

Associations between parental overprotection, maladaptive perfectionism, and chronic and acute stress reactivity

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Long-term stress exposure is associated with a wide range of different negative health consequences. In addition, numerous studies show an influence of maladaptive perfectionism on stress reactivity. A frequently discussed explanation for the development of maladaptive perfectionist attitudes refers to parental influences. In this study, the relationship between parental overprotection, maladaptive perfectionism, and acute and chronic stress reactivity were investigated in a standardized laboratory setting. For this purpose, 67 healthy students (59.7% female, age mean= 21.60; age SD= 3.10) were invited to the laboratory twice to be subjected to an acute stress condition using the Montreal Imaging Stress Task (MIST), or a relaxation condition using relaxation videos. Psychological questionnaires were used to record maladaptive perfectionism, parental criticism, parental overprotection, and vital exhaustion as a measure of chronic stress reactivity. While no significant increase in cortisol was observed in response to the MIST, a significant correlation between maladaptive perfectionism and vital exhaustion was shown with regard to chronic stress. In addition, a mediation effect of parental criticism on the relationship between parental overprotection and maladaptive perfectionism could be demonstrated. These results suggest that parenting styles are associated with the child's personality characteristics, and chronic stress levels later in life. The results are discussed in the light of strengths and limitations of the study.

Keywords: maladaptive perfectionism, stress reactivity, parental overprotection, parental criticism, vital exhaustion, Montreal Imaging Stress Test

Perfectionism is a multidimensional personality disposition characterized by the pursuit of flawlessness and above-average performance standards. As such, perfectionists critically evaluate their own behavior. Based on theoretical considerations, perfectionism has initially been defined as being an extremely neurotic personality disposition without any positive aspects. Yet, recent models focus on both, adaptive and maladaptive aspects of perfectionism (Bieling et al., 2004; Frost et al., 1990; Hewitt & Flett, 1991).

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While adaptive perfectionism has been associated with the achievement of high standards and the experience of associated rewards (Bieling et al., 2004), maladaptive perfectionism has been linked to less life satisfaction and stress (Chang et al., 2004). Furthermore, maladaptive perfectionism

increases the risk for different psychopathologies (Shafran et al., 2002), for example depression (Hewitt & Flett, 1991), anxiety disorders (Antony et al., 1998), eating disorders (Halmi et al., 2000), obsessive-compulsive disorders (Cassidy et al., 1999), chronic sleeping disorders (Vincent & Walker, 2000), and relationship problems (Martin & Ashby, 2004). In this sense, maladaptive perfectionism is both, a risk factor, and a perpetuating factor for various disorders (Egan et al., 2011).

Like maladaptive perfectionism, stress is another important psychopathological risk factor. Acute stress activates various stress systems, e.g., the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis. Consequently, adrenaline and

cortisol provide the organism with the necessary resources to adapt to the situation (Sapolsky, 2000). While adaptive response supports survival, excessive activation of the stress systems caused by chronic stress can wear out the system. As such, the increased allostatic load associated with chronic stress initially leads to hyperactivity of the HPA axis (Juster et al., 2010). At a certain level, however, a breakdown of the stress system occurs, which is accompanied by hypoactivity of the HPA axis (Heim et al., 2000; Van Houdenhove et al., 2009). This results in blunted physiological stress responses and states of vital exhaustion (VE). VE describes extreme fatigue, a severe loss of physical and mental energy, increased irritability, and feelings of demoralization (Appels et al., 1987; B M Kudielka et al., 2006). As such, chronic stress and VE are risk factors for secondary disorders, such as coronary heart disease (Balog et al., 2017; Lupien et al., 2015). Of importance in this context is the mutual connection between maladaptive perfectionism and stress.

The diathesis-stress model of perfectionism explains how stress and maladaptive perfectionism are mutually dependent and can reinforce each other (Hewitt & Flett, 2002). On the one hand, perfectionists might be more frequently exposed to stress through their continuous striving for unattainable standards (Hewitt & Flett, 2002), and more frequent negative social interactions (Dunkley et al., 2006), or interpersonal conflicts (Mackinnon et al., 2012). On the other hand, life circumstances that do not fit the perfectionist's idealized ideas can induce a so-called perfectionist reactivity. Consequently, perfectionists are more vulnerable to maladaptive cognitive, emotional, motivational and behavioral reactions, negative self-evaluation and various forms of psychopathology (Hill, 2016). Accordingly, in relation to the transactional stress model (Lazarus & Folkman, 1984), maladaptive perfectionism plays an important role in primary and secondary stress appraisal. Maladaptive perfectionists generally consider stressful situations as more relevant and threatening (perfectionistic strivings), and think they are not able to adequately cope with the situation (perfectionistic concerns) (Stoeber & Otto, 2006). As an exemplary consequence of this, perfectionist students exhibit more intensive stress symptoms after experiencing academic failure (Rice et al., 2015). What is more, participants with maladaptive perfectionism showed increased sympathetic reactivity by means of alpha-amylase responses to psychosocial stress (McGirr & Turecki, 2009). Consistently, maladaptive perfectionism was

associated with increased endocrine reactivity by means of cortisol responses in male subjects following psychosocial stress (Wirtz et al., 2007). At first glance, this seems to contradict results showing an increased subjective stress reactivity in maladaptive perfectionists, but a lower cortisol reactivity in response to experimental stress (Richardson et al., 2014). One possible explanation are heterogenous operationalizations of perfectionism (Kempke et al., 2016). Yet more importantly, a meta-analysis highlighted the influence of various moderating variables, including the time since stressor onset (Miller et al., 2007). As such, maladaptive perfectionism might initially contribute to a hyperactivity of the HPA axis. On the long run, however, maladaptive perfectionism might increase levels of chronic stress, which wears out the stress system resulting in increased risk for psychopathology (Juster et al., 2010). Consequently, chronic stress experienced by maladaptive perfectionists through their constant pursuit of performance, internal pressure and tendency to brood can be considered a direct health risk (Molnar et al., 2012).

