al., 2008; Booij et al., 2013; Chopra et al., 2009; de Rooij, Schene, Phillips, & Roseboom, 2010; Sun, Deng, Liu, & Tao, 2015). Congruent with the Attenuation hypothesis (Heim et al., 2008; Susman, 2006) and the Allostatic load model (McEwen & Stellar, 1993), it is conceivable that sustained exposure to childhood maltreatment experiences resulted in prolonged periods of elevated activity of the HPA axis, leading subsequently to a shift from higher to lower cortisol response to stress (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Gunnar & Vazquez, 2001). While this change hypothesized to occur over time may acutely protect the body from the persistent effects of stress, it may also mark higher risk for psychopathology in the long run (Shenk, Noll, Putnam, & Trickett, 2010). On the other hand, these results are inconsistent with several other studies suggesting higher HPA axis output in depressed participants according to several HPA axis components (e.g., basal cortisol levels, diurnal rhythm, cortisol response to psychological stress and cortisol reactivity to pharmacological stress) (Burke et al., 2005; Knorr et al., 2010; Lopez-Duran et al., 2009; Staufenbiel, Penninx, Spijker, Elzinga, & van Rossum, 2013; Young et al., 2000). Many factors could theoretically explain this divergence. To begin with, the type of HPA axis output investigated could account for some inconsistencies. Admittedly, the most part of the work

conducted to examine the role of HPA axis activity among depressed individuals concerns cortisol response to pharmacological challenges (i.e., administration of exogenous substances, such as dexamethasone and CRH, that mimic the activity of the HPA axis main components), thus making it difficult to directly compare our results to previous investigations (Burke et al., 2005). Psychological stress test has been hypothesized to assess the activity of the HPA axis, and perhaps, more importantly, its suprahypothalamic input (Burke et al., 2005; Young et al., 2000). Conversely, what is referred to as the cortisol response to exogenous pharmacological challenges reflects an individual's ability to turn off his or her own production of cortisol via negative feedback mechanisms following the stressor (Burke et al., 2005), thus offering a complementary view of the hypothesized impact of maltreatment on HPA axis activity. Nonetheless, other studies that have assessed cortisol response to psychological stress have reported higher cortisol response to stress in depressed individuals (Bagley et al., 2011; Dockray et al., 2009; Lopez-Duran et al., 2009; Weinstein et al., 2010), suggesting that other factors beside the HPA axis component must be implicated.

A second factor that may partly explain the incongruent findings is the severity, the types and the clinical course of depression (de Rooij et al., 2010; Gold & Chrousos, 2002; Höhne et al., 2014). Few studies to date have, however, examined the impact of these indicators on cortisol response to psychosocial stress (Ahrens et al., 2008; Booij et al., 2013; de Rooij et al., 2010; Guerry & Hastings, 2011; Harkness et al., 2011). In regard to depression severity, previous studies revealed a higher cortisol response to stress among depressed individuals with mild/moderate depressive symptoms while lower cortisol response was found in those with moderate/severe depressive symptoms (de Rooij et al., 2010; Harkness et al., 2011). These

findings could be considered consistent with the current study findings, which indicated that a lower cortisol response to stress is related to more severe depressive symptoms, but is at odds with Weinstein and colleagues, who reported greater cortisol response to stress in clinically depressed adults (Weinstein et al., 2010). Alternatively, it could be argued that the association between maltreatment and cortisol response to stress vary as a function of the clinical picture of individuals reporting depressive symptoms. That is, beside the core criterion symptoms of depression – a persistent emotion of sadness and a markedly reduced interest or pleasure in almost all activities – (American Psychiatric Association, 2013), symptoms and experiences may fluctuate from one individual to another (O'Keane, Frodl, & Dinan, 2012). For instance, some depressed patients may display symptoms of insomnia, anorexia and motor agitation (i.e., melancholic depression) whereas others may display hypersomnia, hyperphagia and leaden paralysis (i.e., atypical depression) (American Psychiatric Association, 2013). Accordingly, higher HPA axis activity has been reported in melancholic depression while a blunted stress response was more likely to arise in the context of atypical depression (Gold & Chrousos, 2002; Kaestner et al., 2005; Lamers et al., 2012; Stetler & Miller, 2011; Stewart, Quitkin, McGrath, & Klein, 2005). Additionally, the direction of cortisol response to stress may also depend on the clinical course of depression (Ahrens et al., 2008; Bagley et al., 2011; Booij et al., 2013). For instance, Booij and colleagues found that transient depressive symptomatology is associated with a higher cortisol response to stress whereas more chronic depressive symptomatology is related to a lower cortisol response to stress (Booij et al., 2013), indicating that the persistence of depressive symptoms may be nonlinearly associated with cortisol response to stress. We can only speculate on the factors that may explain the distinct patterns of disrupted cortisol response to stress in regard to depression as no information was collected about the persistence and

clinical presentation of depressive symptoms in our sample. Future studies assessing the severity, the types and the clinical course of depression may thus offer a more complete investigation of the hypothesized role of cortisol response to psychosocial stress.

Second, contrary to our expectations, individual differences in cortisol response to stress did not account for the association between maltreatment and depressive symptoms. As argued by Badanes and colleagues, an atypical cortisol response to stress thought to arise following maltreatment and to persist into adulthood could simply be an indicator of the individual internal representations of the perceived stress as well as his incapacity to actively cope with it (Badanes et al., 2011). The lower cortisol response to stress marginally associated with a greater use of emotion-oriented coping strategies and significantly with a lower use of task-oriented coping strategies in our study offer partial support for this hypothesis. That is, disrupted cortisol response to stress may merely constitute a marker of biological vulnerability for depression without being directly involved in its causal process. This could explain why several investigations also failed to report cortisol as a mediator of the association between maltreatment and internalizing symptoms (Badanes et al., 2011; Busso et al., 2017; Shenk et al., 2015). Alternatively, the current study revealed that a greater cortisol response to stress strengthened the impact of childhood maltreatment on depressive symptoms (moderation model). Interestingly, our results converge with past studies supporting the moderating role of cortisol response to stress in the association between maltreatment and internalizing symptoms (Badanes et al., 2011; Hagan et al., 2014; Kuhlman et al., 2017; Steeger et al., 2017; Suzuki et al., 2014). Our results are also congruent with the Biological sensitivity to context (BSC) theory, which proposed that individuals with high stress reactivity may have a higher sensitivity to the negative

health effects of early life adversity whereas positive health effects may be promoted in stable and low-stress environments (Boyce & Ellis, 2005). However, other studies have also highlighted the enhanced impact of physical abuse on internalizing symptoms among adolescents showing a lower cortisol response to stress (Kuhlman et al., 2017). These differences in findings may partly arise from the developmental period assessed (adolescence compared to young adulthood) as well as the type of childhood adversity evaluated (maltreatment subtype in comparison to the cumulative impact of maltreatment). More importantly, these inconsistent findings underline the importance of systematically assessing the moderating role of cortisol in this association as well as the need for future studies to report nonsignificant findings in order to contextualize when cortisol is expected to affect the association between maltreatment and depressive symptoms and when it does not.

Third, although investigators increasingly acknowledge the need to integrate indicators drawn from multiple pathways hypothesized to explain individual differences in functioning, from normative to pathological, relatively few empirical studies have embarked on this path (Cicchetti, 2006). With this goal in mind, the present study was the first to consider the influence of both cortisol response to stress and coping strategies in the association between maltreatment and depressive symptoms. While existing literature had reported associations between coping strategies and HPA axis activity [for a more in depth review, see Olff, Langeland, & Gersons (2005)], these two processes are not synonymous. That is, considering coping strategies may provide an additional light, enabling us to appreciate how individuals react cognitively and behaviourally to stress, not just physiologically (Lazarus & Folkman, 1984). Converging with prior findings (Choi et al., 2015), we noted that maltreated individuals were more likely to adopt

emotion-oriented coping strategies, which in turn, were related to higher levels of depressive symptoms. Thus, our investigation support prior findings suggesting that these strategies are associated with higher levels of psychological distress (Futa et al., 2003; Leitenberg et al., 1992; Long & Jackson, 1993; O'Leary, 2009; Runtz & Schallow, 1997). Of note, in response to a perceived threat, individuals are thought to select coping strategies that they deem appropriate to terminate the stressful situation (Lazarus & Folkman, 1984). When the event is evaluated as minimally stressful, the three forms of coping strategies will be used at a similar frequency. Conversely, at moderate levels of perceived stress, task-oriented coping strategies predominate while at higher levels of stress, emotion-oriented and avoidance coping strategies are the dominant response (Lazarus & Folkman, 1984). Accordingly, when confronted to maltreatment, children may be more prone to use primitive, desperate emotion-oriented coping strategies to deal with these stressful situations, because focussing on more internal experiences may reduce more effectively distress, shame and helplessness (Hager & Runtz, 2012; Lazarus & Folkman, 1984; Spaccarelli, 1994). However, the use of emotion-oriented coping strategies may become, over time, a predominant response to all types of stressors regardless of the perceived stress level (Hager & Runtz, 2012), because of the maladaptive cognitive schemata induced by childhood maltreatment experiences and thus, to appraise stressful situations in a negative and distortional manner later in life (Beck, 1979, 2008). Such type of appraisals may have a cost, increasing the level of perceived stress of often otherwise benign situations and, accordingly, rely more heavily on emotion-oriented coping strategies to deal with them (Lazarus & Folkman, 1984). As suggested by Hager and Runtz, a greater tendency to adopt emotion-oriented coping strategies to deal with stress while not taking the necessary step to resolve the stressful situation may exacerbate stress and increase risk for a host of stress-related problems, including

depression (Hager & Runtz, 2012). In contrast to prior investigations (Fortier et al., 2009; Merrill et al., 2001; Rosenthal et al., 2005; Steel et al., 2004), however task-oriented and avoidance coping strategies did not explain the impact of maltreatment on depressive symptoms, only emotion-oriented coping did. Task-oriented as well as avoidance coping strategies were neither associated with maltreatment experiences; maltreated and nonmaltreated participants were equally prone to adopt task-oriented and avoidance coping strategies to deal with stress (Hager & Runtz, 2012).

Consistent with studies that have reported that task-oriented coping strategies improved psychological functioning (Bal, Van Oost, De Bourdeaudhuij, & Crombez, 2003; Merrill et al., 2001; Runtz & Schallow, 1997; Steel et al., 2004), we found that maltreated individuals' who had a greater tendency to adopt these strategies to manage stressful situations also report lower levels of depressive symptoms in comparison to those who did not. A more direct comparison of this finding (moderation) is, however, difficult to make because of the paucity of studies that have formally tested this possibility. As a noteworthy exception, one study revealed that the exposure to more stressful environments were related with fewer psychological symptoms among healthy individuals who used task-oriented coping strategies more frequently (Aldwin & Revenson, 1987), suggesting that these strategies may have a buffering effect (i.e., reduce the impact of stress on psychological functioning). Still, not all task-oriented coping strategies seems to protect individuals from the negative effect of stress. Aldwin & Revenson (1987) also reported that the use of negotiation to deal with stressful situations increased, rather than decreased, psychological symptoms under high stress conditions. These results may help to reconcile prior findings indicating that task-oriented coping strategies are associated with both

less (Runtz & Schallow, 1997; Steel et al., 2004) and greater psychological distress (Brand & Alexander, 2003; Daigneault et al., 2006). Accordingly, task-oriented coping strategies may, on the one hand, enhance the active cognitive processing needed to assimilate and resolve controllable stressful events, leading to better psychological adjustment (Wright et al., 2007). On the other hand, stress could also interfere with task-oriented coping strategies functioning (Lazarus & Folkman, 1984) and the perception of achieving ones' goals following their adoption (i.e., coping efficacy), especially considering that their implementation requires substantial resources, inadvertently leading to higher psychological distress (Aldwin & Revenson, 1987)

Unexpectedly, avoidance-oriented coping strategies were not related to either childhood maltreatment or depressive symptoms. These results depart from past investigations suggesting that maltreated individuals have a greater inclination to use these strategies, which in turn, are associated with higher levels of psychological impairment, including depressive symptoms (Choi et al., 2015; Fortier et al., 2009; Merrill et al., 2001; Rosenthal et al., 2005; Steel et al., 2004). We speculate that another pattern of findings may have emerged in female participants because of their greater tendency to engage in avoidance strategies. Specifically, men are reported to rely more on task-oriented coping strategies when confronted to stress whereas women seem to be more prone to adopt emotion-oriented and avoidance coping strategies (Beasley, Thompson, & Davidson, 2003; Cohan, Jang, & Stein, 2006; Folkman & Lazarus, 1980; Pearlin & Schooler, 1978; Stone & Neale, 1984; Tamres, Janicki, & Helgeson, 2002). Congruent with these findings, prior studies have additionally reported that avoidance-oriented coping strategies were not significantly associated with depressive symptoms in male undergraduate students (who reported comparable average use of avoidance as our sample)

while it was related to higher levels of depressive symptoms in female undergraduate students (Cohan et al., 2006; Endler & Parker, 1999). The fact that our participants used avoidance-oriented coping strategies less frequently than the other types of coping strategies or the lack of differences between the maltreated and the nonmaltreated group in their use of avoidance coping strategies may have decreased statistical power, reducing our ability to find significant effects.

