Influences of childhood maltreatment and genetics on emotion regulation:

A twin study

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INFLUENCES OF CHILDHOOD MALTREATMENT AND GENETICS ON EMOTION REGULATION

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Abstract

Childhood maltreatment is widely recognized as a major risk factor for adult psychopathology. Emotion regulation difficulties are increasingly becoming recognized as a central feature in several psychological disorders. The current study therefore investigated the effects of self-reported childhood maltreatment on emotional regulation strategies and difficulties, operationalized through the *Emotion Regulation Questionnaire* and the *Difficulties in Emotion Regulation Scale*, in a sample of mono- and dizygotic twins. Since emotion regulation is partly heritable, another aim of the study was to estimate the heritability of emotion regulation strategies and difficulties. This was done by comparing the correlations for mono- and dizygotic twin pairs. A third aim of the study was to examine the possibility of childhood maltreatment as a pure environmental exposure factor affecting emotion regulation. This was done based on previous research which have found gene-environment interaction and correlations for several psychological disorders. The findings of the current study showed an association of childhood maltreatment and the non-acceptance dimension of emotion regulation. An association of childhood maltreatment and the maladaptive emotion regulation strategy expressive suppression was not found. Moderate heritability was estimated for several of the emotion regulation strategies and difficulties. An effect of childhood maltreatment on the emotion regulation difficulty non-acceptance that was independent of genetics was not found, indicating a possible gene-environment correlation. However, the current study could not confirm this. The findings support previous research on heritability of some of the emotion regulation strategies and adds preliminary support to previous research findings of a gene-environment interplay in the development of emotion regulation difficulties.

Key words: Emotion regulation, Childhood maltreatment, Genetics, Heritability

Introduction

Emotions are adaptive and helpful to us. They help us direct attention to important information in our environment, enhance decision making, prepare behavioral responses, facilitate social interactions and enhance episodic memory (Gross, 2014). However, regulation of emotional responses is often called for in order to make them appropriate in magnitude, duration, or quality for a specific situation (Mcrae et al, 2017). The concept of emotion regulation (ER) includes certain strategies such as monitoring, evaluation and modulation of emotional responses in order to accomplish certain goals (decrease negative emotions and increase positive emotions) and outcomes (i.e. the consequence of using a specific strategy) (Gross, 2014). Emotion regulation has been recognized as a key component in regards to psychological and psychical health (Gross, 2014) and poor emotion regulation as a risk factor in several psychological disorders, including mood-, anxiety-, personality-, eating- and post-traumatic stress disorder (Aldao et al., 2010; Badour & Feldner, 2013; Horowitz, 2011; Stevens et al., 2013). ER difficulties have also been found to mediate the relationship between childhood maltreatment and adult psychological distress (Rudenstine et al, 2019).

Childhood maltreatment (CM) is defined by WHO (2020) as the abuse and neglect that occurs to children under 18 years of age. This includes all types of physical and emotional ill-treatment, sexual abuse, neglect or negligence that may result in actual or potential harm to the child's health, dignity, development or survival in the context of a relationship of responsibility, trust or power. A large body of research points to early trauma exposure and maltreatment as major risk factors in the development of psychopathology, including ER difficulties (Rudenstine et al, 2019; Badour & Feldner, 2013; Kuo, et al., 2015, Weissman, et al., 2019).

Linehan's *Biosocial Theory* (1993) conceptualizes ER difficulties in borderline personality disorder/emotionally unstable personality disorder (BPD/EUPD) as heightened emotional responsivity and difficulties in regulating negative affect arising from a genetic vulnerability and early childhood events which interact with an invalidating environment. Evidence support the high prevalence of CM in this population (Goodman et al., 2004). BPD/EUPD features (e.g. ER difficulties) are not exclusive to this specific clinical population but exist in the general population and other clinical and sub-clinical populations (Trull, 2001). For this reason, it makes it reasonable to consider ER difficulties as a core component in a wide range of psychological states of distress and to screen for and direct interventions addressing these issues to those affected by them (Kuo et al, 2015).

Emotion regulation has been conceptualized and measured in many different ways, due to having been the subject of research in several fields within psychology (John & Eng, 2014). One of these conceptualizations is the process model of emotion regulation developed by Gross (2014) which is derived from the conception of the emotion-generative process, i.e. emotions arise when an emotional cue is appraised or evaluated. When emotional cues are attended to and evaluated they trigger a set of experiential, behavioral and physiological response tendencies, which can be modulated in different ways depending on when in the emotion-generative process they arise. The components of the process are situation, attention, appraisal and response. The strategies for modulation (or regulation) which have been the focus of empirical research in psychology are the ones associated with appraisal and response, that is cognitive reappraisal and expressive suppression, and are typically used for down-regulating emotions of negative valence (e.g. anxiety, fear and anger. Cognitive reappraisal means to construe an emotion-eliciting situation or cue so that it changes its emotional impact. This strategy is focused on the antecedent of the emotional response, thereby determining whether or not the emotional response is triggered, and therefore has

positive effects on affective and social functioning. Expressive suppression is aimed at modulating the response in the emotion-generative process by inhibiting ongoing emotion-expressive behavior. It is directed at emotional responses that have already been triggered and is thereby less effective in attaining affective and social functioning and well-being (John & Eng, 2014). The *Emotion Regulation Questionnaire* (ERQ) was developed on the basis of this conceptualization of ERQ (Gross & John, 2003).