Early theoretical considerations on the origin and development of perfectionism included the parent-child relationship and the associated psychosocial development of the child as primary influencing factors (Blatt et al., 1995; Sorotzkin, 1998). Overprotective behavior, as opposed to autonomy-promoting behavior, presents the child with a threatening image of the world. Thus, parental overprotection promotes avoidance behavior and limits the child's opportunity to develop own abilities and self-confidence (Chorpita & Barlow, 1998; Murray et al., 2009). Indeed, parental overprotection and especially limited autonomy-promoting behavior is associated with anxiety symptoms and disorders in children (van der Bruggen et al., 2008). Overall, an enforcing parenting style and maladaptive perfectionism are strongly related (Kawamura et al., 2002; Rice & Mirzadeh, 2000). Here, the social reaction model postulates that perfectionist attitudes develop as a result of a punitive and critical family environment (Flett et al., 2002). Correspondingly, firstborns or only children are at the highest risk of parental influence on perfectionist attitudes because they are most likely to experience strong parental criticism (Margot & Rinn, 2016; Siegle & Schuler, 2000). Yet, it is still unclear how parental overprotection leads to maladaptive perfectionism. One possible factor that could underlie this association is parental criticism.

The aim of this study was two-fold: On the one hand, we wanted to test the hypothesis that parental overprotection, parental criticism, and maladaptive perfectionism are related. On the other hand, we assumed that maladaptive perfectionism has an impact on markers of health and disease. Here, we were particularly interested in effects of maladaptive perfectionism on both acute and chronic stress reactivity. Together, we wanted to shed light on the origins of maladaptive perfectionism, and its health-related effects.

For this purpose, we recruited a student sample and assessed self-reported parental overprotection, and maladaptive perfectionism. To depict the effects of maladaptive perfectionism on acute stress reactivity, we implemented a within subject design, in which we exposed participants to a stressor (stress condition) on one day, as well as to relaxation videos (relaxation condition) on another day. Stress reactivity was assessed by repeatedly measuring salivary cortisol concentrations. Further, as a measure of chronic stress we assessed self-reported symptoms of vital exhaustion.

Hypothesis: We hypothesized that we would observe associations between maladaptive perfectionism on the one hand and reactivity to acute or chronic stress. We further predicted an association between parental overprotection and maladaptive perfectionism. We also assumed that parental criticism would partly mediate the association between parental overprotection and maladaptive perfectionism. Finally, we hypothesized that maladaptive perfectionism could explain an existing relationship between parental overprotection and altered stress reactivity.

Methods and Material

Participants

Participants were recruited via flyers and the online portal for psychological studies of the University of Konstanz. An online survey was employed to screen for the following exclusion criteria: (1) Age younger than 18, or older than 35 years, (2) BMI below 18.5 or higher than 30 (Wirtz et al., 2008), (3) elevated nicotine consumption (>8 cigarettes/day) (C. Kirschbaum et al., 1994), (4) drug use (Valdez et al., 2003), (5) sleeping disorders, (6) chronic diseases, (7) pregnancy (Obel et al., 2005), (8) clinical depression (measured by Beck

Depression Inventory II sum score > 18) (Heim et al., 2008).

A total of 69 participants took part in the study. Due to incomplete (n=1) or repeated (n=1) participation, 67 subjects (59.7% female, age mean= 21.60; age SD= 3.10) were included in the final analysis (see Appendix Table 1 for a list of the sample sizes included for the respective hypotheses, including essential sample characteristics). Subjects were compensated with either €30 or a certificate of participation (part of their curriculum). Written informed consent was obtained from all participants at the beginning of the experimental procedure. The study was approved by the Ethics committee of the University of Konstanz.

Procedures

Using a within-subject design, eligible participants were exposed to two experimental conditions, which took place on two different days (inter session interval of at least one week) and lasted for approximately 90min each. The order of the experimental conditions was randomized, and the two sessions were conducted by two different investigators. To control for circadian influences on cortisol levels (Dickerson & Kemeny, 2004), all experimental sessions were conducted between 15.30h and 18.30h. Participants were instructed not to consume a meal 2h prior to testing, and not to consume soft drinks, caffeinated beverages or nicotine 1h before testing. The course of the experimental session is shown in *Figure 1*.

During baseline and recovery periods, participants filled in questionnaires. The questionnaire phase was followed by a resting period. For this purpose, participants were instructed to sit in a relaxed position and keep their eyes closed for 6min. Subsequently, during the experimental manipulation (16min overall), either stress was induced using the Montreal Imaging Stress Task (MIST; details below) (Dedovic et al., 2005), or relaxation was induced using short relaxing videos (details below). Both tasks were performed on a laptop (11" Mac Book Air "Core i5" 1.4), while participants wore noise-cancelling headphones (Bose QuietComfort 25 Acoustic Noise Cancelling headphones). After another resting period of 6min, a recovery phase followed. At the end of the second session, participants were thanked, debriefed, and compensated for participation.

Stress condition

The experimental stress induction was performed using the Montreal Imaging Stress Task (MIST) (Dedovic et al., 2005). The MIST has shown high reliability in

inducing physiological stress responses by means of increases in cortisol levels, and decreases in heart rate variability (Dedovic et al., 2005; Marca et al., 2011; Pruessner et al., 2010).