The findings of this study should be considered in light of some limitations. First, our sample consisted of young adult male participants. Sex and gender differences in relation to cortisol (Doom, Cicchetti, & Rogosch, 2014; Power et al., 2012) and coping strategies (Tamres et al., 2002) have been highlighted in previous investigations, suggesting that our findings may not be generalizable to female participants. Second, our study relied on retrospective, self-reported childhood maltreatment experiences, which may be subject to recall biases (Cicchetti & Toth, 2005; Gilbert et al., 2009). However, past research revealed that recollection of childhood maltreatment experiences seems to be reliable in adults (Bifulco, Brown, Lillie, & Jarvis, 1997), with recall bias explaining fewer than 1% of childhood maltreatment variance (Fergusson, Horwood, & Boden, 2011). Third, we did not assess the severity and chronicity of maltreatment experiences. Considering that these factors are thought to influence HPA axis activity (Cicchetti & Rogosch, 2001a), further studies assessing the severity and chronicity of maltreatment experiences are needed in order to gain a more complete understanding of HPA axis activity among maltreated individuals. Fourth, we evaluated the association between coping strategies and depressive symptoms without taking into account the efficacy of coping strategies, that is, if participants perceived the coping strategies adopted as effective in solving the stressful situation. Future studies should consider this factor, as coping efficacy may outline the context

in which coping strategies are associated with well-being and the context in which they are not. Fifth, this study is cross-sectional. Thus, the temporal sequence of events cannot be determined. This is problematic because coping strategies and depressive symptoms may have reciprocal influences on one another. It is possible that the greater is the level of depressive symptoms, the more individuals tend to adopt emotion-oriented coping strategies. Greater use of these strategies may, in turn, further enhance depressive symptoms severity (Aldwin & Revenson, 1987). Because of the nature of the present study, we were unable to test this hypothesis.

Our findings have clinical implications. This study landed support to coping strategies as a target for treatments of depressive symptoms among adults who were maltreated as children and more specifically, to minimize the use of negative emotion-oriented strategies while seeking more often task-oriented coping strategies to find a solution for stressful situations (Steinhardt & Dolbier, 2008). More generally, our findings are compatible with cognitive-behavioural therapeutic programs that highlight the mediating role of cognitive processes in supporting or reducing maladaptive patterns (Butler, Chapman, Forman, & Beck, 2006; Cuijpers et al., 2013; March et al., 2004). Interestingly, Harkness and colleagues reported that depressive patients with severe experiences of maltreatment were more responsive to cognitive-behavioural therapy or antidepressant medication than to interpersonal therapy (Harkness et al., 2012). Also, depressed adults who were exposed to maltreatment benefitted more from cognitive behavioural analysis system of psychotherapy (i.e., cognitive behaviour therapy with elements of interpersonal therapy) than medication (Korotana, Dobson, Pusch, & Josephson, 2016). Although our findings suggested that both task-oriented and emotion-oriented coping strategies are involved in depression, the current study does not inform about the adaptiveness of coping

strategies in the short or the long term. That is, whether these strategies are more beneficial in the short or the long-term. Future longitudinal research needs to examine if the adaptiveness of coping strategies varies as a function of the timeline (short versus long-term), as a better understanding of these factors may provide more informative and readily transferable knowledge to clinicians.

**Conclusion**

To conclude, our findings extend existing research on how disruptions in cortisol stress response and the use of coping strategies may represent pathways by which childhood maltreatment affects later depressive symptoms. Our results suggested that disruptions in cortisol response to stress magnified the impact of maltreatment on depressive symptoms but does not explain the higher level of symptomatology in adults with a history of maltreatment. Conversely, the way individuals cope with stressful situations seems to be more strongly linked with this phenotype. These findings give support to the adoption of a multilevel perspective, including neurophysiological and psychological mechanisms delineating complementary processes among maltreated individuals (Toth & Cicchetti, 2013). Ascertaining the specific role of each hypothesized pathway could eventually be used as leverage to reduce vulnerability and promote resilience in adults exposed to maltreatment as children.

# Discussion

The main objective of this master-degree thesis was to assess the contribution of cortisol response to stress and coping strategies in the association linking childhood maltreatment to depressive symptoms among a sample of young adult men. In this context, two specific objectives were pursued. First, we investigated whether cortisol response to stress and/or coping strategies partly accounted for (explained) the association recurrently noted between maltreatment and depressive symptoms. Second, we evaluated whether participants with a history of maltreatment were more prone to report higher levels of depressive symptoms when they exhibited a dysregulated pattern of cortisol response to stress or if they adopted avoidance or emotion-oriented coping strategies more often. Given the inconsistent findings relative to the impact of task-oriented coping strategies on later adjustment, we abstained from stipulating a hypothesized direction for its modulating role in the association between maltreatment and depressive symptoms. Our results suggested that maltreated participants were more inclined to adopt emotion-oriented coping strategies to deal with stressful situations which, in turn, were associated with higher levels of depressive symptoms. Moreover, among participants who used task-oriented coping strategies less frequently, maltreatment had a stronger impact on depressive symptoms. Although maltreatment was found to be associated with a greater cortisol response to stress, which was additionally related to lesser depressive symptoms, cortisol response to stress did not play a mediating role in this association. However, maltreated participants who evidenced a greater cortisol response to stress reported higher levels of depressive symptoms in comparison to those who showed a lower cortisol response to stress. In line with our objectives, the current master-degree thesis made four contributions to the empirical literature. First, this is the first study to inquire about the role of cortisol response to stress and coping strategies in the association linking childhood maltreatment to depressive

symptoms, thus providing a more complete evaluation of the neurophysiological and psychological pathways that are thought to be involved in this association. Second, we built on the existing literature on coping strategies in the context of maltreatment to expand to adult men exposed to a wide range of maltreatment experiences in childhood. Third, we are among the few first studies to have formally tested the modulating role of HPA axis activity and coping strategies in the association between maltreatment and depressive symptoms. Fourth, to the best of our knowledge, we were the first to provide empirical support for the moderating role of task-oriented coping strategies in the association between maltreatment and depressive symptoms. In this chapter, two noteworthy features of the findings will be thoroughly discussed. Subsequently, we will present some limitations inherent to this study, along with promising directions for future studies. Lastly, we will conclude with the clinical implications of the research findings in regard to mental health.

### 4.1. The Role of the HPA axis in the Association Linking Maltreatment to Depressive Symptoms

Our findings suggested that cortisol response to stress did not explain the association between childhood maltreatment and depressive symptoms, a finding that concord with prior investigations (Badanes et al., 2011; Busso et al., 2017; Shenk et al., 2014). Despite the fact that the present study did not support the mediating role of cortisol response to stress in this association, our results are still compatible with the Allostatic load model (McEwen, 1998; McEwen & Stellar, 1993). Specifically, this theoretical framework highlighted three highly integrated systems, that is, the nervous, the endocrine and the immune systems, as the primary

mediators of allostasis. The joint disruptions of these primary mediators are thought to explain the impact of chronic stress, such as maltreatment, on primary and secondary outcomes, including the wear and tear of organs and tissues in addition to the disorders that may ensue (e.g., cardiovascular disease and depression) (Danese et al., 2009; Flier et al., 1998; McEwen & Stellar, 1993). However, most of prior investigations have primarily focused on one mediator, the HPA axis. Congruent with the propositions of this theory, it is possible that it is not the alterations of one system per se that may explain the association between maltreatment and depressive symptoms. To put it differently, the increased vulnerability to suffer from depressive symptoms reported among maltreated individuals may partly stems from disruptions in the activity of multiple physiological systems (Bauer et al., 2002; Shenk et al., 2010). This hypothesis was supported empirically by a prospective study, which revealed that girls who were exposed to childhood maltreatment showed an asymmetrical physiological response to stress in late adolescence (i.e., participants who experienced an above the median value of vagal withdrawal and below the median value of cortisol reactivity to stress). In turn, the asymmetrical physiological response to stress was found to be associated with higher levels of depressive symptoms in young adulthood (Shenk et al., 2010). It is thus possible that our incapacity to find conclusive results in regard to the mediating role of cortisol response to stress in the association between maltreatment and depressive symptoms could perhaps be because we only evaluated one physiological system as a risk pathway in this association. That is, cortisol response to stress by itself could simply be a marker of biological vulnerability to depression and may not be directly implicated in the causal process of depression. This could partly explain why prior investigations failed to detect the mediating role of cortisol in the association linking maltreatment to internalizing symptoms (Badanes et al., 2011; Busso et al., 2017; Shenk et al.,

2015). As acknowledged by Cicchetti (2006), additional longitudinal studies are needed to identify the risk processes involved in the stress response that operate either additively or work synergistically to account for the increase risk for depression among maltreated individuals. As these systems are interrelated, alterations in allostatic systems should preferably be examined simultaneously in the same sample (Danese et al., 2009).

Consistent with our hypothesis, the current study revealed that a greater cortisol response to stress strengthened the impact of childhood maltreatment on depressive symptoms. Interestingly, our results converge with past studies supporting the moderating role of cortisol response to stress in the association between maltreatment and internalizing as well as depressive symptoms (Badanes et al., 2011; Hagan et al., 2014; Kuhlman et al., 2017; Steeger et al., 2017; Suzuki et al., 2014). For example, Hagan and colleagues reported that childhood maltreatment had a greater impact on internalizing symptoms among young adults who exhibited a higher cortisol response to stress in comparison to those who showed a lower cortisol response to stress. Our findings are congruent with the BSC theory, which proposed that individuals with high stress reactivity may have a higher sensitivity to the negative health effects of early life adversity whereas positive health effects may be promoted in stable and low-stress environments (Boyce & Ellis, 2005). However, others have also reported that lower cortisol response to stress enhanced the impact of physical abuse on parent-rated internalizing symptoms in adolescents (Kuhlman et al., 2017). Additionally, Badanes and colleagues indicated that adolescents who experienced more stressors over time and who showed a lower cortisol response to stress evidenced higher levels of depressive symptoms (Badanes et al., 2011). These differences in findings may partly arise from the developmental period assessed

(children/adolescent in comparison to young adults) as well as the type of adversity evaluated (family stress/maltreatment subtype compared to the cumulative impact of maltreatment), suggesting that variations in methodological strategies need to be considered in future studies. Moreover, these inconsistent findings underline the importance of evaluating the moderating role of cortisol in this association in a systematic manner as well as the need for future studies to report nonsignificant findings in order to contextualize when cortisol is expected to affect the association between maltreatment and depressive symptoms and when it does not.