Another conceptualization of ER is the emotional competence approach (John & Eng, 2014), encompassing a broad set of processes, skills and competencies. It developed from clinical and developmental analyses of what children need to learn in order to become emotionally and socially competent adults. Gratz & Roemer (2004) defined emotion regulation competencies as: awareness and understanding of emotion, acceptance of emotions, and the ability to use situationally appropriate strategies flexibly in order to modulate emotional responses according to goals and situational demands. These competencies constitute the basis of the *Difficulties in Emotion Regulation Scale* (DERS), as constructed by Gratz & Roemer (2004). They defined the core regulatory concept as: “the ability to use situationally appropriate regulation strategies to modulate emotional responses”.

The heritability of ER difficulties is not yet fully determined (Mcrae et al, 2017). A behavioral-genetic study (Weinberg, et al. 2014) reported coefficients from 45% to 50% for genetic contributions for ER. Similar results have been found in studies on emotionality and emotional responding, with an estimation of 40% genetic contribution (Jang et al, 1996; Vernon et al, 2008). Furthermore, studies have estimated the heritability of the ER strategies cognitive reappraisal and emotional suppression and found the former of the two to be more influenced by genetic factors than the latter (Mcrae, et al., 2017). This speaks to the benefit of directing psychological interventions for the improvement of the ER strategies more under environmental influence, such as cognitive reappraisal (Mcrae et al., 2017). Engagement in

the less adaptive ER strategy expressive suppression have been found to be associated with childhood maltreatment in a sample of children and adolescents (Weissman, et al, 2019). The strategy was also found to be interrelated with other maladaptive ER strategies, rumination and emotional reactivity, making it more of a contributing factor in the development of psychopathology (Weissman, et al. 2019). It has also been found to be strongly related to personality variables such as temperament which remains stable over time (Gross & John, 2003). In a study assessing ER difficulties as a risk marker for eating disorders through twin pair analysis, Kanakam el al (2013) found evidence for the influence of shared environment on ER difficulties but not for heritability. A meta-analysis (Aldoa et al, 2009) which examined the relationship of dispositional ER and psychopathology found the maladaptive strategies of avoidance and suppression to be positively associated with overall psychopathology. The predisposing nature of the avoidance-strategy and suppression could be indicative of heritability. However, research investigating the heritability of these ER difficulties is sparse.

Several studies have found significant correlations for ER and childhood maltreatment, as measured through *The Difficulties in Emotion Regulation Scale* (DERS), in psychiatric and non-clinical populations (Burns, et al, 2010; Gratz, et al. 2007, Jennisen, et al. 2016; Rudenstine et al., 2018). Jennisen et al (2016) found that ER difficulties mediate the relationship between childhood maltreatment and adult psychopathology. The authors found a strong correlation for childhood maltreatment and the DERS scales and moderate to large correlations for the DERS scales and psychopathology. Lack of access to strategies for emotion regulation (DERS strategies) was found to be the strongest predictor for adult psychopathology (r=.73, ps<.05) (Jennisen, et al, 2016). Gratz et al (2007) found evidence of increased emotional non-acceptance (along with experiential avoidance) among an adult female sample with a history of childhood maltreatment. This finding adds support to the

Hayes et al. (1996) argument for the avoidance of unwanted internal experiences as a central component in psychopathology such as PTSD and BPD-symptoms.

One way of estimating heritability is through twin studies, which has long been held as "golden standard". This is done based on the fact that identical twins (monozygotic, MZ) share 100% of their genes and non-identical (dizygotic, DZ) 50%, making it possible to estimate the genetic influence on a given variable by comparing the correlation between MZ twin pairs with the correlation between DZ twin pairs. A stronger correlation between MZ twin pairs indicates genetic influence on the variable. The classical twin design builds upon this fact (differences in genetic sharing of MZ and DZ twins) and from this utilizes simultaneous equations to separate the variance on a given variable (phenotype) resulting from additive genetic (A), common or shared environmental (C) and unique or unshared environmental influences (E). Common or shared environment refer to the shared experiences of twins (home environment, socio-economic status, parents, etc.), including intrauterine environment. Unique environmental influences refer to the physical and social environment that are unique to each twin, such as peer influence, psychological and physical trauma etc. (Blokland et al., 2013). One of the underlying assumptions of the classical twin model is that MZ and DZ twins shared environments are equal, i.e. environmental influences contribute equally to the similarity of MZ and DZ twin pairs on a given trait (Blokland et al., 2013). Heritability of ER and related constructs have been estimated using extensions of the classic twin model (McRae et al. 2007; Jang et al, 1996).