The subjects performed time-limited arithmetic tasks on an Apple MacBook Air for 6min. An algorithm was used to adjust the ability, so that all subjects performed

the tasks with an accuracy of between 20 and 45%. After the first run, subjects were given feedback by the investigator that they had not performed as well as expected. Socio-evaluative threat was induced by asking critical questions (e.g., "Do you often have problems with cognitively demanding tasks?"). The subject was subsequently asked to try and improve his

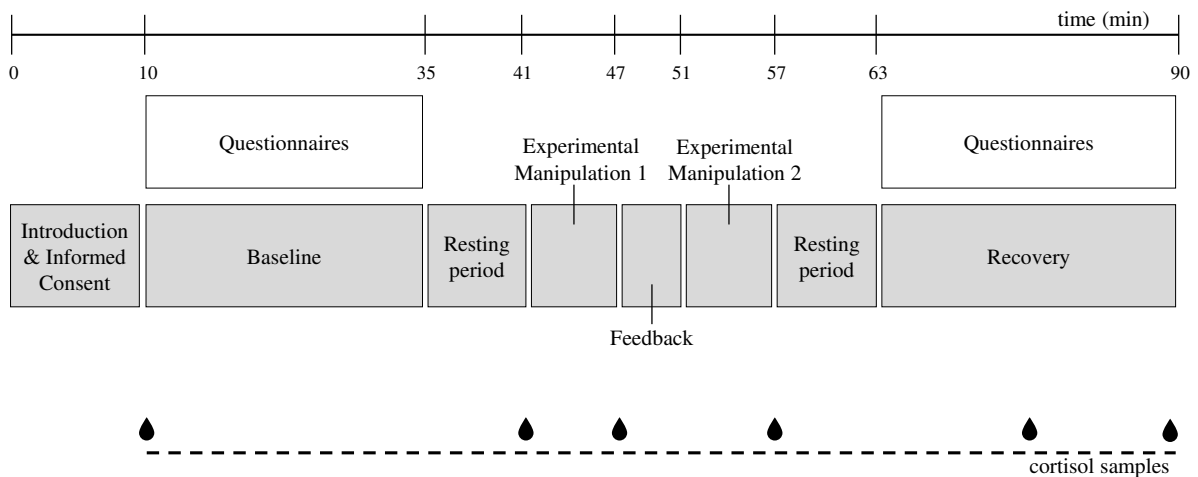


Figure 1. Schematic illustration of the experimental sessions. Each participant took part in two of such sessions, whereby the two blocks of experimental manipulation included the Montreal Imaging Stress Task in the stress condition and watching relaxation videos in the relaxation condition.

or her own performance in a repetition of the task. In the second run, the investigator positioned himself behind the subject and observed the performance, which further increased the socio-evaluative threat and thus aimed to generate psychosocial stress. As an additional element for stress induction, investigators wore a white lab coat during the stress condition.

Relaxation condition

In the relaxation condition, subjects were given four different 6min videos to choose from. The videos contained natural scenes with corresponding background noise: (a) a beach with the sound of waves breaking at the shore, (b) a rain forest with the sound of bird and leaves, (c) a river in a forest, or (4) a fireplace with burning and crackling wood. Previous research has shown that this kind of video induces a relaxation reaction, for example by lowering blood pressure and heart rate (Gladwell et al., 2012; Pilotti et al., 2015). After the first video, subjects were also given feedback to match the procedure in the stress condition, but here the feedback was neutral (e.g., "Which video did you choose to watch?"). The feedback break was followed by watching of the second video.

Physiological measures

To assess acute stress reactivity, saliva samples for the assessment of cortisol were collected at six time points throughout each session using Salivette devices (Sarstedt, Nümbrecht, Deutschland). Participants were instructed to move the synthetic swab from side to side in the mouth for 1min so that it could saturate with saliva, and then to return it to the tube without touching it with their hands. After collection, the samples were stored in a dark and cool place and after the end of testing were transferred to the biochemical laboratory of the Neuropsychology unit of the University of Konstanz, Germany. There, samples were stored at -20°C until biochemical analysis using a competitive enzyme immunosorbent assay (Cortisol Saliva ELISA, RE-52611, IBL International GmbH, Hamburg, Germany; lower detection limit: 0.030 µg/dL, inter-assay CV below 9.3% and intra-assay CV below 7.3% according to manufacturer).

Questionnaires

Parental Bonding Instrument (PBI). A subscale of the PBI was used to assess parental overprotection (Parker et al., 1979). The questionnaire includes 25

items for each parent that retrospectively assess the child's perception of parental attitudes and behavior during the first 16 years of life. The respondent answers on a 4-point Likert scale ranging from "very like" to "very unlike". The PBI comprises two dimensions: care (e.g., „...spoke to me in a warm and friendly voice", Cronbach's $\alpha=0.83$ for father and 0.80 for mother) and overprotection (e.g., „...tried to control everything I did", Cronbach's $\alpha=0.87$ for father and 0.84 for mother). In the present study, the focus was placed on the dimension of overprotection. For statistical analyses, we calculated a total score separately for mother and father (range: 0 (little overprotection) to 39 (strong overprotection)). Furthermore, we calculated a total parental overprotection score by averaging maternal and paternal overprotection and used this in the subsequent statistical analysis.

Frost Multidimensional Perfectionism Scale (FMPS). The German version of the FMPS (Frost et al., 1990) was used to measure perfectionism in six dimensions: concern over mistakes, doubts about actions, parental expectations, parental criticism, personal standards, and organization. The FMPS comprises 35 items that are rated on a 5-point Likert scale. Since we were particularly interested in maladaptive perfectionism in this study, we focused on the subscales concern over mistakes (CM), doubts about actions (D) (Stoeber, 2018, p.8), and calculated a total score for maladaptive perfectionism from those two subscales. In addition, parental criticism was included as a further subscale in the analysis.

Maastricht Vital Exhaustion Questionnaire (MVEQ). To assess levels of chronic stress, we used a German, short version of the MVEQ measuring vital signs of fatigue (Appels et al., 1987; Kopp et al., 1998; Schnorpfeil et al., 2002). The German short version of the questionnaire shows a high degree of agreement with the original version and has been used several times in German-speaking countries (Schnorpfeil et al., 2002). Subjects indicated their agreement with the nine items on a 3-point Likert scale (0=yes; 1=undecided; 2=no), and a sum score (range: 0 to 18) was calculated for statistical analyses. In this study, the scale was recoded so that high scores indicate high vital exhaustion, or chronic stress respectively.

