Name: causal

<Files\\Achterberg & Meulen (2019)> - § 18 references coded [3,34% Coverage]

Reference 1 - 0,19% Coverage

In the current study we therefore provide an overview of MRI
scan quantity and quality in a large developmental twin sample (N=512, 256 twin pairs, aged 7–9), and investigated the genetic and environmental influences on MRI data quantity and quality.

Reference 2 - 0,09% Coverage

In the current study we provide direct estimates of heritability by conducting behavioral genetic analyses on a

Reference 3 - 0,02% Coverage

large childhood twin sample.

Reference 4 - 0,15% Coverage

In addition to trait-like, genetic influences on scan quality, we also investigated the influence of environmentally affected factors, such as emotional state towards the MR scan and MR protocol length.

Reference 5 - 0,13% Coverage

Moreover, we hypothesized that there would be little influence of genetics on scanner related distress, as it is highly influenced by the environment (i.e., the MRI simulation).

Reference 6 - 0,12% Coverage

Next, we evaluated MRI scan quantity by investigating how scan quantity was related to emotional state, and to what extend scan quantity was influenced by genetics.

Reference 7 - 0,12% Coverage

Similar to scan quantity, we investigated whether scan quality was
related to emotional state, and to what extend scan quality was influenced by genetics.

Reference 8 - 0,16% Coverage

By investigating both trait-like genetic influences as well as state-like environmental influences this study can provide insights in factors contributing to scan quantity and quality in developmental samples.

Reference 9 - 0,14% Coverage

Behavioral genetic modeling showed substantial to strong heritability estimates (45–46%) for both scan quantity (number of runs completed) and scan quality (percentage of scans included).

Reference 10 - 0,62% Coverage

These analyses revealed that head motion in fMRI runs was substantially influenced by genetics, with heritability estimates ranging from 29 to 65%, consistent with heritability estimates in adults (CouvyDuchesne et al., 2014). Thus, both the overall measure of scan quality (percentage of scans included), as well as the more sophisticated measure of scan quality in fMRI runs (framewise- and absolute displacement) showed substantial influence of genetics. Together, these findings show evidence for genetic contributions to head motion,

highlighting the need for careful control of motion related artifacts (Caballero-Gaudes and Reynolds, 2017; Power, 2017), specifically for studies in domains where genetic effects might play a strong role, such as in the case of psychiatric disorders that have a genetic basis (Hyman, 2000).

Reference 11 - 0,12% Coverage

Reassuringly, heritability estimates for subtle head motion (after
exclusion based on excessive head motion) were considerably smaller, ranging from 0 to 14%.

Reference 12 - 0,26% Coverage

Thus, in line with previous studies (Couvy-Duchesne et al., 2014; Engelhardt et al., 2017; Van Dijk et al., 2012), we report that excessive head motion is heritable and systematic, but additionally show that, after careful motion correction and exclusion based on excessive head motion, subtle head motion shows little influence of genetics.

Reference 13 - 0,23% Coverage

Indeed, behavioral genetic analyses on quality controlled head motion not only revealed small heritability estimates (0–14%, compared to 29–65% in overall head motion), but also showed that a similar, or even larger, proportion of the variance was explained by shared environmental influences (15–33%).

Reference 14 - 0,10% Coverage

Consistent with findings for quality controlled head movement, reports of emotional states showed little to no influence of genetics

Reference 15 - 0,09% Coverage

These findings suggest that emotional states can be significantly influenced by preparation of the scanner experiences.

Reference 16 - 0,17% Coverage

One aspect that did show influence on scan quality was the length of
the MRI scan session. Results showed that a protocol of>30 min resulted in less than 50% sufficient quality on all scans in this age range of 7-9-year-olds.

Reference 17 - 0,32% Coverage

Consistent with previous studies (Couvy-Duchesne et al., 2014; Engelhardt et al., 2017), the behavioral genetic analyses revealed heritability effects on head motion, with heritability estimates ranging from 29 to 65%. Importantly, however, our results also show that after exclusion based on excessive head motion, heritability estimates declined to 0–14%, indicating that MRI findings of motion corrected and quality-controlled

Reference 18 - 0,33% Coverage

data are not substantially confounded by genetic factors. Moreover, shared environmental influences played a larger role (15–33%) in the variation in quality controlled head motion, suggesting that head motion can be influenced by participant instruction and scanner adjustments. These results provide insight in the genetic and environmental influences on scan quantity and quality and can inform future studies on developmental neuroimaging.

<Files\\Achterberg et al. (2017)> - § 14 references coded [2,35% Coverage]

Reference 1 - 0,17% Coverage

Furthermore, we tested whether peer rejection in children results in behavioral aggression, in a similar way as was previously observed in adults (Chester et al., 2014; Riva et al., 2015; Achterberg et al., 2016).

Reference 2 - 0,12% Coverage

Thus, our aim was to investigate 7–10-year-old children's
responses to social evaluation in terms of neural activity and reactive behavioral aggression.

Reference 3 - 0,21% Coverage

On the behavioral level we expected that the pattern of aggres-
sion after positive, neutral, and negative feedback would be similar across the pilot, test and replication samples, with negative feedback resulting in the highest levels of aggressive behavior.

Reference 4 - 0,17% Coverage

Overall, we found consistent findings over the pilot, test and replication samples for behavioral aggression following negative social feedback, showing significantly more aggression after negative social feedback

Reference 5 - 0,16% Coverage

The neural effects indicated increased activity in the amygdala, insula and mPFC/ACCg after negative feedback, but these effects were only significant in part of the samples and in the meta-analyses.

Reference 6 - 0,11% Coverage

The SNAT showed reliable and consistent behavioral results,
with stronger behavioral aggression (noise blast duration) after social rejection.

Reference 7 - 0,14% Coverage

This study complements the large number of prior studies that focused mainly on withdrawal, as we showed that social rejection feedback also elicits aggression in children.

Reference 8 - 0,15% Coverage

In the pilot sample, we found significant heightened activation in the insula after negative vs. neutral social feedback, similar to the effects reported in adults (Achterberg et al., 2016).

Reference 9 - 0,20% Coverage

Moreover, although heightened activation in the visual cortex (including the fusiform gyrus) after positive compared to negative and neutral feedback was consistent over the pilot and test sample, we could not confirm this in the replication sample

Reference 10 - 0,19% Coverage

The region described as the ACC gyrus (ACCg; located adjacent and dorsal to the genu of the corpus callosum in humans) shows overlap with the region that showed increased activation after negative social feedback in children (this study)

Reference 11 - 0,10% Coverage

Moreover, the meta-analysis showed that the anterior insula was more active after negative compared to neutral feedback,

Reference 12 - 0,11% Coverage

In addition, the meta-analysis showed significantly more activa-
tion in the amygdala after negative feedback compared to positive feedback.

Reference 13 - 0,15% Coverage

Thus, although we found that behaviorally children reacted in a similar way to social evaluation as adults do, the similarities in neural findings between children and adults are more mixed.

Reference 14 - 0,38% Coverage

Negative social feedback resulted in the highest levels of behavioral aggression. Moreover, meta-analyses on predefined ROIs revealed that negative social feedback resulted in more neural activation in the amygdala (compared to positive feedback) and in the anterior insula and mPFC/ACCg (compared to neutral feedback). Exploratory whole brain analyses confirmed heightened activation in the medial prefrontal cortex (mPFC) after negative relative to neutral social feedback

<Files\\Achterberg et al. (2018)> - § 19 references coded [2,73% Coverage]

Reference 1 - 0,09% Coverage

To date, the size of
environmental and genetic contributions to limbic/subcortical-PFC connectivity has not been examined in children.

Reference 2 - 0,05% Coverage

and the heritability of these connections in 7-to-9-year-old twins (N ¼ 220).

Reference 3 - 0,13% Coverage

and additionally explore genetic and environmental influences on that connectivity, thereby providing important insights in the underlying mechanisms of functional brain connectivity in childhood.

Reference 4 - 0,05% Coverage

and (2) the heritability of these connections in 7-to-9-year-old twins (N¼ 220).

Reference 5 - 0,05% Coverage

and 2) to examine the heritability of these connections comparing MZ and DZ twins.

Reference 6 - 0,37% Coverage

To address the second question, concerning the heritability of limbic
connectivity, we compared MZ and DZ twin pairs using ACE modeling. This decomposition model provides an estimate of the proportions of the variance in the data that are attributed to heritable, shared environmental, and unshared/unique environmental factors. Previous studies have shown both influences of genetics (Richmond et al., 2016) and environmental contributions (Tottenham and Galvan, 2016), indicating that there could be an interplay between genetics and environment (Yang et al., 2016).

Reference 7 - 0,09% Coverage

the current findings fit with the hypothesized top-down control of dorsal lateral prefrontal cortex over the limbic subcortical brain regions

Reference 8 - 0,12% Coverage

Variance in the majority of connections from the ventral striatum to the prefrontal cortex was best described by genetics, with moderately strong heritability factors (up to 67%).

Reference 9 - 0,09% Coverage

Interestingly, connectivity from the ventral striatum to the vACC and thalamus was mostly influenced by shared and unique environmental factors,

Reference 10 - 0,06% Coverage

while short range thalamic and vACC connectivity is more influenced by environmental factors.

Reference 11 - 0,24% Coverage

With the exception of ventral striatum-thalamic connectivity, limbic/
subcortical-subcortical connectivity was notably influenced by genetics, with heritability estimates ranging from 32 to 42%. For instance, we found heritability for amygdala-hippocampus connectivity (A ¼ 32%), indicating that this emotional memory network (Phelps, 2004)is influenced by genetic factors.

Reference 12 - 0,26% Coverage

Moreover, while ventral striatum-prefrontal cortex connective showed large genetic influences, amygdala-prefrontal cortex connectivity showed mostly effects of the environment, with high estimates of the E component (up to 92%). There were two exceptions to this general pattern. First, in line with the ventral striatum, amygdala-vACC connectivity showed influences of the shared environment.

Reference 13 - 0,06% Coverage

Secondly, 54% of the variance in amygdala-OFC connectivity was explained by genetic influences.

Reference 14 - 0,12% Coverage

Our study is the first to show that variance in amygdala-OFC functional connectivity in childhood is explained by genetic factors. This finding has important implications for intervention

Reference 15 - 0,06% Coverage

research: Certain genetic profiles might be more susceptible to environmental influences than others,

Reference 16 - 0,18% Coverage

Overall, the patterns of genetic and environmental influences for
ventral striatum and amygdala were distinct: Long-range PFC connectivity with the ventral striatum was genetically influenced, whereas longrange amygdala connectivity was mostly environmentally influenced.

Reference 17 - 0,12% Coverage

Fourth, some of our genetic analyses of neural responses resulted in
high estimates for the E component (up to 92%), reflecting influences from the unique environment and measurement error.

Reference 18 - 0,39% Coverage

Taken together, this study was the first to investigate twin effects in
subcortical-subcortical and subcortical-cortical RS-fMRI in children, providing important insights in genetic and environmental influences on childhood brain connectivity. The behavioral genetic analyses showed moderate

to substantial heritability of striatum-prefrontal cortex brain connectivity, and environmental influences on amygdala-orbitofrontal cortex connectivity, with implications for our understanding of the etiology of disorders that are associated with disrupted connectivity, such as drug abuse and depression.

Reference 19 - 0,19% Coverage

The current findings provide the first step in laying the groundwork for understanding genetic and environmental influences in shaping brain connectivity and may be the starting point for a better understanding of how brain development is both biologically based and environmentally driven.

<Files\\Achterberg, Duijvenvoorde, Bakermans-Kranenburg, & Crone (2016)> - § 11 references coded [2,82% Coverage]

Reference 1 - 0,16% Coverage

The first goal of this study was to disentangle effects of positive and negative feedback in a social evaluation paradigm (Somerville et al., 2006).

Reference 2 - 0,27% Coverage

Based on prior research, we expected that positive social feedback would result in increased activation in the subgenual ACC (Somerville et al., 2006) and the ventral striatum (Guyer et al., 2009; Davey et al., 2010; Gunther Moor et al., 2010).

Reference 3 - 0,12% Coverage

The second goal of this study was therefore to examine how individuals respond to negative social feedback,

Reference 4 - 0,21% Coverage

On a behavioral level, we hypothesized that negative social feedback would trigger reactive aggression, i.e. longer noise blasts (Twenge et al., 2001; Reijntjes et al., 2011; Riva et al., 2015).

Reference 5 - 0,18% Coverage

At the neural level, conjunction analyses showed that both negative and positive social feedback resulted in increased activity in the mPFC and the bilateral insula.

Reference 6 - 0,39% Coverage

Comparing the conjunction analyses with the separate contrasts of negative and positive vs neutral feedback showed that positive feedback resulted in increased activity in the striatum and the ventral mPFC, whereas negative feedback activation merely overlapped with dorsal mPFC and insula activation observed following both positive and negative feedback

Reference 7 - 0,24% Coverage

In this study we found that, consistent with prior studies (Guyer et al., 2009; Davey et al., 2010; Gunther Moor et al., 2010), there was increased activity in the ventral mPFC and the striatum after positive feedback.

Reference 8 - 0,38% Coverage

The current findings, which show enhanced insula and mPFC activity following both positive and negative feedback (relative to neutral feedback), suggest that the insula and mPFC in social evaluation paradigms might work as a salience network, and signal events that are socially relevant (Guroglu et al., 2010[TQ1]; Van den Bos et al., 2011[TQ1]).

Reference 9 - 0,43% Coverage

Our study shows that receiving negative social feedback is also followed by more aggressive behavior (i.e. by a longer noise blast toward the peer). In addition, we show that more activity in the right dlPFC is related to 'less' aggression after negative social feedback (compared with neutral feedback), indicating that the lateral PFC is an important neural regulator of social aggression.

Reference 10 - 0,23% Coverage

This study is the first study to investigate aggressive responses after positive, neutral and negative feedback, and shows a role of the dlPFC in individual differences in the regulation of aggressive behavior.

Reference 11 - 0,20% Coverage

In conclusion, we found evidence that the insula and mPFC
generally respond to socially salient feedback, with no significant differentiation between negative and positive feedback.

<Files\\Achterberg, Duijvenvoorde, Meulen, Bakermans-Kranenburg, & Crone (2018)> - § 14 references coded [2,04% Coverage]

Reference 1 - 0,24% Coverage

At the same time, there is a gap in our understanding of the genetic and environmental influences of brain responses to social feedback and regulatory responses. In this study, we therefore investigated the neural underpinnings and heritability of social feedback processing and subsequent aggression in middle childhood.

Reference 2 - 0,10% Coverage

We aimed to explore whether neural reactions to social feedback that could elicit aggression show similar heritability estimates.

Reference 3 - 0,25% Coverage

In this study, we therefore used a large developmental twin sample (N5509 7- to 9-year-olds), to investigate (a) the heritability of behavioral aggression following social evaluation; (b) the neural underpinnings of social evaluation and their relation to behavioral aggression; and (c) the heritability of these neural underpinnings.

Reference 4 - 0,11% Coverage

We hypothesized that negative social feedback would result in
behavioral aggression (Achterberg et al., 2016, 2017; Chester et al., 2014).

Reference 5 - 0,20% Coverage

Consistent with prior studies, negative social feedback resulted in behavioral aggression (Achterberg et al., 2016, 2017). Behavioral genetic modeling revealed that aggression following negative feedback (negative–positive and negative–neutral) was influenced

Reference 6 - 0,14% Coverage

by genetic as well as shared and unique environmental influences. Genetic influences ranged from 10% to 20%, whereas $7% of the variance was explained by shared environmental influences.

Reference 7 - 0,17% Coverage

It shows that environmental factors are important predictors of reactive aggressive behaviors. In line with our results, longitudinal stability in reactive aggression has been shown to be influenced by environmental effects

Reference 8 - 0,13% Coverage

Our results show that activation of regions coding social saliency is present already in childhood, indicating this might be a core social motivational mechanism in humans.

Reference 9 - 0,17% Coverage

Positive feedback, on the other hand, resulted in heightened activation in the caudate, SMA and bilateral DLPFC, which is consistent with previous social evaluation paradigms that reported increased activation in striatum

Reference 10 - 0,12% Coverage

Genetic modeling showed that genetics played a role in activation
in the DLPFC, the SMA, and the right caudate, with 10%–14% of the variance explained by genetics.

Reference 11 - 0,09% Coverage

Taken together, our results suggest that the processing of social
feedback is partly explained by genetic factors,

Reference 12 - 0,10% Coverage

and the level of behavioral aggression following these evaluations are related to genetics and shared environmental influences.

Reference 13 - 0,08% Coverage

Our findings underscore that the way children react to positive and negative social feedback is influenced by

Reference 14 - 0,15% Coverage

environmental factors. This stresses the important role of environmental inputs on observed behavior, such as parents and teachers, and points to an important role for parenting programs and interventions.

<Files\\Becht et al. (2017)> - § 13 references coded [3,11% Coverage]

Reference 1 - 0,50% Coverage

However, little is known about how different aspects of identity uncertainty (reconsidering current identity commitments and fluctuations in the strength of commitments) and identity commitment formation are affecting each other at the within-person level from early to late adolescence. Following the identity status paradigm to study identity formation, the aim of the current study was to test theoretical assumptions concerning longitudinal linkages between certainty and uncertainty dynamics of identity formation at the withinperson level in two salient identity domains (i.e., interpersonal and educational identity) across adolescence.

Reference 2 - 0,46% Coverage

Although tentative predictions concerning the developmental order in certainty and uncertainty can be based on research on identity status transitions, it remains unclear how the underlying dynamics between certainty and uncertainty dimensions of the identity formation process affect each other over time within the same adolescents. Hence, our main aim was to test reciprocal within-person longitudinal linkages between

adolescents' reconsideration of identity commitments, fluctuations in commitment and identity commitment levels within the interpersonal and educational identity domains.

Reference 3 - 0,15% Coverage

Furthermore, we investigated in both identity domains whether certainty and uncertainty aspects of identity formation affect each other equally in strength from early to late adolescence.

Reference 4 - 0,31% Coverage

First, we found support for identity's theory's (Erikson, 1968) key assumption that adolescents increasingly reconsider current identity commitments and consider alternatives before they make strong commitments within the interpersonal identity domain. Second, within the educational identity domain we found that the certainty and uncertainty dynamics in the process of identity formation operated

Reference 5 - 0,28% Coverage

differently, in such a way that adolescents' increasing commitment level and commitment fluctuations predicted decreasing uncertainty of their identity commitments over time. Thus, our findings support elements of identity theory but revealed that how certainty and uncertainty aspects of identity formation affect each other differs across identity domains

Reference 6 - 0,17% Coverage

These results provide the first empirical evidence that adoles-
cents' uncertainty in identity precedes commitment making at the within-person level from early to late adolescence for interpersonal identity (Erikson, 1968).

Reference 7 - 0,27% Coverage

Whereas prior longitudinal research has already found that college students' increasing consideration of identity preceded new commitment making at the between-person level (Luyckx, Goossens, & Soenens, 2006), our findings show that this dynamic in identity formation processes also applies to the adolescent period at the within-person level.

Reference 8 - 0,11% Coverage

In our study, we also show that the dynamic between certainty and uncertainty aspects of identity formation was different across identity domains.

Reference 9 - 0,11% Coverage

However, when adolescents increased in the strengths of their commitments to school, they reconsidered these identity commitments less over time.

Reference 10 - 0,19% Coverage

Our findings also confirm earlier suggestions that fluctuations in certainty about adolescents' current identity commitments reflect on ongoing evaluative process (Luyckx, Goossens, & Soenens, 2006), which strengthens future identity commitments.

Reference 11 - 0,10% Coverage

First, for interpersonal identity more identity fluctuations were related to less reconsideration of commitments, concurrently.

Reference 12 - 0,31% Coverage

Second, within the educational identity domain increasing commitment fluctuations predicted less reconsideration of commitments over time. Our results suggest that when adolescents fluctuate in the strengths of their commitments, this is not necessarily bad but instead might reflect a process of identity consolidation, evidenced by decreasing uncertainty about their commitments across adolescence.

Reference 13 - 0,15% Coverage

We investigated these longitudinal associations on a within-person level. When adolescents have increasingly reconsidered and explore identity alternatives, strong commitments are likely to follow.

<Files\\Becht et al. (2018)> - § 1 reference coded [0,12% Coverage]

Reference 1 - 0,12% Coverage

The purpose of the present research was to
investigate self-reported and neural processes underlying adolescents' identity in two separate studies.

<Files\\Becht2015> - § 2 references coded [0,30% Coverage]

Reference 1 - 0,16% Coverage

In addition, we found some support for the goodness-of-fit
model (Thomas & Chess, 1977) by showing that the effect of overreactivity on aggression and rule breaking was dependent in some cases upon the fit between the parenting environment and the child's personality traits.

Reference 2 - 0,13% Coverage

Specifically, we found that parental overreactivity decreased the likelihood of following the high increasing aggression trajectory compared to the high decreasing aggression trajectory, but only for highly expressive children.

<Files\\Bos et al. (2017)> - § 10 references coded [2,02% Coverage]

Reference 1 - 0,17% Coverage

We developed a novel neuroimaging paradigm to test
approach behavior and cognitive control to cues that reflected participants' preferences compared to stimuli that participants identified as non-interests

Reference 2 - 0,13% Coverage

We predicted that participants would have faster reaction times to their interests and would be more impulsive to these cues compared to their non-interests.

Reference 3 - 0,15% Coverage

We also predicted that successful inhibition to these cues would engage increased activity in cognitive control regions such as the inferior frontal gyrus (IFG) (Aron et al.,2014).

Reference 4 - 0,13% Coverage

Consistent with our hypotheses, there was a behavioral
trend that adults were more accurate to respond to their preferred interest versus non-interest.

Reference 5 - 0,66% Coverage

There were no differences between interests versus non-interests in mean or mu RT, however, participants were slower to respond to both interests and non-interests relative to colors, suggesting that the colors were easier stimuli to process compared to the visually more complex images of interests and non-interests. There was no difference in accuracy to interests versus colors, which also highlights a potential attentional bias towards these visually complex, yet preferred stimuli. There was increased activation to interests as compared to
colors in left anterior insula which has consistently been associated with the processing of positive versus negative emotional experiences (Kurth et al., 2010; Duerden et al., 2013) and differentiating emotions (Gorno-Tempini et al., 2001).