In addition to estimating the effects of genetic and environmental influences upon a trait, gene-environment interaction and correlation effects (i.e. the joint effect of genes and environment) are possible to estimate through extensions of the classical twin design (Dick, 2011). Understanding the genetic and environmental risk factors and how they interact is of importance for a better understanding of the etiology of psychiatric disorders and other

psychological outcomes (Dick, 2011). Distel et al, found gene-environment interaction and correlation effects for BPD/EUPD features and negative life events. This means that the effects of genes and environment on certain outcomes are not independent of each other, such that environmental factors can mediate genetic risks for psychopathology (Rutter et al., 2001). The effect of genes and environmental factors can however also be independent of each other, but the possibility of the association of environmental risk factors and psychopathology being there because of shared genetic factors influencing both environment and outcome has been shown. A gene-environment correlation, in which the genes exert influence on the environment, can occur through three ways (Plomin et al., 1977; Scarr & McCartney, 1983); 1) passive gene-environment correlation, referring to the correlation of e.g., a child's genotype and home environment, 2) evocative gene-environment correlation, in which an individual's temperament or disposition influence the response they receive from others, and 3) active correlation, in which an individual with a certain disposition selects certain environments accordingly.

Evidence of genetic contribution to ER strategy expressive suppression (McRae, et al. 2017) and an association with CM (Weissman et al., 2019) has thus been shown. Heritability of the dimensions of ER measured by the DERS scale is however sparse. However, some evidence points to the non-acceptance of emotions and lack of strategies for ER as a predisposing and predictive factor for adult psychopathology (Aldoa, et al. 2009; Jennisen et al., 2016). The association of ER difficulties (as measured by the DERS scale) and CM is well established (Burns et al, 2010; Gratz et al. 2007; Jennisen et al. 2016; Rudenstine et al., 2018). Evidence of the combined effect of genetics and negative life events (as an interaction effect and correlation of the two) have been found for BPD/EUPD features (Distel et al., 2011), other personality disorders (Bulbena-Cabre et al., 2018) and depression (Kendler et al., 1995). Gene-environment interaction and correlation effects have been documented

across many clinical disorders and their effects are closely intertwined. Therefore, research aiming at identifying risk factors and etiological pathways need to take into account the contribution of both (Thapar, 2007). Accounting for the possibilities of combined effects (as gene-environment interaction and correlation effects) and parsing out the effects of these on ER is thus motivated.

Research purpose

Given the vast amount of evidence for the negative consequences of childhood maltreatment on emotion regulation difficulties, which are an essential part of psychological well-being, this study aims to investigate the etiological basis of ER difficulties through a twin design. This makes it possible to estimate the heritability and environmental contribution of ER strategies and also to explore the possibility of interactions and correlation effects of these. Childhood maltreatment is in the current study regarded as an environmental exposure. A better understanding of the etiological pathways underlying ER difficulties, i.e. estimating the effect of CM independently of genetics, could contribute to the development of new interventions and the effectiveness of existing ones. The current study intended to examine the effect of CM on ER difficulties, by calculating the correlations between the CM total scale and the ERQ and DERS-scales. Based on previous research a positive association between CM and ERQ scale expressive suppression and CM and DERS nonacceptance and lack of strategies was hypothesized. The second aim of the study was to estimate the heritability of ER difficulties through MZ and DZ twin pair correlations, operationalized through DERS and ERQ. The strategy expressive suppression was hypothesized to be more heritable than cognitive reappraisal. The nonacceptance and strategies scales were hypothesized to be more heritable than the other ER difficulties measured by the DERS. A third aim of the study was to test if CM had an effect on ER independent of genetics. Since both genes and environment contribute to the development of traits and psychopathology

(Dick, 2011; Thapar, 2007), investigating the nature and degree of their interconnectedness and identifying etiological pathways is of importance. One way to do so is to assess the influence of CM on ER independent of genetics, thereby accounting for the possibility of gene-environment interaction and correlations. The estimated effects of CM on ER were explored through correlation analyses. The current study hypothesize the following statements:

H1: There is a correlation between CM and DERS scores and CM and ERQ scores (i.e. there is an association between childhood maltreatment and ER).

H1a: Emotion regulation strategy expressive suppression is associated positively with CM.

H1b: Emotion regulation difficulties nonacceptance and lack of strategies is associated with CM.

H2: There is a genetic contribution to emotion regulation difficulties, as measured through DERS and ERQ-scales.

H2a: Expressive suppression is more heritable than cognitive reappraisal.

H2b: Nonacceptance and lack of strategies are more heritable than other ER difficulties as measured by the DERS scales.

H3: There is an effect of CM on ER independent of genetics.

Method

Participants

The current study used data from the *TwinfMRI* research project of 2016-2017. The participants were recruited through the Swedish twin registry. A total of 5000 twins were contacted for participation of which *n*=646 accepted to participate. Of these *n*=333 were excluded after a screening procedure. Exclusion criteria were a history of severe genetic or

neurological syndromes, agoraphobia or severe mental disorder. Furthermore, additional exclusion criteria were used for the MR scans. From the total set of 307 MZ and DZ twin-pairs, a number of pairs (*n*= 26) were excluded from the analyses due to missing answers in the current study. Another amount of five participants (*n*=5) were excluded from the analysis for being singletons (i.e. missing a twin sibling). Only DZ twin pairs of the same sex were recruited, in order to minimize effect of sex in comparison with the MZ twin pairs. The groups were equally distributed on sex, DERS-and ERQ scores (see table 1 for descriptive statistics and tests for significance). For the age-variable, the distributions differed between the groups with DZ twin pairs showing a median age of 20-25 and MZ twin pairs 30-35 years of age.