Statistical analysis

Statistical analyses were conducted using IBM SPSS Statistics 24 (IBM Deutschland GmbH, Ehningen, Germany). Significance testing was performed on a two-sided basis and a significance level

of $\alpha=.05$ was set. Effect sizes are reported using partial eta square η^2 (Field, 2017). For interpretation, a $\eta^2=.01$ or greater was considered a small effect, a $\eta^2=.06$ or greater a medium effect, and a $\eta^2=.14$ or greater a large effect. Sex was included as a control variable due to its influence on the endocrinological stress response (Kudielka et al., 2012). The area under the curve with respect to increase (AUCi) was calculated to determine cortisol changes after stress induction (Pruessner et al., 2003). To index changes in cortisol concentration as a function of stress induction specifically, we only focused on the time after stressor start (samples 3-6). Cortisol samples 1 and 2 were classified as anticipation phase.

A manipulation check to verify mean differences in cortisol reactivity between the stress and the relaxation condition was performed using a two-factorial repeated-measures ANOVA with Greenhouse Geisser correction and the factors condition (2 levels: stress/relaxation) and time (4 levels: sampling 3-6). In addition, a t-test was performed to determine mean differences in cortisol AUCi's between conditions (stress/relaxation). The hypotheses were tested using Pearson product-moment correlations, t-tests, partial correlations, single factor ANOVAS, and mediation analyses. The mediation analyses were performed using PROCESS (Hayes, 2018). The linearity was verified by visual inspection of the matrix diagrams of the variables with LOESS smoothing. The ratio of the variables was approximately linear in all analyses. To calculate confidence intervals and inferential statistics, bootstrapping with 5000 iterations was used together with heteroskedasticity-consistent standard errors. If the confidence interval did not include zero, effects were considered to be statistically significant.

In an exploratory manner, a k-means cluster analysis was performed to identify different cortisol response patterns.

Results

The reliability of the psychological questionnaires in this sample ranged from excellent to good values for Cronbach's alpha (see Appendix Table 2 for information on internal consistency, range and average scale value of the questionnaires used).

To test the effectiveness of the experimental manipulation, we used a two-factorial repeated-measure ANOVA with Greenhouse Geisser correction including the cortisol samples 3-6. There was no significant interaction between time and experimental condition ($F(1.92, 119.21)=1.76$, $p=.15$, $\eta^2=.03$)

There was also no significant main effect of experimental condition ($F(1,62)=1.29$, $p=.26$, $\eta^2=.02$). A significant main effect was found for the factor time ($F(1.90, 117.87)=11.67$, $p<.05$, $\eta^2=.16$), indicating a significant change in cortisol level over

sampling 3-6 in both stress and experimental condition. Changes in cortisol levels over time for the two experimental conditions are depicted in Figure 2.

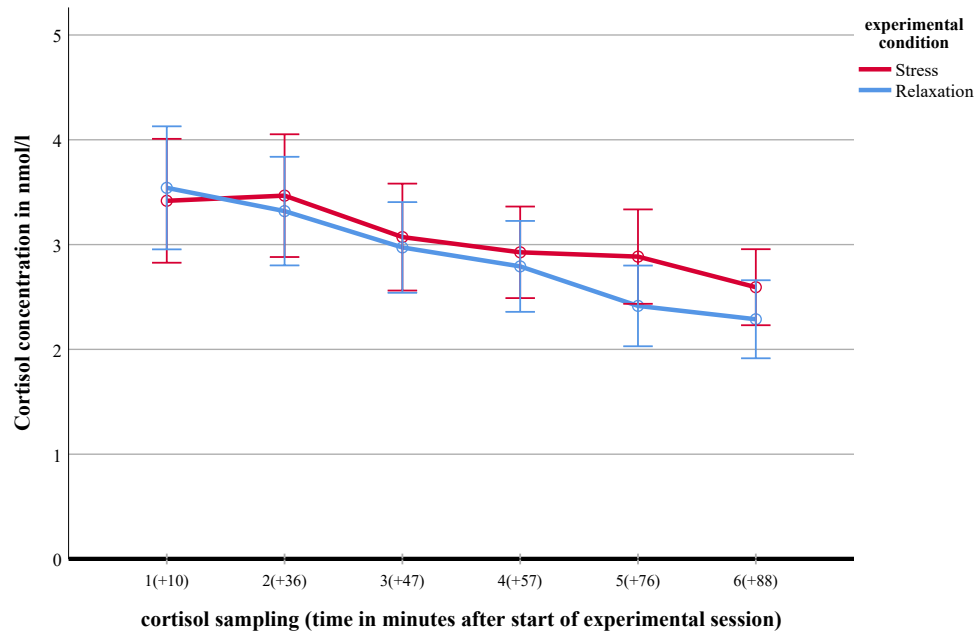


Figure 2. Mean cortisol concentration (nmol/l) for the stress and relaxation condition over time. A total of six cortisol values were collected for each condition, sampling 1 (+10min), sampling 2 (+36min), sampling 3 (+47min), sampling 4 (+57min), sampling 5 (+76min), and sampling 6 (+88min) after the start of the experimental session. The error lines depict the 95% confidence interval. The grey blocks mark the experimental manipulation: either time during the Montreal Imaging Stress Task in the stress condition, or time during watching relaxation videos in the relaxation condition.

Accordingly, using a t-test for dependent samples to compare cortisol AUCi's in the stress (mean=-.38, SD=2.47) and relaxation condition (mean=-.97, SD=2.36) showed no significant effect ($t(61)=1.34$, $p=.18$).

Overall, we did not find a significant increase in cortisol stress levels in response to the MIST in the stress condition, and results concerning acute stress reactivity should be interpreted with caution.