### 4.3. The Role of Coping strategies in the Association Between Maltreatment and Depressive Symptoms

Congruent with the Transactional model of stress and coping (Lazarus & Folkman, 1984) and the Allostatic load model (McEwen & Stellar, 1993), the current study offers additional support for the role of coping strategies in the association between maltreatment and depressive symptoms. However, our results indicated that maltreated children who were more likely to adopt emotion-oriented coping strategies were, in turn, those who reported higher levels of depressive symptoms. In a normative context, the three forms of coping strategies are adopted at the same level of frequency when individuals are confronted to mild stressful situations. At moderate levels of stress, however, task-oriented coping strategies are expected to occur at a greater frequency whereas at higher levels of stress, emotion-oriented and avoidance-oriented coping strategies are more prevalent (Lazarus & Folkman, 1984). Because maltreatment is considered as triggering high levels of stress (Shonkoff, 2010; Shonkoff, Richter, van der Gaag, & Bhutta, 2012), maltreated children are expected to be more prone to use emotion-oriented

coping strategies (Lazarus & Folkman, 1984). Effectively, emotion-oriented coping strategies may seem like an appropriate choice in this context because they may help reduce distress, shame and helplessness (Hager & Runtz, 2012). However, with time, emotion-oriented coping strategies may become the predominant response to challenging situations, irrespective of the level of perceived stress of the situation (Lazarus & Folkman, 1984). Specifically, maltreatment experiences may lead to the development of maladaptive schemata (i.e., negative attitudes and biases about the self that are later integrated into cognitive structures), which may predispose these children to appraise stressful situations in a negative and distortional manner (Beck, 1979, 2008). Considering the high level of stress that maltreatment may trigger, these children may be more inclined to adopt emotion-oriented and avoidance-oriented coping strategies to deal with stress than task-oriented coping strategies (Lazarus & Folkman, 1984). Emotion-oriented could thus be crystallized as a preferred coping strategies to manage stressful situations even in adulthood (Lazarus & Folkman, 1984). While these coping strategies may be adaptive in a dangerous context such as maltreatment, a more internalized approach to coping with stress may be costly, increasing the susceptibility to suffer from higher levels of depressive symptoms (Futa et al., 2003; Long & Jackson, 1993; O'Leary, 2009; Runtz & Schallow, 1997). In sum, our findings suggest that maltreatment experiences may have long-lasting impact on the manner in which individuals cope with stress, which in turn, may confer a greater vulnerability to manifest depressive symptoms. However, contrary to previous findings (Fortier et al., 2009; Merrill et al., 2001; Rosenthal et al., 2005; Steel et al., 2004), task-oriented and avoidance-oriented coping strategies did not account for the association between maltreatment and depressive symptoms, suggesting that these strategies may not be involved in the causal process of depression.

Although the present findings highlighted the detrimental effects of emotion-oriented coping strategies, it is important to note that coping strategies are not inherently good or bad (Lazarus & Folkman, 1984). Austenfeld and Stanton, (2004) have indeed challenged the bad reputation of emotion-oriented coping strategies, arguing that the recurrently noted association between these strategies and dysfunctional outcomes is related to the way they are measured. Specifically, investigators typically assess emotion-oriented coping strategies (e.g., self-blame, preoccupation, fantasizing) that are reported to be a risk factor for psychological adjustment, what could thus be named negative emotion-oriented strategies. However, they often do not measure alternative emotion-oriented strategies that actively attempt to explore meanings, to acknowledge and to come to an understanding of one's emotions (i.e., emotional processing) and that actively attempts to express one's emotions (i.e., emotional expression). These latter emotion-oriented strategies have been found to be associated with well-being (positive emotion-oriented strategies) (Austenfeld & Stanton, 2004). For example, Runtz and Schallow (1997) have reported that expressing emotion (i.e., talking to family and friends about your feelings, giving yourself permission to feel your feelings and considering any feelings to be ok) was associated with higher levels of psychological adjustment among maltreated women. However, as no information was collected concerning positive emotion-oriented coping strategies in this study, we could not explore the impact of these strategies on the association linking maltreatment to depressive symptoms. The assessment of a broader range of emotion-oriented coping strategies could not only provide a more accurate view of the coping strategies typically used by maltreated individuals, but would also be more informative for clinicians. Specifically, future studies could examine whether emotional processing and emotional expression partly explain the impact of maltreatment on depressive symptoms. They could also test the mediating

role of positive and negative emotion-oriented coping strategies simultaneously, allowing to evaluate whether the mediating impact of negative emotion-oriented coping strategies still subsist when controlling for the use of positive emotion-oriented coping strategies.

Task-oriented coping strategies were also found to dampen the impact of maltreatment on depressive symptoms. In other words, individuals with a history of maltreatment showed lower levels of depressive symptoms when they reported a greater inclination to adopt task-oriented coping strategies to deal with stress. Adopting task-oriented strategies such as outlining their priorities, determining a course of action to follow and adjusting their priorities, may increase the chances of resolving stressful situations, thus reducing the negative effect of stress on psychological functioning in maltreated individuals (Aldwin & Revenson, 1987; Lazarus & Folkman, 1984). A comparison to prior findings is difficult, given the dearth of studies examining the moderating role of task-oriented coping strategies in the association between maltreatment and depressive symptoms. Nevertheless, increased reliance on task-oriented coping strategies was reported to modulate the impact of stress on psychological symptoms among a sample of healthy participants. This study revealed that participants undergoing high levels of stress who adopted task-oriented coping strategies more frequently reported fewer psychological symptoms (Aldwin & Revenson, 1987). However, our findings are inconsistent with previous investigations suggesting that these strategies are related to greater psychological distress (Brand & Alexander, 2003; Daigneault et al., 2006). The adaptiveness of task-oriented coping strategies may vary as a function of a third, often forgotten factor, namely, coping efficacy (i.e., the perception that the coping strategies adopted was successful in achieving the individuals' goals) (Aldwin & Revenson, 1987). Effectively, coping researchers have often

measured the association between coping strategies and psychological distress (i.e., coping effectiveness) assuming that coping strategies will have the same effect on this outcome, irrespective of the individual's strength and difficulties, the situation and the very execution of the strategy (Aldwin & Revenson, 1987). It is possible that in a challenging situation where the adoption of task-oriented coping strategies is perceived as effective (i.e., helping to resolve the situation), the adoption of these strategies is associated with well-being. Conversely, when the use of these strategies is perceived as ineffective, especially when they required a lot of resources to put into action, they may be related to higher levels of psychological distress, including depressive symptoms. For instance, higher use of negotiation was associated with lower levels of psychological symptoms when these strategies were perceived as effective. Inversely, these strategies were related to higher psychological distress when they were perceived as ineffective (Aldwin & Revenson, 1987). However, given that we did not assess coping efficacy in this sample, we were unable to determine whether this factor affected the modulating impact of task-oriented on the association between maltreatment and depressive symptoms (moderated moderation model). Further investigations on the impact of task-oriented coping strategies on depressive symptoms should evaluate coping efficacy to better contextualize when these strategies are expected to lead to adaptation and when they are not.

More generally, the present findings underscore the importance of considering the psychological stress response in studies evaluating the physiological stress response in order to have a more complete appreciation of an individual response to challenging situations. Effectively, our results suggested that coping strategies played a critical role in the association between maltreatment and depressive symptoms, which was not shown to be the case for the cortisol

response to stress. As acknowledged by prior theorists, the normative stress response includes cognitive and behavioural components whereby individuals interpret and react to a perceived stressful situation in complement to the neurophysiological stress response (Lazarus & Folkman, 1984; McEwen & Stellar, 1993). While these two processes interact, they are not identical. That is, coping strategies provide an additional viewpoint, allowing to appreciate the way an individual reacts cognitively and behaviourally to stress (Lazarus & Folkman, 1984). However, these two bodies of research have often been analyzed separately, without much effort to integrate existing findings (Olff et al., 2005). Future studies could close this gap in knowledge by systematically assessing coping strategies when the physiological stress response is being evaluated.

### 4.4. Limitations

These findings should be considered in light of a number of limitations. First, we did not measure current stress. As acknowledged by Tarullo & Gunnar (2006), the effect of childhood maltreatment on the HPA axis may be confounded with current life stress. More specifically, while disruptions of HPA axis activity may be the consequence of maltreatment, there is also a possibility that they may reflect a history of life stress or the interaction of these experiences (Rao et al., 2008). Controlling for life stress would have allowed to reduce the risk of ascribing a contributing status to a risk factor when other relevant co-occurring risk factors are omitted from the analysis (Shenk et al., 2015). We attempted to control this bias by evaluating the impact of several sociodemographic and lifestyle characteristics (offering a proxy of the type of environment that the individuals lived in) on cortisol response to stress and subsequently controlled for factors uniquely associated with cortisol response to stress. Second, it is possible

that the cross-sectional nature of our study diminished our capacity to accurately test the indirect effect of cortisol response to stress and coping strategies in the association linking maltreatment to depressive symptoms. Specifically, childhood maltreatment was assessed using a retrospective self-reported measure, which may have been affected by recall bias (Cicchetti & Toth, 2005; Gilbert et al., 2009). However, previous studies indicated that recall bias account for less than 1% of maltreatment variance (Fergusson et al., 2011), suggesting that the recollections of maltreatment experiences are reliable in adulthood (Bifulco et al., 1997). Furthermore, we were unable to consider the temporal sequence of events. This is problematic considering that coping strategies and depressive symptoms may be mutually influencing. That is, with increasing depressive symptomatology, individuals may be more prone to adopt emotion-oriented coping strategies to deal with stress. In turn, greater use of these strategies may increase depressive symptoms severity (Aldwin & Revenson, 1987). However, given the cross-sectional nature of the current study, we did not evaluate this hypothesis. Third, we did not assess the severity and chronicity of maltreatment experiences. Considering that these factors are thought to influence HPA axis activity (Cicchetti & Rogosch, 2001a), further studies assessing the severity and chronicity of maltreatment experiences are needed in order to gain a more complete understanding of HPA axis activity among maltreated individuals. Fourth, our sample was composed of young adult men. Past studies have reported sex and gender differences in respect to the prevalence of maltreatment experiences (Briere & Elliott, 2003; Finkelhor, 1994; Gilbert et al., 2009), cortisol (Doom et al., 2014; Power et al., 2012), coping strategies (Tamres et al., 2002), and depression (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Weissman & Klerman, 1977), indicating that our findings may not be generalizable to adult women. More particularly, previous investigations suggest that women are more likely to be

sexually abused in comparison to men, who are, however, at a greater risk for physical abuse and neglect (Briere & Elliott, 2003; Cawson, May-Chahal, Brooker, & Kelly, 2000; Edwards, Holden, Felitti, & Anda, 2003; Sobsey, Randall, & Parrila, 1997), although see (Scher, Forde, McQuaid, & Stein, 2004). Further investigations are thus needed to elucidate the contribution of cortisol response to stress and coping strategies in the association linking maltreatment to depressive symptoms among young adult women.

## 4.5. Directions for future studies

While this investigation has extended prior knowledge concerning the role of the HPA axis and coping strategies in the association linking maltreatment to depressive symptoms, it also raised several interesting avenues for future studies. Some of these questions will be presented here.