Reference 6 - 0,13% Coverage

Thus, the heightened activation observed for interests supports a role for the left anterior insula in an attentional bias to positive, motivating cues.

Reference 7 - 0,13% Coverage

However, similar to what was observed in the social condition, the non-social stimuli induced greater impulsivity relative to the control condition of colors.

Reference 8 - 0,37% Coverage

The facial expressions elicited behavioral and neural activa-
tion patterns that were consistent with an extensive literature using variations of this task (Hare and Casey, 2005; Shafritz et al., 2006; Schulz et al., 2009). Participants were slower to respond to calm faces relative to happy faces, as reported previously (Hare and Casey, 2005; Schulz et al., 2007; Somerville et al., 2011), suggesting adults demonstrated a greater bias towards

Reference 9 - 0,01% Coverage

happy faces.

Reference 10 - 0,14% Coverage

Although no differences were observed in response inhibition, we observed a subtle attentional bias and increased activity in the anterior insula to one's interests.

<Files\\Brouwer et al. (2015)> - § 6 references coded [0,97% Coverage]

Reference 1 - 0,30% Coverage

The twin sample allows (Boomsma et al. 2002) assess-
ing whether associations among measures of grey matter density in the brain and hormone levels can be attributed to variations in the genome (genetic pleiotropy) or can be attributed to environmental factors that influence both phenotypes.

Reference 2 - 0,21% Coverage

In this study we measured the influence of pubertal hormones on brain structure between 9 and 12 years of age and the extent to which these associations might be due to genetic and environmental influences.

Reference 3 - 0,07% Coverage

These associations were driven by unique environmental factors.

Reference 4 - 0,11% Coverage

The associations between estradiol and grey matter densities were driven by common environmental influences.

Reference 5 - 0,12% Coverage

Our results now suggest that maybe FSH is responsible for hippocampal growth in girls in the early phases of puberty.

Reference 6 - 0,16% Coverage

The association between grey matter density and estra-
diol was mainly explained by common environmental factors shared by twins raised in the same family.

<Files\\Cao et al. (2018)> - § 3 references coded [0,42% Coverage]

Reference 1 - 0,09% Coverage

We particularly focused on the interactive effects between the DRD2 gene and maternal parenting on the longitudinal development ofdepressive symptoms.

Reference 2 - 0,13% Coverage

Second, we assessed the extent to which the DRD2 gene polymorphisms (i.e., the TaqIA andA241G polymorphisms) and maternal positive parenting additively and interactively explained trajectory variance ofdepressive symptoms.

Reference 3 - 0,20% Coverage

This longitudinal study is the first to demonstrate that the
A241G polymorphism, but not the TaqIA polymorphism, interacts with maternal positive parentinginpredictingtrajectory membership ofdepressive symptoms from early to mid-adolescence. This is promising, given the potential modifiable nature ofmaternal parenting.

<Files\\Collin, Kahn, De Reus, Cahn, & Van den Heuvel (2014)> - § 5 references coded [0,84% Coverage]

Reference 1 - 0,28% Coverage

Extending previous find-
ings, this study now reveals intermediate levels of rich club connectivity in unaffected siblings of schizophrenia patients, suggesting that impaired rich club connectivity in patients is likely to have a familiar, possibly genetic, component.

Reference 2 - 0,24% Coverage

The currently observed
abnormalities in rich club organization in these individuals seem to suggest that rich club dysconnectivity includes a neurodevelopmental vulnerability for the illness, which may be mediated by genetic factors.

Reference 3 - 0,18% Coverage

These studies and our current findings converge on the notion that connectome abnormalities in schizophrenia reflect, at least in part, an inherited susceptibility to the disease.9

Reference 4 - 0,03% Coverage

possibly reflecting genetic,

Reference 5 - 0,11% Coverage

Our findings emphasize a central role for abnormal rich club organization in the etiology of schizophrenia.

<Files\\Collin, Scholtens, Kahn, HIllegers, & Heuvel (2017)> - § 5 references coded [1,17% Coverage]

Reference 1 - 0,18% Coverage

thus provides an opportunity to assess the impact of genetic vulnerability for psychotic illness on brain development well before the age at which psychosis typically manifests.

Reference 2 - 0,32% Coverage

In all, previous studies and our current findings suggest that genetic predisposition for SZ versus BD may have a differential effect on the developmental formation of the connectome's central RC system. Specifically, RC deficits appear to be a unique connectome disturbance in those affected by, or at elevated risk for, SZ.

Reference 3 - 0,26% Coverage

RC connectivity was found to show a negative correlation
with functional connectome modularity in SZ offspring, suggesting that deficits in anatomical connectivity among central brain hubs may reduce the brain's capacity for intermodular functional integration.

Reference 4 - 0,20% Coverage

Our current study suggests that abnormal developmental formation of the central RC system in youths at risk for SZ may form the anatomical substrate for disturbed functional integration, which may in turn

Reference 5 - 0,21% Coverage

id not find evidence for RC deficits in offspring of BD patients, in line with previous findings in adult patients, suggesting a differential influence of genetic predisposition for SZ versus BD on connectome development.

<Files\\Cosijn, Benthem, Schee, & Spijkerman (2015)> - § 5 references coded [0,73% Coverage]

Reference 1 - 0,19% Coverage

Moreover, we hypothesized that individual differences in cognitive control would moderate the relationship between motivational processes (attentional bias, approach bias, craving) and amount of cannabis use and severity of cannabis-related problems.

Reference 2 - 0,15% Coverage

In contrast to our hypothesis, cognitive control did not significantly moderate the relationship between motivational processes and amount of cannabis use and severity of cannabis-related problems,

Reference 3 - 0,17% Coverage

The current findings suggest that craving plays an equally important role in adolescent CUDs compared to adult CUDs and other SUDs, implicating a crucial role for the aforementioned brain areas in the course of adolescent CUDs.

Reference 4 - 0,15% Coverage

A clear limitation of the current study is the lack of a control group. However, the cannabis bias was significantly higher than the alcohol bias, suggesting a cannabis specific attentional bias.

Reference 5 - 0,08% Coverage

These findings support an important role for craving in the course of adolescent cannabis use and dependence.

<Files\\Cousijn, Koolschijn, Zanolie, Kleibeuker, & Crone (2014)> - § 6 references coded [0,99% Coverage]

Reference 1 - 0,19% Coverage

Given the strong relationship between age and gray matter morphology, it was also investigated if age group (adolescents vs. adults) moderated the relationship between divergent thinking (on a behavioral and neural level) and gray matter morphology.

Reference 2 - 0,22% Coverage

We predicted that cortical thickness in the main brain areas activated during the AUT would be positively related to AUT behavioral scores and AUT brain activity, irrespectively of age under the assumption that neural structure is an underlying factor that aids in task performance [31].

Reference 3 - 0,16% Coverage

Moreover, age group moderated the relationship between AUT flexibility and cortical thickness of the left SMG, showing a negative relation between cortical thickness and flexibility in adolescents only.

Reference 4 - 0,14% Coverage

The current results indicate an important role of a widespread brain network involved in flexible visual processing of spatial relationships [64]in visuo-spatial divergent thinking.

Reference 5 - 0,14% Coverage

A tentative but speculative explanation may be that a more mature SMG (as reflected by cortical thinning during mid adolescence; [16, 65]) is beneficial to flexible thinking.

Reference 6 - 0,13% Coverage

The results support an important role of a widespread brain network involved in flexible visual processing of spatial relationships in visuospatial divergent thinking.

<Files\\Cousijn, Luijten, & Wiers (2014)> - § 6 references coded [1,19% Coverage]

Reference 1 - 0,18% Coverage

Both heavyandoccasional drinkers completed the task allowing us to investigate whether the presence of emotional primes influenced the expression of an alcohol-approach bias in heavy drinkers.

Reference 2 - 0,20% Coverage

To the best of our knowledge, previous studies have not inves-
tigated the relationship between emotional primes and alcoholapproach action tendencies. Besides investigating the effects of different emotional primes,

Reference 3 - 0,12% Coverage

role that coping and enhancement drinking motives have on the relationship between alcohol-approach bias and emotional primes.

Reference 4 - 0,41% Coverage

The findings showed that the primed emotional context influenced the approach bias in both occasional and heavy drinkers. More specifically, the alcohol-approach bias increased in the negative context relative to the appetitive (non-alcoholic) control context. This context effect was driven by slower avoidance (not faster approach) suggesting that the avoidance of alcohol-related cues may generally be more difficult in a negative emotional context.

Reference 5 - 0,11% Coverage

Unexpectedly, the effect of the negative context was smaller in drinkers with higher levels of alcoholrelated problems.

Reference 6 - 0,17% Coverage

However, the influence of emotional context on alcoholrelated action tendencies may become smaller when alcohol use becomes more problematic, which is in line with habit accounts of addiction

<Files\\Cousijn, Zanolie, Munsters, Kleibeuker, & Crone (2014)> - § 13 references coded [2,07% Coverage]

Reference 1 - 0,07% Coverage

and how this network is sensitive to adaption due to training of flexibility in thinking in adolescents.

Reference 2 - 0,06% Coverage

and (2) the effects of a divergent thinking training on RS connectivity in adolescents.

Reference 3 - 0,20% Coverage

We therefore tested the hypothesis that core regions involved in divergent thinking (MTG [extending into AG], SMG, and MeFG [Medial Frontal Gyrus] as part of the PFC) would show stronger connectivity at rest after a divergent thinking training, relative to a control group.

Reference 4 - 0,07% Coverage

Moreover, divergent thinking was expected to improve in adolescents following the AUT-training.

Reference 5 - 0,08% Coverage

(2) training did not improve divergent thinking or change RS functional connectivity in the training group,

Reference 6 - 0,20% Coverage

The connection between MTG, a region previously implicated in divergent thinking [8–10], and postcentral gyrus may aid in simulating possible ways in which objects can be used across cognitive and somatosensory domains, and as such may be helpful in thinking of more alternative uses.

Reference 7 - 0,42% Coverage

The pre-test to post-test comparison offluency scores in the scanner resulted in the expected training group x time interaction, however, the findings were in a different direction than anticipated. That is, the experimental group outperformed the active control group at the post test, but this was due to a decrease in performance in the active control group at posttest compared to pre-test. A closer inspection of the training data showed that when evaluating performance across the home training sessions, the AUT-training did not improve in divergent thinking fluency scores.

Reference 8 - 0,07% Coverage

A tentative explanation is that divergent thinking generally decreases over time without training.

Reference 9 - 0,12% Coverage

Providing active guidance during the training, including information about the creativity construct and the goal ofthe training may therefore be an essential component of

Reference 10 - 0,11% Coverage

a successful divergent thinking training. Hence, including active guidance and an incentive to maintain high motivation are recommended in future studies.

Reference 11 - 0,17% Coverage

We also tested the effects of divergent thinking training on a
near transfer task (AUT-brick) and a far transfer task (CAT), but the results showed that performance did not differ between the experimental and control group at post-test.

Reference 12 - 0,28% Coverage

Contrary to our predictions, the divergent thinking training as compared to the rule-switching (LGT) training was not significantly related to changes in seed-based or whole-brain RS functional connectivity over time. Thus, in comparison to the control group, we did not observe a significant change in seedbased and whole-brain RS functional connectivity patterns after the 8-session AUT-training.

Reference 13 - 0,22% Coverage

While not predicted in advance, this finding may indicate that a stronger functional segregation between the SMG and occipital cortex may benefit the possibility to think of more alternative ways to use objects for a similar interpretation of stronger connectivity predicting negative training outcome

<Files\\Crocetti et al. (2016)> - § 15 references coded [3,72% Coverage]

Reference 1 - 0,20% Coverage

In line with these considerations, in this study we sought to
examine whether adolescent empathy moderates the effects of strategies that parents can use to monitor their children (Kerr and Stattin, 2000) on adolescent antisocial behaviors.

Reference 2 - 0,17% Coverage

We hypothesized that adolescent empathy could moderate the effects of parental monitoring on adolescent antisocial behaviors, and tested this using a multi-informant multi-method longitudinal design.

Reference 3 - 0,16% Coverage

In line with this reasoning, in this study we examined whether
different dimensions of empathy could moderate associations between parental monitoring and adolescent antisocial behaviors.

Reference 4 - 0,39% Coverage

In fact, drawing from the basic principle that empathy makes adolescents more attuned to relationships (Hoffman, 2000) we can hypothesize that adolescents with higher empathy (a) might be better able to see parents' good intentions in solicitation and control; or (b) might be more sensitive to the potentially intrusive

aspects ofsolicitation and control in a period of adolescence in which both have to decline to match youth autonomy needs (Racz and McMahon, 2011).

Reference 5 - 0,12% Coverage

We sought to test both competing hypotheses to clarify if and for which adolescents parental monitoring behaviors are more or less beneficial.

Reference 6 - 0,74% Coverage

In synthesis, the aim of the present longitudinal multi-method and multi-informant study was to examine if the effects of parental monitoring on adolescent antisocial behaviors would be moderated by adolescent empathy. In line with recent developments of the empathy literature (e.g., Decety and Cowell, 2014; Van der Graaff et al., 2016), we tested for the moderating effects of trait and state dimensions of empathy considering both empathic concern and perspective taking. More specifically, as schematized in Figure 1, we investigated whether the effects of parental control and solicitation reported by fathers and mothers at T1 (when adolescents were 17-year-old) were differently related to relative changes (controlled for prior levels) in adolescent antisocial behaviors at T2 (1 year later, when adolescents were 18-year-old), for different levels of adolescent empathy at T1.

Reference 7 - 0,31% Coverage

We found that both affective (empathic concern) and cognitive (perspective taking) state empathy moderate the effects of parental solicitation on adolescent antisocial behaviors. Results highlighted that solicitation had unfavorable effects on adolescent antisocial behaviors in adolescents with high empathy whereas the opposite effect was found for adolescents with low empathy.

Reference 8 - 0,12% Coverage

We found that the impact ofparental solicitation on children's
antisocial behaviors differed for adolescents with high and low state empathy.

Reference 9 - 0,38% Coverage

First, results indicate that adolescents with higher empathic skills were more likely to react aversively to high levels of

parental solicitation. Thus, it is more important for parents to attune their monitoring to the developing autonomy needs of adolescents when adolescents have high interpersonal skills and are, therefore, more attentive to the potential intrusive nature of these behaviors.

Reference 10 - 0,09% Coverage

Second, we found beneficial effects of parental solicitation
for adolescents with low state empathic concern.

Reference 11 - 0,18% Coverage

Akin, in this study we found that parental solicitation is more beneficial for low empathic adolescents that might need a stronger parental presence for regulating their behavior and reduce involvement in antisocial acts.

Reference 12 - 0,11% Coverage

On the other hand, state (but not trait) dimensions of empathy moderated the impact of parental solicitation on antisocial behaviors.

Reference 13 - 0,23% Coverage

Finally, we found a significant moderation for both affective
and cognitive dimensions of state empathy. For affective empathy, we found differential significant effects at high and low levels, whereas for cognitive empathy, we detected a significant effect only at high levels.

Reference 14 - 0,43% Coverage

In this prospective longitudinal study, we found that parental solicitation behaviors aimed at eliciting adolescents' disclosure of information had detrimental effects for adolescents with high state empathy, while they were more positive for those with low empathy. These results have relevant practical implications, suggesting that parents need to become aware that their behaviors might elicit different effects for different children. Thus, they need to modulate their solicitation behaviors, using them with

Reference 15 - 0,10% Coverage

caution with high empathic children that can experience them as a violation of their privacy and a lack of trust.

<Files\\Damsteegt, IJzendoorn, Out, & Bakermans-Kranenburg (2014)> - § 1 reference coded [0,20% Coverage]

Reference 1 - 0,20% Coverage

Our findings with regard to pre-adoption living arrangements, TMT asymmetry, and sleep problems suggest that TMT is sensitive to early environmental influences

<Files\\De Zeeuw et al. (2017)> - § 8 references coded [2,07% Coverage]

Reference 1 - 0,26% Coverage

In the present study a direct causal effect of ADHD on educational achievement is tested taking potential genetic confounding into account in a large population-based sample of children in primary school. What must be noted is that a direct causal effect can only be refuted and not proven.

Reference 2 - 0,27% Coverage

A first prediction is that if ADHD symptoms have a
direct causal effect on educational achievement, ADHD symptoms will have an effect on educational achievement even when taking into account genetic and environmental factors with a pleiotropic influence on ADHD symptoms and educational achievement.

Reference 3 - 0,20% Coverage

A second prediction is that the association between ADHD symptoms and educational achievement exists longitudinally and that there is a direct path from ADHD symptoms at an earlier age to educational achievement at a later age.

Reference 4 - 0,20% Coverage

A third prediction is that, within monozygotic (MZ) twin pairs, differences in ADHD symptoms should be associated with differences in educational achievement because confounding by genes and a shared environment is excluded.

Reference 5 - 0,09% Coverage

A fourth prediction is that lowering of the number of ADHD symptoms will improve educational achievement.

Reference 6 - 0,18% Coverage

A significantly higher educational achievement test score in children with ADHD who use methylphenidate than children with ADHD who never used methylphenidate would be consistent with the causal hypothesis.

Reference 7 - 0,10% Coverage

Taken together, the tests were consistent with a direct causal effect of ADHD symptoms on educational achievement.

Reference 8 - 0,76% Coverage

To conclude, a direct causal effect seems to, at least
partly, underlie the association between ADHD and low educational achievement. A practical implication is that, when the prescription of medication, e.g. methylphenidate, leads to a reduction in ADHD symptoms, it can also, as a positive side effect, have an enhancing influence on educational achievement. There is also some evidence that psychological interventions, e.g. behavioral therapy, parent training and social skills training, have beneficial effects on ADHD symptoms in school-ages children (Serrano-Troncoso et al. 2013), in which case an improvement in educational achievement is also expected. One must keep in mind that the effect on educational achievement may be larger for children displaying inattentive symptoms compared to children mainly demonstrating hyperactive symptoms

<Files\\De Zeeuw et al. (2019)> - § 11 references coded [2,87% Coverage]

Reference 1 - 0,34% Coverage

Therefore, the present study investigates the interplay between genes and SES in relation to EA in the Netherlands, a more egalitarian country, where nearly all schools are state-supported and adhere to the same governmentally imposed standards and curriculum, regardless of whether they are private or state schools.

Reference 2 - 0,16% Coverage

The aim of the current study was to explore the effects of SES
on EA, in a large sample of 12-year-old twins from the Netherlands Twin Register (NTR).

Reference 3 - 0,19% Coverage

First, we assessed the moderating effect of SES on the mean of EA and on the genetic and environmental variance components underlying differences in EA (within-family design).

Reference 4 - 0,41% Coverage

of the genetic variance, as captured by a PGS, depended on SES, (ii) the association between PGS and EA differed across SES groups (between-family design) and (iii) the DZ twin with the higher PGS scored higher on EA than their co-twin with the lower PGS, to determine the effect of a child's PGS on EA while accounting for the effect of parental SES on EA (within-family design).

Reference 5 - 0,02% Coverage

To a large extent,

Reference 6 - 0,39% Coverage

differences between children in EA were attributable to genetic differences. SES accounted for a small, but significant, portion of the common environmental variance in EA. In the higher SES groups, the phenotypic

variance in EA was smaller and this could be attributed to a decrease in both the genetic and the environmental variance component with increasing SES.

Reference 7 - 0,67% Coverage

The association between PGS and EA might be confounded by a genotype-SES correlation, i.e., children who grow up in higher SES families have, on average, a higher PGS. In the current study, we addressed this potential confounding by employing a withinfamily design. In families with DZ twin offspring, the twin with the higher PGS scored higher on the EA compared with the co-twin with the lower PGS. This demonstrates that a child's PGS was related to EA even when taking into account the possible genotype-SES correlation confounding effect of parental SES as SES is shared by siblings growing up in the same household.

Reference 8 - 0,23% Coverage

More importantly, even in relatively egalitarian societies SES still has an effect when taking into account the effect of genetic differences between people, as also observed in other societies than the Netherlands.

Reference 9 - 0,12% Coverage

Differences between children from the same SES group were
partly due to differences in genetic predisposition.

Reference 10 - 0,23% Coverage

Policies to enhance EA could in the future focus on increasing the mean level of EA in these children through, for example, extra educational programs, to reduce inequality in educational outcomes and to ensure that

Reference 11 - 0,10% Coverage

all children are able to develop the academic skills that are needed to succeed in society.

<Files\\De Zeeuw, Beijsterveldt, Hoekstra, Bartles, & Boomsma (2017)> - § 8 references coded [2,09% Coverage]

Reference 1 - 0,33% Coverage

Using a multiple rater twin design in a large sample of general population preschool twins, this study aims to (a) estimate the contribution of genetic and environmental factors to autistic traits, controlling for the possible effects of rater bias, (b) explore possible sex differences in etiology of autistic traits

Reference 2 - 0,16% Coverage

In this large study of nearly 19,000 twin pairs, heritability estimates of autistic traits in 3-year-olds were high in both boys (78%) and girls (83%).

Reference 3 - 0,19% Coverage

The present study also found a small influence of these common environmental effects, but only on the parent-specific view on autistic traits and not on the shared behavioral view.

Reference 4 - 0,52% Coverage

This study found no evidence for the existence of differences in genes responsible for individual differences in autistic traits (qualitative sex differences), but did find some differences in the magnitude of the contribution of genes to variation in autistic traits (quantitative sex differences). However, these quantitative sex

differences were rather subtle and only significant because of the large sample size. Sex differences in the etiology of autistic traits thus appear relatively minor.

Reference 5 - 0,19% Coverage

The finding of negligible sex differences in our general population twin study suggests that the influence of common genetic variants on ASD is likely to be similar in boys and girls.

Reference 6 - 0,29% Coverage

The results of this study are in keeping with the consistent evidence for a modest, but potentially crucial role for environmental effects unique to each twin, although there may also be rare monozygotic pairs who are discordant for posttwinning de novo CNVs (Ehli et al., 2012).