Regarding the relationship between maladaptive perfectionism and acute stress, a partial correlation with sex as a control variable showed no significant association between maladaptive perfectionism and cortisol AUCi's in the stress condition ($r=-.18$, $p=.17$). Looking at the relation between maladaptive perfectionism and chronic stress, we found a significant positive Pearson product-moment correlation between the total score for maladaptive perfectionism and the total score for VE ($r=.27$; $p<.05$). The correlation is illustrated in Figure 3.

A partial correlation with sex as a control variable showed a significant positive correlation between total parental overprotection and maladaptive perfectionism ($r=.31$, $p<.05$). A mediation analysis showed an effect of parental overprotection on maladaptive perfectionism ($B=.48$, $p<.05$). After inclusion of the mediator, parental overprotection significantly predicted the mediator (parental criticism) ($B=.27$, $p<.05$), which in turn significantly predicted maladaptive perfectionism ($B=.87$, $p<.05$). Thus, the relationship between parental overprotection and maladaptive perfectionism is completely mediated by parental criticism, indirect effect $ab=.27$, 95%-CI[.04, .47]. Figure 4 illustrates the mediation graphically.

An effect of parental overprotection on the AUCi's of the stress condition could not be found in the mediation analysis ($B=-.45$, $p=.15$). Thus, the direct effect c is not given and the condition for mediation is not fulfilled.

The Pearson product-moment correlation of parental overprotection and cortisol AUCi's in the stress condition also showed no significant correlation ($r=-.17$, $p=.18$).

In a mediation analysis, no effect of parental overprotection on chronic stress by means of VE was

found ($B=.25$, $p=.12$). Thus, the direct effect c is not given and the prerequisite for a mediation is not fulfilled. The Pearson product-moment correlation of parental overprotection and VE also showed no significant correlation ($r=.18$, $p=.15$).

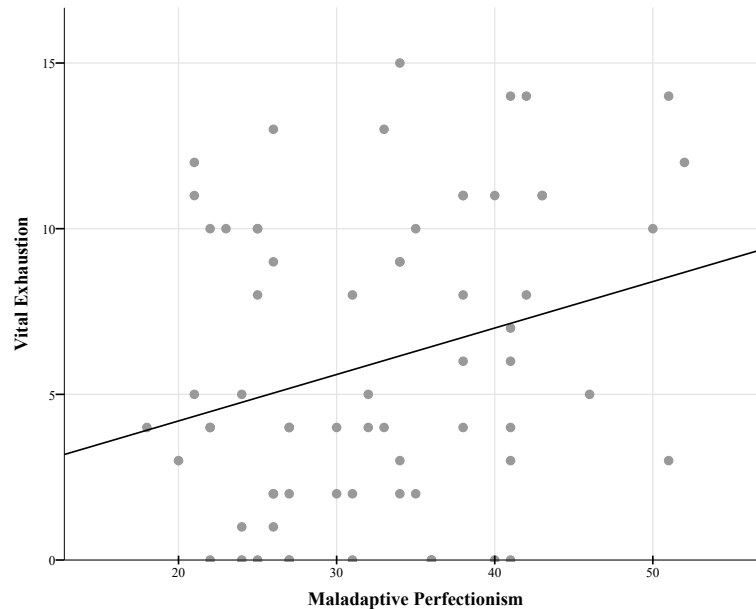


Figure 3. Scatterplot to illustrate the relationship between vital exhaustion and maladaptive perfectionism.

To further explore cortisol changes in response to our experimental stress manipulation, an explorative cluster analysis was performed to determine whether separate groups with different reaction patterns were present within the sample. For this purpose, the cortisol levels of sample 3-6 of the stress condition were grouped in a k-means cluster analysis. Starting with a cluster number of $k=4$, groups with different reaction patterns could be identified. With lower cluster numbers, no meaningful allocation became apparent. Since one subject significantly differed in its reaction pattern from the other groups and thus made up one of the four clusters, this subject was excluded from further analyses and finally three clusters ($n(C1)=32$; $n(C2)=9$; $n(C3)=22$) were identified and examined for possible group differences considering the hypotheses of this study.

The sample characteristics of the three clusters are listed in Appendix Table 3 and cortisol trajectories in the stress condition for the three clusters are graphically illustrated in Figure 5.

Furthermore, the three clusters were examined for potential mean differences in maladaptive

perfectionism. An ANOVA with cluster affiliation as a factor indicated no significant differences in MP between the clusters ($F(2,60)=1.46$, $p=.24$).

The clusters were also examined for possible mean differences in relation to parental overprotection using one-way ANOVAs. Depending on the cluster affiliation, significant mean differences were found with regard to both maternal ($F(2,60)=6.66$, $p<.05$, $\eta^2=.18$) and paternal overprotection ($F(2,57)=6.11$, $p<.05$, $\eta^2=.18$) between the participants.

With respect to maternal overprotection C3 (the cluster with a tendency to slightly decreasing cortisol concentration over time) showed the strongest expression. Paternal overprotection was most pronounced in C2, the cluster with an increase in cortisol concentration between samplings 1 and 2 followed by a strong decrease. C1, the cluster with stable low cortisol levels over time, showed the lowest overall parental overprotection. The results are graphically illustrated in Appendix Figure 1.

Discussion

The present study aimed to explore potential associations between maladaptive perfectionism and acute and chronic stress, considering the role of parental overprotection during childhood as a possible influencing factor. While no acute increase in cortisol

was observed in response to the manipulation, a significant correlation between maladaptive perfectionism and chronic stress as operationalized using vital exhaustion (VE) was found. Additionally, a mediation effect of parental criticism on the correlation between parental overprotection and maladaptive perfectionism could be demonstrated.