### *4.5.1. A more in-depth study of homogeneous dimensions of depression*

Inconsistent findings have been reported regarding the association between HPA axis activity and depression (Burke et al., 2005; Ciufolini et al., 2014; Lopez-Duran et al., 2009; Young et al., 2000). Depression is a heterogeneous disorder (American Psychiatric Association, 2013). Therefore, depressed individuals may show different patterns of depressive symptoms in terms of mood, appetite, arousal and sleep patterns (Buitelaar, 2013; Wardenaar et al., 2011). As such, overall measures of depression severity may not be an optimal clinical phenotype for HPA axis studies (Wardenaar et al., 2011). Buitelaar (2013) has argued that, to move forward, researchers should examine phenotypically more homogeneous dimensions of depression, indicated by the Tripartite model (Clark & Watson 1991). This model covers symptoms of general distress (e.g.,

pessimism and feeling of guilt), anhedonic symptoms (e.g., lack of positive affect and emotionality) and anxious arousal symptoms (e.g., sweating and palpitations). One prior study revealed that the association between the dimensions of depression and morning cortisol took the form of an inverted U shape, that is, both high and low levels of severity of the general distress dimension and the anhedonic dimension were associated, respectively, with a lower CAR in comparison to intermediate levels of severity (Wardenaar et al., 2011). However, very few studies have assessed symptoms-specific associations between homogeneous dimensions of depression and HPA axis activity. Additional investigations are thus necessary to evaluate the association between multiple index of HPA axis activity (i.e., cortisol response to psychological and pharmacological challenges, basal and diurnal cortisol levels) and more homogeneous dimensions of depression. These empirical advances may provide unprecedented and essential insights in the HPA axis activity of depressed individuals.

*4.5.2. Importance of considering the timing and the nature of maltreatment*

It has been hypothesized that patterning of the HPA axis dysregulation may be affected by maltreatment subtypes, as well as the timing and duration of exposure to maltreatment (Gonzalez, 2013; Heim, Newport, et al., 2000; McLaughlin, Sheridan, & Lambert, 2014; Twardosz & Lutzker, 2010; Voorhees & Scarpa, 2004). Despite the co-occurring nature of maltreatment subtypes, researchers have argued that they can be assessed separately and that they may have unique effects on the HPA axis (Bernard, Frost, Bennett, & Lindhiem, 2017; McLaughlin et al., 2014). Indeed, existing findings suggest that emotional abuse and sexual abuse are associated with higher cortisol levels (Bruce et al., 2009; Bublitz & Stroud, 2012; Cicchetti & Rogosch, 2001a; Nicolson et al., 2010; Şimşek et al., 2016), whereas physical abuse

is related to lower cortisol levels (Cicchetti & Rogosch, 2001a; Flory et al., 2009; Joyce et al., 2007; Kohrt, Hruschka, et al., 2015; Mello et al., 2015). Moreover, neglect and physical neglect have been found to be linked to higher cortisol levels (Bick et al., 2015; Fries et al., 2008; Gerra et al., 2009, 2010; Gerra et al., 2016; Lu, Gao, Huang, Li, & Xu, 2016). However, incongruent findings are also documented (Bruce et al., 2009; Heim et al., 2009; Kuhlman, Geiss, Vargas, & Lopez-Duran, 2015; Yeung et al., 2016). In contrast, studies addressing the association between different types of maltreatment and cortisol response to stress are scarce. Nonetheless, their results seem to parallel those reported by studies evaluating basal and diurnal cortisol levels, with exposure to sexual abuse and severe neglect associated with a greater cortisol response to stress (Bick et al., 2015; Fries, Shirtcliff, & Pollak, 2008). Conversely, physical abuse by itself, or in combination with sexual abuse, have recurrently been found to be related with a blunted cortisol response to stress (Carpenter et al., 2007, 2010; Trickett et al., 2014). However, nonsignificant findings also exist (Bugental, Martorell, & Barraza, 2003; Kempke et al., 2015; Kuhlman et al., 2015). Overall, several studies suggest that exposure to divergent maltreatment experiences may be associated with different patterns of disruptions of HPA axis activity. However, few empirical studies have assessed this hypothesis and to date, existing findings are inconsistent. Further studies delineating the impact of different types of maltreatment on HPA axis activity in basal and stressful contexts are thus needed in order to have a more in-depth understanding of the HPA axis activity in maltreated individuals.

Considering the plastic nature of the brain in early life, childhood maltreatment may result in either lower or higher cortisol response to stress as a function of the timing of the exposure to maltreatment (Lupien et al., 2009). While investigators have often acknowledged the

importance of assessing this hypothesis, very few studies to date have examined the effect of maltreatment onset on the HPA axis activity in basal and stressful contexts (Bernard et al., 2017; Heim, Newport, et al., 2000; Voorhees & Scarpa, 2004). Preliminary findings suggest that children who were exposed to physical and sexual abuse prior to age 5—in infancy, toddlerhood or preschool developmental periods with concurrent internalizing symptoms—showed a flattening of cortisol levels across the day in comparison to maltreated children who did not experience early physical or sexual abuse and nonmaltreated children (Cicchetti, Rogosch, Gunnar, & Toth, 2010). However, Cicchetti and colleagues also revealed that the developmental period of maltreatment was not related to cortisol levels in another study (Cicchetti & Rogosch, 2001a). Therefore, although animal studies support the effect of the timing of early adversity on brain outcomes (Gonzalez, 2013; Lupien et al., 2009), due to the scarceness of available findings, this association remains inconclusive in humans. The next phase of research, necessary to move toward a more detailed understanding of HPA axis activity in maltreated individuals, is to examine the timing and duration of exposure to maltreatment experiences in further studies.

*4.5.3. A more in-depth study of protective pathways*

Although researchers are increasingly recognizing that several maltreated individuals do not manifest depression over time, there are relatively few studies examining resilience among individuals with a history of maltreatment. However, studying these factors has the potential to identify targets for prevention of depression at a very early age (Thapar, Collishaw, Pine, & Thapar, 2012). Individual factors (e.g., high intelligence, emotion-regulation capacities and coping strategies), good quality interpersonal relations (e.g., parental warmth, acceptance, and low parental control), as well as quality of peer support have been reported to protect against

the development of depression in high-risk individuals (Jaffee, Takizawa, & Arseneault, 2017; Thapar et al., 2012). While these findings are promising, there are still very few studies that focused on resilience factors, and even fewer have assessed the underlying mechanisms leading to resilient outcomes among maltreated individuals (Thapar et al., 2012).

### 4.6. Clinical Implications

The current study gave support to coping strategies as a target for interventions aiming to prevent or reduce depressive symptoms among maltreated individuals. More specifically, treatments that minimize the use of emotion-oriented coping strategies while inviting clients to frequently engage in task-oriented coping strategies to deal with stressful situations, may lead to lower levels of depressive symptoms in individuals who were exposed to childhood maltreatment (Steinhardt & Dolbier, 2008). Our findings converge with cognitive-behavioural therapeutic programs that support the mediating role of cognitive processes in sustaining or reducing maladaptive patterns (Butler et al., 2006; Cuijpers et al., 2013; March et al., 2004). Past studies indeed support the beneficial effect of cognitive-behavioural therapy among depressive patients with a history of maltreatment (Harkness et al., 2012; Korotana et al., 2016). Although our findings highlighted the impact of coping strategies on depressive symptoms, considering the cross-sectional nature of this study, our results do not inform about whether the association between coping strategies and depressive symptoms vary as a function of timing (i.e., the short versus the long-term). It is possible that emotion-oriented are associated with well-being in the short term. Conversely, they may increase depressive symptoms severity in the long term (Steinhardt & Dolbier, 2008; Wright et al., 2007). Additional knowledge about

these factors could be very informative for clinicians, outlining the context in which these strategies are adaptive and the context in which they are not.

### 4.7. Conclusion

This master-degree thesis aimed to assess the contribution of cortisol response to stress and coping strategies in the association linking maltreatment to depressive symptoms. The current study highlighted the mediating and modulating role of coping strategies as well as the moderating role of cortisol response to stress in this association. These findings support coping strategies as a target for prevention and intervention efforts aiming to minimize depressive symptoms among adults exposed to maltreatment in childhood. Although this study extended prior findings, we only examined two mechanisms hypothesized to be involved in the association between maltreatment and depressive symptoms. Considering that both maltreatment and depression are often related to a broad range of psychosocial difficulties, it is critical that future investigations identify mechanisms that explain or modulate the impact of maltreatment on depressive symptoms, in order to reach a more in-depth understanding of this association. This endeavour should preferably be pursued in a multiple-level perspective, assessing complementary risk and protective processes among maltreated individuals. Such an approach may eventually yield information that can be used to identify amenable targets that have the potential to optimize evidence-based interventions for depressed individuals exposed to maltreatment.

# Bibliographie


Ahrens, T., Deuschle, M., Krumm, B., van der Pompe, G., den Boer, J. A., & Lederbogen, F. (2008). Pituitary-adrenal and sympathetic nervous system responses to stress in women remitted from recurrent major depression. *Psychosomatic Medicine*, *70*(4), 461–467.

Aldwin, C. M., & Revenson, T. A. (1987). Does coping help? A reexamination of the relation between coping and mental health. *Journal of Personality and Social Psychology*, *53*(2), 337.

Alink, L. R., Cicchetti, D., Kim, J., & Rogosch, F. A. (2012). Longitudinal associations among child maltreatment, social functioning, and cortisol regulation. *Developmental Psychology*, *48*(1), 224.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.

Austenfeld, J. L., & Stanton, A. L. (2004). Coping through emotional approach: a new look at emotion, coping, and health-related outcomes. *Journal of Personality*, *72*(6), 1335–1364.

Badanes, L. S., Watamura, S. E., & Hankin, B. L. (2011). Hypocortisolism as a potential marker of allostatic load in children: Associations with family risk and internalizing disorders. *Development and Psychopathology*, *23*(03), 881–896.

Bagley, S. L., Weaver, T. L., & Buchanan, T. W. (2011). Sex differences in physiological and affective responses to stress in remitted depression. *Physiology & Behavior*, *104*(2), 180–186.

Bal, S., Van Oost, P., De Bourdeaudhuij, I., & Crombez, G. (2003). Avoidant coping as a mediator between self-reported sexual abuse and stress-related symptoms in adolescents. *Child Abuse & Neglect*, *27*(8), 883–897. https://doi.org/10.1016/S0145-2134(03)00137-6

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*(6), 1173.

Beasley, M., Thompson, T., & Davidson, J. (2003). Resilience in response to life stress: the effects of coping style and cognitive hardiness. *Personality and Individual Differences*, *34*(1), 77–95.

Beck, A. T. (1979). *Cognitive therapy of depression*. Guilford press.

Beck, A. T. (2008). The evolution of the cognitive model of depression and its neurobiological

correlates. *American Journal of Psychiatry*, *165*(8), 969–977.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Beck depression inventory-II. *San Antonio*, *78*(2), 490–8.

Bernard, K., Frost, A., Bennett, C. B., & Lindhiem, O. (2017). Maltreatment and diurnal cortisol regulation: A meta-analysis. *Psychoneuroendocrinology*, *78*, 57–67.

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., … Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse & Neglect*, *27*(2), 169–190. https://doi.org/10.1016/S0145-2134(02)00541-0

Bicanic, I. A., Postma, R. M., Sinnema, G., De Roos, C., Olff, M., Van Wesel, F., & Van de Putte, E. M. (2013). Salivary cortisol and dehydroepiandrosterone sulfate in adolescent rape victims with post traumatic stress disorder. *Psychoneuroendocrinology*, *38*(3), 408–415.

Bick, J., Nguyen, V., Leng, L., Piecychna, M., Crowley, M. J., Bucala, R., … Grigorenko, E. L. (2015). Preliminary associations between childhood neglect, MIF, and cortisol: Potential pathways to long-term disease risk. *Developmental Psychobiology*, *57*(1), 131–139. https://doi.org/10.1002/dev.21265

Bifulco, A., Brown, G. W., Lillie, A., & Jarvis, J. (1997). Memories of childhood neglect and abuse: corroboration in a series of sisters. *Journal of Child Psychology and Psychiatry*, *38*(3), 365–374.