Table 1.

Descriptive statistics for MZ- and DZ twin pairs and test for significance between groups

Variable	MZ *n*=136	DZ n=140	x^2/t	*p*
Sex [male/female]	54/82	54/86	0.037	.85[a]
Age (years)	34.4 (8.3)	33.7 (11.9)	-0.55	.58[b]
CM-total	0.98 (1.7)	0.85 (1.5)	-0.67	.50[b]
DERS-total	70 (19.6)	69.6 (20.3)	-0.15	.88[b]
DERS-nonaccept	11.7 (5.5)	11.7 (5)	0.012	.99[b]
DERS-goals	12.3 (4.4)	11.7 (4.5)	-1.001	.32[b]
DERS-impulse	9.7 (3.7)	9.6 (3.6)	-0.241	.81[b]
DERS-awareness	14.1 (4.3)	14.1 (4.9)	0.097	.92[b]
DERS-strategies	13.6 (5.5)	13.8 (5.7)	0.236	.81[b]
DERS-clarity	8.5 (3.1)	8.5 (3.4)	0.146	.88[b]
ERQ-total	41.2 (8)	41.4 (8.3)	0.242	.81[b]
ERQ-cognitive.	28.9 (6.1)	28.7 (7.2)	-0.334	.74[b]
ERQ-expressive.	12.2 (5)	12.7 (5.1)	0.831	.41[b]
Education [1/2/3]	8/21/107	4/35/101		

Note: Levels of education: 1= primary school, 2= secondary school, 3= post-secondary education. Values presented in frequency, sex and education or mean *m(sd)* for age and DERS- and ERQ-scale.
[a] tested with chi-square test
[b] tested with independent samples t-test

Material

The *Difficulties in Emotion Regulation Scale* (DERS) is a 36-item questionnaire assessing emotion regulation across six dimensions; (a) lack of emotional awareness, (b) lack of emotional clarity, (c) non-acceptance of negative emotions, (d) limited access to emotion regulation strategies, (e) difficulties controlling impulsive behavior when experiencing negative emotions and (f) inability to engage in goal-directed behavior when experiencing negative emotions. Each item is rated on 5-point Likert scale ranging from 1 (*almost never*) to 5 (*almost always*). Gratz & Roemer (2004) estimated a high internal consistency for the different items (Cronbach's alpha .93) and adequate internal consistency between its subscales (Cronbach's alpha >.8) in a non-clinical sample of under-graduates in their initial validation paper. The DERS total scale showed good test-retest reliability and the subscales showed adequate test-retest reliability. Hallion et al. (2018) showed good internal consistency for all scales except awareness in a clinical population. The Swedish validation paper (Bjureberg et al., 2015) showed a similar internal consistency, *Cronbach's alpha r*anging .92 to .95. The DERS has been shown to have adequate construct and predictive validity (Graetz & Roemer, 2003).

The *Emotion Regulation Questionnaire* (ERQ; Gross & John, 2003) is a 10-item questionnaire comprised of the two scales Reappraisal and Suppression. Both the reappraisal ($\alpha = 0.84$) and the expressive suppression subscales ($\alpha = 0.75$) demonstrated adequate internal consistency in a non-clinical sample. The ERQ has showed good discriminant and convergent validity (Gross & John, 2003).

The CM measurement was comprised of items from the original questionnaire in the *TwinfMRI* project. The items addressed the above mentioned categories of CM: sexual abuse had two items: (1) *Have you ever been touched, or forced to touch someone in a sexual manner* and (2) *Have you ever had sex because you felt forced, or felt that you or someone*

else were threatened?. Physical abuse and neglect had two items: (1) *Have you ever been physically neglected? For example not been given food, not given proper clothing or been left to take care of yourself when you felt that you were too young or unable to do so,* and (2) *Have you ever been physically abused? For example beaten, almost choked, burned, or severely punished, by someone you knew well for example a parent*. Emotional neglect and abuse had one item; *Have you ever been emotionally abused or neglected? For example been told off, embarrassed, ignored or told that you are not good enough.* Each item was scored based on frequency of occurrence before 18 years of age, ranging from zero to three; 0 representing non-occurrence of the item, 1 representing "One time", 2 representing "A few times" and 3 representing "Often". There was also a "Don't know/don't want to answer"-alternative. The total score of the items were summed up to measure the total CM variable. The maximum score on the scale was 12. The scale was not a validated questionnaire and the most reliable measure that could be calculated was the total CM score. Hence, only the total scale was used.