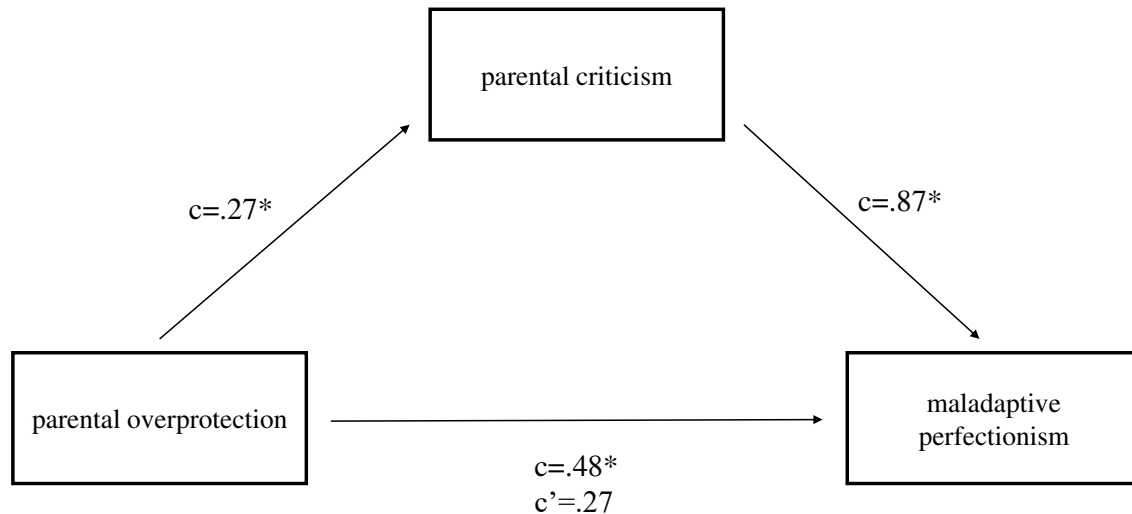


Figure 4. Graphical illustration of the mediation between parental overprotection and maladaptive perfectionism by parental criticism

We found no significant difference between the cortisol reactions in the stress and relaxation condition. This result indicates that the goal of inducing an acute stress response through the MIST was not achieved in this study. In a follow-up, explorative cluster analysis of the stress trajectories showed that three different cortisol response patterns could be observed. Based on visual inspection, the first group (C1) showed a relatively low cortisol concentration that was constant across all samples, the second group (C2) showed an increase between sampling 1 and sampling 2, which can be interpreted as an anticipatory reaction to the stress manipulation, but then a constant decrease between sampling 2 and sampling 6. The third group (C3) showed a slight decrease in cortisol concentration across all samples, but at a higher level than C1 overall. A possible cause for these differences is discussed with regard to research question 4.

The aim of the first research question was to investigate potential associations between maladaptive perfectionism and reactivity to acute and chronic stress. With regard to acute stress reactivity, no significant correlation between maladaptive perfectionism and

cortisol increases during the stress condition was found. Further, no mean differences between the groups identified in the cortisol cluster analysis regarding maladaptive perfectionism could be found. However, this finding must be interpreted with caution since the experimental stress induction in this study did not lead to a significant increase in salivary cortisol. Yet, previous studies also provided mixed results regarding this particular research question. For example, some studies show an association between maladaptive perfectionism and increased cortisol reactivity to acute stress (Wirtz et al., 2007), while other results associate lower cortisol reactivity with maladaptive perfectionism (Richardson et al., 2014). Previous research suggests the influence of various intervening variables on the change from hyper- to hypoactivity of the HPA axis induced by chronic stress, including time since stressor onset (Miller et al., 2007). Further empirical evidence is required to be able to make clear statements about the extent to which maladaptive perfectionism contributes to altered cortisol reactivity.

In terms of chronic stress reactivity, a significant positive correlation between maladaptive perfectionism and VE was shown. This finding is consistent with previous studies suggesting a relationship between maladaptive perfectionism and stress-related diseases and symptoms (Kempke et al., 2016; Stoeber & Rennert, 2008; Zhang et al., 2007). The diathesis-stress model of perfectionism provides a theoretical explanation (Hewitt & Flett, 2002), which

postulates that persons with maladaptive perfectionism are more vulnerable to perfectionist reactivity to stressors and thus more prone to maladaptive cognitive, emotional, motivational, and behavioral responses that promote negative stress symptoms such as VE. As such, our results are in line with findings suggesting that higher levels of maladaptive perfectionism are associated with higher chronic stress levels.

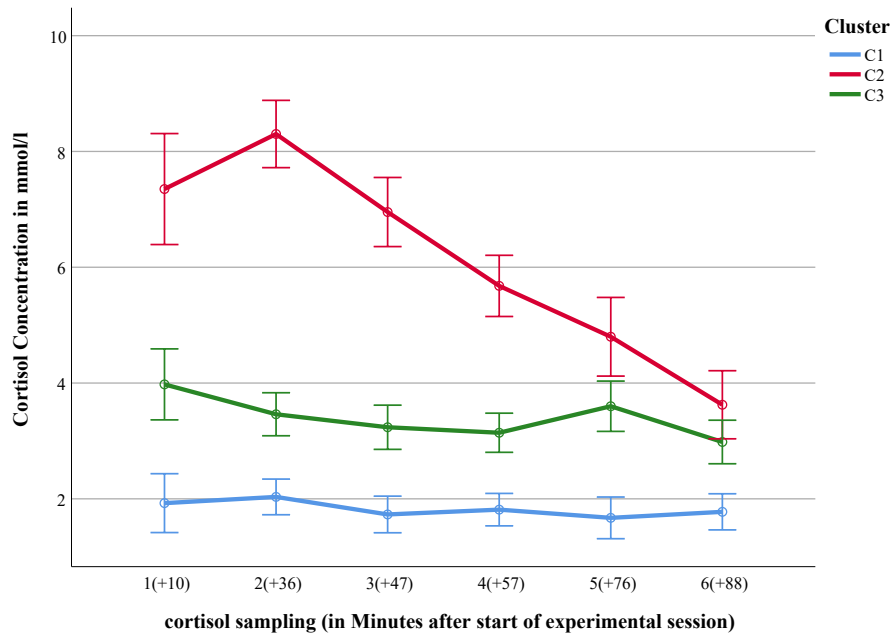


Figure 5. The mean cortisol concentration (nmol/l) of the three identified clusters during the stress condition. A total of six cortisol values were collected for each condition, sampling 1 (+10min), sampling 2 (+36min), sampling 3 (+47min), sampling 4 (+57min), sampling 5 (+76min), and sampling 6 (+88min) after the start of the experimental session. The error lines depict the 95% confidence interval. The grey blocks mark the time during the stressor (Montreal Imaging Stress Task).