Reference 7 - 0,17% Coverage

Autistic traits, as measured more reliably with multiple informants, are as heritable in early childhood as has been previously reported for older children and adults.

Reference 8 - 0,24% Coverage

There were only very small differences in the underlying etiology of autistic traits in boys and girls. • Environmental effects that are unique to a child also play a modest role, but the family environment seems to be less important.

<Files\\Derks, Krugers, Hoogenraad, Joëls, & Sarabdjitsingh (2016)> - § 14 references coded [2,60% Coverage]

Reference 1 - 0,18% Coverage

In line with this hypothesis, we expect MD animals to show efficient synaptic plasticity when tested under high-stress conditions -mimicked in vitro by CORT incubation- as opposed to conditions characterized by low CORT levels.

Reference 2 - 0,13% Coverage

In the current study we investigated when MD affects synaptic plasticity ofCA1 hippocam-
pal pyramidal neurons throughout development, in bothmale and female rats.

Reference 3 - 0,22% Coverage

Specifically, we studied 1) the effect ofMD on baseline field excitatory postsynaptic potential (fEPSP) properties and synaptic plasticity at different stages ofdevelopment, 2) whether these effects are sexdependent, and 3) how hippocampal fEPSP properties respond to high-stress CORT

Reference 4 - 0,13% Coverage

There is some support for the notion that MD causes quicker maturation of synaptic plasticity in males, because LTP was significantly enhanced by MD at P22-24.

Reference 5 - 0,10% Coverage

In our hands, MD induced alterations in hippocampal synaptic plasticity without accompanying long-
termneuroendocrine effects.

Reference 6 - 0,28% Coverage

The immediate reduction in bodyweight after MDwas rapidly restored, which indicates that the animal is well able to cope with the initial metabolic challenge. In addition, the absence of large MD effects on stress-sensitive parameters allows us to compare the two treatment conditions without confounding effects of the state of the endocrine system.

Reference 7 - 0,39% Coverage

However, in addition to this developmental effect,MD facilitated LTP induction in P22-24 males and in P85-95 females. It should be noted that all animals were weaned at P21, just before recordings on P22-24.Weaning is highly stressful for young rats, and we cannot exclude that the effect ofMD in the male adolescent group was amplified by 'weaning stress' one to three days before recording. The higher basal CORT levels in P22-24 rats (when compared to adult animals) seem to support this;

Reference 8 - 0,23% Coverage

In our statistical models, the situation predicting an increase in LTP up to adulthood in controls and a plateau in the (male) MD groups yielded the highest Bayes factor, which we carefully interpret as support for an acceleratedmaturation after MD in males, at least regarding LTP.

Reference 9 - 0,31% Coverage

The pattern ofMD interference with development of synaptic plasticity in females was different from that in males. Control females reached a plateau in synaptic strength already at P22-24, similar to the MDmales. Acceleration of synaptic plasticitymaturation by MDwas not observed; in contrast, MD affected synaptic plasticity in adult females by further increasing the level of induced LTP.

Reference 10 - 0,05% Coverage

We observeda CORT-induced suppression ofLTP induction in adult males,

Reference 11 - 0,13% Coverage

Plasma CORT from trunk blood and induced %LTP were correlated in the P85-95 male rats, where we observed (in agreement with the group averages) a negative correlation

Reference 12 - 0,07% Coverage

Ofnote, the suppressive effect by CORT on LTP in adult males was observedwith very high CORT

Reference 13 - 0,15% Coverage

levels, an order ofmagnitude above the levels seen in our control (MD and no-MD) rats, because the latter were always killed under rest and in the morning when plasma CORT levels are low.

Reference 14 - 0,23% Coverage

In conclusion, our study indicates that MD affects the development of synaptic plasticity in the CA1 hippocampus in a sex-dependentmanner. This implies that the optimal timing for treatments against the detrimental effects of early life stress is likely to be different in males and females.

<Files\\Eichelsheim, Blokland, Meeus, & Branje (2019)> - § 2 references coded [0,48% Coverage]

Reference 1 - 0,30% Coverage

Moreover, based on the framework proposed by Collins (2003), we would expect that background variables may also have an effect on the features of early adulthood romantic relationships. Among them are

socioeconomic status (SES; Collins, 2003), whether respondents already had romantic relationship experience (Boisvert & Poulin, 2016), and whether they experienced the divorce of parents in the family of origin (Amato & Booth, 2001; Cui & Fincham, 2010).

Reference 2 - 0,18% Coverage

As high-quality romantic relationships facilitate desistance from crime, the current findings suggest that persistence in antisocial behavior in the adult years may be partly contributed to both the nature of adolescent family relations and the extent of adolescent delinquency.

<Files\\Fox, Entink, & Timmers (2014)> - § 1 reference coded [0,05% Coverage]

Reference 1 - 0,05% Coverage

student ability and working speed influence feedback behavior.

<Files\\Hawk, Becht, & Branje (2016)> - § 2 references coded [0,24% Coverage]

Reference 1 - 0,10% Coverage

The results suggest that parents' suspicions and
anxieties about their children, and not the actualities of youths' behaviors, might be the prime moti-

Reference 2 - 0,14% Coverage

vator of snooping. Notably, the negative link between snooping and parents' perceived efficacy underscores the temptation to use such tactics when they lack confidence about their own skills or relationships.

<Files\\Hawk, Ter Bogt, Van den Eijnden, & Nelemans (2015)> - § 15 references coded [2,75% Coverage]

Reference 1 - 0,19% Coverage

Based on prior studies examining power, power motivation, and risk-taking, we hypothesized that high-narcissism youths who perceive a lack of social power might compensate with increasingly exhibitionistic SNS behavior (Buss & Chiodo, 1991).

Reference 2 - 0,14% Coverage

We tested this main hypothesis across two studies, in which we examined whether perceived social power moderates the link between adolescents' narcissism and disclosures on SNSs.

Reference 3 - 0,10% Coverage

our research elaborates upon power-risk processes that have previously only been demonstrated in artificial laboratory settings.

Reference 4 - 0,16% Coverage

We examined both types of SNS disclosure in this research, in order to further compare their relative frequencies and to examine whether the same psychological processes might underlie the two behaviors.

Reference 5 - 0,43% Coverage

Finally, researchers examining both narcissistic SNS exhibition-
ism (Bergman et al., 2011) and adolescents' online risk behaviors (Baumgartner, Valkenburg, & Peter, 2010) have pleaded for experiments supporting existing correlational and longitudinal studies. Doing so provides greater confidence when targeting particular factors in education and intervention. We therefore employed an

experimental manipulation of power Study 2, to provide causal evidence of how experiences of power(lessness) affect youths with differing levels of narcissism.

Reference 6 - 0,16% Coverage

as we expected, highernarcissism youths not only engage in more self-promotional SNS behavior, generally speaking, but also amplify their exhibitionism when they perceive less social power than they desire.

Reference 7 - 0,27% Coverage

These results suggest that the direct link between narcissism
and SNS activity widely found in prior studies only holds for certain (normative) types of self-disclosure, whereas other (problematic) forms of information sharing are linked to an interaction between this personality characteristic and the adolescents' social environment.

Reference 8 - 0,26% Coverage

We hypothesized that an experimental manipulation of social
power would produce differential inclinations toward SNS disclosure for lower- and higher-narcissism adolescents. We examined perceptions of disclosure risks in order to investigate whether differences in social power actually precipitate greater levels of SNS
disclosure

Reference 9 - 0,19% Coverage

These results indicate a causal role for power in the previously established link between narcissism and problematic SNS activity, with perceptions of low social power apparently inclining narcissistic youths toward more online exhibitionism.

Reference 10 - 0,15% Coverage

This suggests that youths higher in narcissism are prone to engage in more frequent and severe SNS exhibitionism when their social realities conflict with their desires for power over others.

Reference 11 - 0,14% Coverage

In Study 2, the effects of a fairly modest power manipulation upon risk assessments were substantially stronger for higher-narcissism youths than for lower-narcissism youths.

Reference 12 - 0,11% Coverage

If needs for validation are being met (i.e., they feel powerful), narcissistic adolescents may be more careful to avoid incriminating disclosures.

Reference 13 - 0,25% Coverage

Our results also suggest that parents or educators discussing
responsible online behavior might consider addressing narcissistic youths' responses to a perceived lack of social power, and how such experiences might elevate attention-seeking and statusenhancement motives to engage in problematic SNS disclosures.

Reference 14 - 0,14% Coverage

Adolescents scoring higher in narcissism appear to be prone to
engaging in different levels of problematic SNS disclosure, depending on whether they possess or lack social power.

Reference 15 - 0,07% Coverage

and possible mechanisms to address when attempting to promote responsible online behavior.

<Files\\Hessels, Cornelissen, Hooge, & Kemner (2017)> - § 4 references coded [0,49% Coverage]

Reference 1 - 0,31% Coverage

Although participants were asked to make eye contact while monitored by the experimenter (and recorded on video for later coding), there was no objective measurement that participants indeed looked at the eyes of the other. Where do people look when they (are instructed to) look toward each other? Is there still a bias for fixating the eye region when there is the possibility of social interaction (cf. Laidlaw et al., 2011)? We address these questions in the present study.

Reference 2 - 0,03% Coverage

More specifically, is there interaction be-

Reference 3 - 0,09% Coverage

tween the two partners in a duo with regard to looking at the eyes, such that the time spent looking at each other's eyes is correlated?

Reference 4 - 0,07% Coverage

We expected that if one partner looked longer into the eyes of
the other, the second partner should follow.

<Files\\Hessels, Hooge, & Kemner (2016) [1]> - § 5 references coded [1,24% Coverage]

Reference 1 - 0,08% Coverage

Q1.2 (Additional): Is time to target localization dependent on target and nontarget dissimilarity?

Reference 2 - 0,16% Coverage

Moreover, we noted that saccadic search performance was dependent on the target: Saccadic search performance was best for the 608 target, followed by the 308 target, and finally the 908 target.

Reference 3 - 0,09% Coverage

We return to this point momentarily, but conclude that saccadic search performance is at least target-dependent.

Reference 4 - 0,03% Coverage

and performance that is targetdependent

Reference 5 - 0,87% Coverage

However, we found that the 908 target was generally

fixated less often than the 308 and 608 targets, irrespective of target eccentricity. We therefore suggest that the 908 target may either constitute a less conspicuous target than the 308 and 608 targets, or infants do not consider the 908 line to be a target. This may be due to the 908 target being horizontal, and not slanted as the 308 and 608 target are. If infants categorize slanted objects from horizontal and vertical objects, as has been

suggested by earlier research (Quinn & Bhatt, 1998; Treisman & Gormican, 1988), subjective target–nontarget similarity for the 908 target is expected to be higher than for slanted objects. Future research into infant visual search performance should benefit from using a smaller range of target angle (i.e., excluding the horizontal target) in order to calculate search slopes as function of target–nontarget similarity, or adopt different set sizes to calculate search slopes.

<Files\\Hessels, Hooge, & Kemner (2016) [2]> - § 6 references coded [1,37% Coverage]

Reference 1 - 0,08% Coverage

Q1.2 (Additional): Is time to target localization dependent on target and nontarget dissimilarity?

Reference 2 - 0,16% Coverage

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Reference 3 - 0,09% Coverage

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Reference 4 - 0,03% Coverage

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Journal of Vision (2016) 16(8):10, 1–14 Hessels, Hooge, & Kemner
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Reference 6 - 0,13% Coverage

Infants search for discrepant items in the absence of instruction, and saccadic search performance is dependent on target and nontarget dissimilarity.

<Files\\Junge, Rooijen, & Raijmakers (2018)> - § 8 references coded [2,95% Coverage]

Reference 1 - 0,19% Coverage

This study examines whether frequency distributions of novel objects can guide infants' perceptual categorization.

Reference 2 - 0,26% Coverage

This study therefore tests whether frequency distributions shape object categoriza-
tion by adopting the experimental design from Maye et al. (2002).

Reference 3 - 0,73% Coverage

We hypothesized that if the frequency distribution of exemplars affects object categorization, infants with a bimodal distribution would prefer alternating over nonalternating trials, whereas infants who observed a unimodal distribution do not show any preference. If the hypothesized group effects appear short-lived, this would further indicate that infants' novel categories are fragile and can be adjusted with additional input.

Reference 4 - 0,18% Coverage

Our results show that the initial frequency distribution of objects can affect infant category formation.

Reference 5 - 0,15% Coverage

Yet, it was only in the first test block that we observe the hypothesized group differences.

Reference 6 - 0,35% Coverage

It appears that with additional input, infant category formation can change accordingly such that at the end of the testing phase, infants from the unimodal condition are also discriminating between tokens.

Reference 7 - 0,22% Coverage

In this paper, we set out to test whether distributional learning is a plausible
domain-general mechanism for categorization.

Reference 8 - 0,87% Coverage

Our results on object perception corroborate that this is likely so as it couples with positive evidence in the domains of speech perception (e.g., Maye et al., 2002) and face perception (Altvater-Mackensen et al., 2017). Yet although category formation rapidly emerges based on initial frequency distributions of observed objects, it is far from fixed as it remains susceptible to additional exposures. Clearly, categorization is an unfolding process and is continually shaped and updated through new experiences.

<Files\\Kanatsou et al. (2015)> - § 7 references coded [1,53% Coverage]

Reference 1 - 0,27% Coverage

Since sex-differences in MR function appear to exist in humans and rodents, we examined in this study whether forebrain-specific overexpression of MRs in female mice affects contextual memory formation, emotional memory formation and anxiety.

Reference 2 - 0,23% Coverage

Translating these findings from humans into rodent models,
we expected MR overexpression in female mice to reduce anxiety-like behavior, increase fear memory formation and context-depend memory formation.

Reference 3 - 0,12% Coverage

This suggests that MR overexpression possibly causes a compensatory downregulation ofcorticosterone levels.

Reference 4 - 0,13% Coverage

Therefore, we conclude that also the discriminative ability is not affected by overexpression of MR in female mice.

Reference 5 - 0,13% Coverage

Here we report that MR overexpression did not affect memory formation in a non-aversive contextual learning task.

Reference 6 - 0,34% Coverage

Taken together, testing female mice with forebrain-specific MR overexpression in several behavioral tasks revealed no effect on unconditioned anxiety, fear memory, the ability to discriminate between the threatening cue and the relatively safe cue-offperiod, and non-aversive contextual memory formation.

Reference 7 - 0,30% Coverage

Although we cannot exclude that effects of MR overexpression may be apparent in some of the tasks under different testing conditions, the current data suggest that MR overexpression does not substantially alter performance offemale mice in these behavioral domains.

<Files\\Kanatsou et al. (2017)> - § 10 references coded [2,16% Coverage]

Reference 1 - 0,34% Coverage

The aim of the current study was to examine the potential
causality between enhancedMRactivity and resilience to early life adversity. Therefore we investigated, in mice, whether transgenic overexpression ofMRs (Lai et al., 2007) is able to protect against earlier reported (Brunson et al., 2005; Rice et al., 2008; Naninck et al., 2015) adverse effects ofchronic early life stress on cognitive function and relevant cellular endpoints for memory formation.

Reference 2 - 0,41% Coverage

Here we report that transgenic forebrain overexpression of MRs from postnatal day 15 (Lai et al., 2007) in mice alleviates the effects of ELS on non-stressful contextual memory formation at adult age. Moreover, we observed an interaction between ELS and MR overexpression on the number of DCX positive cells in the DG and the frequency of mEPSCs and mIPSCs in DG granule cells. These results suggest that MR overexpression may partly prevent or reverse ELS-induced changes in hippocampusassociated behavior as well as hippocampal structural and functional plasticity.

Reference 3 - 0,17% Coverage

While other studies have reported that low levels of maternal
care and early life adversity enhance contextual fear memory formation (Champagne et al., 2008; Oomen et al., 2010) these effects were not found in our current study.

Reference 4 - 0,20% Coverage

Thus, in the same ELS paradigm we found that ELS results in enhanced expression of fear during cue-off periods in an auditory fear conditioning paradigm (Arp et al., 2016), indicating that ELS does impair the ability ofanimals to appreciate ‘safe’ moments in-between cue exposure.

Reference 5 - 0,07% Coverage

Importantly, our current results indicate that enhanced MR activation may overcome effects ofELS.

Reference 6 - 0,16% Coverage

Yet, we did see an interaction effect with regard to the number of DCX-positive cells, possibly related to the fact that MR-tg mice displayed increased numbers ofDCX positive (and Ki67) positive cells, particularly after ELS.

Reference 7 - 0,09% Coverage

Thus, MR overexpression may enhance the generation and maturation of newborn cells in the DG, but not their survival.

Reference 8 - 0,31% Coverage

This finding is in line with the current observation that overexpression ofMRs enhances proliferation in the DG. Our observation that ELS enhances the number ofDCXpositive cells specifically in MR-tg mice is more unexpected. Thus, enhanced proliferation –based on the result with Ki67 staining- in combination with (slightly) enhanced survival –as inferred from BrdU staining- were observed both in control and in ELS MR-tg mice.

Reference 9 - 0,10% Coverage

Our data reveal that MR overexpression may confer resilience to the effects of stress exposure during early life on contextual memory formation.

Reference 10 - 0,30% Coverage

Regardless, the fact that changes in MR expression starting after ELS has taken place result in hippocampal function that is indistinguishable from that seen in non-stressed wildtypes has interesting translational potential. These studies may provide new avenues to interfere with vulnerability factors (such as CRH) or resilience factors (such as MR) to promote behavioral performance after early life adversity.

<Files\\Kentrop et al. (2016)> - § 12 references coded [2,77% Coverage]

Reference 1 - 0,37% Coverage

Therefore we now set out to specifically study the long-
term effects of 24 h MD at PND 3 on behavior in the 5-choice SRTT, an operant task in which both attention and behavioral inhibition can be measured (Robbins, 2002). Given the effect of early life stress on HPA-axis dysregulation, we were also interested in the ability of the non-selective GR antagonist mifepristone to reverse potential effects of early life stress on behavior.

Reference 2 - 0,31% Coverage

We reasoned that the effects of acute mifepristone administration on behavior might be particularly effective when brain areas involved in impulsive behavior are still highly plastic, such as during (early) adolescence. We therefore aimed to reverse putative behavioral effects of MD through a 3-day early adolescent treatment with mifepristone between PND 26 and 28.

Reference 3 - 0,15% Coverage

We hypothesized that attention and behavioral inhibition, critically depending on PFC functioning, are affected by MD and sensitive to mifepristone treatment during early adolescence.

Reference 4 - 0,15% Coverage

We demonstrated that 24 h MD on PND 3 significantly reduces behavioral inhibition, as evident from a modest increase in premature responses when the inter-trialinterval was prolonged.

Reference 5 - 0,13% Coverage

A 3-day Mif treatment during early adolescence did not mitigate the effects of MD on behavioral control, but by itself slightly reduced perseverative behavior.

Reference 6 - 0,16% Coverage

It is not unlikely that the observed effects of MD are at least in part caused by a combination of the two factors and with the current protocol these two factors cannot be addressed separately.

Reference 7 - 0,45% Coverage

The effect of MD on body weight was not
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affected by Mif treatment on PND 26–28. Because from week 12 onwards animals were food restricted and maintained on 90–95% of their body weight and their body weights at the start of training was no longer a reflection of their natural free-fed weight. We cannot entirely exclude that body weight differences might have affected acquisition of the 5-choice task, but this seems unlikely since all animals acquired the task equally well.

Reference 8 - 0,10% Coverage

i.e., we also report no effects of early life stress on impulsive responding during acquisition and baseline performance.

Reference 9 - 0,07% Coverage

It was exactly in this condition that in our experiments MD effects became apparent.

Reference 10 - 0,36% Coverage

Interestingly, while Mif was ineffective in moderating the
effects of MD, we did find an effect of Mif treatment by itself on perseverative responses; i.e., Mif treated animals made less perseverative responses than Veh treated animals. A high number of perseverative responses in this task is thought to reflect compulsive behavior (Dalley et al., 2011) and this would suggest that Mif might act to prevent this type of behavior.

Reference 11 - 0,30% Coverage

Overall, the results of this study suggest that a separation
from the mother for 24 h on PND 3, during the stress hyporesponsive period, impacts on impulsivity, but does not influence attention as measured in the 5-choice SRTT; blockade of GRs for 3 days during early adolescence was ineffective to counteract the behavioral changes caused by early life stress.

Reference 12 - 0,21% Coverage

If so, this would support the notion that early life adverse conditions do not necessarily cause behavioral deficits in adulthood, but that multiple ‘‘hits’’ may compromise the adaptive capacity ofindividuals (McEwen, 1998; Daskalakis et al., 2013).

<Files\\Koenis et al. (2015)> - § 9 references coded [1,40% Coverage]

Reference 1 - 0,16% Coverage

Using structural equation modeling [Boker et al., 2011] and network connectivity analyses [Rubinov and Sporns, 2010], we estimated the heritability and development of the brain network in adolescence at a three-year interval.

Reference 2 - 0,23% Coverage

Both increases in white matter integrity (FA) and reorganization of the network contribute to an increase in FA-based global and local efficiency. At the same time, local increases and decreases in streamline count and a

reorganization of the network both contribute to a net decrease in streamline count-based network global efficiency.

Reference 3 - 0,12% Coverage

This effect seemed stronger in FA based efficiency, which showed a clear pattern of frontal and temporal increases of local efficiency that were related to an increase IQ.

Reference 4 - 0,16% Coverage

We report moderate to high heritability for efficiency of
the brain's structural network (locally up to 74% for FAbased networks; up to 64% for streamline count based networks) in early adolescence. For both average local and

Reference 5 - 0,18% Coverage

global efficiency, the genetic factor influencing the efficiency measure remained stable during development in adolescence. Besides, the relation between IQ and FAbased local network efficiency was completely explained by genes shared by both phenotypes.

Reference 6 - 0,24% Coverage

The positive associations between IQ and FA-based local efficiency were particularly prominent in frontal and temporal nodes, emphasizing the relevance of frontal and temporal regions for intelligence [Haier et al., 1988, 2004], in accordance with the P-FIT regions (parieto-frontal integration theory of intelligence) regions [Jung and Haier, 2007].