Booij, S. H., Bouma, E. M., de Jonge, P., Ormel, J., & Oldehinkel, A. J. (2013). Chronicity of depressive problems and the cortisol response to psychosocial stress in adolescents: The TRAILS study. *Psychoneuroendocrinology*, *38*(5), 659–666.

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, *17*(2), 271–301.

Brand, B. L., & Alexander, P. C. (2003). Coping with incest: The relationship between recollections of childhood coping and adult functioning in female survivors of incest. *Journal of Traumatic Stress*, *16*(3), 285–293.

Briere, J., & Elliott, D. M. (2003). Prevalence and psychological sequelae of self-reported childhood physical and sexual abuse in a general population sample of men and women. *Child*

*Abuse & Neglect*, *27*(10), 1205–1222. https://doi.org/10.1016/j.chiabu.2003.09.008

Bruce, J., Fisher, P. A., Pears, K. C., & Levine, S. (2009). Morning cortisol levels in preschool-aged foster children: differential effects of maltreatment type. *Developmental Psychobiology*, *51*(1), 14–23.

Bublitz, M. H., & Stroud, L. R. (2012). Childhood sexual abuse is associated with cortisol awakening response over pregnancy: preliminary findings. *Psychoneuroendocrinology*, *37*(9), 1425–1430.

Bugental, D. B., Martorell, G. A., & Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones and Behavior*, *43*(1), 237–244. https://doi.org/10.1016/S0018-506X(02)00008-9

Buitelaar, J. K. (2013). *The role of the HPA-axis in understanding psychopathology: cause, consequence, mediator, or moderator?* Springer.

Burke, H. M., Davis, M. C., Otte, C., & Mohr, D. C. (2005). Depression and cortisol responses to psychological stress: a meta-analysis. *Psychoneuroendocrinology*, *30*(9), 846–856.

Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology*, *63*(3), 452.

Busso, D. S., McLaughlin, K. A., & Sheridan, M. A. (2017). Dimensions of adversity, physiological reactivity, and externalizing psychopathology in adolescence: deprivation and threat. *Psychosomatic Medicine*, *79*(2), 162–171.

Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: a review of meta-analyses. *Clinical Psychology Review*, *26*(1), 17–31.

Cantón-Cortés, D., & Cantón, J. (2010). Coping with child sexual abuse among college students and post-traumatic stress disorder: The role of continuity of abuse and relationship with the perpetrator. *Child Abuse & Neglect*, *34*(7), 496–506.

Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., … Price, L. H. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry*, *62*(10), 1080–1087.

Carpenter, L. L., Shattuck, T. T., Tyrka, A. R., Geracioti, T. D., & Price, L. H. (2010). Effect of childhood physical abuse on cortisol stress response. *Psychopharmacology*, *214*(1), 367–375.

https://doi.org/10.1007/s00213-010-2007-4

Carrion, V. G., Weems, C. F., Ray, R. D., Glaser, B., Hessl, D., & Reiss, A. L. (2002). Diurnal salivary cortisol in pediatric posttraumatic stress disorder. *Biological Psychiatry*, *51*(7), 575–582.

Cawson, P., May-Chahal, C., Brooker, S., & Kelly, G. (2000). Child Maltreatment in the United Kingdom: a Study of the Prevalence of Abuse and Neglect.

Choi, K. W., Sikkema, K. J., Velloza, J., Marais, A., Jose, C., Stein, D. J., … Joska, J. A. (2015). Maladaptive coping mediates the influence of childhood trauma on depression and PTSD among pregnant women in South Africa. *Archives of Women's Mental Health*, *18*(5), 731–738.

Chopra, K. K., Ravindran, A., Kennedy, S. H., Mackenzie, B., Matthews, S., Anisman, H., … Levitan, R. D. (2009). Sex differences in hormonal responses to a social stressor in chronic major depression. *Psychoneuroendocrinology*, *34*(8), 1235–1241.

Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders: overview of physical and behavioral homeostasis. *Jama*, *267*(9), 1244–1252.

Cicchetti, D. (2006). Development and Psychopathology. In *Developmental Psychopathology, Second Edition* (Second Edition, Vol. 1, pp. 1–23). Wiley & Sons, Inc.

Cicchetti, D., & Rogosch, F. A. (2001a). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology*, *13*(3), 677–693.

Cicchetti, D., & Rogosch, F. A. (2001b). The impact of child maltreatment and psychopathology on neuroendocrine functioning. *Development and Psychopathology*, *null*(04), 783–804. https://doi.org/null

Cicchetti, D., Rogosch, F. A., Gunnar, M. R., & Toth, S. L. (2010). The differential impacts of early physical and sexual abuse and internalizing problems on daytime cortisol rhythm in school-aged children. *Child Development*, *81*(1), 252–269.

Cicchetti, D., & Toth, S. L. (2005). Child Maltreatment. *Annual Review of Clinical Psychology*, *1*(1), 409–438. https://doi.org/10.1146/annurev.clinpsy.1.102803.144029

Cicchetti, D., & Valentino, K. (2006). An Ecological-Transactional Perspective on Child Maltreatment: Failure of the Average Expectable environment and Its Influence on Child Maltreatment. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental Psychopathology: Risk, Disorder, and Adaptation* (Vol. 3). Retrieved from

http://ca.wiley.com/WileyCDA/WileyTitle/productCd-0471237388.html?0471237388=

Ciufolini, S., Dazzan, P., Kempton, M. J., Pariante, C., & Mondelli, V. (2014). HPA axis response to social stress is attenuated in schizophrenia but normal in depression: evidence from a meta-analysis of existing studies. *Neuroscience & Biobehavioral Reviews*, *47*, 359–368.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, *100*(3), 316.

Clément, M.-È., Bernèche, F., Fontaine, C., & Chamberland, C. (2013). *La violence familiale dans la vie des enfants du Québec, 2012: les attitudes parentales et les pratiques familiales*. Institut de la statistique du Québec.

Coffey, P., Leitenberg, H., Henning, K., Turner, T., & Bennett, R. T. (1996). The relation between methods of coping during adulthood with a history of childhood sexual abuse and current psychological adjustment. *Journal of Consulting and Clinical Psychology*, *64*(5), 1090.

Cohan, S. L., Jang, K. L., & Stein, M. B. (2006). Confirmatory factor analysis of a short form of the Coping Inventory for Stressful Situations. *Journal of Clinical Psychology*, *62*(3), 273–283. https://doi.org/10.1002/jclp.20211

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, 385–396.

Conradi, H. J., Ormel, J., & De Jonge, P. (2011). Presence of individual (residual) symptoms during depressive episodes and periods of remission: a 3-year prospective study. *Psychological Medicine*, *41*(6), 1165–1174.

Cuijpers, P., Berking, M., Andersson, G., Quigley, L., Kleiboer, A., & Dobson, K. S. (2013). A meta-analysis of cognitive-behavioural therapy for adult depression, alone and in comparison with other treatments. *The Canadian Journal of Psychiatry*, *58*(7), 376–385.

Daigneault, I., Hébert, M., & Tourigny, M. (2006). Attributions and coping in sexually abused adolescents referred for group treatment. *Journal of Child Sexual Abuse*, *15*(3), 35–59.

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior*, *106*(1), 29–39.

Danese, A., Moffitt, T. E., Harrington, H., Milne, B. J., Polanczyk, G., Pariante, C. M., … Caspi, A. (2009). Adverse childhood experiences and adult risk factors for age-related disease: depression, inflammation, and clustering of metabolic risk markers. *Archives of Pediatrics &*

*Adolescent Medicine*, *163*(12), 1135–1143.

De Bellis, M. D., Baum, A. S., Birmaher, B., Keshavan, M. S., Eccard, C. H., Boring, A. M., … Ryan, N. D. (1999). Developmental traumatology part I: Biological stress systems. *Biological Psychiatry*, *45*(10), 1259–1270.

de Rooij, S. R., Schene, A. H., Phillips, D. I., & Roseboom, T. J. (2010). Depression and anxiety: Associations with biological and perceived stress reactivity to a psychological stress protocol in a middle-aged population. *Psychoneuroendocrinology*, *35*(6), 866–877.

Dean, J., & Keshavan, M. (2017). The neurobiology of depression: An integrated view. *Asian Journal of Psychiatry*, *27*, 101–111. https://doi.org/10.1016/j.ajp.2017.01.025

Dickerson, S. S., & Kemeny, M. E. (2004). Acute Stressors and Cortisol Responses: A Theoretical Integration and Synthesis of Laboratory Research. *Psychological Bulletin*, *130*(3), 355–391. https://doi.org/10.1037/0033-2909.130.3.355

Dienes, K. A., Hazel, N. A., & Hammen, C. L. (2013). Cortisol secretion in depressed, and at-risk adults. *Psychoneuroendocrinology*, *38*(6), 927–940.

DiLillo, D., Long, P. J., & Russell, L. M. (1994). Childhood coping strategies of intrafamilial and extrafamilial female sexual abuse victims. *Journal of Child Sexual Abuse*, *3*(2), 45–66.

Dockray, S., Susman, E. J., & Dorn, L. D. (2009). Depression, cortisol reactivity, and obesity in childhood and adolescence. *Journal of Adolescent Health*, *45*(4), 344–350.

Doom, J. R., Cicchetti, D., & Rogosch, F. A. (2014). Longitudinal patterns of cortisol regulation differ in maltreated and nonmaltreated children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *53*(11), 1206–1215.

Doom, J. R., Cicchetti, D., Rogosch, F. A., & Dackis, M. N. (2013). Child maltreatment and gender interactions as predictors of differential neuroendocrine profiles. *Psychoneuroendocrinology*, *38*(8), 1442–1454. https://doi.org/10.1016/j.psyneuen.2012.12.019

Dozier, M., Manni, M., Gordon, M. K., Peloso, E., Gunnar, M. R., Stovall-McClough, K. C., … Levine, S. (2006). Foster Children's Diurnal Production of Cortisol: An Exploratory Study. *Child Maltreatment*, *11*(2), 189–197. https://doi.org/10.1177/1077559505285779

Edwards, V. J., Holden, G. W., Felitti, V. J., & Anda, R. F. (2003). Relationship Between Multiple Forms of Childhood Maltreatment and Adult Mental Health in Community Respondents: Results From the Adverse Childhood Experiences Study. *American Journal of*

*Psychiatry*, *160*(8), 1453–1460. https://doi.org/10.1176/appi.ajp.160.8.1453

Elzinga, B. M., Roelofs, K., Tollenaar, M. S., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse events. *Psychoneuroendocrinology*, *33*(2), 227–237. https://doi.org/10.1016/j.psyneuen.2007.11.004

Endler, N. S., & Parker, J. D. A. (1994). Assessment of multidimensional coping: Task, emotion, and avoidance strategies. *Psychological Assessment*, *6*(1), 50–60. https://doi.org/10.1037/1040-3590.6.1.50

Endler, N. S., & Parker, J. D. A. (1999). Coping Inventory for Stressful Situations: Manual (Second Edition). Multi-Health Systems.

Fergusson, D. M., Horwood, L. J., & Boden, J. M. (2011). Structural equation modeling of repeated retrospective reports of childhood maltreatment. *International Journal of Methods in Psychiatric Research*, *20*(2), 93–104. https://doi.org/10.1002/mpr.337

Ferrari, A. J., Charlson, F. J., Norman, R. E., Patten, S. B., Freedman, G., Murray, C. J., … Whiteford, H. A. (2013). Burden of depressive disorders by country, sex, age, and year: findings from the global burden of disease study 2010. *PLoS Med*, *10*(11), e1001547.