Analysis

The association of CM and ER and ER difficulties was assessed by calculating the correlation coefficients for the CM, DERS and ERQ-scales. The genetic contribution to DERS and ERQ total score and subscale scores were estimated by comparing MZ and DZ correlation coefficients ($r_{MZ,}$ r_{DZ}). Genetic contribution would be assumed if the correlations for MZ twins exceeded the correlations for the DZ twins (Blokland, et al., 2008). The heritability (h^2) was estimated using Falconer's formula (Mayhew & Meyre, 2017) in which r_{MZ} twin pairs was subtracted with r_{DZ} twin pairs and multiplied by two, $h^{2=}2*(r_{MZ}-r_{DZ})$. MZ share 100% of their genetics, therefore h^2 can never exceed $r_{MZ,}$ h^2 =min [r_{MZ}, $2*(r_{MZ}-r_{DZ})$]. For the calculations where h^2 exceeded $r_{MZ,}$ h^2 was adjusted to $r_{MZ.}$ For the calculations were

r_{DZ} exceeded r_{MZ}, h^2 was adjusted to zero (0). Based on the results from the H1 analysis (i.e. CM-ER correlations), the scales which showed a significant correlation with CM (i.e. DERS nonacceptance) were further assessed in the H3 analysis (i.e. tests for an effect of CM independently of genetics). Consequently, a t-test was performed for the CM-discordant MZ twin pairs (i.e. one twin exposed to CM and the other not exposed) to assess the effect of CM (as an environmental factor or exposure) on ER independent of genetics. The cut-off for CM was a score of 1 on the CM total scale. To test for a genetic influence on CM (i.e. self-reports of CM), h^2 was calculated for MZ and DZ twin pairs for the CM total scale.

Due to the highly skewed distribution of the CM scale, Spearman's *rho*-coefficient ($r_{S)}$ was used to calculate the correlations between CM and DERS/ERQ scales. Spearman's *rho* was also used for other twin pair correlations where DERS scores were non-normally distributed. Pearson's *r* were also calculated and reported for these analyses for transparency reasons. To minimize effects of extreme values which could lead to spurious findings, outliers were excluded using Box-plots (labeling data points outside the 25-75% quartile range) for the between twin pair correlations. All statistical analyses were computed in JASP (0.16.1., 2022)

Ethics

The *TwinfMRI* research project ethics (and thereby the current study's) was tested and approved by the Regional Review Board in Uppsala (registration number 2016/171). All participants gave their written consent to participate in the study, in accordance with the Helsinki Declaration. The consent included that the data could be used in future research. Data used in the current study has been obtained as de-identified and only data relevant to answer the research questions has been assessed. The procedure is therefore considered to be in line with research ethical guidelines.

Results

Estimation of effect of childhood maltreatment on emotion regulation difficulties

To test the first hypothesis of the study (H1), estimation of CM effects on ER difficulties, a correlation analysis was performed. The analysis yielded one significant correlation between subscale nonaccept and CM scale (r_{s276}=.13, p= .026). The ERQ-total and subscales correlations with CM were also calculated, yielding no significant results. See table 2 and 3.

Table 2

Correlation coefficients for DERS-scales and Childhood maltreatment scale

	CM-total				
Variable	*n*	*r*	*p*	*rs*	*p*
DERS-total	276	.05	.378	.11	.076
DERS-nonaccept	276	.09	.145	.13*	.026
DERS-goals	276	-.05	.451	.05	.404
DERS-impulse	276	.01	.931	.08	.171
DERS-awareness	276	.06	.352	-.00	.977
DERS-strategies	276	.04	.474	.09	.124
DERS-clarity	276	.09	.154	.08	.181

Note. Correlation coeffiecents between DERS (total score and subscale scores), Pearson's correlation coefficient (r), Spearman's rho (r_s) and significance level (p). *p<.05

Table 3

Correlation coefficients for ERQ-scales and Childhood maltreatment scale

Variable	*CM-total*				
	n	*r*	*p*	*rs*	*p*
1. ERQ-total	267	.14	.021	.02	.708
ERQ- cognitive	267	.07	.244	.01	.860
ERQ-expressive	267	.13	.031	.07	.278

Note. Correlation coefficients between ERQ (total and subscales), Pearson's correlation coefficient (*r*), Spearman's rho (*rs*) and significance level (*p*).

Estimation of genetic contribution to emotion regulation

To test the second hypothesis (H2), which predicted a genetic contribution to emotion regulation, correlation analyses for the MZ and DZ twin pairs were performed for the total scores and subscale scores of the DERS and ERQ. *S*pearman's *rho* coefficient was used due to the non-normal distribution of the scales (Pearson's *r* included for transparency). The heritability (h^2) was estimated to r_{MZ} for the scales where h^2 exceeded r_{MZ} since r_{MZ} is consider the upper limit for heritability. The correlation analyses showed positive correlations which were higher for MZ twin pairs than for DZ twin pairs on each scale except for DERS clarity which, when outliers were excluded, showed a DZ correlation coefficient (rs_{64}=.34, p=.007) higher than the MZ correlation coefficient (rs_{64}=.24, p=.058). The r_{MZ} was however only near-significant. Positive significant r_{MZ} were found for all other scales and h^2 could be estimated for: DERS-total (rs_{67}=.50, p=<.001, h^2=.48, 48%), nonacceptance (r_{s64}=.41, p=.001, h^2=.41, 41%), goals (rs_{68}=.33, p=.006, h^2=.28, 28%), impulse (r_{s62}=.48, p=<.001, .*h*2=.48, 48%), awareness (r_{s67}=.247, p=.004, h^2=.18, 18%) and strategies (r_{s63}=.52, p=<.001, h^2=.52, 52%).