Reference 7 - 0,15% Coverage

Our finding suggests that during this time of rapid intellectual development, plasticity of the brain's network is an important contributing, if not necessary, factor to maintain and possibly gain in intellectual capacity.

Reference 8 - 0,01% Coverage

We also found

Reference 9 - 0,15% Coverage

that variation in the topology of both FA and streamlinebased networks of young adolescents is partly due to genetic variation, and that genes shaping FA-based connectivity organization also benefit intelligence.

<Files\\Kok et al. (2015) [1]> - § 10 references coded [2,15% Coverage]

Reference 1 - 0,20% Coverage

Moreover, no studies on the influence of caregiving on child brain structure have used repeated measures of the quality of both maternal and paternal caregiving in early childhood.

Reference 2 - 0,27% Coverage

First, we examined the relation between early parenting and child brain structure in a large and relatively homogeneous sample of healthy children (N ¼ 191), thus extending previous results to nondisadvantaged families with fewer confounders.

Reference 3 - 0,20% Coverage

Third, we investigated the association of maternal and paternal sensitivity separately and explored whether differences exist in their respective influences on child brain structure.

Reference 4 - 0,20% Coverage

Fourth, we adjusted our analyses for head size at 6 weeks of age and thus accounted for a proxy of brain development immediately after birth and limited the risk of reversed causality.

Reference 5 - 0,07% Coverage

a global effect of sensitivity on the brain seems plausible.

Reference 6 - 0,30% Coverage

our results suggest that fathers'
sensitivity is no less important for child brain development than mothers' sensitivity. Our results appear to imply that the quality of caregiving is most important for brain development and not dependent on the person providing this care.

Reference 7 - 0,10% Coverage

Our findings emphasize the importance of the whole family system for optimal child development.

Reference 8 - 0,53% Coverage

Parental sensitivity was a significant predictor of child
brain volume independent of infant head size, suggesting that the association cannot be explained by underlying biological vulnerability. Due to the design with only 1 measure of child brain volume, we cannot preclude reverse causality, but our findings are in accordance with recent experimental studies showing that parental sensitivity can result in differences in brain development in both infants and adolescents.14,23

Reference 9 - 0,12% Coverage

Second, although we controlled for a number of potential confounders, we cannot preclude residual confounding.

Reference 10 - 0,16% Coverage

These findings are in line with robust evidence that caregiving is essential for child development in cognitive, behavioral, and social domains.

<Files\\Kok et al. (2015) [2]> - § 11 references coded [2,50% Coverage]

Reference 1 - 0,20% Coverage

Moreover, no studies on the influence of caregiving on child brain structure have used repeated measures of the quality of both maternal and paternal caregiving in early childhood.

Reference 2 - 0,27% Coverage

First, we examined the relation between early parenting and child brain structure in a large and relatively homogeneous sample of healthy children (N ¼ 191), thus extending previous results to nondisadvantaged families with fewer confounders.

Reference 3 - 0,20% Coverage

Third, we investigated the association of maternal and paternal sensitivity separately and explored whether differences exist in their respective influences on child brain structure.

Reference 4 - 0,20% Coverage

Fourth, we adjusted our analyses for head size at 6 weeks of age and thus accounted for a proxy of brain development immediately after birth and limited the risk of reversed causality.

Reference 5 - 0,34% Coverage

We do not expect to find differences in the relation between maternal versus paternal sensitivity and child brain structure because both maternal and paternal sensitivity are related to more favorable child outcomes, and the quality of care may be more influential than whether it is provided by mother or father.

Reference 6 - 0,07% Coverage

a global effect of sensitivity on the brain seems plausible.

Reference 7 - 0,30% Coverage

our results suggest that fathers'
sensitivity is no less important for child brain development than mothers' sensitivity. Our results appear to imply that the quality of caregiving is most important for brain development and not dependent on the person providing this care.

Reference 8 - 0,10% Coverage

Our findings emphasize the importance of the whole family system for optimal child development.

Reference 9 - 0,53% Coverage

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Reference 10 - 0,12% Coverage

Second, although we controlled for a number of potential confounders, we cannot preclude residual confounding.

Reference 11 - 0,16% Coverage

These findings are in line with robust evidence that caregiving is essential for child development in cognitive, behavioral, and social domains.

<Files\\Kolijn et al. (2017)> - § 9 references coded [3,49% Coverage]

Reference 1 - 0,23% Coverage

The current protocol presents a randomized controlled trial in which we aim to examine the neurocognitive mechanisms through which intervention effects on parenting behavior might be established. The focus will be on assessing the underlying neural activity of two

Reference 2 - 0,09% Coverage

constructs that may be important in parenting behavior: emotion recognition and inhibitory control.

Reference 3 - 0,36% Coverage

We will investigate neural processing of emotional facial expressions and the neural correlates of inhibitory control as it is plausible that these processes are important for parental sensitivity. More specifically, the two neurocognitive processes of interest may be affected by the intervention since key elements of the intervention involve parental coping with children's displays of (negative) emotionality.

Reference 4 - 0,17% Coverage

In the current study we aim to test whether the intervention will affect the N170 in response to children' emotional faces in a large non-clinical sample of mothers of young same-sex twins.

Reference 5 - 0,24% Coverage

As inhibitory control plays an important role in emotion regulation and thereby modulates parental reactions to children's behavior [46], we aim to test whether the intervention enhances N2 amplitudes as well as the efficiency of response inhibition in a stop signal paradigm.

Reference 6 - 0,19% Coverage

For the current study we aim to investigate whether the intervention lowers stress in parents, as reflected in self-reported stress and in lower cortisol levels, which in turn may facilitate parental sensitivity.

Reference 7 - 0,80% Coverage

Our primary aim is to investigate intervention effects on the neural correlates of inhibitory control and the neural processing of emotional facial expressions. First, we will examine whether the intervention affects the neural processing of children's emotional faces as reflected in the N170 component. We expect that N170 amplitudes in response to emotional faces will be enhanced in parents in the intervention condition compared to parents in the control condition. In addition, we will explore potential latency and differential emotion effects as well. Second, we will examine whether the intervention affects the N2 during a response inhibition (stop signal) task. Compared to parents in the control condition, we expect N2 amplitudes in response to stop signals to be enhanced in parents in the intervention condition. In addition, we will explore whether the intervention affects latency of the N2.

Reference 8 - 1,09% Coverage

Our secondary aim is to investigate the neurobiological mechanisms through which intervention effects on parenting behavior are established. More specifically, we will investigate whether the intervention results in changes in these neurocognitive processes which in turn contribute to observable effects on parenting behavior. We will examine whether intervention effects on parenting behavior are mediated by intervention effects on the N170 and N2. The expectation is that the intervention positively affects the neural processing of children's emotional faces and inhibitory control mechanisms, as indicated by enhances amplitudes of the N170 and the N2, which in turn will promote parental sensitivity and sensitive discipline during parent– child interactions. In addition, we will examine whether intervention effects on sensitive parenting behavior are mediated by the stress hormone cortisol. It is expected that the intervention reduces stress levels in parents which in turn promotes parental sensitivity and sensitive discipline.
3) Our tertiary aim is to explore whether intervention effects on parenting behavior and on N170 and N2 amplitudes are moderated by patterns of asymmetric frontal cortical activity (see Fig. 1).

Reference 9 - 0,33% Coverage

The current protocol presents a study design of a randomized controlled trial in which we aim to investigate neural and hormonal mechanisms that may be involved in the intervention effects of the VIPP on parenting

behavior. More specifically, we hope to gain insight in the mediating mechanisms through which intervention effects on parenting behavior are brought about.

<Files\\Kretschmer et al. (2015)> - § 2 references coded [0,30% Coverage]

Reference 1 - 0,11% Coverage

Peer-based prevention and intervention programs should aim to counteract negative peer effects while simultaneously build on positive facets of adolescents' individual peer contexts.

Reference 2 - 0,19% Coverage

Despite these limitations, this study contributes to the continuous effort to understand adolescents' peer experiences and the interplay between different interpersonal systems in adolescents' social environments, and adds to the literature on transmission processes between parents and peers.

<Files\\Kretschmer et al. (2018)> - § 18 references coded [3,54% Coverage]

Reference 1 - 0,18% Coverage

Besides examining direct effects between bullying involve-
ment and mastery ofdevelopmental tasks, we attend to recent calls for studies into potential buffers ofconsequences ofbullying involvement by testing whether supportive relationships with parents diminish the risk for negative outcomes.

Reference 2 - 0,38% Coverage

Aiming to consolidate these divergent findings and contrib-
uting to a still small number ofstudies that incorporate perpetrators of bullying, we concentrated on parental support as moderator on links between early adolescent victimization and perpetration and mastery of early adult developmental tasks. Although it is plausible that negative effects of victimization and perpetration can best be diminished if social support is available at time of exposure, we were particularly interested in support during the transition from adolescence through adulthood because of its temporal proximity to the outcomes under study.

Reference 3 - 0,33% Coverage

High levels of support are needed to finish school, find a job, and limit engagement in substance use, and those young adults who are supported in taking up new roles and develop adult competencies might suffer less from the consequences of earlier social insults. The role of parental support is less straightforward for perpetrators but if bullying is understood as a facet of a broader externalizing type, its developmental stability and link with early adult outcomes should also be diminished by particularly positive relationships with parents.

Reference 4 - 0,14% Coverage

We therefore examined gender-specificity in associations between bullying victimization and perpetration and mastery of early adult tasks and explored whether potential moderation effects by parental support depend on gender.

Reference 5 - 0,19% Coverage

Childhood demographics and child intelligence are impor-
tant predictors ofadult development and have served as control variables in other studies on outcomes ofbullying involvement (Takizawa et al. 2014;Wolke etal. 2013), we thus estimated all models both unadjusted as well as adjusted for these potential confounders.

Reference 6 - 0,44% Coverage

Moreover, childhood psychopathology might be an important explanatory mechanisms for associations between bullying involvement and mastery of developmental tasks as bullying victimization and, to a lesser extent, perpetration are linked to psychopathology, concurrently and over time. In other words, those involved in bullying as victims or perpetrators are perhaps less likely to master developmental tasks because psychopathological symptoms prevent them from doing so. Thus, we also computed models in which internalizing and externalizing symptoms in childhood were added. We expected that confounders and childhood psychopathology reduce the strengths of associations between bullying involvement and mastery ofdevelopmental tasks.

Reference 7 - 0,08% Coverage

Second, we examined potential moderating effects of pa-
rental support as buffer against the negative sequelae of bullying involvement.

Reference 8 - 0,17% Coverage

This question has hardly been researched, especially when outcomes refer to developmental tasks, but we tentatively expected that individuals who receive plenty of support while being confronted with early adult tasks would likely be less affected by previous bullying involvement.

Reference 9 - 0,18% Coverage

Associations ofvictimization with education/work and finan-
cial competence were detected in the TRAILS sample, though only the link between victimization and welfare dependence remained statistically significant after controlling for a range of potential confounders and when associations between child

Reference 10 - 0,04% Coverage

psychopathology and developmental tasks were estimated simultaneously.

Reference 11 - 0,14% Coverage

In RADAR-young, victimized teens were at greater risk to smoke at least occasionally by early adulthood though this link weakened to non-significance when adjusted for childhood demographics, intelligence, and psychopathology.

Reference 12 - 0,15% Coverage

Moreover, adjusting for childhood demographics and psychopathology reduced effect sizes, partly considerably, which suggests that in light ofvarious other childhood risks, bullying victimization is less crucial for mastery ofearly adult developmental tasks.

Reference 13 - 0,25% Coverage

Only the reduced likelihood for a law-abiding live in bullying perpetrators was retained in TRAILS, tentatively suggesting that even in light of various childhood risks for crime involvement, bullying perpetration increased the risk for an antisocial lifestyle (Hussong et al. 2004). Programs focused on helping victims deal with their plight should thus also keep an eye on the development of the perpetrators.

Reference 14 - 0,06% Coverage

These measures thus appear to function as suppressors but the changes in effect sizes are negligible.

Reference 15 - 0,09% Coverage

Either way, links between bullying involvement and mastery ofdevelopmental tasks were modest, there is thus little effect that needs to be buffered.

Reference 16 - 0,36% Coverage

Family SES in early adolescence predicted whether or not young adults in both RADAR-young and TRAILS participated in the labor market or tertiary or continuing education and, at least in TRAILS, family instability contributed to risk for low educational attainment, lack of financial competence, and welfare dependence. These were anticipated associations, yet, the impact of these childhood demographics on mastery of developmental tasks almost a decade later is still striking, particularly considering that these links remained largely stable when child psychopathology was added to the models.

Reference 17 - 0,14% Coverage

Finally, in TRAILS, higher intelligence and higher SES in
early adolescence increased the likelihood for alcohol and cannabis use in early adulthood, the link between intelligence and alcohol use was also detected inRADAR-young.

Reference 18 - 0,23% Coverage

The strengths of associations was reduced when adjusted for other childhood risks and when childhood psychopathology was examined as potential explanation for links between bullying involvement and mastery of developmental tasks, which demonstrates that, relative to other childhood risks, bullying involvement is less strongly implied in mastery of early adult developmental tasks.

<Files\\Kretschmer, Barker, Dijkstra, Oldewinkel, & Veenstra (2015)> - § 2 references coded [0,31% Coverage]

Reference 1 - 0,13% Coverage

We further examined whether peer victimization con-
tributed to change in maladjustment using latent transition analyses.

Reference 2 - 0,18% Coverage

Notably, peer victimization may actually cushion the risk for externalizing behavior, which contradicts studies that found this association [4, 5] in single-outcome models.

<Files\\Kretschmer, Vollebergh, & Oldehinkel (2017)> - § 11 references coded [1,47% Coverage]

Reference 1 - 0,18% Coverage

In trying to understand how parent–child relationship quality
and later romantic relationship characteristics are linked, some have argued that parent–child negativity impairs a young person’s development of social competence (Rauer et al., 2013), something that

Reference 2 - 0,05% Coverage

can later be reflected in compromised relationships with romantic partners.

Reference 3 - 0,04% Coverage

Nonlinear associations may support our view that different

Reference 4 - 0,06% Coverage

mechanisms drive the association at different levels ofparent–child relationship quality.

Reference 5 - 0,13% Coverage

Extending this work, we examined social skills more generally and explored indirect paths from parent–child relationship quality to romantic involvement, commitment, and satisfaction via social skills.

Reference 6 - 0,06% Coverage

Do possible association between parent–child and romantic relationships run via social skills?

Reference 7 - 0,15% Coverage

Moreover, we examined whether associations are partly accounted for by social skills but given the lack of studies on parent–child and romantic relationships and general social skills, these analyses were of exploratory nature.

Reference 8 - 0,15% Coverage

The nonlinear effect suggests that, despite this overall trend, individuals who had experienced particularly high or low levels of positivity, were somewhat more committed, the latter possibly reflecting compensation.

Reference 9 - 0,25% Coverage

Parents make an important contribution to their offspring's development of interpersonal relationships but the nature of this contribution seems more complex than prior studies indicate. In other words, it is not necessarily the case that extremely positive parenting is reflected in particularly well-functioning romantic relationships as indicated by the nonlinear trend.

Reference 10 - 0,21% Coverage

Cooperation and self-control were stably nonlinearly associated with parent–child positivity suggesting that not only low parent–child positivity is detrimental to cooperation and self-control; highly positive parenting–child relationship quality appears to be problematic for social skill development as well.

Reference 11 - 0,18% Coverage

In fact, the amount of variance explained in commitment increased remarkably with gender, parental SES and relationship duration in the model, suggesting that individual and demographic factors may at least be as important as experiences in other interpersonal relationships.

<Files\\La Roi, Kretschmer, Dijkstra, Veenstra, & Oldehinkel (2016)> - § 12 references coded [1,34% Coverage]

Reference 1 - 0,12% Coverage

Aiming to fill these gaps, we examine from which developmental period disparities in depressive symptoms between heterosexual and LGB youth begin to occur and which factors act as catalysts of these disparities.

Reference 2 - 0,09% Coverage

Therefore, we study whether parental rejection and peer victimization mediate the potential association between sexual orientation and depressive symptoms.

Reference 3 - 0,15% Coverage

In this study, we will also focus on the effect of peer victimization and parental rejection on depressive symptom levels of LGB youth and expect that these interpersonal mechanisms explain the association between sexual orientation and depressive symptoms at least partly.

Reference 4 - 0,15% Coverage

The aims of this study were to examine from what developmental period onwards disparities in depressive symptoms between heterosexual and LGB youth start to occur, how these disparities develop over time and what factors act as catalysts ofthese disparities.

Reference 5 - 0,08% Coverage

In short, we expect further pubertal development to lead to an increase in depressive symptom disparities between heterosexual and LGB youth (H2).

Reference 6 - 0,08% Coverage

We will test this mechanism and expect that peer victimization mediates the association between sexual orientation and depressive symptoms (H3).

Reference 7 - 0,08% Coverage

Based on this literature, we expect that parental rejection mediates the association between sexual orientation and depressive symptoms (H4).

Reference 8 - 0,15% Coverage

To examine the developmental stability of associations, we additionally tested whether peer victimization in early adolescence (wave 2) and parental rejection in late adolescence (wave 4) mediated the association between sexual orientation and depressive symptoms.

Reference 9 - 0,16% Coverage

The intercept differences in depressive symptoms by sexual orientation were partially mediated by self-identified peer victimization, as well as parental rejection. For girls, we were thus able to detect mechanisms in line with the Minority Stress Framework, already at age 11.

Reference 10 - 0,08% Coverage

For boys, we did however detect an indirect effect of sexual orientation on depressive symptoms, via self-reported peer victimization.

Reference 11 - 0,16% Coverage

The intercept differences in depressive symptoms by sexual orientation were partially mediated by self-identified peer victimization, as well as parental rejection. Also for bisexuals, we were thus able to detect mechanisms in line with the Minority Stress Framework, already at age 11.

Reference 12 - 0,05% Coverage

These differences were partly mediated by peer victimization and parental rejection.

<Files\\Laceulle, Jeronimus, Aken, & Ormel (2015)> - § 21 references coded [4,44% Coverage]

Reference 1 - 0,24% Coverage

In this study, we test whether individual differences in perceived relationship affection mediate the prospective association between temperament and the evocation of stressful social events in adolescents. A demonstration of mediation by perceived relationship affection could propel the exploration and understanding of the mechanisms that drive the social selection principle.

Reference 2 - 0,29% Coverage

We hypothesise that temperamental differences lead to differences in perceived affect, which in turn result in the evocation of stressful social events. Specifically, in the current study, we tested whether perceived relationship affection—that is, adolescents subjective experience of care, protection, comfort and approval provided by significant others (i.e. parents, peers)—mediates the prospective association between temperament and stressful social events.

Reference 3 - 0,18% Coverage

For example, we tested whether individuals high (versus low) on temperamental frustration are more likely to perceive low relationship affection, because high frustration may be related to more anger, frustration and withdrawal, resulting in more conflicts with significant others.

Reference 4 - 0,11% Coverage

To recapitulate, in the current study, we verify whether adolescent's perceived relationship affection mediates the association between adolescent temperament and evocation of

Reference 5 - 0,04% Coverage

stressful social events (see Figure 1 for a conceptual model).

Reference 6 - 0,18% Coverage

To test for spill-over effects between different social domains, we examined mediation effects of both perceived parental affection and perceived peer affection in the associations
between temperament and stressful social events in the parental, peer and romantic partner domain.

Reference 7 - 0,43% Coverage

Additionally, we hypothesised that prospective associations between temperament and subsequent stressful social events are partially mediated by perceived relationship affection (H2). More specific, we hypothesised domain-specific associations, viz. perceived parental affection as the primary mediator of stressful social event evocation effects in the parental domain (H3a) and perceived peer affection as the mediator of stressful social event evocation effects in the peer domain (H3b). Lastly, we tested whether associations between temperament and subsequent stressful social events in the romantic partner domain were mediated by perceived either parental or peer affection.

Reference 8 - 0,44% Coverage

Our results support the hypothesis that temperamental differences are manifested in differences in stressful social event evocation, in line with previous research. Our study innovated by the observation that perceived relationship affection mediates a modest part of this association. In other words, temperaments colour the way adolescents perceive received affection, which, in turn, influences the probability of subsequent stressful social events. Our distinction between three social domains (i.e. parents, peers and romantic partners) yielded support for both generic and domain-specific effects, which exemplifies the challenge of isolating mechanisms behind the stress selection principle.

Reference 9 - 0,11% Coverage

Hence, extraversion (being out-going, sociable etc.) seems more influential when engaging with romantic partners than in the more persistent relationships with parents and peers.

Reference 10 - 0,08% Coverage

Indeed, perceived relationship affection mediated several of the associations between temperament and stressful social events.

Reference 11 - 0,08% Coverage

More specifically, our results suggest that perceived relationship affection mediated part of the studied associations.

Reference 12 - 0,28% Coverage

Our results align with the interpretation that individuals
develop internal working models based on their temperamental characteristics, which, in turn, modulates adolescents' perceptions of their affective relationships with others, as outlined in the introduction. Hence, perceived relationship affection, a rather complex intrapsychic characteristic, can have real-world consequences in terms of subsequent stressful social events.

Reference 13 - 0,27% Coverage

Moreover, the findings confirm and extent previous research showing that people's perceptions of their relationships with others (e.g. expected rejection) can become a self-fulfilling prophecy when people start behaving in ways (e.g. withdrawal and aggression) that elicit stressful social interaction (e.g. conflicts, rejection and breakup; see Bradbury & Fincham, 1990; Downey et al., 1998; London et al., 2007; Sroufe, 1990).

Reference 14 - 0,33% Coverage

We hypothesised that mediation by perceived relation affection would be largely domain-specific (H3). Indeed, parental affection was the primary mediator of temperamental stressful social event evocation in the parental domain (H3a), whereas perceived peer affection mediated the evocation of stressful events in the peer domain (H3b). Perceived parental affection mediated part of the association between affiliation and events in the parental domain, but surprisingly, no effect was found for the other temperamental traits.

Reference 15 - 0,27% Coverage

With regard to perceived peer affection, several media-
tion effects were found. Adolescents lower on frustration or shyness and/or higher on affiliation or effortful control reported more peer affection, which, in turn, predicted less subsequent stressful social events in the peer domain. This suggests that, as hypothesised, adolescent's perceived affection received from their peers is important in the association

Reference 16 - 0,04% Coverage

between temperament traits and events in the peer domain.