Field, A. (2013). *Discovering statistics using IBM SPSS statistics*. Sage.

Filipas, H. H., & Ullman, S. E. (2006). Child Sexual Abuse, Coping Responses, Self-Blame, Posttraumatic Stress Disorder, and Adult Sexual Revictimization. *Journal of Interpersonal Violence*, *21*(5), 652–672. https://doi.org/10.1177/0886260506286879

Finkelhor, D. (1994). Current information on the scope and nature of child sexual abuse. *The Future of Children*, 31–53.

Flier, J. S., Underhill, L. H., & McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, *338*(3), 171–179.

Flory, J. D., Yehuda, R., Grossman, R., New, A. S., Mitropoulou, V., & Siever, L. J. (2009). Childhood trauma and basal cortisol in people with personality disorders. *Comprehensive Psychiatry*, *50*(1), 34–37.

Folkman, S., & Lazarus, R. S. (1980). An analysis of coping in a middle-aged community sample. *Journal of Health and Social Behavior*, 219–239.

Fortier, M. A., DiLillo, D., Messman-Moore, T. L., Peugh, J., DeNardi, K. A., & Gaffey, K. J. (2009). Severity of child sexual abuse and revictimization: The mediating role of coping and

trauma symptoms. *Psychology of Women Quarterly*, *33*(3), 308–320.

Frick, P. J. (2004). The inventory of callous-unemotional traits. *Unpublished Rating Scale*.

Fries, A. B. W., Shirtcliff, E. A., & Pollak, S. D. (2008). Neuroendocrine dysregulation following early social deprivation in children. *Developmental Psychobiology*, *50*(6), 588–599.

Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D. H. (2005). A new view on hypocortisolism. *Psychoneuroendocrinology*, *30*(10), 1010–1016.

Futa, K. T., Nash, C. L., Hansen, D. J., & Garbin, C. P. (2003). Adult Survivors of Childhood Abuse: An Analysis of Coping Mechanisms Used for Stressful Childhood Memories and Current Stressors. *Journal of Family Violence*, *18*(4), 227–239. https://doi.org/10.1023/A:1024068314963

Garnefski, N., & Kraaij, V. (2007). The cognitive emotion regulation questionnaire. *European Journal of Psychological Assessment*, *23*(3), 141–149.

Gerra, G., Zaimovic, A., Castaldini, L., Garofano, L., Manfredini, M., Somaini, L., … Donnini, C. (2010). Relevance of perceived childhood neglect, 5-HTT gene variants and hypothalamus–pituitary–adrenal axis dysregulation to substance abuse susceptibility. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, *153*(3), 715–722.

Gibbs, M. S. (1989). Factors in the victim that mediate between disaster and psychopathology: A review. *Journal of Traumatic Stress*, *2*(4), 489–514.

Gibson, L. E., & Leitenberg, H. (2001). The impact of child sexual abuse and stigma on methods of coping with sexual assault among undergraduate women. *Child Abuse & Neglect*, *25*(10), 1343–1361.

Giese-Davis, J., Wilhelm, F. H., Conrad, A., Abercrombie, H. C., Sephton, S., Yutsis, M., … Spiegel, D. (2006). Depression and stress reactivity in metastatic breast cancer. *Psychosomatic Medicine*, *68*(5), 675–683.

Gilbert, R., Widom, C. S., Browne, K., Fergusson, D., Webb, E., & Janson, S. (2009). Burden and consequences of child maltreatment in high-income countries. *The Lancet*, *373*(9657), 68–81.

Gold, P. W., & Chrousos, G. P. (2002). Organization of the stress system and its dysregulation in melancholic and atypical depression: high vs low CRH/NE states. *Molecular Psychiatry*, *7*(3), 254.

Gonzalez, A. (2013). The impact of childhood maltreatment on biological systems: Implications for clinical interventions. *Paediatrics & Child Health*, *18*(8), 415–418.

Green, B. L., Wilson, J. P., & Lindy, J. D. (1985). Conceptualizing post-traumatic stress disorder: A psychosocial framework. *Trauma and Its Wake*, *1*, 53–69.

Griffing, S., Lewis, C. S., Chu, M., Sage, R., Jospitre, T., Madry, L., & Primm, B. J. (2006a). The process of coping with domestic violence in adult survivors of childhood sexual abuse. *Journal of Child Sexual Abuse*, *15*(2), 23–41.

Griffing, S., Lewis, C. S., Chu, M., Sage, R., Jospitre, T., Madry, L., & Primm, B. J. (2006b). The process of coping with domestic violence in adult survivors of childhood sexual abuse. *Journal of Child Sexual Abuse*, *15*(2), 23–41.

Guerry, J. D., & Hastings, P. D. (2011). In search of HPA axis dysregulation in child and adolescent depression. *Clinical Child and Family Psychology Review*, *14*(2), 135–160.

Gunnar, M. R., & Vazquez, D. (2006). Stress neurobiology and developmental psychopathology.

Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology*, *13*(03), 515–538.

Hagan, M. J., Roubinov, D. S., Mistler, A. K., & Luecken, L. J. (2014). Mental health outcomes in emerging adults exposed to childhood maltreatment: the moderating role of stress reactivity. *Child Maltreatment*, *19*(3–4), 156–167. https://doi.org/10.1177/1077559514539753

Hager, A. D., & Runtz, M. G. (2012). Physical and psychological maltreatment in childhood and later health problems in women: An exploratory investigation of the roles of perceived stress and coping strategies. *Child Abuse & Neglect*, *36*(5), 393–403.

Harkness, K. L., Michael, R., & Kennedy, S. H. (2012). Childhood maltreatment and differential treatment response and recurrence in adult major depressive disorder. *Journal of Consulting and Clinical Psychology*, *80*(3), 342–353. https://doi.org/10.1037/a0027665

Harkness, K. L., Stewart, J. G., & Wynne-Edwards, K. E. (2011). Cortisol reactivity to social stress in adolescents: role of depression severity and child maltreatment. *Psychoneuroendocrinology*, *36*(2), 173–181.

Hayes, A. F. (2012). *PROCESS: A versatile computational tool for observed variable mediation,*

*moderation, and conditional process modeling*.

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. Guilford Press.

Hébert, M., Tremblay, C., Parent, N., Daignault, I. V., & Piché, C. (2006). Correlates of behavioral outcomes in sexually abused children. *Journal of Family Violence*, *21*(5), 287–299.

Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, *25*(1), 1–35.

Heim, C., Nater, U. M., Maloney, E., Boneva, R., Jones, J. F., & Reeves, W. C. (2009). Childhood trauma and risk for chronic fatigue syndrome: association with neuroendocrine dysfunction. *Archives of General Psychiatry*, *66*(1), 72–80.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., … Nemeroff, C. B. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Jama*, *284*(5), 592–597.

Heim, C., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B. (2008). The link between childhood trauma and depression: insights from HPA axis studies in humans. *Psychoneuroendocrinology*, *33*(6), 693–710.

Heim, C., Newport, D. J., Wagner, D., Wilcox, M. M., Miller, A. H., & Nemeroff, C. B. (2002). The role of early adverse experience and adulthood stress in the prediction of neuroendocrine stress reactivity in women: A multiple regression analysis. *Depression and Anxiety*, *15*(3), 117–125. https://doi.org/10.1002/da.10015

Höhne, N., Poidinger, M., Merz, F., Pfister, H., Brückl, T., Zimmermann, P., … Ising, M. (2014). Increased HPA axis response to psychosocial stress in remitted depression: the influence of coping style. *Biological Psychology*, *103*, 267–275. https://doi.org/10.1016/j.biopsycho.2014.09.008

Hulme, P. A., McBride, C. L., Kupzyk, K. A., & French, J. A. (2015). Pilot study on childhood sexual abuse, diurnal cortisol secretion, and weight loss in bariatric surgery patients. *Journal of Child Sexual Abuse*, *24*(4), 385–400.

Hyman, S. M., Paliwal, P., & Sinha, R. (2007). Childhood maltreatment, perceived stress, and stress-related coping in recently abstinent cocaine dependent adults. *Psychology of Addictive Behaviors*, *21*(2), 233.

Jaffee, S. R., Takizawa, R., & Arseneault, L. (2017). Buffering effects of safe, supportive, and nurturing relationships among women with childhood histories of maltreatment. *Psychological Medicine*, 1–12.

Janoff-Bulman, R. (1985). The aftermath of victimization: Rebuilding shattered assumptions. *Trauma and Its Wake*, *1*, 15–35.

Johnson, D. M., Sheahan, T. C., & Chard, K. M. (2004). Personality disorders, coping strategies, and posttraumatic stress disorder in women with histories of childhood sexual abuse. *Journal of Child Sexual Abuse*, *12*(2), 19–39.

Joyce, P. R., Williamson, S. A., McKenzie, J. M., Frampton, C. M., Luty, S. E., Porter, R. J., & Mulder, R. T. (2007). Effects of childhood experiences on cortisol levels in depressed adults. *Australian & New Zealand Journal of Psychiatry*, *41*(1), 62–65.

Judd, L. L. (1997). The clinical course of unipolar major depressive disorders. *Archives of General Psychiatry*, *54*(11), 989–991.

Kaestner, F., Hettich, M., Peters, M., Sibrowski, W., Hetzel, G., Ponath, G., … Rothermundt, M. (2005). Different activation patterns of proinflammatory cytokines in melancholic and non-melancholic major depression are associated with HPA axis activity. *Journal of Affective Disorders*, *87*(2), 305–311.

Kempke, S., Luyten, P., De Coninck, S., Van Houdenhove, B., Mayes, L. C., & Claes, S. (2015). Effects of early childhood trauma on hypothalamic–pituitary–adrenal (HPA) axis function in patients with Chronic Fatigue Syndrome. *Psychoneuroendocrinology*, *52*, 14–21.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., … Wang, P. S. (2003). The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R). *Jama*, *289*(23), 3095–3105.

Kessler, R. C., McGonagle, K. A., Swartz, M., Blazer, D. G., & Nelson, C. B. (1993). Sex and depression in the National Comorbidity Survey I: Lifetime prevalence, chronicity and recurrence. *Journal of Affective Disorders*, *29*(2), 85–96. https://doi.org/10.1016/0165-0327(93)90026-G

Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine*, *61*(2), 154–162.

Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'–a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*(1–2), 76–81.

Knorr, U., Vinberg, M., Kessing, L. V., & Wetterslev, J. (2010). Salivary cortisol in depressed patients versus control persons: a systematic review and meta-analysis. *Psychoneuroendocrinology*, *35*(9), 1275–1286.

Kohrt, B. A., Hruschka, D. J., Kohrt, H. E., Carrion, V. G., Waldman, I. D., & Worthman, C. M. (2015). Child abuse, disruptive behavior disorders, depression, and salivary cortisol levels among institutionalized and community-residing boys in Mongolia. *Asia-Pacific Psychiatry*, *7*(1), 7–19.

Kohrt, B. A., Worthman, C. M., Ressler, K. J., Mercer, K. B., Upadhaya, N., Koirala, S., … Binder, E. B. (2015). Cross-cultural gene– environment interactions in depression, post-traumatic stress disorder, and the cortisol awakening response: FKBP5 polymorphisms and childhood trauma in South Asia. *International Review of Psychiatry*, *27*(3), 180–196. https://doi.org/10.3109/09540261.2015.1020052

Korotana, L. M., Dobson, K. S., Pusch, D., & Josephson, T. (2016). A review of primary care interventions to improve health outcomes in adult survivors of adverse childhood experiences. *Clinical Psychology Review*, *46*, 59–90. https://doi.org/10.1016/j.cpr.2016.04.007

Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). HPA axis responses to laboratory psychosocial stress in healthy elderly adults, younger adults, and children: impact of age and gender. *Psychoneuroendocrinology*, *29*(1), 83–98.

Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: A review. *Biological Psychology*.

Kuhlman, K. R., Geiss, E. G., Vargas, I., & Lopez-Duran, N. (2017). HPA-axis activation as a key moderator of childhood trauma exposure and adolescent mental health. *Journal of Abnormal Child Psychology*, 1–9.

Kuhlman, K. R., Geiss, E. G., Vargas, I., & Lopez-Duran, N. L. (2015). Differential associations between childhood trauma subtypes and adolescent HPA-axis functioning. *Psychoneuroendocrinology*, *54*, 103–114.

Lamers, F., Rhebergen, D., Merikangas, K. R., De Jonge, P., Beekman, A. T. F., & Penninx, B.

(2012). Stability and transitions of depressive subtypes over a 2-year follow-up. *Psychological Medicine*, *42*(10), 2083–2093.

Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York, NY, US: McGraw-Hill.

Lazarus, R. S., & Folkman, S. (1984a). Stress. *Appraisal, and Coping*, *725*.

Lazarus, R. S., & Folkman, S. (1984b). *Stress, Appppraisal and Coping* (Springer Publishing Company). New York: Springer Publishing Company.

Leitenberg, H., Gibson, L. E., & Novy, P. L. (2004). Individual differences among undergraduate women in methods of coping with stressful events: The impact of cumulative childhood stressors and abuse. *Child Abuse & Neglect*, *28*(2), 181–192.

Leitenberg, H., Greenwald, E., & Cado, S. (1992). A retrospective study of long-term methods of coping with having been sexually abused during childhood. *Child Abuse & Neglect*, *16*(3), 399–407.

Li, M., D'Arcy, C., & Meng, X. (2015). Maltreatment in childhood substantially increases the risk of adult depression and anxiety in prospective cohort studies: systematic review, meta-analysis, and proportional attributable fractions. *Psychological Medicine*, 1–14.

Littleton, H., Horsley, S., John, S., & Nelson, D. V. (2007). Trauma coping strategies and psychological distress: a meta-analysis. *Journal of Traumatic Stress*, *20*(6), 977–988.

Liu, R. T. (2017). Childhood adversities and depression in adulthood: Current findings and future directions. *Clinical Psychology: Science and Practice*.

Livneh, H., & Martz, E. (2007). An introduction to coping theory and research. In *Coping with chronic illness and disability* (pp. 3–27). Springer.

Long, P. J., & Jackson, J. L. (1993). Childhood coping strategies and the adult adjustment of female sexual abuse victims. *Journal of Child Sexual Abuse*, *2*(2), 23–39.

Lopez-Duran, N. L., Kovacs, M., & George, C. J. (2009). Hypothalamic–pituitary–adrenal axis dysregulation in depressed children and adolescents: A meta-analysis. *Psychoneuroendocrinology*, *34*(9), 1272–1283.

Lopez-Duran, N. L., McGinnis, E., Kuhlman, K., Geiss, E., Vargas, I., & Mayer, S. (2015). HPA-axis stress reactivity in youth depression: evidence of impaired regulatory processes in depressed boys. *Stress*, *18*(5), 545–553.

Luby, J. L., Heffelfinger, A., Mrakotsky, C., Brown, K., Hessler, M., & Spitznagel, E. (2003). Alterations in stress cortisol reactivity in depressed preschoolers relative to psychiatric and no-disorder comparison groups. *Archives of General Psychiatry*, *60*(12), 1248–1255.

Lupien, S. (2010). *Par amour du stress*. Éditions au Carré.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*(6), 434–445.

Lupien, S. J., Ouellet-Morin, I., Hupbach, A., Tu, M. T., Buss, C., Walker, D., … McEwen, B. S. (2006). Beyond the stress concept: Allostatic load--a developmental biological and cognitive perspective.

MacMillan, H. L., Georgiades, K., Duku, E. K., Shea, A., Steiner, M., Niec, A., … Schmidt, L. A. (2009). Cortisol Response to Stress in Female Youths Exposed to Childhood Maltreatment: Results of the Youth Mood Project. *Biological Psychiatry*, *66*(1), 62–68. https://doi.org/10.1016/j.biopsych.2008.12.014

March, J., Silva, S., Petrycki, S., Curry, J., Wells, K., Fairbank, J., … Vitiello, B. (2004). Fluoxetine, cognitive-behavioral therapy, and their combination for adolescents with depression: Treatment for Adolescents With Depression Study (TADS) randomized controlled trial. *Jama*, *292*(7), 807–820.

McCrory, E., De Brito, S. A., & Viding, E. (2012). The link between child abuse and psychopathology: a review of neurobiological and genetic research. *Journal of the Royal Society of Medicine*, *105*(4), 151–156.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *840*(1), 33–44.

McEwen, B. S. (2007). Physiology and Neurobiology of Stress and Adaptation: Central Role of the Brain. *Physiological Reviews*, *87*(3), 873–904. https://doi.org/10.1152/physrev.00041.2006

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: mechanisms leading to disease. *Archives of Internal Medicine*, *153*(18), 2093–2101.

McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity and neural development: Deprivation and threat as distinct dimensions of early experience. *Neuroscience & Biobehavioral Reviews*, *47*, 578–591. https://doi.org/10.1016/j.neubiorev.2014.10.012

Mello, A. F., Juruena, M. F., Maciel, M. R., Cavalcante-Nobrega, L. P., Cividanes, G. C., Fossaluza, V., … de Jesus Mari, J. (2015). Factors related to the cortisol awakening response of children working on the streets and siblings, before and after 2 years of a psychosocial intervention. *Psychiatry Research*, *225*(3), 625–630.

Merrill, L. L., Guimond, J. M., Thomsen, C. J., & Milner, J. S. (2003). Child sexual abuse and number of sexual partners in young women: the role of abuse severity, coping style, and sexual functioning. *Journal of Consulting and Clinical Psychology*, *71*(6), 987.

Merrill, L. L., Thomsen, C. J., Sinclair, B. B., Gold, S. R., & Milner, J. S. (2001). Predicting the impact of child sexual abuse on women: The role of abuse severity, parental support, and coping strategies. *Journal of Consulting and Clinical Psychology*, *69*(6), 992.

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*(1), 25.

Monroe, S. M., & Harkness, K. L. (2005). Life stress, the" kindling" hypothesis, and the recurrence of depression: considerations from a life stress perspective. *Psychological Review*, *112*(2), 417.

Muthén, L. K., & Muthén, B. O. (2012). Mplus statistical modeling software: Release 7.0. *Los Angeles, CA: Muthén & Muthén*.

Nanni, V., Uher, R., & Danese, A. (2012). Childhood maltreatment predicts unfavorable course of illness and treatment outcome in depression: a meta-analysis. *American Journal of Psychiatry*.

Nestler, E. J., Barrot, M., DiLeone, R. J., Eisch, A. J., Gold, S. J., & Monteggia, L. M. (2002). Neurobiology of depression. *Neuron*, *34*(1), 13–25.

Nicolson, N. A. (2008). Measurement of cortisol. *Handbook of Physiological Research Methods in Health Psychology*, 37–74.

Nicolson, N. A., Davis, M. C., Kruszewski, D., & Zautra, A. J. (2010). Childhood maltreatment and diurnal cortisol patterns in women with chronic pain. *Psychosomatic Medicine*, *72*(5), 471–480.

Norman, R. E., Byambaa, M., De, R., Butchart, A., Scott, J., & Vos, T. (2012). The long-term health consequences of child physical abuse, emotional abuse, and neglect: a systematic review

and meta-analysis. *PLoS Medicine*, *9*(11), e1001349. https://doi.org/10.1371/journal.pmed.1001349

O'Keane, V., Frodl, T., & Dinan, T. G. (2012). A review of Atypical depression in relation to the course of depression and changes in HPA axis organization. *Psychoneuroendocrinology*, *37*(10), 1589–1599.

O'Leary, P. J. (2009). Men who were sexually abused in childhood: Coping strategies and comparisons in psychological functioning. *Child Abuse & Neglect*, *33*(7), 471–479.

Olff, M., Langeland, W., & Gersons, B. P. (2005). Effects of appraisal and coping on the neuroendocrine response to extreme stress. *Neuroscience & Biobehavioral Reviews*, *29*(3), 457–467.

Ouellet-Morin, I., Odgers, C. L., Danese, A., Bowes, L., Shakoor, S., Papadopoulos, A. S., … Arseneault, L. (2011). Blunted cortisol responses to stress signal social and behavioral problems among maltreated/bullied 12-year-old children. *Biological Psychiatry*, *70*(11), 1016–1023.

Patten, S. B. (2009). Accumulation of major depressive episodes over time in a prospective study indicates that retrospectively assessed lifetime prevalence estimates are too low. *BMC Psychiatry*, *9*(1), 19.

Patten, S. B., Wang, J. L., Williams, J. V., Currie, S., Beck, C. A., Maxwell, C. J., & el-Guebaly, N. (2006). Descriptive epidemiology of major depression in Canada. *The Canadian Journal of Psychiatry*, *51*(2), 84–90.

Pearlin, L. I., & Schooler, C. (1978). The structure of coping. *Journal of Health and Social Behavior*, 2–21.

Perreault, S. (2015). *La victimisation criminelle au Canada, 2014* (No. 28) (p. 45). Canada: Statistique Canada. Retrieved from http://www.statcan.gc.ca/pub/85-002-x/2015001/article/14241-fra.htm#n14

Power, C., Thomas, C., Li, L., & Hertzman, C. (2012). Childhood psychosocial adversity and adult cortisol patterns. *The British Journal of Psychiatry*, bjp. bp. 111.096032.

Preacher, K. J., Wichman, A. L., MacCallum, R. C., & Briggs, N. E. (2008). *Latent growth curve modeling. Quantitative applications in the social sciences*. Sage Publications, Inc., Thousand Oaks, CA.

Raine, A., Dodge, K., Loeber, R., Gatzke-Kopp, L., Lynam, D., Reynolds, C., … Liu, J. (2006).

The reactive–proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. *Aggressive Behavior*, *32*(2), 159–171.

Rao, U., Hammen, C., Ortiz, L. R., Chen, L.-A., & Poland, R. E. (2008). Effects of early and recent adverse experiences on adrenal response to psychosocial stress in depressed adolescents. *Biological Psychiatry*, *64*(6), 521–526.

Rao, U., & Morris, M. C. (2015). Cortisol Responses to Psychosocial Stress: The Role of Childhood Maltreatment and Depression. *International Journal of Public Mental Health and Neurosciences*, *2*(1).

Rogosch, F. A., Dackis, M. N., & Cicchetti, D. (2011). Child Maltreatment and Allostatic Load: Consequences for Physical and Mental Health in Children from Low-Income Families. *Development and Psychopathology*, *23*(4), 1107–1124. https://doi.org/10.1017/S0954579411000587

Rosenthal, M. Z., Hall, M. L. R., Palm, K. M., Batten, S. V., & Follette, V. M. (2005). Chronic avoidance helps explain the relationship between severity of childhood sexual abuse and psychological distress in adulthood. *Journal of Child Sexual Abuse*, *14*(4), 25–41.

Runtz, M. G., & Schallow, J. R. (1997). Social support and coping strategies as mediators of adult adjustment following childhood maltreatment. *Child Abuse & Neglect*, *21*(2), 211–226.

Sapolsky, R. M. (2004). *Why zebras don't get ulcers: The acclaimed guide to stress, stress-related diseases, and coping-now revised and updated*. Macmillan.