The analysis of the ERQ scales yielded significant results for the MZ twin pairs in all of the scales: ERQ total (r_{s65}=.26, p=.039), ERQ cognitive reappraisal (r_{s65}=.37 p=.003) and ERQ expressive suppression (r_{s68}=.45, p=<.001). For the DZ twin pairs, the expressive

suppression scale correlation was significant (r_{s70}=.24, p=.048). The heritability (h^2) was estimated to 43% for expressive suppression and 37% for cognitive reappraisal. See table 4 and 5.

Table 4

Correlation coefficients for MZ and DZ twin pairs for DERS scales

	MZ twin pairs			DZ twin pairs			
Variable	*n*	*rs(r)*	*p*	n	*rs(r)*	*p*	h^2
DERS-total	67	.50(.48)	<.001***(.001)	67	.26(.27)	.037(.0.27)	.48(.43)
DERS-nonacceptance	64	.41(.35)	<.001***(.005)	67	.14(18)	.249(.141)	.41(.33)
DERS-goals	68	.33(.34)	.006**(.005)	69	.19(.19)	.115(.124)	.28(.30)
DERS-impulse	62	.48(.40)	<.001***(.001)	64	.01(.10)	.955(.414)	.48(.40)
DERS-awareness	67	.25(.22)	.044*(.076)	69	.16(.13)	. 195(.287)	.18(.18)
DERS-strategies	63	.52(.48)	<.001***(.001)	64	.22(.26)	.082(.038)	.52(.44)
DERS-clarity	64	.24(.22)	.058(.075)	64	.34(.39)	.007(.001)	0(0)

Note: Correlation for MZ- and DZ twin pairs respectively for DERS scales, number of participants (*n*) after removal of outliers, Spearman's correlation coefficient (*rs*), Pearson's correlation coefficient (*r*), significance (*p*) and estimated heritability (h^2).
*p<.05, **p<.01, ***p<.001

Table 5

Correlation coefficients for MZ and DZ twin pairs for ERQ scales

	MZ twin pairs			DZ twin pairs			
Variable	*n*	*rs(r)*	*p*	n	*rs(r)*	*p*	h^2
ERQ-total	65	.26(.24)	.039*(.056)	66	.07(.11)	.57(.365)	.37(.25)
ERQ-cognitive	65	.37(.37)	.003**(.002)	69	.08(.09)	.512(.462)	.37(.37)
ERQ-expressive	68	.45(.47)	<.001***(.001)	70	.24(.22)	.048*(.068)	.43(.47)

Note: Correlation for MZ- and DZ twin pairs respectively for ERQ scales, number of participants (*n*) after removal of outliers, Spearman's correlation coefficient (*rs*), Pearson's correlation coefficient (*r*), significance (*p*) and estimated heritability (h^2).
*p<.05, **p<.01, ***p<.001

Estimation of CM effect on ER difficulties independent of genetics

To assess whether CM had an effect on DERS-nonacceptance that was independent of genetics, a T-test between CM discordant MZ twins was performed with CM exposed twins in one group and the non-exposed co-twins in the other. The test yielded a non-significant result, $t(42)=-.210$), p=.835. This indicated that there is no effect of CM separate from genetics. To test for a genetic influence on CM, $r_{MZ,}$ r_{DZ} and h^2 for CM was calculated. The r_{MZ} was weak but significant (r_s=.32, p=.009) and h^2 was estimated to r_{MZ}. This indicated a higher r_{MZ} than r_{DZ} for CM. See table 6 and 7.

Table 6

T-test for MZ twins discordant for CM

	MZ twins CM		MZ twins no CM		*t*(42)	*p*
Variable	*M*	*SD*	*M*	*SD*		
DERS-nonacceptance	14	5.69	13.59	7.18	-0.210	.835

Note: T-test for significant differences MZ discordant for CM on DERS-nonacceptance scores, mean (M), standard deviation (SD) and *t*(degrees of freedom).

Table 7

Correlation coefficients for MZ and DZ twin pairs for CM total scale

	MZ twin pairs			DZ twin pairs			
Variable	*n*	*rs(r)*	*p*	n	*rs(r)*	*p*	h^2
CM-total	66	.32(.37)	.009**(.002)	69	.07(.13)	.55(.30)	.32(.37)

Note: Correlation for MZ- and DZ twin pairs respectively for CM total scale, number of participants (*n*) after removal of outliers, Spearman's correlation coefficient (*rs*), Pearson's correlation coefficient (*r*), significance (*p*) and estimated heritability (h^2).
*p<.05, **p<.01, ***p<.001

Discussion

The current study aimed to investigate the associations of CM and ER strategies and difficulties. It also estimated the heritability of these. Furthermore, it intended to examine if CM had an effect on ER that was independent of genetics. The results showed an association of CM and the non-acceptance dimension of ER and support for the heritability of ER strategies and difficulties as measured by the ERQ and DERS respectively. An effect of CM on ER independent of genetics was not found.