Reference 17 - 0,23% Coverage

Notably, for both frustration and effortful control, no direct effects were found on stressful social events in the peer domain. This indicates that perceived peer affection mediates part of the stress-evocation effects of frustration and effortful control, whereas extraversion-driven evocation effects tend to be more independent of perceived affection.

Reference 18 - 0,44% Coverage

Findings suggested some spill-over effects. Adolescents lower on frustration and/or higher on affiliation or effortful control reported more affection from parents and peers, which, in turn, predicted subsequent stressful social events in the romantic partner domain. Whereas more perceived parental affection predicted fewer events in the romantic partner domain (in line with the negative association between perceived parental affection and events in the parental domain), more perceived peer affection predicted more events in the romantic partner domain (a positive association, diametrical to the negative association between perceived peer affection and events in the peer domain)

Reference 19 - 0,17% Coverage

Moreover, the spill-over effects of perceived parental affection to the romantic domain may reflect that young adolescents use their perceptions of their parents to guide their behaviour in interaction with their (first) romantic partners (Linder, Crick, & Collins, 2002).

Reference 20 - 0,10% Coverage

Results showed that the effects of affiliation and frustration remained in these more conservative analyses, bolstering the robustness of the findings.

Reference 21 - 0,13% Coverage

Findings indicate that individuals may evoke subsequent stressful social events based on their temperament and that this associations is partially mediated by adolescents' perceived relationship affection.

<Files\\Loi (2017)> - § 12 references coded [1,83% Coverage]

Reference 1 - 0,39% Coverage

In view of the well-established effects of stress hormones, such as corticosteroids, on cognitive function (Aisa et al., 2007; Lupien et al., 2009), it is conceivable that a dysfunctional HPA axis contributes to the development of behavioral deficits and thus may form a target for intervention. To test this idea, we exposed rats to 24-h maternal deprivation (MD) on postnatal day (PND)3, examined their ability to form hippocampus-dependent contextual memories in young adulthood (approximately three months of age) and examined whether transient mifepristone (MIF) treatment, which among others blocks GRs, (van Haarst et al, 1996) at PND26-PND28 normalizes the expected behavioral deficits.

Reference 2 - 0,13% Coverage

To underpin the behavioral observations, we examined neuronal activity by c-Fos staining in brain structures likely contributing to the behavioral tasks, particularly hippocampal, prefrontal, and striatal areas (van den Bos et al., 2014).

Reference 3 - 0,21% Coverage

The main finding of this study is that transiently reduc-
ing GR activation during a critical developmental period prevents precipitation of cognitive impairments in adulthood associated with early life adversity. These behavioral effects were accompanied by changes in neuronal activity of principal neurons in the dorsal CA1 hippocampus and dorsomedial striatum.

Reference 4 - 0,08% Coverage

The lower body weight and higher basal CORT level at
PND26 following 24-h MD at PND3 support the efficacy of the early life adverse conditions.

Reference 5 - 0,12% Coverage

Even in the hippocampus, where CORT levels were increased at PND26 after MD, in the absence of changes in GR expression, temporary MIF treatment may help to restore or prevent the development of behavioral deficits.

Reference 6 - 0,23% Coverage

We now show that the reverse is not true: MD affected spatial learning since rats did not discriminate empty from baited arms (despite the presence of cues) and probably as a consequence reward-like learning was compromised. While the deficits in spatial learning were completely restored by MIF, treatment did not completely reverse deficits in reward learning, and seemed to have effects per se (see below).

Reference 7 - 0,12% Coverage

Supporting this and underpinning the behavioral observations, we found, using c-Fos expression and electrophysiology, that MD affected dorsal hippocampus and dorsomedial striatal functioning, which was restored by MIF.

Reference 8 - 0,19% Coverage

We furthermore observed that MD changed c-Fos expression in the right shell of the nucleus accumbens and insular cortex, which was restored by MIF. These changes may also have contributed to the observed behavioral changes and point to a role of MD in reward processing in addition to spatial learning (in line with van Hasselt et al., 2012b).

Reference 9 - 0,06% Coverage

Yet, spontaneous glutamatergic transmission in the dorsomedial striatum was increased after MD and restored by MIF.

Reference 10 - 0,08% Coverage

Collectively, these data suggest that early life stress targets glutamatergic transmission, an effect that can be restored by temporary MIF treatment.

Reference 11 - 0,07% Coverage

The current results provide a promising basis for new intervention strategies in humans exposed to early life adversity.

Reference 12 - 0,14% Coverage

In conclusion, transient prepubertal treatment with MIF
fully normalizes hippocampus-striatum-dependent contextual memory/spatial learning deficits in male rats exposed to early life adversity, likely involving glutamatergic transmission.

<Files\\Loi (2017a)> - § 10 references coded [1,12% Coverage]

Reference 1 - 0,06% Coverage

We therefore set out to study the effects of 24-h MD at
PND 3 in three cognitive domains and on hippocampal structural measures.

Reference 2 - 0,18% Coverage

The peripubertal period is considered a critical time

window in which programming of the brain and HPA axis can be primed or ameliorated, depending on the intervention (Tsoory and Richter-Levin, 2006). We tested the latter possibility, by treating ELS and control female rats with the glucocorticoid receptor antagonist mifepristone (MIF) twice daily during PND 26–28.

Reference 3 - 0,20% Coverage

Given that earlylife adversity is a common denominator in the vulnerability to mood- and anxiety disorders and that many of these disorders are more prevalent in females, we expected to see clear behavioral effects in this severe model of early-life adversity, and wondered whether this could possibly be ameliorated by a brief intervention with the powerful glucocorticoid receptor antagonist MIF.

Reference 4 - 0,04% Coverage

We therefore have no reason to doubt the effectiveness of the early-life intervention.

Reference 5 - 0,08% Coverage

The main outcome of the study is that 24-h MD at
PND3 does not seem to change performance in any of the behavioral tasks to which the female rats were subjected.

Reference 6 - 0,15% Coverage

In males, the effects of corticosteroids seem to affect particularly the later stage of the task, when animals switch from an exploratory phase that involves affective and motivational circuits, to a cognitive control loop that depends (among other things) on prefrontal areas (De Visser et al., 2011).

Reference 7 - 0,05% Coverage

Third, while female (non-MD) rats were able to learn the ORT, we observed no consistent effects of MD or MIF.

Reference 8 - 0,16% Coverage

While the numbers we found here were largely in line with studies on rats of related ages (Oomen et al., 2010, 2011; Klomp et al., 2014), MD did not affect neurogenesis in female rats 17 weeks of age, indicating that the decreased neurogenesis found earlier in MD females at PND 21 (Oomen et al., 2009) does not last into later ages.

Reference 9 - 0,05% Coverage

Lack of any effect of MD on the behavioral paradigms
that we applied came somewhat as a surprise to us.

Reference 10 - 0,14% Coverage

Our own data and the overview of existing
rodent literature reveal that particularly non-stressful (hippocampus-dependent) learning tasks are less vulnerable to early-life adversity in female than male subjects, which is interesting in the light of several recent studies in humans.

<Files\\Loi, Koricka, Lucassen, & Joëls (2014) [1]> - § 1 reference coded [0,18% Coverage]

Reference 1 - 0,18% Coverage

In this paper, we will highlight the effects of the early life environment on hippocampal neurogenesis (see below), focusing on stress experienced just prior to, or during, the first 2 postnatal weeks.

<Files\\Loi, Koricka, Lucassen, & Joëls (2014) [2]> - § 5 references coded [1,08% Coverage]

Reference 1 - 0,18% Coverage

In this paper, we will highlight the effects of the early life environment on hippocampal neurogenesis (see below), focusing on stress experienced just prior to, or during, the first 2 postnatal weeks.

Reference 2 - 0,21% Coverage

If corticosteroid over-exposure is indeed an essential step in
the cascade, one would expect to see beneficial effects of treatment with pharmacological agents that block the GR, i.e., the receptor most prominently activated after stress.

Reference 3 - 0,27% Coverage

As shown in Figure 4, the number ofKi67-positive cells was sig-
nificantlyhigher in the hilus (but not in theDGas awhole, data not shown) in MD rats treated with mifepristone compared to those treated with vehicle, whereas the drug did not affect the number of Ki67-positive cells in non-deprived rats.

Reference 4 - 0,27% Coverage

Similarly, mifepristone treatment tended to cause higher levels of DCX-positive cells in the dentate supra-pyramidal blade of MD rats compared to vehicle treated MD controls, although this did not reach significance ($p = 0.08$); mifepristone did not affect the number of DCX-positive cells in non-deprived rats.

Reference 5 - 0,16% Coverage

Nevertheless, the results are generally in line with earlier findings that brief treatment with the GR-antagonist mifepristone can quickly normalize effects of stress on neurogenesis (77).

<Files\\Maciejewski et al. (2019)> - § 1 reference coded [0,08% Coverage]

Reference 1 - 0,08% Coverage

This suggests that low mood variability may be protective against psychopathology by being related to decreasing depressive and delinquency symptoms.

<Files\\Mastrotheodoros, Van der Graaff, Dekovic, Meeus, & Branje (2019b)> - § 5 references coded [0,92% Coverage]

Reference 1 - 0,10% Coverage

Therefore, this study investigated the order of effects between interparental conflict and parent–adolescent relationships across adolescence.

Reference 2 - 0,20% Coverage

Therefore, in this study we applied a six-wave longitudinal and multi-informant design that spans across adolescence to examine the longitudinal effects of interparental conflict resolution on parent–adolescent relationship quality, according to mother, father, and adolescent reports.

Reference 3 - 0,14% Coverage

First, we applied random-intercept cross-lagged panel models (Hamaker et al., 2015; Keijsers, 2016), which allow investigating the order of effects and distinguishing between-person and within-person effects.

Reference 4 - 0,32% Coverage

However, the few significant effects on the within-person level were in support of the spillover hypothesis, which posits that conflict
in one family subsystem might lead to conflict in another family subsystem. These within-person effects were found only for fathers, echoing recent studies that fathers are more spillover prone than mothers (e.g., Chung et al., 2009; Elam, Chassin, Eisenberg, & Spinrad, 2017; Kouros, Papp, Goeke-Morey, &Mark, 2014).

Reference 5 - 0,17% Coverage

In this study we found that for the most part, whether parents increase or decrease their conflict management strategies they apply in conflicts with their spouses does not induce changes in the quality of parent–adolescent relationship.

<Files\\Moorman, Gobes, Van de Kamp, Zandbergen, & Bolhuis (2015)> - § 1 reference coded [0,16% Coverage]

Reference 1 - 0,16% Coverage

We hypothesized that playbacks of the target song (tutor song) during the day would trigger memory consolidation, which might occur during sleep.

<Files\\Nelemans et al. (2014)> - § 3 references coded [0,87% Coverage]

Reference 1 - 0,32% Coverage

Regarding adolescent depressive symptomatology, our
results on the longitudinal association between persistent heightened CAR and adolescent depressive symptoms, which remained significant even after controlling for sex as a covariate, provide further evidence for the potentially important role of adrenal cortisol secretory activity in the neurobiology of adolescent depression.

Reference 2 - 0,20% Coverage

These findings suggest that CAR, and thereby potentially certain aspects of the human physiological stress response system, may play a more prominent and consistent role in the development of depressive symptoms than anxiety disorder symptoms.

Reference 3 - 0,34% Coverage

A related implication of these findings is that although persistent heightened CAR appeared to be specifically associated with higher levels of PD and SepAD symptoms and not with GAD and SAD symptoms, suggesting potential differences in stress activation may underlie different anxiety disorder symptoms, sex appeared to play a more important role in the development of all anxiety disorder symptoms than CAR.

<Files\\Nelemans et al. (2016)> - § 1 reference coded [0,23% Coverage]

Reference 1 - 0,23% Coverage

For example, our results also suggested that adolescents who do not share their father's negative view of the parent–adolescent relationship are somewhat ''protected'' with respect to depressive symptoms (in line with findings regarding adolescent and parent reports of the family in association with adolescent anxiety; Ohannessian and De Los Reyes 2014).

<Files\\Nelemans, Hale III Branje, Meeus, & Rudolph (2018)> - § 7 references coded [0,99% Coverage]

Reference 1 - 0,08% Coverage

The goals of this study were (a) to examine the impact of
the middle school transition on the continuity versus discontinuity of anxiety over time,

Reference 2 - 0,08% Coverage

Hence, the first goal of this study was to examine the impact of the middle school transition on anxiety trajectories from childhood to adolescence.

Reference 3 - 0,10% Coverage

First, we aimed to examine the impact ofthe middle school transition on developmental trajectories of anxiety symptoms from second to eighth grade (middle childhood to middle adolescence).

Reference 4 - 0,16% Coverage

Rather, the middle school transition appeared to be a turning point (Graber & Brooks-Gunn, 1996; Rutter, 1996; Seidman & French, 2004), reflecting a time of environmental stress or opportunity (as suggested by Shell et al., 2014) that could alter anxiety trajectories for better or for worse in some youth.

Reference 5 - 0,25% Coverage

For this minority of youth, the middle school transition appeared to have a substantial impact on their anxiety trajectories, considering that after fifth grade youth in the increasing after transition anxiety trajectory experienced an average increase in anxiety symptoms of 8.9 points on a 28-point scale (32%) and youth in the decreasing after transition anxiety trajectory experienced an average decrease in anxiety symptoms of 9.4 points on a 28-point scale (34%).

Reference 6 - 0,18% Coverage

Therefore, even though these youth started out with higher anxiety symptoms, they were confronted with few transition-related stressors along with high support from their friends and parents. For these youth, the middle school transition appeared to create opportunities for better adjustment, allowing them to recover across this transition.

Reference 7 - 0,13% Coverage

This complex interplay resulted in continuity or discontinuity in certain anxiety trajectories, with contextual factors after the middle school transition seemingly impacting anxiety trajectories of some youth in a for-better or for-worse manner.

<Files\\Nelemans, Hale III, Branje, Hawk, & Meeus (2014)> - § 26 references coded [5,69% Coverage]

Reference 1 - 0,08% Coverage

Therefore, in this study we aimed to examine the direction of effects between parental criticism and

Reference 2 - 0,18% Coverage

In the present study, we therefore examined whether adolescent perceptions of parental criticism mediated the potential bidirectional associations between mother-reported criticism and adolescents' anxiety and depressive symptoms over time.

Reference 3 - 0,09% Coverage

In the present 6-year longitudinal community study, we aimed to examine the direction of effects (i.e., parent effects, child

Reference 4 - 0,15% Coverage

effects, or reciprocal effects) between maternal criticism and adolescent depressive and GAD symptoms, including adolescents' perceptions of criticism as a potential mediator in the associations.

Reference 5 - 0,09% Coverage

In a reciprocal effects model, we would expect to find both of the aforementioned parent and child effects over time.

Reference 6 - 0,13% Coverage

Secondly, we hypothesized that the longitudinal association
between maternal criticism and adolescent depression/GAD wouldbemediatedbyadolescents' perceptions of criticism.

Reference 7 - 0,54% Coverage

In a parent effects model, mediation ofadolescent perceived EE in the longitudinal association from maternal EE to adolescent depression/GAD would suggest that maternal criticism negatively affects adolescents' perceptions of the emotional climate or mother-adolescent interaction patterns, which in turn affect adolescents' depressive and GAD symptoms over time. In contrast, mediation of adolescent perceived EE in a child effects model would suggest that adolescents' depressive and GAD symptoms affect their perceptions of the emotional climate or mother-adolescent interaction patterns, which in turn affect maternal criticism. In a reciprocal effects model, both of the aforementioned processes maybetakingplace.

Reference 8 - 0,38% Coverage

Finally, based on recent studies that have found stronger
support for child effects than parent effects in associations between different aspects ofparenting and adolescent anxiety and depressive symptoms (e.g., Branje et al. 2010; Van Eijck et al. 2012; Wijsbroek et al. 2011), we also hypothesized stronger longitudinal child effects from adolescents' depression and GAD to maternal criticism, compared to the longitudinal parent effects from maternal criticism to adolescents' depression and GAD.

Reference 9 - 0,23% Coverage

Our results provide preliminary support for a reciprocal effects model (i.e., both parent effects and child effects) with respect to maternal criticism and adolescent depressive symptoms, mediated by adolescent perceived criticism (Fig. 1). As expected, we found stronger child effects mediation than parent

Reference 10 - 0,02% Coverage

effects mediation.

Reference 11 - 0,15% Coverage

Furthermore, our results provide preliminary support for a child effects model only with respect to maternal criticism and adolescent GAD symptoms, via adolescent perceived criticism (Fig. 2).

Reference 12 - 0,12% Coverage

In other words, adolescent GAD symptoms were predictive of later maternal criticism and this association was mediated by adolescent perceived criticism.

Reference 13 - 0,19% Coverage

Overall, our results suggest stronger support for child effects than parent effects in the longitudinal associations between maternal criticism, adolescent perceived criticism, and adolescent symptoms of depression and GAD from early to late adolescence.

Reference 14 - 0,14% Coverage

In our study, we found stronger support for child effects than
parent effects in the longitudinal associations between maternal criticism and both adolescent depression and GAD.

Reference 15 - 0,18% Coverage

In addition, our results further emphasize the importance of examining potential child effects in addition to parent effects in associations between aspects of the parent– child relationship and adolescent psychopathological symptoms.

Reference 16 - 0,11% Coverage

while at the same time adolescent depressive symptoms indirectly reinforce a later negative emotional climate between parents and their adolescents.

Reference 17 - 0,12% Coverage

The results seem to suggest a slightly different pattern with respect to adolescent GAD symptoms, where adolescent symptoms mainly appear to deteriorate the

Reference 18 - 0,47% Coverage

These results could have potential clinical implications, but we should be cautious in discussing these because this study
was conducted in a community sample. Even though the term “child effects” may seem to imply that adolescents themselves should be the focus of intervention practices, adolescents’ anxiety and depressive symptoms appear to have an important negative effect on the parent-adolescent relationship. Therefore, including parents in interventions for adolescent problems of anxiety and depression may be recommended to ameliorate both negative family dynamics and adolescent anxiety and depressive symptoms.

Reference 19 - 0,23% Coverage

In line with previous reasoning (Millikan et al. 2002;Powers et al. 1994; Rohner et al. 2005; Yahav 2007), our results suggest that adolescents’ perceptions of parental criticism are a critical mediating variable in associations between maternal criticism and adolescent symptoms of depression and GAD.

Reference 20 - 0,24% Coverage

For adolescent depressive symptoms, this suggests that adolescents’ perceptions of the parenting they receive are important to consider, as adolescents’ subjective experiences of criticism appeared to have a stronger influence on their symptoms of depression than the criticism they received according to their mothers.

Reference 21 - 0,23% Coverage

However, since we found stronger support for child effects

than for parent effects in associations between maternal criticism and both adolescent depressive and GAD symptoms, this also points towards a critical role of adolescents' subjective experiences ofcriticism in mothers' views oftheir own criticism.

Reference 22 - 0,31% Coverage

In addition, we may have to consider this longitudinal
prediction of maternal self-reported criticism by adolescent perceived criticism less from a parent or child effects model, and instead more with respect to entire estimated models (i.e., top part of Figs. 1 and 2), as there appeared to be an interesting dynamic interplay between maternal criticism and adolescents' perceived criticism over time.

Reference 23 - 0,24% Coverage

This continuous reinforcement ofthe negative mother-adolescent emotional climate or interaction pattern over time, fueled by adolescent symptoms ofdepression or GAD, may indicate that a process of"spillover" occurs betweenmothers' and adolescents' (negative) appreciations of the parent-adolescent emotional climate.

Reference 24 - 0,15% Coverage

Most importantly, our results on the mediating role of adolescent perceived criticism suggest that researchers should recognize the importance of adolescents' subjective perceptions of parenting.

Reference 25 - 0,34% Coverage

Concluding, in this 6-year longitudinal community study we found preliminary support for a reciprocal effects model (i.e., both parent effects and child effects) with respect to maternal criticism and adolescent depressive symptoms, and a child effects model with respect to maternal criticism and adolescent GAD symptoms. Furthermore, we found adolescent perceived criticism to act as a significant longitudinal mediator in these associations.

Reference 26 - 0,59% Coverage

Thus we found stronger support for child effects than parent effects in the longitudinal associations between maternal criticism and adolescent internalizing symptoms, including adolescents' perception of parental criticism as a critical mediating variable. This study thereby further emphasizes the importance of examining potential child effects in addition to parent effects in associations between aspects of the parent–child relationship and adolescent psychopathological symptoms, and suggests that higher levels ofmaternal criticism and higher levels of adolescent anxiety and depression do not only co-occur (i.e., a co-occurrence of problems), but also that higher levels of adolescent depressive and GAD symptoms appear to erode the parent–child relationship over time.

<Files\\Neumann et al. (2017)> - § 8 references coded [1,19% Coverage]

Reference 1 - 0,15% Coverage

In the present study, we aimed to quantify the SNP heritability of
cortisol, i.e the variance jointly explained by common autosomal single nucleotide polymorphisms.

Reference 2 - 0,19% Coverage

The low heritability of plasma cortisol in two large samples esti-
mated by two different approaches strongly suggests that plasma cortisol is not substantially affected by the additive effects of autosomal SNPs.

Reference 3 - 0,15% Coverage

The same conclusion can be drawn for morning saliva cortisol, which was also estimated by two analytical approaches, and to a lesser extent for diurnal cortisol.

Reference 4 - 0,08% Coverage

This notion is supported by the low heritability of the diurnal cortisol measurements.

Reference 5 - 0,16% Coverage

This conclusion is further supported by the small effect adjusting for time-of-day had on the plasma cortisol estimates and the low heritability of awakening saliva cortisol.

Reference 6 - 0,14% Coverage

Furthermore, after excluding participants with plasma cortisol measurements after 11am and corticosteroid use, heritability estimates remained under 1%.

Reference 7 - 0,15% Coverage

The negative findings in addition to the convergent evidence from the smaller saliva cortisol samples suggest that acute cortisol measures have low SNP heritability.