The second research question was aimed at identifying possible links between parental overprotection and maladaptive perfectionism. Initially, a significant relationship was found between maternal and paternal overprotection. This result is consistent with previous PBI results, which overall showed a small variance between the ratings for mother and father (Xu et al., 2018). The overall score for parental overprotection calculated on the basis of this fulfilled condition showed a significant correlation with maladaptive perfectionism. This finding is consistent with previous results that show connections between a critical, demanding, and authoritarian parenting style and maladaptive perfectionism (Kawamura et al., 2002).

The third question focused on identifying possible mechanisms behind the connection between parental

overprotection and maladaptive perfectionism. The mediation analysis revealed a significant mediator effect for parental criticism on the association between parental overprotection and maladaptive perfectionism. The overall conclusion from this result is that overprotective parents are more likely to criticize their children's behavior, thereby promoting the presence of maladaptive perfectionist attitudes. Albeit the correlative study design does not allow conclusions to be drawn about causal relationships, but the temporal sequence of the factors may suggest a directional relationship, meaning that overprotective parental behavior during the first 16 years of life leads to increased maladaptive perfectionism in young adulthood. This finding supports the social reaction model (Flett et al., 2002), which postulates that perfectionism emerges as a reaction to a punishing and

critical family environment: Children in a critical parental environment develop perfectionist tendencies as a coping strategy to avoid negative feedback, shame, or other negative consequences of not achieving the expectations placed on them.

The fourth research question was aimed at testing maladaptive perfectionism as a possible mediator between parental overprotection and chronic and acute stress reactivity. In terms of acute stress reactivity, the mediation analysis also showed no direct effect of both maternal and paternal overprotection on the AUCi's of cortisol concentration in the stress condition. As already mentioned, the experimental stress induction was not successful and therefore the cortisol values of the test persons cannot be considered to reflect stress reactivity. However, analysis of the identified reactivity clusters showed that parental overprotection seems to be related to the reaction pattern of the cortisol response, as the clusters differed significantly with respect to parental overprotection. Depending on the sex of the parent, a slightly different distribution among the clusters was found, but C1 showed the lowest overall parental overprotection. A possible explanation for this observation is that students who experience higher parental overprotection feel more pressure in their studies and therefore perceive their studies as a stronger stressor. Since the subjects of this study were exclusively students who took part in the experiment in the temporal context of everyday university life, it is conceivable that higher parental overprotection is accompanied by a higher general stress level and thus a higher overall cortisol release which could be reflected in the different reaction patterns of the clusters. However, further research is needed to verify this explanatory approach or other conceivable mechanisms behind this observation. For example, studies with successful stress induction will be able to show the extent to which different subgroups differ in their cortisol response patterns and how these differences are related to parental overprotection more clearly.

Previous findings provide evidence that maladaptive perfectionism is both promoted by parental influences (Flett et al., 2002) and increases the risk of chronic stress symptoms such as VE (Dunkley et al., 2016; Hewitt & Flett, 2002). A theoretical consolidation of these findings suggests that parental overprotection is indirectly associated with chronic stress and that this association is mediated by maladaptive perfectionism. However, this assumption could not be confirmed in the present study. The mediation analysis showed no direct effect of parental

overprotection on VE. Thus, the requirement for maladaptive perfectionism as a mediator variable was not fulfilled. One possible explanation is that the study design does not sufficiently control for the actual current experience of stress. As such, current stress experienced by the subjects (e.g., due to a high workload in the study or critical life events) might have masked possible effects of parental overprotection on VE. Future study designs should take this factor into account by either including the current stress experience as a control variable, or by limiting the sample to clinically stressed patients (such as those with chronic fatigue syndrome) (Kempke et al., 2016).

There are some limitations to our study that should be considered when interpreting the results. Our sample consisted of healthy students aged 18 to 30 years, which implies a lowered external validity, as the effects found cannot be transferred to the entire population. A further limitation exists regarding the stress protocol used (Montreal Imaging Stress Task; Dedovic et al., 2005), which did not trigger a reliable stress response in this study. Since it has been used successfully before, one possible explanation is that some of the test participants may already have had prior knowledge of the methodology. Future studies should thus screen for prior experiences with the stressor. It is also possible that the investigators were not sufficiently convincing in their negative feedback, so that the social-evaluative threat was not credibly generated as a consequence. The reason why we chose the MIST was that it does have some advantages in comparison to other well-established stress protocols such as the Trier Social Stress Test for Groups (Kirschbaum et al., 1993): less manpower is needed to conduct the experiment. Further research should be carried out to better define the prerequisites to reliably induce stress using the MIST outside of the MRI scanner. Despite its limitations, the present study provides some important overall findings which provide informative implications for numerous fields of application as well as for future research. First, maladaptive perfectionism was associated with higher VE. This finding confirms the assumption that maladaptive perfectionism can be considered a direct health risk (Molnar et al., 2012). In an organizational context, and particularly in relation to occupational groups with an increased risk of stress-related diseases, this finding may provide evidence for prophylactic interventions. Previous findings suggest that the risk of burnout is significantly increased in occupational groups with high occupational ideals and a high degree of occupational social interaction (e.g., teachers, nurses, etc.) (Shirom, 2003). If the results of the present

study are taken into account, one explanation for this cumulation is that those jobs entail high performance standards while at the same time performance is difficult to measure and errors are often unavoidable, which means that according to the diathesis-stress model of perfectionism (Hewitt & Flett, 2002) maladaptive perfectionism can lead to chronic stress. However, the extent to which this explanatory approach applies should be empirically tested in further studies. Psychological interventions for the treatment and prevention of stress-related diseases should consider and treat perfectionism as a contributing factor. Evidence shows that therapeutic treatment of perfectionism is associated with a reduction in psychopathological symptoms and an improved response to therapeutic treatments, which reinforces the recommendation that clinicians should routinely question and treat perfectionism in the context of mental health. Cognitive interventions could also break maladaptive perfectionist patterns of thought resulting from critical education (Stoeber, 2018). In the future, the development and implementation of preventive approaches targeting (maladaptive) perfectionism should thus be the focus in the clinical and organizational context.