The first hypothesis of the study was that there should be a positive correlation between CM and the expressive suppression strategy and nonacceptance and lack of strategies dimensions of ER. DERS nonacceptance and CM showed a weak but significant positive correlation, indicating an effect of CM on this dimension of ER. This is in line with previous research which found positive associations for the DERS nonacceptance scale and post-traumatic stress symptom severity (Burns et al., 2010; Tull et al., 2007) in populations exposed to CM. The nonacceptance scale intends to measure avoidance of unwanted internal experiences (Gratz & Roemer, 2003), which has been found to be a common factor across psychological disorders (Aldoa et al, 2009). The current result of an association between CM and nonacceptance can therefore be argued to add to the previous findings of ER difficulties as a central component of psychopathology in CM exposed individuals since CM is well established as a risk factor for psychopathology (Badour & Feldner, 2013; Rudenstine et al., 2018). Furthermore, Gratz et al. (2007) found emotional non-acceptance to mediate the relationship between CM and experiential avoidance, adding evidence to the Hayes et al. (1996) theory of experiential avoidance as a central element in abuse-related psychopathology. Considering CM to be an exposure which increases the risk for adult psychopathology, the results could be interpreted to support the environmental influence of CM on this dimension of ER. This adds to the previous findings supporting the utility of directing interventions to this dimension of ER in trauma-associated psychological disorders (Tull et al., 2016; Rudenstine et al., 2019; Gratz et al., 2007). No significant association of CM and expressive suppression was found. This is contrary to previous research which found the expressive suppression strategy to be correlated with CM (Weissman et al., 2019). However, the previous association was found in a sample of children and adolescents aged 8-16. The use of this strategy has been found to decrease from childhood to middle adolescence to then stabilize in adulthood (Gullone et al., 2010; Gullone & Taffee, 2012). A lack of

association of CM on this strategy of ER in the current adult sample could be explained by this. However, since expressive suppression is associated with adult psychopathology (Aldoa et al., 2009), it would be undue to not consider it as being associated with the negative psychological consequences of CM. McRae et al (2017) suggest that since expressive suppression was more heritable than cognitive reappraisal (and as heritable as emotionality more generally), it would be less influenced by environmental factors than cognitive reappraisal. This could explain the current results.

The second hypothesis was that there is a genetic contribution to ER and, more specifically, a larger heritability of nonacceptance, lack of strategies and expressive suppression. The results of the analysis showed the highest heritability for the lack of strategies scale, estimated to 52%. The heritability of the nonacceptance scale was estimated to 41%, which was lower than the total scale and the impulse scale. A genetic influence would be assumed if the correlation for MZ twin pairs is approximately twice the size of the correlation of the DZ twin pairs. DERS clarity showed a DZ twin pair correlation coefficient higher than the MZ twin pair correlation coefficient. This points to a higher contribution of environmental factors to this dimension of ER and therefore making it a reasonable focus of psychological interventions (Mcrae et al., 2017). Results should however be interpreted cautiously since MZ correlation was only near significant. Furthermore, this dimension of ER was not associated with CM and therefore it is beyond the scope of this study to draw conclusions of the nature of the environmental influences for it. The highest heritability was found for the DERS strategies scale. Given the previous finding that this ER dimension best predicted adult psychopathology (Jennisen, et al, 2016), this result could be expected.

The heritability (h^2) of the ERQ scales was estimated to 37% for ERQ-total, 37% for cognitive reappraisal and 43% for expressive suppression. These findings are in line with McRae et al. (2017) which found expressive suppression to have higher heritability than

cognitive reappraisal. This finding adds support to the utility of making the cognitive reappraisal strategy a focus of psychological intervention.

The effect of CM on the nonacceptance scale independently from genetics was assessed by comparing the means of MZ twin pairs which were discordant for CM. The test yielded no significant effect of CM when genetics where controlled for. In other words, no effect of CM on the nonacceptance dimension of ER that were separate from genetic effects could be found. This result points to CM as not an entirely "pure" exposure effect on the nonacceptance dimension in this sample, i.e. a genetic effect on CM might be assumed. This prompted further analysis in order to assess whether there is a genetic influence on CM. The results showed a higher (but weak) correlation for MZ twin pairs than DZ twin pairs and a heritability was estimated; a genetic influence on CM could therefore be assumed. The association of the CM scores and the nonacceptance scale and the estimated heritability of CM could indicate that the same genetic factor influencing CM (liability) also influences the non-acceptance dimension of ER. However, the current sample was too small to perform the analyses needed to confirm this.

The results could be (assuming there is a genetic factor influencing the CM variable) explained by previous research on gene-environment correlations in twin studies (Thapar, 1996; Plomin et. al. 1977) showing how individuals actively or passively expose themselves to different environments or evoke other individuals responses to them. An example of this is from Distel et al. (2011) where they found that the genes that influence BPD/EUPD features also increased likelihood of being exposed to certain life events, such as divorce and violent or sexual assault. This was demonstrated through a twin design and the gene-environment correlation of BPD/EUPD features and adverse life events is explained drawing from Kendler (2003), which found common familial factors to be predisposing for both neuroticism (a

personality trait associated with BPD/EUPD) and environmental adversity. These familial factors were estimated to be partly genetic.