Reference 8 - 0,16% Coverage

However, the evidence is less clear for day-time profiles. These were only available in a small sample and have very wide confidence intervals, thus firm conclusions cannot be made.

<Files\\Ormel et al. (2017)> - § 5 references coded [1,33% Coverage]

Reference 1 - 0,36% Coverage

The study reported herein sought not only to replicate the findings of Copeland and colleagues with regard to the relevance of psychiatric history for current functioning, but also to expand those findings by additionally investigating the potential influence of type of disorder (internalizing v. externalizing) and age of onset (childhood v. adolescence).

Reference 2 - 0,25% Coverage

Thus, the present study aimed to examine whether psychiatric history explains variation in functioning at age 19 – the time of entry in adulthood – over and above current mental health status in a representative Dutch population sample of 2230 youths.

Reference 3 - 0,31% Coverage

The study reported herein sought to examine whether psychiatric history, in terms of internalizing v. externalizing disorder and age of onset, explains additional variation in functioning at age 19 – the time of entry in adulthood- over and above current mental health status. The answer is definitely yes.

Reference 4 - 0,18% Coverage

This suggests that at least part of the disadvantage has occurred long before the transition into adulthood and that disorder remission has not resulted in catching up completely.

Reference 5 - 0,23% Coverage

Collectively, the findings strongly stress the need to
improve prevention and treatment of mental disorder in childhood and adolescence, especially ‘generalized’ psychopathology rather than single disorders (Weisz et al. 2005).

<Files\\Pappa et al. (2015)> - § 4 references coded [0,59% Coverage]

Reference 1 - 0,21% Coverage

and perhaps it is this overlap that has a common genetic influence. However, the pathway analysis revealed no significant pathways associated with attachment security.

Reference 2 - 0,05% Coverage

Our findings indicate that attachment

Reference 3 - 0,11% Coverage

disorganization may be partially influenced by multiple genes with small effects.

Reference 4 - 0,22% Coverage

For attachment security, our study showed weaker evidence of genetic influences, indicating distinct neurobiological pathways for attachment disorganization and security.

<Files\\Pas, Munkhof, Plessis, & Vink (2017)> - § 8 references coded [2,63% Coverage]

Reference 1 - 0,36% Coverage

The current study aims to investigate the role of the
striatum during reactive response inhibition, and test whether its activation may in part reflect anticipatory processing triggered by previous contextual cues (Zandbelt and Vink, 2010).

Reference 2 - 0,28% Coverage

With this subjective measurement, we could investigate the effect on proactive inhibition of both an objective stop-signal probability and the participant’s interpretation of these cues.

Reference 3 - 0,20% Coverage

In our current study, we will use the same paradigm to investigate the role of the striatum during the response period of the task.

Reference 4 - 0,31% Coverage

As the striatum is thought to modulate motor cortical responses, we also predict a corresponding diminished motor cortex activation for expected stops, compared to unexpected stops (Zandbelt and Vink, 2010).

Reference 5 - 0,12% Coverage

Our current findings stress the important role of the striatum in expectation.

Reference 6 - 0,50% Coverage

We have demonstrated the involvement of the striatum in forming expectations during proactive inhibition, and its subsequent contribution to reactive inhibition. For the first time we show how this involvement is

exhibited during actual stopping, with greater striatal activation for expected stops when compared to unexpected stops.

Reference 7 - 0,26% Coverage

It suggests that the striatum not only plays a role during the initial phase of assessing probability of inhibition, but continues this involvement during actual stopping.

Reference 8 - 0,60% Coverage

These results allow us to build toward a more complete model of response inhibition, delineating the roles of reactive and proactive processes in the brain. Furthermore, shedding light on the contribution of the striatum to response inhibition allows for a better understanding of psychopathology revolving around striatal dysfunction, such as seen in schizophrenia, impulsivity or addictive behaviors.

<Files\\Rekker, Keijsers, Branje, Koot, & Meeus (2017)> - § 14 references coded [2,80% Coverage]

Reference 1 - 0,18% Coverage

This longitudinal study on Dutch adolescents (age 12e18) therefore examined the interplay of parental monitoring with arguably the most classic risk factor for problem behavior: a low socioeconomic status (Merton, 1968).

Reference 2 - 0,30% Coverage

Specifically, we investigated whether the association of minor delinquency with parental solicitation and control would be more beneficial for low-SES adolescents. Additionally, we examined whether this hypothesized moderation is specific for parents' monitoring efforts, or whether it also applies to adolescent disclosure of information (Stattin & Kerr, 2000).

Reference 3 - 0,08% Coverage

Therefore, we examined monitoring effects before controlling for disclosure in this study.

Reference 4 - 0,28% Coverage

The present studyexamined such a moderating role ofSES within individuals over time. Ifmonitoring is indeedmore important for youthswho are at risk for problembehavior, wemayexpect that especially low-SES adolescents offend less during periods in which their parents solicit and control more than during other periods with less monitoring.

Reference 5 - 0,25% Coverage

In sum, the present study examined the interplay of SES with parental monitoring both between and within individuals.
We hypothesized (H4) that the expected beneficial association ofsolicitation and control with delinquency would be stronger for low-SES adolescents than for high-SES adolescents.

Reference 6 - 0,09% Coverage

Contrarily, we did not expect such a moderating effect of SES for adolescents' disclosure of information.

Reference 7 - 0,13% Coverage

In conclusion, this study examined the interplay between monitoring and SES in predicting delinquency, both between adolescents and within adolescents

Reference 8 - 0,14% Coverage

Finally, we hypothesized that the beneficial association of solicitation and control, but not disclosure, with delinquency would be stronger among low-SES adolescents (H4).

Reference 9 - 0,09% Coverage

Results revealed that this interplay is different within adolescents across time than between adolescents.

Reference 10 - 0,28% Coverage

Within individuals, higher levels of parental control were unexpectedly associated with higher levels of adolescent delinquency, but this relationwas dependent on SES: Low-SES adolescents, but not high-SES adolescents, offended more during periods in which their parents exercised more control than during other periods with less control.

Reference 11 - 0,11% Coverage

Instead, the present study revealed that monitoring interacted with SES only within individuals and in the opposite direction.

Reference 12 - 0,53% Coverage

Despite this uncertainty about the explanatory mechanism, this study's within-individual analyses suggest that moni-
toring could be less effective in low-SES contexts. Although such causal inferences remain speculative without an experimental design, this study's within-individual analyses may come slightly closer to causality than between-individual models by for example controlling for all time-constant third variables (Molenaar & Campbell, 2009; Voelkle et al., 2014). Hence, monitoring could contribute to the relation between SES and delinquency by being both less often exercised and less effective for low-SES adolescents.

Reference 13 - 0,19% Coverage

Despite these limitations, this study challenged the idea that monitoring is more effective in high-risk contexts by
demonstrating a within-individual interaction between parental control and SES in the opposite direction

Reference 14 - 0,14% Coverage

Low-SES adolescents, but not high-SES adolescents, offended more during periods in which their parents exercised more control than during other periods with less control.

<Files\\Richards et al. (2016)> - § 16 references coded [2,03% Coverage]

Reference 1 - 0,18% Coverage

In the present cross-sectional study we set out to investigate possible main and interaction
effects ofcandidate genes and the social environment along with the possible moderating role ofage on brain volumes in a large sample of children, adolescents, and young adults with and without an ADHD diagnosis.

Reference 2 - 0,11% Coverage

In addition, we investigated whether these effects depended on ADHD severity. We aimed to advance previous studies on brain volumes by investigating positive and negative environmental influences.

Reference 3 - 0,06% Coverage

As expected, few (i.e., one) main effect was observed, of deviant peer affiliation on left caudate volume.

Reference 4 - 0,12% Coverage

However, none ofthe current findings were moderated by ADHD severity, suggesting they contribute to total GM, putamen, and caudate volumes in a more general manner, similar for individuals with and without ADHD.

Reference 5 - 0,07% Coverage

These opposite effects are in line with the differential effects ofDAT1 variants we found on total GM versus putamen volumes.

Reference 6 - 0,29% Coverage

Besides different effects ofthe same gene, differential effects ofpositive peer affiliation were found for carriers of specific gene variants as well, i.e., participants scoring low on positive peer affiliation with the DAT1 9-repeat or two HTTLPR long alleles had larger total GM volume, while those with low positive peer affiliation and the DRD4 7-repeat allele had smaller putamen volumes with age, with the opposite pattern found in participants scoring high on positive peer affiliation.

Reference 7 - 0,12% Coverage

What our findings most consistently show is that (for carriers of specific gene variants) the direction of associations between certain social environments and brain volumes depends on developmental stage.

Reference 8 - 0,05% Coverage

Such differential effects were found in each ofthe reported two- and three-way interactions.

Reference 9 - 0,25% Coverage

In agreement, our group has shown that associations between the 5-HTT, DAT1, DRD4 and neurocognitive functioning, such as inhibition and working memory, depended on age as well [82]. These findings highlight the importance ofincluding age when studying genetic and environmental effects on the neural architecture ofchildren, adolescents and young adults, as the direction of associations likely depend on developmental stage.

Reference 10 - 0,04% Coverage

For caudate volume, one developmentally stable main effect was found.

Reference 11 - 0,06% Coverage

All but one ofthe reported effects in the current study were found in relation to peer affiliation.

Reference 12 - 0,12% Coverage

Besides peer presence and peer verbal abuse investigated in the aforementioned studies [45, 92, 93], our results indicate that the type of peers seems to be relevant for structural brain differences.

Reference 13 - 0,10% Coverage

In conclusion, beside a main effect ofdeviant peer affiliation on caudate volume, we found multiple developmentally sensitive GxE effects on total GM and putamen volume.

Reference 14 - 0,10% Coverage

Despite previously reported differences in total GM, caudate, and putamen volumes between individuals with ADHD and healthy controls, our results were independent ofADHD severity.

Reference 15 - 0,20% Coverage

Both children, adolescents, and young adults with and without ADHD showed differential sensitivity to environmental influences, depending on genotype and age. This suggests that interactions between genes and the social environment contribute in a general way to the included cortical and subcortical brain volumes and are not specific for ADHD.

Reference 16 - 0,15% Coverage

While it is clear that our complex findings are in need ofreplication, our results stress the importance ofa developmentally sensitive approach when investigating genetic and social environmental influences on interindividual brain volume variability.

<Files\\Richards et al. (2019)> - § 12 references coded [3,00% Coverage]

Reference 1 - 0,12% Coverage

We expanded upon prior research by (a) not only considering long-term main effects, but also the interplay between family and peer environments,

Reference 2 - 0,14% Coverage

To our knowledge, this is one of the first studies to focus on the long-term effects of cross-context interactions on multidimensional functioning in young adulthood.

Reference 3 - 0,24% Coverage

Our findings are in agreement with the McMaster model of family functioning, which considers affective responsiveness and involvement as core functions of families; with inappropriate responses and involvement viewed as detrimental for a child's development (Epstein et al., 1978).

Reference 4 - 0,58% Coverage

In line with ecological and social systems theories, we found
that the effects of peer contexts can depend on the family context and vice versa (Bronfenbrenner, 1979; Hartup, 1989). For example, the negative effect of peer fighting was absent in case of high parental control or low parental rejection. In addition, the negative effect of parental overprotection was no longer significant at high levels of peer affection. This suggests both family and peer contexts may function as 'buffers', in line with previous studies on adolescent functioning (Gauze et al., 1996; Lansford et al., 2003; Gaertner et al., 2010; Sentse et al., 2010; Véronneau and Dishion, 2010; Trudeau et al., 2012).

Reference 5 - 0,67% Coverage

Finally, we found that the positive effect of peer status was only
present in the absence of negative family experiences, including low parental rejection and family dysfunction. As such the positive effect of peer status does not appear to be able to compete with negative family experiences. Together these findings suggest that family and peer experiences can interact in different ways. Risk factors in one context can be exacerbated by negative experiences or buffered by positive experiences in the other context, and protective factors in one context can be overshadowed by negative experiences in the other context. Furthermore, interactions between parent and peer contexts appear to be circumscribed, that is, holding for some aspects of family and peer experiences, but not for others.

Reference 6 - 0,10% Coverage

When comparing the relative contribution of family and peers
on future functioning, we primarily found family effects.

Reference 7 - 0,12% Coverage

As such, our findings suggest that adolescent family experiences may be more important for future functioning than adolescent peer relations.

Reference 8 - 0,09% Coverage

In agreement with the latter, our findings stress the role of family experiences at both ages 11 and 16.

Reference 9 - 0,25% Coverage

Most effects appeared similar for both sides of functioning. However, effects of parental warmth and parental problem solving – the only two positively framed family experiences – seemed to be specific for positive functioning, while parental anger specifically predicted negative functioning.

Reference 10 - 0,25% Coverage

In all, our findings are in line with the idea that mental health problems and wellbeing can have both overlapping and distinctive correlates (e.g. Kinderman et al., 2015; Patalay and Fitzsimons, 2016), and suggests that targeting predictors of one domain shall not necessarily benefit the other.

Reference 11 - 0,20% Coverage

The findings further suggest that certain family and peer experiences are interdependent in their prediction of later functioning, which highlights the importance of considering the relative effects of one context in relation to the other.

Reference 12 - 0,24% Coverage

Nevertheless, despite a comprehensive assessment, adolescent family and peer experiences played a modest role in predicting young adult functioning. Thus, having negative family or peer experiences during adolescence does not necessarily mean one will function worse in young adulthood.

<Files\\Riem et al. (2017)> - § 17 references coded [2,82% Coverage]

Reference 1 - 0,18% Coverage

In the current studies, we examine whether massage stimulates endogenous oxytocin release in men and women and whether enhanced oxytocin levels after massage affect responses to infant crying and laughter.

Reference 2 - 0,36% Coverage

The current study is the first to examine whether massage stimulates endogenous oxytocin release through mechanically-delivered massage in a standardized setting. First, we examine whether massage applied by a massage seat cover influences salivary oxytocin levels in a pilot study with N= 20 healthy female participants (Study 1). We expected that massage stimulates endogenous oxytocin release and results in

Reference 3 - 0,29% Coverage

In Study 2, we examine whether effects of massage in N= 46 healthy male participants depend on experiences of emotional maltreatment. In addition, we examine whether enhanced oxytocin levels after massage affect the use of excessive handgrip force in response to infant crying and laughter as measured with a handgrip dynamometer.

Reference 4 - 0,19% Coverage

We expected that enhanced oxytocin levels after massage are related to decreased handgrip force in response to infant signals, but that oxytocin-releasing effects of massage are influenced by emotional maltreatment.

Reference 5 - 0,13% Coverage

We found some evidence that, in females, massage applied by the seat cover increased salivary oxytocin levels compared to the control condition.

Reference 6 - 0,08% Coverage

Thus, human touch seems not an essential component in the oxytocin enhancing effects of massage.

Reference 7 - 0,15% Coverage

Compared to a control condition during which participants sat on a comfortable chair without massage, we found that the massage affected oxytocin levels in both men and women.

Reference 8 - 0,24% Coverage

Our finding extends previous studies investigating the effects of massage by showing that massage without human touch results in elevated oxytocin levels compared to a control condition and that human touch is not an essential component in the oxytocin enhancing effects of massage.

Reference 9 - 0,12% Coverage

Together, these findings may indicate that massage promotes a more sensitive response to infant signals by stimulating oxytocin release.

Reference 10 - 0,11% Coverage

Thus, our findings point to increased oxytocin release as one of the mechanism underlying sensitivity enhancing effects of massage.

Reference 11 - 0,14% Coverage

The current study indicates that the stimulation of endogenous oxytocin release through massage may be another way of promoting positive parent-infant interactions.

Reference 12 - 0,07% Coverage

Our findings may have implications for the development of par-
enting interventions.

Reference 13 - 0,09% Coverage

However, stimulation of endogenous oxytocin release might improve troubled parent-child relationships.

Reference 14 - 0,18% Coverage

Thus, individuals with negative childhood experiences do not only show abnormal sensitivity to exogenous oxytocin administration, but also a different response to stimulation of endogenous oxytocin release.

Reference 15 - 0,20% Coverage

Our findings seem to indicate that massage
0.06 −0.23
−0.01 0.18
0.01 0.26
−0.47* 0.12
0.14 −0.09 0.06 −0.26 SE ß delta R2 0.18*
−0.02 0.05
0.01 0.27
−0.48* 0.03
0.05 Handgrip Ratio Crying B SE ß delta R2 0.14†

Reference 16 - 0,11% Coverage

stimulates oxytocin release and that enhanced oxytocin levels after massage promote a more sensitive response to infant signals.

Reference 17 - 0,18% Coverage

Although massage did not increase oxytocin levels in men with experiences of emotional maltreatment, it normalized excessive handgrip force in response to infant crying and laughter in these individuals.

<Files\\Riem, Alink, Out, Van IJzendoorn, & Bakermans-Kranenburg (2015)> - § 11 references coded [1,62% Coverage]

Reference 1 - 0,09% Coverage

The first study examines the effects of maltreatment experiences reported during the Adult Attachment Interview (AAI) on hippocampal volume in female twin pairs.

Reference 2 - 0,08% Coverage

In addition, we explore the moderating influence of time after exposure to the abuse, the timing of the abuse, and the severity of abuse.

Reference 3 - 0,15% Coverage

We expect that the association of child maltreatment with hippocampal volume is stronger in adults compared to children, and we expect stronger associations when the maltreatment occurred early in childhood and when individuals experienced multiple types of maltreatment.

Reference 4 - 0,11% Coverage

These findings indicate that stress can induce laterality
changes in the hippocampus, possibly because of neuroanatomical and neurochemical asymmetries in the hippocampus (Zach et al., 2010).

Reference 5 - 0,06% Coverage

In the empirical study, we found a reduction of hippocampal volume as a function of childhood maltreatment.

Reference 6 - 0,18% Coverage

In line with the empirical study, our meta-analysis confirmed that experiences of childhood maltreatment are associated with a reduction in hippocampal volume and that the associations with maltreatment are more pronounced when the maltreatment occurred in middle childhood compared to early or late childhood and adolescence.

Reference 7 - 0,32% Coverage

In the current meta-analysis, we found that the effects of
maltreatment were more pronounced in individuals with maltreatment experiences at ages 0 to 12 years compared to individuals with maltreatment experiences at ages 0 to 5 years, ages 7to18years,and before age18years.Thisindicates that the association between maltreatment and hippocampal volume reduction is stronger when the maltreatment occurred in middle childhood, which is not consistent with the suggestion the hippocampus has a relatively early sensitive period (Andersen et al., 2008; Rao et al., 2010).

Reference 8 - 0,15% Coverage

Although the contrast between these combined effect sizes was not significant, the difference between children and adults is consistent with the suggestion that the effects of childhood maltreatment on hippocampal development may be delayed (Teicher & Samson, 2013).

Reference 9 - 0,35% Coverage

In addition to time after exposure to maltreatment, our meta-analysis shows that changes in hippocampal volume are dependent on the severity of the maltreatment. We found that, in particular, the combination of multiple types of abuse affects hippocampal volume. No significant influences on hippocampal volume were found for experiences of solely sexual abuse, emotional abuse, or institutionalized deprivation. Although the contrast between the effect sizes was not significant, these results seem to indicate that the combination of multiple types of abuse is most severe, leading to profound hippocampal changes.

Reference 10 - 0,10% Coverage

In addition, we found that the neurobiological effects of maltreatment are most profound when maltreatment was reported to have occurred in middle childhood, rather than in early

Reference 11 - 0,02% Coverage

childhood or adolescence.

<Files\\Riem, Van IJzendoorn, De Carli, Vingerhoets, & Baermans-Kranenburg (2017) [1]> - § 7 references coded [5,34% Coverage]

Reference 1 - 0,35% Coverage

The current fMRI study aims to connect these subfields and examines differential neural processing of infant versus adult tears.

Reference 2 - 0,58% Coverage

Because of their affective relevance and salience, tears seem to evoke strong responses in the visual cortex, pointing to the important role of tears in the communication of distress (Rottenberg & Vingerhoets, 2012).

Reference 3 - 0,19% Coverage

infant tears elicited more activity in visual regions than adult tears.

Reference 4 - 0,79% Coverage

our study indicates that infant crying is not only an acoustic signal, but that visual aspects are also important for the communication of distress. Infant tears appear to be highly salient even compared to adult tears and this salience may facilitate caregiving responses to infant distress.

Reference 5 - 0,99% Coverage

Compared to adult tears, infant tears elicited more
activity in brain regions involved in mentalizing, including the precuneus and the middle frontal gyrus, and in the pre- and postcentral gyrus, regions that are part of the somatosensory cortex. To our surprise, no activity in response to infant or adult tears was found in the insula or anterior cingulate cortex.

Reference 6 - 0,86% Coverage

Interestingly, our study shows that the preand postcentral gyrus responds to infant tears even in the absence of a somatic cause for the distress. This may indicate that we actually feel the distress of the crying infant and that infant tears evoke more empathic distress than even a tearful crying adult can trigger.

Reference 7 - 1,57% Coverage

In conclusion, our study indicates that infant tears
uniquely activate somatosensory pain regions and trigger more brain activity than adult tears. This may indicate that infant tears elicit feelings of shared distress, which could stimulate actions directed at the elimination of the source of pain. Shedding tears may be a strong means to elicit the parent's sharing of the infant's feelings. This may improve the infant's chance of survival because it ameliorates the aversive impact of the cry acoustics and facilitates a sensitive caregiving response to the distressed infant.

<Files\\Riem, Van IJzendoorn, De Carli, Vingerhoets, & Baermans-Kranenburg (2017) [2]> - § 8 references coded [5,84% Coverage]

Reference 1 - 0,35% Coverage

The current fMRI study aims to connect these subfields and examines differential neural processing of infant versus adult tears.

Reference 2 - 0,51% Coverage

Since infant tears may play a role in the establishment of a bond with the infant, we hypothesize that infant tears elicit more brain activity in empathy related regions than adult tears.

Reference 3 - 0,58% Coverage

Because of their affective relevance and salience, tears seem to evoke strong responses in the visual cortex, pointing to the important role of tears in the communication of distress (Rottenberg & Vingerhoets, 2012).