Appendix

Supplementary information is available at the end of this article (p. 14).

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Appendix

Appendix Table 1. Sample characteristics for the respective hypotheses.

Hypothesis	N	Demographic Data <i>M(SD)</i>		Psychological Questionnaires <i>M(SD)</i>				
		Age in years	BMI in kg/m ²	MP (FMPS-D)	VE (MVEQ)	Maternal Overprotection (PBI)	Paternal Overprotection (PBI)	Parental Criticism (FMPS-D)
1.1	67	21.60 (3.10)	21.82 (2.19)	32.54 (8.54)	6.00 (4.50)	10.67(6.72)	7.83(6.16)	7.55 (3.36)
1.2*	62	21.52 (2.91)	21.82 (2.22)	32.24 (8.57)	5.61 (4.37)	10.25(6.77)	7.80 (6.30)	7.42 (3.28)
2	67	21.60 (3.10)	21.82 (2.19)	32.54 (8.54)	6.00 (4.50)	10.67(6.72)	7.83(6.16)	7.55 (3.36)
3	67	21.60 (3.10)	21.82 (2.19)	32.54 (8.54)	6.00 (4.50)	10.67(6.72)	7.83(6.16)	7.55 (3.36)
4.1	67	21.60 (3.10)	21.82 (2.19)	32.54 (8.54)	6.00 (4.50)	10.67(6.72)	7.83(6.16)	7.55 (3.36)
4.2*	62	21.52 (2.91)	21.82 (2.22)	32.24 (8.57)	5.61 (4.37)	10.25(6.77)	7.80 (6.30)	7.42 (3.28)

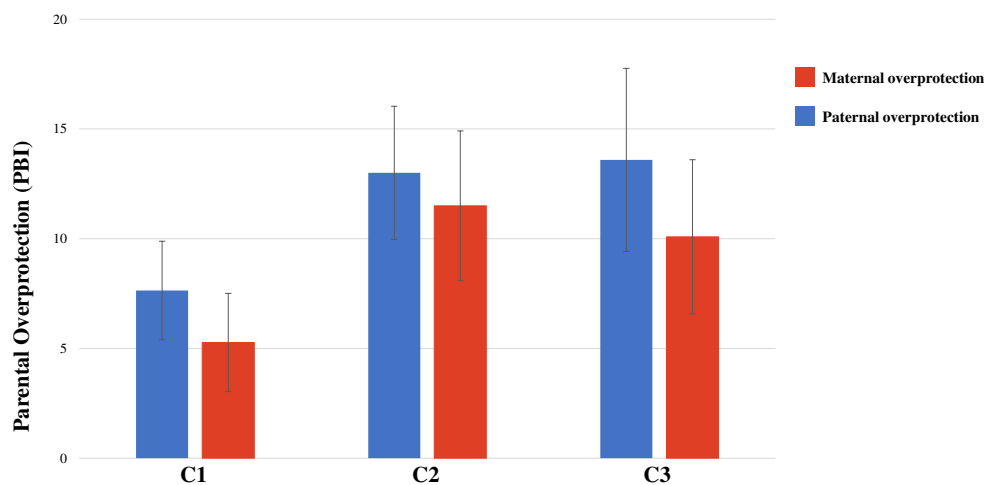
Note. M = mean, SD = standard deviation, BMI = Body Mass Index, MP = Maladaptive Perfectionism, FMPS-D = Frost Multidimensional Perfectionism Scale, VE = Vital Exhaustion, MVEQ = Maastricht Vital Exhaustion Questionnaire, PBI = Parental Bonding Instrument.

Appendix Table 2. Internal consistency, range and average scale value.

	N	Interne Konsistenz (a)	Spannweite	Durchschnittlicher Skalenwert (+/- SD)
FMPS-D Total	67	.89	68-138	98.16 (17.94)
FMPS-D (MP)	67	.85	18-52	32.54 (8.54)
FMPS-D (parental criticism)	67	.73	4-16	7.55 (3.36)
MVEQ Total	67	.76	0-15	6.00 (4.50)
PBI (maternal overprotection)	67	.86	2-26	10.67(6.72)
PBI (paternal overprotection)	64	.85	0-26	7.83(6.16)

Appendix Table 3. Sample characteristics of the identified clusters.

Cluster	N	Demographic Data		Psychological Questionnaires				
		M(SD)		M(SD)				
		Age in years	BMI in kg/m ²	MP (FMPS-D)	VE (MVEQ)	Maternal Overprotection (PBI)	Paternal Overprotection (PBI)	Parental Criticism (FMPS-D)
C1	32	21.53 (2.60)	21.76 (2.61)	31.19 (8.08)	4.72 (3.93)	7.64 (4.49)	5.27 (4.48)	6.69 (2.61)
C2	9	21.22 (3.03)	21.84 (2.25)	36.67 (7.45)	8.33 (3.87)	13.00 (6.06)	11.50 (6.82)	9.22 (4.09)
C3	22	21.50 (2.92)	21.83 (1.64)	32.05 (9.51)	5.82 (4.85)	13.60 (8.37)	10.09 (7.02)	8.09 (3.62)



Appendix Figure 1. Mean parental overprotection as measured using the maternal overprotection subscale of the Parental Bonding Instrument in the cortisol response clusters (C1, C2 and C3) extracted using an exploratory k-means cluster analysis.