Scher, C. D., Forde, D. R., McQuaid, J. R., & Stein, M. B. (2004). Prevalence and demographic correlates of childhood maltreatment in an adult community sample. *Child Abuse & Neglect*, *28*(2), 167–180. https://doi.org/10.1016/j.chiabu.2003.09.012

Shenk, C. E., Griffin, A. M., & O'Donnell, K. J. (2015). Symptoms of major depressive disorder subsequent to child maltreatment: Examining change across multiple levels of analysis to identify transdiagnostic risk pathways. *Development and Psychopathology*, *27*(4 0 2), 1503.

Shenk, C. E., Noll, J. G., Putnam, F. W., & Trickett, P. K. (2010). A prospective examination of the role of childhood sexual abuse and physiological asymmetry in the development of psychopathology. *Child Abuse & Neglect*, *34*(10), 752–761.

Shenk, C. E., Putnam, F. W., Rausch, J. R., Peugh, J. L., & Noll, J. G. (2014). A longitudinal study of several potential mediators of the relationship between child maltreatment and

posttraumatic stress disorder symptoms. *Development and Psychopathology*, *26*(01), 81–91. https://doi.org/10.1017/S0954579413000916

Shonkoff, J. P. (2010). Building a new biodevelopmental framework to guide the future of early childhood policy. *Child Development*, *81*(1), 357–367.

Shonkoff, J. P., Richter, L., van der Gaag, J., & Bhutta, Z. A. (2012). An integrated scientific framework for child survival and early childhood development. *Pediatrics*, *129*(2), e460–e472.

Sigmon, S. T., Greene, M. P., Rohan, K. J., & Nichols, J. E. (1997). Coping and adjustment in male and female survivors of childhood sexual abuse. *Journal of Child Sexual Abuse*, *5*(3), 57–75.

Şimşek, Ş., Kaplan, I., Uysal, C., Yüksel, T., & Alaca, R. (2016). The levels of cortisol, oxidative stress, and DNA damage in the victims of childhood sexual abuse: a preliminary study. *Journal of Child Sexual Abuse*, *25*(2), 175–184.

Şimşek, Ş., Yüksel, T., Kaplan, İ., Uysal, C., & Alaca, R. (2015). Examining the levels of BDNF and cortisol in children and adolescent victims of sexual abuse—a preliminary study. *Comprehensive Psychiatry*, *61*, 23–27.

Sobsey, D., Randall, W., & Parrila, R. K. (1997). Gender differences in abused children with and without disabilities. *Child Abuse & Neglect*, *21*(8), 707–720. https://doi.org/10.1016/S0145-2134(97)00033-1

Spaccarelli, S. (1994). Stress, appraisal, and coping in child sexual abuse: a theoretical and empirical review. *Psychological Bulletin*, *116*(2), 340.

Staufenbiel, S. M., Penninx, B. W., Spijker, A. T., Elzinga, B. M., & van Rossum, E. F. (2013). Hair cortisol, stress exposure, and mental health in humans: a systematic review. *Psychoneuroendocrinology*, *38*(8), 1220–1235.

Steeger, C. M., Cook, E. C., & Connell, C. M. (2017). The Interactive Effects of Stressful Family Life Events and Cortisol Reactivity on Adolescent Externalizing and Internalizing Behaviors. *Child Psychiatry & Human Development*, *48*(2), 225–234.

Steel, J., Sanna, L., Hammond, B., Whipple, J., & Cross, H. (2004). Psychological sequelae of childhood sexual abuse: Abuse-related characteristics, coping strategies, and attributional style. *Child Abuse & Neglect*, *28*(7), 785–801.

Steinhardt, M., & Dolbier, C. (2008). Evaluation of a resilience intervention to enhance coping

strategies and protective factors and decrease symptomatology. *Journal of American College Health*, *56*(4), 445–453.

Stetler, C., & Miller, G. E. (2011). Depression and hypothalamic-pituitary-adrenal activation: a quantitative summary of four decades of research. *Psychosomatic Medicine*, *73*(2), 114–126.

Stewart, J. W., Quitkin, F. M., McGrath, P. J., & Klein, D. F. (2005). Defining the boundaries of atypical depression: evidence from the HPA axis supports course of illness distinctions. *Journal of Affective Disorders*, *86*(2), 161–167.

Stone, A. A., & Neale, J. M. (1984). New measure of daily coping: Development and preliminary results. *Journal of Personality and Social Psychology*, *46*(4), 892.

Storch, E. A., Roberti, J. W., & Roth, D. A. (2004). Factor structure, concurrent validity, and internal consistency of the beck depression inventory—second edition in a sample of college students. *Depression and Anxiety*, *19*(3), 187–189. https://doi.org/10.1002/da.20002

Sun, Y., Deng, F., Liu, Y., & Tao, F.-B. (2015). Cortisol response to psychosocial stress in Chinese early puberty girls: possible role of depressive symptoms. *BioMed Research International*, *2015*.

Susman, E. J. (2006). Psychobiology of persistent antisocial behavior: Stress, early vulnerabilities and the attenuation hypothesis. *Neuroscience & Biobehavioral Reviews*, *30*(3), 376–389.

Suzuki, A., Poon, L., Papadopoulos, A. S., Kumari, V., & Cleare, A. J. (2014). Long term effects of childhood trauma on cortisol stress reactivity in adulthood and relationship to the occurrence of depression. *Psychoneuroendocrinology*, *50*, 289–299.

Takaki, J., Nishi, T., Shimoyama, H., Inada, T., Matsuyama, N., Kumano, H., & Kuboki, T. (2005). Possible interactive effects of demographic factors and stress coping mechanisms on depression and anxiety in maintenance hemodialysis patients. *Journal of Psychosomatic Research*, *58*(3), 217–223.

Tamres, L. K., Janicki, D., & Helgeson, V. S. (2002). Sex Differences in Coping Behavior: A Meta-Analytic Review and an Examination of Relative Coping. *Personality and Social Psychology Review*, *6*(1), 2–30. https://doi.org/10.1207/S15327957PSPR0601_1

Tarullo, A. R., & Gunnar, M. R. (2006). Child maltreatment and the developing HPA axis. *Hormones and Behavior*, *50*(4), 632–639.

Thabet, A. A. M., Tischler, V., & Vostanis, P. (2004). Maltreatment and coping strategies among male adolescents living in the Gaza Strip. *Child Abuse & Neglect*, *28*(1), 77–91.

Thapar, A., Collishaw, S., Pine, D. S., & Thapar, A. K. (2012). Depression in adolescence. *The Lancet*, *379*(9820), 1056–1067.

Toth, S. L., & Cicchetti, D. (2013). A developmental psychopathology perspective on child maltreatment. *Child Maltreatment*, *18*(3), 135–139.

Tremblay, C., Hébert, M., & Piché, C. (1999). Coping strategies and social support as mediators of consequences in child sexual abuse victims. *Child Abuse & Neglect*, *23*(9), 929–945.

Trickett, P. K., Gordis, E., Peckins, M. K., & Susman, E. J. (2014). Stress reactivity in maltreated and comparison male and female young adolescents. *Child Maltreatment*, 1077559513520466.

Trickett, P. K., Noll, J. G., Susman, E. J., Shenk, C. E., & Putnam, F. W. (2010). Attenuation of cortisol across development for victims of sexual abuse. *Development and Psychopathology*, *22*(01), 165–175.

Trocmé, N., Fallon, B., MacLaurin, B., Sinha, V., Black, T., Fast, E., … Holroyd. (2010). *Étude d'incidence canadienne 2008 | Canadian Child Welfare Research Portal* (p. 128). Ottawa: Agence de la santé publique du Canada. Retrieved from http://cwrp.ca/fr/eci-2008

Twardosz, S., & Lutzker, J. R. (2010). Child maltreatment and the developing brain: A review of neuroscience perspectives. *Aggression and Violent Behavior*, *15*(1), 59–68.

van der Vegt, E. J. M., van der Ende, J., Huizink, A. C., Verhulst, F. C., & Tiemeier, H. (2010). Childhood Adversity Modifies the Relationship Between Anxiety Disorders and Cortisol Secretion. *Biological Psychiatry*, *68*(11), 1048–1054. https://doi.org/10.1016/j.biopsych.2010.07.027

van der Vegt, E. J. M., van der Ende, J., Kirschbaum, C., Verhulst, F. C., & Tiemeier, H. (2009). Early neglect and abuse predict diurnal cortisol patterns in adults: A study of international adoptees. *Psychoneuroendocrinology*, *34*(5), 660–669. https://doi.org/10.1016/j.psyneuen.2008.11.004

Voorhees, E. van, & Scarpa, A. (2004). The Effects of Child Maltreatment on the Hypothalamic-Pituitary-Adrenal Axis. *Trauma, Violence, & Abuse*, *5*(4), 333–352. https://doi.org/10.1177/1524838004269486

Walsh, K., Fortier, M. A., & DiLillo, D. (2010). Adult coping with childhood sexual abuse: A theoretical and empirical review. *Aggression and Violent Behavior*, *15*(1), 1–13. https://doi.org/10.1016/j.avb.2009.06.009

Wardenaar, K. J., Vreeburg, S. A., van Veen, T., Giltay, E. J., Veen, G., Penninx, B. W., & Zitman, F. G. (2011). Dimensions of depression and anxiety and the hypothalamo-pituitary-adrenal axis. *Biological Psychiatry*, *69*(4), 366–373.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063.

Weinstein, A. A., Deuster, P. A., Francis, J. L., Bonsall, R. W., Tracy, R. P., & Kop, W. J. (2010). Neurohormonal and inflammatory hyper-responsiveness to acute mental stress in depression. *Biological Psychology*, *84*(2), 228–234.

Weissbecker, I., Floyd, A., Dedert, E., Salmon, P., & Sephton, S. (2006). Childhood trauma and diurnal cortisol disruption in fibromyalgia syndrome. *Psychoneuroendocrinology*, *31*(3), 312–324.

Weissman, M. M., & Klerman, G. L. (1977). Sex differences and the epidemiology of depression. *Archives of General Psychiatry*, *34*(1), 98–111.

Widom, C. S., DuMont, K., & Czaja, S. J. (2007). A prospective investigation of major depressive disorder and comorbidity in abused and neglected children grown up. *Archives of General Psychiatry*, *64*(1), 49–56.

World Health Organization. (2016, September). World Health Organization. Retrieved August 30, 2017, from http://www.who.int/mediacentre/factsheets/fs150/en/

Wright, M. O., Crawford, E., & Sebastian, K. (2007). Positive resolution of childhood sexual abuse experiences: The role of coping, benefit-finding and meaning-making. *Journal of Family Violence*, *22*(7), 597–608.

Yeung, E. W., Davis, M. C., & Ciaramitaro, M. C. (2016). Cortisol Profile Mediates the Relation Between Childhood Neglect and Pain and Emotional Symptoms among Patients with Fibromyalgia. *Annals of Behavioral Medicine*, *50*(1), 87–97.

Young, E. A., Lopez, J. F., Murphy-Weinberg, V., Watson, S. J., & Akil, H. (2000). Hormonal evidence for altered responsiveness to social stress in major depression.

*Neuropsychopharmacology*, *23*(4), 411–418.

Zeidner, M., & Endler, N. S. (1996). *Handbook of coping: Theory, research, applications* (Vol. 195). John Wiley & Sons.

# Annexe 1. Regression analysis assessing the impact of confounding variables on cortisol response to stress

| Variables | B | SE (B) | β | *t* | p |
|---|---|---|---|---|---|
| Age | .002 | .002 | .071 | .917 | .361 |
| Smoking habits | -.071 | .017 | -.325 | -4.151 | .000 |
| Alcohol consumption per week | .024 | .014 | .126 | 1.656 | .10 |
| Drugs consumption | -.023 | .015 | -.120 | -1.514 | .132 |
| Flu in the past month | -.043 | .015 | -.219 | -2.935 | .004 |

Notes. B = unstandardized beta coefficient, SE(B) = standard error, β = standardized coefficient, *t* = *t*-test statistic, *p* = significance value