Reference 4 - 0,19% Coverage

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Reference 5 - 0,79% Coverage

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Reference 8 - 1,57% Coverage

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<Files\\Riem, Van IJzendoorn, De Carli, Vingerhoets, & Baermans-Kranenburg (2017a)> - § 15 references coded [2,11% Coverage]

Reference 1 - 0,12% Coverage

Here, we examine the perception of and responding to infant and
adult tears in individuals with varying childhood caregiving experiences.

Reference 2 - 0,19% Coverage

We examined whether responses to tears were affected by experiences of maternal use of love withdrawal, a disciplinary strategy that involves withholding love and affection when a child misbehaves or fails at a task.

Reference 3 - 0,21% Coverage

We predict (a) that individuals with experiences of maternal love withdrawal show blunted amygdala, insula, and approach-related responses to adult tears because they may experience less empathy and feel less connected with a crying adult,

Reference 4 - 0,08% Coverage

(b) that experiences of maternal love withdrawal do not affect responses to infant tears,

Reference 5 - 0,15% Coverage

(c) that context effects induced by the approach-avoidance task are less pronounced for responses to infant tears than adult tears because of the salience of infant tears.

Reference 6 - 0,13% Coverage

Our study indicates that visual aspects of infant crying do matter in the communication of infant distress and that infant tears are highly salient.

Reference 7 - 0,14% Coverage

Our finding that amygdala and insula reactivity to infant tears are not affected by context and personal characteristics is consistent with this suggestion.

Reference 8 - 0,19% Coverage

No effects of maternal love withdrawal were found for amygdala reactivity to infant tears, and love withdrawal was more strongly related to amygdala reactivity to adult tears than to amygdala reactivity to infant tears.

Reference 9 - 0,12% Coverage

Maternal love withdrawal did not affect behavioral responding to infant or adult tears, as measured with the approach-avoidance task.

Reference 10 - 0,16% Coverage

Furthermore, individuals responded faster to adult tears in the approach condition compared with the avoidance condition, indicating that adult tears facilitate approach behavior.

Reference 11 - 0,15% Coverage

However, the approach condition did not facilitate responding to infant tears: individuals responded fast to infant tears, regardless of approach or avoidance condition.

Reference 12 - 0,10% Coverage

This indicates that love withdrawal also affects the perception of neutral adult faces without signs of distress.

Reference 13 - 0,12% Coverage

Contrary to our expectations, experiences of maternal love with-
drawal did not affect performance during the approach-avoidance task.

Reference 14 - 0,17% Coverage

Thus, although individuals with experiences of love withdrawal showed less amygdala and insula reactivity to adult tears, this did not lead to differences in approach-related responses to tears.

Reference 15 - 0,08% Coverage

In addition, we found that infant tears elicit fast responses that are not affected by context.

<Files\\Rijlaarsdam et al. (2017)> - § 9 references coded [1,74% Coverage]

Reference 1 - 0,18% Coverage

Here we investigate the main and interactive effects of stress exposure, oxytocin receptor (OXTR) rs53576 genotype, and OXTR methylation on child autistic traits.

Reference 2 - 0,22% Coverage

Theobjectiveofthe current study wastoexamine
OXTR rs53576 allele-specific sensitivity for OXTR methylation in relation to (1) prenatal maternal stress exposure, and (2) child autistic traits at age 6.

Reference 3 - 0,23% Coverage

Second, we investigated the extent to which prenatal maternal stress exposure and neonatal OXTR methylation combined either additively or interactively with OXTR rs53576 genotype to influence child autistic traits.

Reference 4 - 0,24% Coverage

Our main finding was that OXTR rs53576 genotype and methylation of the OXTR CpG island contributed interactively, but not additively, to child autistic traits in general and social communication problems in particular.

Reference 5 - 0,14% Coverage

OXTR methylation and OXTR rs53576 genotype were
not interrelated but combined interactively to influence child autistic traits.

Reference 6 - 0,16% Coverage

Interestingly, the observed OXTR methylation 3 OXTR rs53576 genotype interaction did not differ between boys and girls and extended to child PDP.

Reference 7 - 0,21% Coverage

This candidate gene study focused on the specific hypothesis that OXTR rs53576 genotype and methylation of the OXTR CpG island contributed interactively to child autistic traits at age 6.

Reference 8 - 0,21% Coverage

We observed a significant OXTR rs53576 genotype x OXTR methylation interaction in the absence of main effects, suggesting that opposing effects on child social problems cancelled each other out.

Reference 9 - 0,15% Coverage

Indeed, OXTR methylation increased the risk for social problems in OXTR rs53576 G-allele homozygous children but not in Aallele carriers.

<Files\\Rippe et al. (2016)> - § 5 references coded [1,12% Coverage]

Reference 1 - 0,17% Coverage

In the current study we examined the influence of SES and ethnicity on hair cortisol levels in 2484 6year-old children, and we tested hair characteristics as additional covariates or potential confounders.

Reference 2 - 0,44% Coverage

The aim of this paper is to explore the influence of socio-
economic and other demographic variables on hair cortisol levels in a large, multi-ethnic sample of homogeneous age. Furthermore, the influence of various characteristics of hair such as hair color, use of hair products, hair washing, and amount of sun light (in hours) in the month of visit on hair cortisol levels will be examined, in order to present a set of potential confounders to be taken into account in substantive studies on hair cortisol and cortisone.

Reference 3 - 0,21% Coverage

In the current study we examined the influence of SES and other family characteristics, as well as individual characteristics and ethnicity on hair cortisol levels in 2484 6-year-old children, and we tested hair characteristics as potential confounders.

Reference 4 - 0,13% Coverage

In this large cohort study, we identified a number of child and
family characteristics influencing hair cortisol and cortisone levels in 6-year-old children.

Reference 5 - 0,16% Coverage

Hair color is an important potential confounder to account for in substantive analyses, since different pigmentations independently of ethnicity influence cortisol and cortisone levels.

<Files\\Sarabdjitsingh et al. (2014a)> - § 8 references coded [1,69% Coverage]

Reference 1 - 0,37% Coverage

By using high-resolution imaging and electrophysiological approaches, we report that a single pulse of CORT to hippocampal networks causes synaptic enrichment of glutamate receptors and increased responses to spontaneously released glutamatergic vesicles, collectively abrogating the ability to subsequently induce synaptic long-term potentiation.

Reference 2 - 0,22% Coverage

We here examined how glutamatergic transmission in the hippocampus is affected when cells are exposed to a second pulse of CORT 1 h after the first, mimicking the ultradian pattern of endogenous CORT release (6).

Reference 3 - 0,45% Coverage

We show that a second pulse of CORT, hitting the cells at a time
point when the effects of the first pulse have already developed, can fully normalize all investigated aspects of glutamatergic transmission in the hippocampus and restore responsiveness to high-frequency dependent encoding. The system does so by triggering an entirely different signaling pathway after the second pulse than the one involved in the first pulse.

Reference 4 - 0,07% Coverage

The observation that limbic cells respond differently to a sec-

Reference 5 - 0,06% Coverage

ond pulse of CORT than to the first is not unprecedented.

Reference 6 - 0,20% Coverage

Here we show that, in the hippocampus, metaplastic responses can be observed not only at the level of mEPSC amplitude, but also with respect to AMPAR trafficking and synaptic plasticity.

Reference 7 - 0,14% Coverage

In summary, we show that pulsatile exposure to CORT has the potential to maintain glutamatergic transmission in the hippocampal

Reference 8 - 0,17% Coverage

CA1 area at a stable level, preventing a “fixed” situation that may arise after a single pulse.

<Files\\Sarabdjitsingh et al. (2014b)> - § 11 references coded [2,67% Coverage]

Reference 1 - 0,45% Coverage

We therefore tested the hypothesis that pharmacological inhibition of 11b~HSD1 acutely prevents the effect of stress on hippocampal synaptic plasticity and that this is reflected in reduced contextual fear conditioning, a

learning paradigm that is sensitive to circulating levels of glucocorticoid hormones (Pugh et al., 1997a, b; Zhou et al., 2010).

Reference 2 - 0,27% Coverage

Our results show that inhibiting 11b~HSD1 acutely prevents the effects of stress on hippocampal synaptic plasticity and reduces contextual fear conditioning, while leaving cue fear conditioning undisturbed.

Reference 3 - 0,17% Coverage

Our current data suggest that regeneration ofcorticosterone from the inert 11-keto metabolite is a critical step in this process.

Reference 4 - 0,35% Coverage

Regardless ofthe site ofaction, we conclude that a single injection of the 11b~HSD1 inhibitor UE2316 prevents the impairing effect of stress on subsequent hippocampal synaptic plasticity, i.e. prevents the elevation in threshold for synaptic strengthening of novel input.

Reference 5 - 0,07% Coverage

Preventing enzymatic regeneration of corticosterone by

Reference 6 - 0,28% Coverage

administering UE2316 did not significantly affect basal hippocampal transmission, implying that the enzyme (and hence the level of corticosterone under non-stressed conditions) is not crucial for basal transmission.

Reference 7 - 0,17% Coverage

Moreover, administration ofUE2316 did not affect hippocampal synaptic plasticity in mice that were not exposed to novelty stress.

Reference 8 - 0,30% Coverage

Our observation that inhibition of 11b~HSD1 impairs contextual fear conditioning in a mild stimulation paradigm implies that this enzyme and its regeneration of corticosterone are indeed important for contextual fear conditioning.

Reference 9 - 0,20% Coverage

Interestingly, inhibiting 11b~HSD1 did not affect memory for-
mation in a cue fear conditioning paradigm, which critically depends on amygdala function.

Reference 10 - 0,16% Coverage

In summary, we show that a single peripheral injection with the 11b~HSD1 inhibitor UE2316 is sufficient to prevent stress-

Reference 11 - 0,27% Coverage

In anticipation of traumatic events (e.g. in professionals with a high risk on trauma exposure) 11b~HSD1 inhibitors may provide an interesting tool for new strategies aimed at prevention of psychopathology.

<Files\\Sarabdjitsingh, & Joëls (2014)> - § 9 references coded [2,93% Coverage]

Reference 1 - 0,47% Coverage

Therefore, in this study we aimed to examine (1) the rapid ef-
fect of corticosterone on LTP formation in the BLA and (2) whether this is affected by recent stress exposure. Furthermore, we extended previous studies on the role of the b-adrenoceptors in synaptic plasticity. Hereto we studied (3) whether the rapid effects of corticosterone depend on the activation of the b-adrenoceptor under control or stress conditions.

Reference 2 - 0,13% Coverage

In the current study we additionally investigated the effect of restraint stress in the CA1 region of the hippocampus.

Reference 3 - 0,27% Coverage

In the current study we demonstrate that corticosterone can
rapidly modulate and supress synaptic plasticity in the BLA. This suppression and loss of persistence particularly occurs in animals that have recently experienced restraint stress.

Reference 4 - 0,24% Coverage

We found that blocking b-adrenergic transmission with propranolol before stress exposure also results in transient LTP, an effect that is even more pronounced when tissue is subsequently exposed to corticosterone.

Reference 5 - 0,19% Coverage

In line with previous work from our lab (Pu et al., 2009), we first
showed that acute administration of corticosterone can rapidly decrease synaptic potentiation in the BLA.

Reference 6 - 0,62% Coverage

To some extent we did observe different responses to cortico-
sterone regarding LTP when the organism had earlier been exposed to restraint stress. In particular the stability of the LTP evoked in the presence of corticosterone turned out to be sensitive to earlier stress exposure. While a single pulse of corticosterone (i.e. in tissue from non-stressed mice) rapidly attenuated LTP but still yielded considerably elevated synaptic transmission 60 min later, recent exposure to stress gradually led to a much stronger suppression of LTP by corticosterone.

Reference 7 - 0,26% Coverage

Regardless of the mechanism, however, the present results do indicate that BLA cells and circuits respond differently to corticosterone when the organism has recently experienced a stressor, compared to undisturbed control conditions.

Reference 8 - 0,49% Coverage

We here show that such a situation affects LTP in the BLA differently than when corticosterone is given against an undisturbed background, i.e. by reducing the BLA ability to express sustained synaptic plasticity. Our results suggest a protective role for renewed corticosteroid exposure, by resetting BLA excitability and synaptic plasticity and hence preventing the stress response to cause inappropriate or excessive memory consolidation.

Reference 9 - 0,25% Coverage

In conclusion, we have demonstrated that corticosterone and b-
adrenergic transmission are major players in the long-term maintenance and persistence of stress-induced increases in synaptic plasticity in the mouse BLA.

<Files\\Staats, Valk, Meeus, & Branje (2018)> - § 9 references coded [1,77% Coverage]

Reference 1 - 0,01% Coverage

By using a

Reference 2 - 0,18% Coverage

longitudinal design (Cole & Maxwell, 2003), we further explored whether there is (partial) mediation of adolescent–parent conflict management between inter-parental conflict management and adolescent conflict management in relationships with friends and romantic partners.

Reference 3 - 0,13% Coverage

Therefore, we also took into account bidirectional relationships between inter-parental and adolescent–parent conflict management on the one hand and adolescent–friend conflict management on the other hand.

Reference 4 - 0,14% Coverage

Moreover, we examined (3) whether adolescent–parent conflict management mediates the relation between inter-parental conflict management and adolescent conflict management with both friends and romantic partners.

Reference 5 - 0,37% Coverage

For the entire sample, we found that adolescents transmit the conflict management style used in conflicts with parents to their conflicts with friends and romantic partners for positive problem solving and conflict engagement. Although the majority of findings were replicated across the two RADAR subsamples, we found a few differences concerning the transmission of positive problem solving and conflict engagement, the reasons for which remain unclear. Overall, the results of this study underscore the relevance of adolescents' experiences in relationships with parents.

Reference 6 - 0,30% Coverage

In line with our expectations and in agreement with previous findings (Reese-Weber & Bartle-Haring, 1998; Van Doorn et al., 2011), the results of this study indicated spillover (Erel & Burman, 1995; Larson & Almeida, 1999) from adolescent–parent conflict management styles to adolescent–friend and adolescent–partner relationships, in addition to a concurrent relation at Time 1, and correlated changes, between adolescent conflict management with parents and friends.

Reference 7 - 0,17% Coverage

These results suggest that adolescents transmit internal relationship models and conflict management skills learned based on experiences with their parents to their relationships with friends and romantic partners, as proposed by attachment theory (Bowlby, 1969).

Reference 8 - 0,24% Coverage

These results suggest that adolescent interpersonal functioning in relationships with friends and romantic partners is not just a function of modeling relationships observed in the family, and underscore the relevance of experiences in adolescents' own reciprocal relationships with parents for the development of interpersonal skills, such as conflict management skills.

Reference 9 - 0,24% Coverage

As adolescents' conflict management style is prospectively related to their psychosocial and relational functioning, it is important to monitor and address adolescent conflict management in relationships with parents, so that constructive conflict management styles are utilized by adolescents in relationships with parents and in later friendships and romantic relationships.

<Files\\Swagerman et al. (2017)> - § 9 references coded [1,90% Coverage]

Reference 1 - 0,21% Coverage

We fitted a model with A, E, F and D parameters, including cul-
tural transmission to be able to test our primary hypothesis regarding cultural versus genetic transmission.

Reference 2 - 0,08% Coverage

In this study we aimed to test if the family resemblance which

Reference 3 - 0,13% Coverage

has been reported for reading ability and disability is caused by genetic or cultural transmission.

Reference 4 - 0,16% Coverage

We found that individual differences in reading ability were mainly caused by genetic factors, both additive and non-additive.

Reference 5 - 0,24% Coverage

Environmental factors that are shared between parents and children did not contribute to familial resemblance and no evidence was found for cultural transmission from parents to their offspring.

Reference 6 - 0,25% Coverage

From the model-fitting analysis it can be concluded that familial resemblance is caused by genetic factors: the broad sense heritability (variance due to additive + non-additive genetic factors) is 64%.

Reference 7 - 0,27% Coverage

In conclusion, after taking into account the genetic liability that
is passed on from parent to child and assortative mating, there is no additional effect of parental reading ability to offspring reading ability.

Reference 8 - 0,18% Coverage

In the case of children with reading disability, we would advise that interventions should focus on the child's, and not the parents' reading skills.

Reference 9 - 0,38% Coverage

Our results suggest that the precursors for reading disability observed in familial risk studies are caused by genetic, not environmental, liability from parents. That is, having family risk does not reflect experiencing a less favorable literacy environment, but receiving less favorable genetic variants.

<Files\\Teeuw et al. (2019)> - § 16 references coded [2,30% Coverage]

Reference 1 - 0,06% Coverage

We investigated the spatiotemporal dynamics of genetic and environmental influences on cortical thickness.

Reference 2 - 0,09% Coverage

Specifically, we address the question whether different genetic factors influencing cortical thickness at different stages of childhood and adolescent brain development.

Reference 3 - 0,13% Coverage

In addition, we extend on our previous findings on heritability of changes in cortical thickness between age 9 and 12 years (van Soelen et al. 2012b)with new finding on heritability of changes in cortical thickness between age 12 and 17 years.

Reference 4 - 0,18% Coverage

We find a single genetic factor that affects the acceleration of overall cortical thinning across childhood and adolescent development with increasing heritability of changes in mean global cortical thickness: ages 9 and 12 years, and
hΔ =21%12 2
hΔ =53%23 2
(<p
(=p 0.154; [n.s.]) between 0.001) between ages 12
and 17 years.

Reference 5 - 0,27% Coverage

Locally, we again find a core genetic factor influencing cortical thickness and a second genetic factor involved in innovation explaining changes in local cortical thickness during different stages of childhood and adolescent development; areas with the highest estimates include the anterior cingulate cortex (=h 71%2
in cortical thick-
ness change between age 9 and 12 years) and the superior medial frontal cortex (=h 70%2
in cortical thickness change
between age 12 and 17 years).

Reference 6 - 0,25% Coverage

We find moderate to high heritability estimates of cortical thickness for most of the cortex, corroborating the evidence that the cortex is under strong genetic control (Lenroot et al. 2009; Blokland et al. 2012; Schmitt et al. 2014). Estimating the genetic overlap in cortical thickness between lobar regions of the cortex revealed a strong core genetic factor affecting overall cortical thickness across childhood and adolescent development (Fig. 4b).

Reference 7 - 0,07% Coverage

We now add to these findings that the same genetic factor is responsible for cortical thinning during childhood and adolescence

Reference 8 - 0,13% Coverage

Using the longitudinal twin model setup, we show there is a single
genetic factor that dominates across childhood and adolescence which is involved in cortical thickness and cortical thickness change—and thus involved in cortical thinning.

Reference 9 - 0,12% Coverage

Indeed, decomposition of genetic influences on cortical plas-

ticity revealed a second genetic factor, representing genetic innovation, that is influencing cortical thickness during childhood and adolescent development.

Reference 10 - 0,17% Coverage

The areas where genetic innovation occurred were most prominent in the frontal cortex, involving the anterior cingulate cortex (with a heritability of changes in cortical thickness of 71% between age 9 and 12 years) and superior medial frontal cortex (heritability of 70% between age 12 and 17 years).

Reference 11 - 0,20% Coverage

Together, the two genetic factors involved in changes in cortical thickness explain the strong positive correlations between homotopic regions across the hemispheres and across age, evident from the diagonal banding in the correlation matrices (Fig. 4) and as reported from cross-sectional twin studies at both lobar and local level (Chen et al. 2013; Wen et al. 2016).

Reference 12 - 0,10% Coverage

These results suggest that cortical thickness during childhood and adolescence is primarily driven by a core genetic component with secondary regional-specific genetic influences.

Reference 13 - 0,08% Coverage

We found negligible influences of sex, handedness, and age at scan on heritability of (changes) in cortical thickness in qualitative post-hoc analyses.

Reference 14 - 0,21% Coverage

Quantitative evaluation of sex and handedness effects on the means and variance of (changes in) global cortical thickness confirmed the absence of sex or handedness effects. Although sex effects for mean and variance of changes in cortical thickness between ages 12 and 17 years were approaching significance, no discernible effects were observed in the qualitative evaluation.

Reference 15 - 0,16% Coverage

In conclusion, cortical thickness development during child-
hood and adolescence is under strong genetic control and although it is largely driven by a single genetic factor, the influence exerted by this core genetic factor varies with age and its influence seems to decrease towards adulthood.

Reference 16 - 0,08% Coverage

In addition, new genetic factors influence regional cortical thickness development during different stages of childhood and adolescent development.

<Files\\Thijssen et al. (2017)> - § 10 references coded [1,09% Coverage]

Reference 1 - 0,09% Coverage

Here, we examined whether the development of the amygdala–mPFC circuit is modulated by typical variation in parental care.

Reference 2 - 0,12% Coverage

In the present study we found that the association between age and amygdala–mPFC connectivity from age 6 to 10 years was modulated by normal variation in parental care.

Reference 3 - 0,08% Coverage

Our results, therefore, suggest that less sensitive parenting accelerates amygdala–mPFC circuit development.

Reference 4 - 0,17% Coverage

Here, we show that in a population-based sample, variation in caregiving experience modulates the development of amygdala– mPFC coupling, such that lower levels of parental care seem to accelerate amygdala–mPFC circuit development.

Reference 5 - 0,10% Coverage

Our exploratory analyses suggest that parental sensitivity
affects the development of amygdala–mPFC connectivity in daughters only.

Reference 6 - 0,19% Coverage

In the present study, the Sensitivity~Age interaction effect for the combined parental sensitivity measure had a smaller p value (on face value) than the interaction effect for the maternal sensitivity measure, implying that both maternal and paternal sen-

Reference 7 - 0,03% Coverage

sitivity contribute to the observed effect.

Reference 8 - 0,09% Coverage

Moreover, results of the exploratory three-way interaction analysis suggest similar effects for mothers and fathers.

Reference 9 - 0,12% Coverage

Our results, therefore, imply that the quality of parental caregiving is more important for brain development than the sex of the person providing this care.

Reference 10 - 0,10% Coverage

In conclusion, the present population-based study suggests
that parenting quality may moderate the development of amygdala–mPFC coupling.

<Files\\Treur et al. (2018)> - § 14 references coded [4,11% Coverage]

Reference 1 - 0,29% Coverage

The objective of the present study was twofold: to explore the influence of being exposed to smoking in the family environment, over and above genetic factors, and to explore the interaction between such exposure to smoking and genetic factors.