The assumption of equal shared environments of MZ- and DZ twins of the classical twin model would be violated if there was a correlation between genes and environment on the relevant environmental measure and if the environment showing that correlation had an effect within MZ pairs (Rutter et al., 2001). The gene-environment correlation may be understood as a passive correlation. However, if this was true, the correlation would not be higher for MZ than for DZ twins (since they both share the same home environment). For this to be the case, the assumption of shared home environment would not hold true. This assumption has been challenged by findings showing MZ twins being treated more similar than DZ twins (Blokland, et al. 2013). This would be an example of an evocative correlation. However, other research has not been able to prove that greater environmental similarity results in greater phenotypic similarity (Blokland et. al, 2013). For the current study, this would be similarity in self-reported CM. Another possible explanation may be that there is a greater similarity in reported CM due to greater similarity in predisposition to select certain unique environments (active correlation), e.g. being exposed to negative life events such as abuse. Kendler & Baker (2006) reported, in their meta-analysis on studies of genetic influences on environment, a heritability of being exposed by way of active correlation. A greater similarity in non-shared environment would thus indicate genetic influences on this variable.

One factor that might have influenced participants' responses is age. The t-test for MZ and DZ twins' age showed no significant difference for mean age. However, the median age was 20-25 for DZ twins while 30-35 for MZ twins. The lower median age of the DZ twins could have affected their responses on the DERS and ERQ scales in relation to the MZ twins, considering that the use of some ER strategies varies with age (Gullone & Hughes, 2010;

Gullone & Taffee, 2012). The age span that might be of interest in the current sample is 20-35 years of age. Therefore it would make sense to consider changes in ER that takes place from late adolescence to adulthood. The changes in ER that takes place *during* adolescence (due to increased ability for cognitive control, neurophysiological development and socioemotional changes etc.) are however more comprehensive (Riedeger & Klipker, 2014). The current study however, found no statistically significant differences in responses between the MZ and DZ twins for the ER scales. This could be interpreted to support the finding that some dimensions of ER tend to stabilize in adulthood (Gullone & Hughes, 2010).

Put together, the results of the study indicate that both genes and CM contribute to ER strategies and ER difficulties. They also indicate that there is a gene-environment correlation (and possibly interaction) of CM and genetics on the non-acceptance dimension of ER. Although this could not be confirmed, the current study concluded that there is no effect of CM on ER independent of genetics. The lack of association of CM to the expressive suppression scale in addition to the estimated heritability of this strategy could indicate that this strategy is less affected by environmental factors.

Limitations

There are several limitation to this study worth mentioning. First of all, the estimations of heritability of ER and CM should be interpreted cautiously since the correlation coefficients for DZ twin pairs were non-significant. This would in itself not render h^2 invalid since it is based on r_{MZ} being higher than $r_{DZ.}$ However, further analysis would be needed to statistically secure the estimated heritability of these measures. To make these analyses, a structural equation model would be used (Blokland et al., 2013). It is also worth noting that the estimated associations of CM and ER difficulties and strategies are based on correlations; making any claims about causality would thus be undue.

The CM scale was not validated and tested for internal consistency. Therefore, the operationalization of CM may have differed from that of previous research on the subject. It also makes the inconsistency of interpretation of the items a possible measurement error. Furthermore, the items were not specifically concerning abuse in the home environment and therefore the question of shared or non-shared environmental influences on ER (i.e. if CM was part of the home environment) remained unresolved. The items did however ask about participant's age at the time of the incidents. Taking this into account would have given an indication of shared vs. non-shared environments.

Another limitation is the self-report nature of all measurements used in the current study. This means that participants' responses could be influenced by their accuracy in their assessment of their behavior and their willingness to report incidents of maltreatment (Burns et al., 2010; Gratz et al., 2007). The responses for the CM scale could in addition to this be influenced by retrospective bias (Gratz et al., 2007).

Conclusions

The results of this study adds support to previous research of joint influences of childhood maltreatment and genetics on emotion regulation difficulties. It also adds support to previous studies' estimations of heritability of specific strategies of emotion regulation as well as preliminary support to which emotion regulations difficulties measured by the DERS are more heritable. To understand the interplay of genetics and environment (exposure to childhood maltreatment more specifically) is of importance for a better understanding of the etiology of psychiatric disorders of which emotion regulation difficulties is a central component. This plays a role in the development of new, and the improvement of existing, psychological interventions. Future research should examine the possibility of gene-environment interaction effects on emotion regulation and the gene-environment correlation of genetics and childhood maltreatment on the non-acceptance dimension of emotion

regulation, .i.e. if the same genes that affect the likelihood of being exposed to childhood maltreatment also affects the non-acceptance of negative emotions. In addition to this, an aim for further research could be to examine the effect of different types of maltreatment on emotion regulation difficulties. For example, studies have found emotional abuse and neglect to have considerable impact on emotion regulation (Burns et al. 2010; Jennisen et al., 2016).

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The author of the current study has not partaken in the collection of data for the above mentioned research project. Research questions have been formulated by the author and revised in consultation with the supervisors. Statistical analyses has been done by the author. Another thesis was written within framework of the project which analyzed other variables than the current thesis with the exception of child maltreatment

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