Reference 2 - 0,20% Coverage

By comparing smoking behavior between these four categories of children of twins, we explore the effect of family environment, on top of genetic factors, on smoking.

Reference 3 - 0,12% Coverage

In a second research design, we therefore specify genetic
and environmental factors for smoking.

Reference 4 - 0,23% Coverage

By analyzing the PRS (genetic risk) together with exposure to smoking during childhood (family environment), we test their individual effect on smoking behavior, as well as their interaction.

Reference 5 - 0,56% Coverage

The present study utilizes two research designs to assess genetic and familial environmental influences on smoking: (1) in a CoT design, including 723 children of 712 twins, the influence of genetic factors, environmental factors, and their interaction on smoking initiation are investigated and (2) in 4072 adults, the influence of PRS on smoking, exposure to smoking during childhood, and their interaction on smoking initiation and smoking heaviness are investigated.

Reference 6 - 0,24% Coverage

With a CoT design, there was evidence for familial environmental influence on smoking initiation in addition to genetic risk, while statistical power was too low to test gene environment interaction (G×E).

Reference 7 - 0,26% Coverage

When specifying genetic risk with a PRS and environmental influence with a question on exposure to smoking during childhood, there was also evidence for an added influence of familial environment but no evidence for G×E.

Reference 8 - 0,28% Coverage

For smoking heaviness, there was some evidence for G×E such that PRS for smoking heaviness were only associated with smoking heaviness when participants were exposed to smoking during childhood, not when participants were not exposed.

Reference 9 - 0,38% Coverage

This confirms the effect of genetic influences on smoking initiation but also implies an effect of the shared family environment in addition to genetic risk. Neither of the research designs showed evidence for an interaction between genetic risk and shared family environment in their influence on smoking initiation.

Reference 10 - 0,18% Coverage

We now show that the influence of exposure to smoking during childhood on whether a person becomes a heavy smoker depends on that person’s genetic make-up.

Reference 11 - 0,21% Coverage

These findings corroborate those from the present study in showing that genetic risk for heaviness of smoking can interact with exposure to smoking by parents, siblings, or peers.

Reference 12 - 0,46% Coverage

Overall, this study showed the combined importance of genetic and familial environmental influences on smoking behavior. More specifically, we demonstrated that exposure to smoking in the family home has a unique effect, over and above genetic factors. For smoking heaviness, genetic risk only played a role for those individuals who were previously exposed to smoking during childhood.

Reference 13 - 0,40% Coverage

This could have important implications. It suggests that individuals who have been exposed to smoking in their childhood and who are genetically vulnerable to heavy smoking, are at higher risk of becoming a heavy smoker than are individuals who carry the same genetic risk variants but who were not exposed to smoking during childhood.

Reference 14 - 0,29% Coverage

The group that is genetically susceptible and exposed to smoking should therefore be particularly targeted with preventive measures. It also gives additional incentive to recommending parents not to expose their children to cigarette smoking.

<Files\\Van Bergen et al. (2018)> - § 13 references coded [2,61% Coverage]

Reference 1 - 0,21% Coverage

In the current study, we apply direction of causal-
ity models to infer the causal relations (if any) between reading ability and print exposure in a large sample (N > 11,000) of 7-year-olds.

Reference 2 - 0,28% Coverage

Given the robust association between our traits of interest, the larger impact of environmental differences on print exposure than on reading ability, and the use of latent variables to account for measurement error, direction of causality modelling should work well.

Reference 3 - 0,23% Coverage

We found evidence for a causal influence of reading ability on print exposure, consistent with previous findings from behavioural studies (Aarnoutse & van Leeuwe, 1998; Harlaar et al., 2011; Lepp€anen et al., 2005).

Reference 4 - 0,16% Coverage

Our findings refute the common belief that there is an influence of print exposure on reading ability, or that there are reciprocal influences between them.

Reference 5 - 0,20% Coverage

The finding that reading ability is the driver of
print exposure does not, of course, imply that exposure to print and thus exposure to orthographic forms is irrelevant to learning to read.

Reference 6 - 0,16% Coverage

We demonstrate here that whether children choose to read for themselves depends, in part, on their reading ability, underlining the fact that poor

Reference 7 - 0,19% Coverage

In fact, we found that reading ability accounted for 16% of the variance in print exposure. In addition, other influences, both genetic and shared environmental, are also at play.

Reference 8 - 0,09% Coverage

By studying causality in the natural situation, we demonstrated that reading ability

Reference 9 - 0,03% Coverage

drives print exposure.

Reference 10 - 0,32% Coverage

By using direction of causality modelling, we
extend what is known about the context in which children's reading skills develop. We show that it is a useful technique for understanding individual differences in reading attainment and the factors which determine the enjoyment (or dislike) of reading.

Reference 11 - 0,25% Coverage

We endorse previous findings of a genetic influence on word-level reading and extend this to show that the same genetic factors influence print exposure causatively and this, in turn, depends on additional genetic and environmental factors.

Reference 12 - 0,18% Coverage

We confirmed that individual differences in reading ability were mostly due to genetic differences, while print exposure was equally genetic and environmental in origin.

Reference 13 - 0,30% Coverage

Importantly, we found evidence that children's reading level fuels how much they choose to read – it follows, as practitioners know, that children tend to avoid reading if they find it difficult. Interventions should focus not only on promoting reading skills but also motivation to read.

<Files\\Van Bommel, Van der Giessen, Van der Graaff, Meeus, & Branje (2019)> - § 15 references coded [1,77% Coverage]

Reference 1 - 0,08% Coverage

Therefore, we expect mothers to have a leading role in regulating emotions and driving transitions during conflict interactions.

Reference 2 - 0,09% Coverage

The main aim of this study is to examine the role of the mother in making transitions in emotions during conflict interactions with adolescents.

Reference 3 - 0,06% Coverage

We expect that mothers are more important than adolescents in driving transitions toward positivity.

Reference 4 - 0,07% Coverage

Strongest effects are expected for negativity, since negativity is found to be more contagious than positivity.

Reference 5 - 0,11% Coverage

By examining the moderating role of maternal emotion regulation problems, we aim to identify adaptive and nonadaptive transitions in emotional states during conflict interactions.

Reference 6 - 0,08% Coverage

Therefore, the present study contributes to the literature by focusing on the role of mothers in conflict interactions with adolescents.

Reference 7 - 0,13% Coverage

By performing internal replication analyses, the present study rigorously indicated that mothers play a more important role than adolescents in preventing conflict interactions from becoming rigidly negative.

Reference 8 - 0,10% Coverage

No support was found for mothers high in internalizing problems driving less adaptive interaction patterns compared to mothers low in internalizing problems.

Reference 9 - 0,11% Coverage

However, we did find that adolescents with mothers low in internalizing problems were more likely to reciprocate negativity than adolescents with mothers high in internalizing problems.

Reference 10 - 0,14% Coverage

Overall, our findings underline the importance of mothers' active role in regulating negative emotions into positive emotions because adolescents are more likely to maintain rigid and dysfunctional interaction patterns of negativity.

Reference 11 - 0,11% Coverage

For this reason, mothers should take an active role in resolving conflicts and modeling effective regulation strategies, instead of sitting out the negative moods of their adolescents.

Reference 12 - 0,07% Coverage

while we expected that maternal internalizing problems would mainly influence emotional responses of the mother.

Reference 13 - 0,27% Coverage

To conclude, the present study extended our knowledge about real-time conflict interaction processes between mothers and adolescents (1) by directly testing and showing that mothers have a role distinct from adolescents in driving transitions in emotions and (2) by demonstrating the importance of acknowledging within-person transitions in emotional states in order to get an accurate understanding of real-time interaction processes.

Reference 14 - 0,07% Coverage

we found a possible trend of maternal internalizing problems influencing the reciprocation of negativity by mothers.

Reference 15 - 0,28% Coverage

Perhaps the most important lesson to be learned from this study is that, whereas adolescents tend to maintain negativity, mothers have a leading role in regulating negative emotions toward positive emotions. This indicates that an active role of mothers in regulating conflict interactions with adolescents is desirable: conflicts are more likely to get stuck in maladaptive cycles of negativity if adolescents rather than mothers have to initiate positivity.

<Files\\Van den Boomen, Jonkman, Jaspers-Vlamings, Cousijn, & Kemner (2015)> - § 1 reference coded [0,21% Coverage]

Reference 1 - 0,21% Coverage

We suggest that the development of LSF processing would drive developmental changes in selective processing ofHSF versus LSF until 10 years ofage [7–10].

<Files\\Van der Meulen et al. (2017a)> - § 8 references coded [1,81% Coverage]

Reference 1 - 0,28% Coverage

We tested if incongruent situations in which the peer feedback did not resemble the girls' own perception of the media model (i.e., too thin vs. normal or normal vs. too thin) compared to congruent situations (i.e., too thin vs. too thin and normal vs. normal) would lead to increased activity in the ACC and insula.

Reference 2 - 0,16% Coverage

Therefore, our second study (behavioral study), in a different late adolescent female sample, tested if participants changed their ratings of the models after receiving peer feedback.

Reference 3 - 0,05% Coverage

A third goal was to test for the role ofself-esteem.

Reference 4 - 0,33% Coverage

These findings imply that girls with lower self-esteem are not only more sensitive for body image concerns but are also more responsive toward feedback on media portrayals. Therefore, we expected to find stronger neural activity for incongruent feedback in participants with lower self-reported self-esteem, as well as a larger change in behavior following peer feedback.

Reference 5 - 0,12% Coverage

Finally, we performed an exploratory analysis to the role of body dissatisfaction in the influence of peer feedback on rating media models.

Reference 6 - 0,18% Coverage

Interestingly, this effect was stronger in the ACC when participant ratings of a media model as being of normal body size were followed by peer feedback indicating that the media model is too thin.

Reference 7 - 0,49% Coverage

Finally, in Experiment 1 we found that especially late ado-
lescent girls with lower self-esteem were impacted more by incongruent feedback from peers when directed toward what should be considered normal body shapes, as indicated by associations between neural activity in dmPFC/ACC and bilateral insula, and self-esteem. This effect was independent of their own BMI and mirrors previous findings that girls with lower self-esteem were more affected by peer feedback on ultrathin images compared to those higher in self-esteem (Veldhuis et al., 2014a).

Reference 8 - 0,20% Coverage

Subsequently, the results hold implications for using peer feedback in (media-based) interventions in order to set the young women open to discussion ofwhich body images and sizes are healthy and normal, and which are not.

<Files\\Van der Meulen et al. (2017b)> - § 3 references coded [0,89% Coverage]

Reference 1 - 0,57% Coverage

On a behavioral level we hypothesized that observing social ex-
clusion would lead to prosocial compensating behavior (Riem et al., 2013; van der Meulen et al., 2016; Vrijhof et al., 2016). On a neural level we expected that both experiencing self-exclusion and self-inclusion would result in activity in dACC and bilateral insula (Cacioppo et al., 2013; Dalgleish et al., 2017; Eisenberger et al., 2003; Rotge et al., 2015). Furthermore, we expected that engaging in prosocial compensating behavior would lead to activity in dACC and bilateral insula (Masten et al., 2013, 2010) and TPJ, and NAcc, similar to what has been found in adults (van der Meulen et al., 2016).

Reference 2 - 0,11% Coverage

As expected, the exclusion of a fourth player by two others resulted in increased ball tossing by the participant to the excluded

Reference 3 - 0,21% Coverage

Interestingly, we found no strong evidence for specific neural activity related to prosocial compensating behavior towards the excluded player, but robust evidence was found for neural contributions to feelings of self-inclusion and –exclusion.

<Files\\Van der Meulen Steinbeis, Achterberg, Van IJzendoorn, & Crone (2018)> - § 14 references coded [1,79% Coverage]

Reference 1 - 0,10% Coverage

Therefore it remains an important question whether these processes are more sensitive to genetic or environmental influences. This study therefore had two goals:

Reference 2 - 0,10% Coverage

II) To examine the heritability of social processes in brain regions that are involved in possible self-exclusion, selfinclusion, and prosocial compensating.

Reference 3 - 0,08% Coverage

Therefore we investigated the genetic versus environmental influences on brain activity in middle childhood using a twin design.

Reference 4 - 0,11% Coverage

whereas experiencing inclusion was expected to lead to activation in bilateral insula/ACC (Menon and Uddin, 2010; Seeley et al., 2007) and the striatum (Van der Meulen et al., 2016).

Reference 5 - 0,11% Coverage

Third, we expected that social brain areas (mPFC, precuneus, TPJ and STS) would be activated when acting prosocially (Guroglu et al., 2014; van der Meulen et al., 2016).

Reference 6 - 0,11% Coverage

Finally, we tested the different influences of genetics, shared environment and unique environment on social exclusion sensitivity and prosocial behavior in these brain regions.

Reference 7 - 0,17% Coverage

Therefore, we also tested the effects of genetics, shared environment and unique environment on total brain volume, a brain measure that has shown consistent heritability in adults (for reviews see Batouli et al., 2014; Peper et al., 2007) and children (Teeuw et al., 2018).

Reference 8 - 0,10% Coverage

We therefore expected to observe mainly genetic influences on total brain volume in the current sample (see Teeuw et al., 2018, including 9-year-old children).

Reference 9 - 0,08% Coverage

Analyses of heritability revealed only unique environmental and/or measurement error influences on prosocial behavioral differences.

Reference 10 - 0,14% Coverage

When we investigated heritability of the neural reactions towards
social exclusion, we found that across all ROIs differences in activity were best accounted for by unique environmental factors and measurement error.

Reference 11 - 0,20% Coverage

In the current study, ACE models showed significant genetic contributions in two out of our nine ROIs: We found that differences in activity in IFG and smPFC during the experience of possible self-exclusion were best accounted for by genetic (estimated 29–33%) and unique environmental factors/measurement error.

Reference 12 - 0,11% Coverage

Our control analysis on heritability of total brain volume showed
that differences in total brain volume were accounted for by genetic (86%) and unique environmental factors.

Reference 13 - 0,10% Coverage

The current study builds upon the existing literature by showing
that children show prosocial compensating behavior when they observe social exclusion.

Reference 14 - 0,28% Coverage

Further, although we note that certain conclusions are based on reverse inference, our findings suggest that children experience possible social self-exclusion as a negative event (as indicated by activity in IFG, smPFC and amygdala), inclusion as a positive and salient event (as indicated by striatum and ACC-insula activity) and that prosocial compensating behavior is partly driven by mentalizing capacities (as indicated by activity in PCC).

<Files\\Van Doeselaar, Meeus, Koot, & Branje (2016)> - § 1 reference coded [0,13% Coverage]

Reference 1 - 0,13% Coverage

To disentangle the hypothesized influence effects from these distorting factors, we differentiated between stable and unstable friendships.

<Files\\Van Wijk et al. (2019)> - § 11 references coded [2,21% Coverage]

Reference 1 - 0,19% Coverage

In the current study, we examined the associations among Fear, EC, and resting FA and explored whether temperamental features and FA are influenced by distinct or overlapping genetic and environmental factors in early childhood.

Reference 2 - 0,19% Coverage

Therefore, we investigated the genetic and environmental factors accounting for variation in Fear and EC, as well as in Fear and resting FA, in a sample of 4- to 6-year-old same-sex twins using bivariate behavioral genetic modeling.

Reference 3 - 0,17% Coverage

Because Fear and EC both are related to child temperament and studies have shown associations between the two traits, we tested whether the same genetic and/or environmental factors are involved in Fear and EC.

Reference 4 - 0,24% Coverage

We were specifically interested in estimating genetic and environmental influences in early childhood given that both Fear and EC individual differences are found to be stable from around 3 years of age (Gullone, 2000; Kochanska et al., 2000; Rothbart & Bates, 2006; Tiberio et al., 2016).

Reference 5 - 0,08% Coverage

We examined whether the same genetic and/or environmental factors are involved in Fear and FA.

Reference 6 - 0,12% Coverage

In the current study, we examined the possible modulating role of EC by computing partial correlations between Fear and FA while controlling for EC.

Reference 7 - 0,15% Coverage

Substantial differences between the bivariate correlations (between Fear and FA) and the partial correlations would indicate an influence of EC on the association between Fear and FA.

Reference 8 - 0,34% Coverage

Because previous research has suggested associations between Fear and EC and between Fear and FA (Cole et al., 2016; Fox et al., 2001; Hill-Soderlund & Braungart-Rieker, 2008; Howarth et al., 2016; Kiff et al., 2011; Rothbart, 2011; Schmidt, 2008), we were interested in the extent to which the same and/or different genetic and environmental factors account for variation in these temperamental characteristics.

Reference 9 - 0,19% Coverage

Results showed that individual differences in parent-reported Fear and EC, as well as children's FA, were best explained by genetic factors (for about one quarter) and by unique environmental factors (for about three quarters).

Reference 10 - 0,32% Coverage

In line with previous studies (Anokhin et al., 2006; Clifford et al., 2015; Goldsmith et al., 1997; Lemery-Chalfant et al., 2008; Smit et al., 2007; Van Houtem et al., 2013), we found that Fear and EC and Fear and FA were best explained by genetic and unique environmental factors (AE models). Still, most of the variation between individuals was explained by unique environmental factors.

Reference 11 - 0,24% Coverage

In sum, our findings indicate that individual differences in young children's temperament-related traits are best explained by a combination of genetic factors and unique environmental factors. Unique environmental factors in particular accounted for a large proportion of the variance.

<Files\\Voorthuis, Bakermans-Kranenburg, & Van IJzendoorn (2019)> - § 10 references coded [2,06% Coverage]

Reference 1 - 0,34% Coverage

In the present study we aim to shed more light on individual differences in T reactivity in response to caring for a crying infant simulator. In particular, we examined whether oral contraceptive use, basal cortisol levels and childhood caregiving experiences each or in concert played a role in T reactivity in response to caring for a crying infant simulator.

Reference 2 - 0,47% Coverage

In the present study we tested whether and how basal CORT, basal T, OC use and childhood experiences of maternal love
withdrawal influence T reactivity in response to infant crying. First, we examined T reactivity in response to infant crying and caretaking, and we explored the role of OC use. Second, we examined the role of basal CORT in relation to T reactivity, and we explored the influence of childhood experiences of maternal love withdrawal on T reactivity taking basal CORT into account.

Reference 3 - 0,09% Coverage

Young women showed a decrease in T levels in response to caring for a crying infant simulator.

Reference 4 - 0,18% Coverage

This result supports the theoretical
framework of the Steroid/Peptide Theory of Social Bonds (Van Anders et al., 2011) in that nurturing parental behavior is related to decreasing T levels.

Reference 5 - 0,17% Coverage

In line with earlier findings (Dabbs & De la Rue, 1991; Liening et al., 2010), menstrual cycle did not affect T or CORT levels, and
time of day had an effect on CORT but not on T.

Reference 6 - 0,29% Coverage

When basal CORT was taken into account, a marginally significant interaction between basal CORT and T reactivity was found in participants who did not use OC; participants with high basal CORT tended to show higher initial T levels and a larger decrease of T compared to individuals with low basal CORT.

Reference 7 - 0,08% Coverage

No main or interaction effects were found for participants with oral contraception use.

Reference 8 - 0,08% Coverage

Experiences of love withdrawal did not predict or moderate T reactivity to caregiving.

Reference 9 - 0,25% Coverage

Our results show that not only the levels of T differ between women with and without OC use; the interaction between T and CORT is also significantly different. Thus, when examining T in women, OC use should always be taken into account as a potential moderator.

Reference 10 - 0,12% Coverage

Controlling for contraceptive use, we found some support for a decrease of T in women who had to take care of a crying infant.

<Files\\Werner, Graaff, Meeus, & Branje (2016)> - § 11 references coded [1,98% Coverage]

Reference 1 - 0,09% Coverage

Moreover, (4) effects from adolescents' depressive symptoms on maternal psychological control were examined.

Reference 2 - 0,18% Coverage

It was expected that throughout adolescence, maternal empathic concern and perspective taking expresses itself in mothers' use of psychological control, and is thus indirectly related to adolescents' depressive symptoms.

Reference 3 - 0,06% Coverage

Moreover, adolescent-effects are expected to be present throughout adolescence.

Reference 4 - 0,15% Coverage

This implies that, although girls remain to experience psychological control and depressive symptoms, mothers' psychological control is no longer ofimpact on girls' depressive symptoms.

Reference 5 - 0,08% Coverage

This indirect effect was found for boys throughout adolescence, and for girls until middle adolescence.

Reference 6 - 0,31% Coverage

The absence ofa direct association between mothers' empathic concern and perspective taking, and adolescents' depressive symptoms, suggests that mothers' empathic tendencies are only important for predicting adolescents' depressive symptoms when they are expressed in concrete parenting behaviors such as psychological control, which in turn predict adolescents' depressive symptoms.

Reference 7 - 0,12% Coverage

For boys, adolescent-effects were present throughout adolescence. However, for girls, adolescent-effects were only present in early adolescence.

Reference 8 - 0,21% Coverage

Despite the lack of direct associations between mothers' empathy and adolescents' depressive symptoms, this study is the first to show over a 6-year period that both aspects of mothers' empathy are indeed important in predicting boys' and girls' depressive

Reference 9 - 0,08% Coverage

symptoms in adolescence, with mothers' use ofpsychological control as the underlying mechanism.

Reference 10 - 0,54% Coverage

At the same time, the use of a six wave longitudinal design in this study helps to disentangle the direction ofthese effects, and shows the unique importance ofboys and girls in shaping the parenting they receive across adolescence. Moreover, these findings give important insight in potential factors for intervention when adolescents experience depressive symptoms. Although adolescents' depressive symptoms are predicted bymothers' parenting behaviors, being psychological control, this study shows that both aspects ofmothers'

empathic tendencies precede mothers' psychological control use, thus providing additional information on where to intervene.

Reference 11 - 0,15% Coverage

Additionally, as a community sample was used, the findings provide useful information on factors to address in screening and prevention programs for depressive symptoms in adolescence.

<Files\\Wildeboer et al. (2015) [1]> - § 1 reference coded [0,11% Coverage]

Reference 1 - 0,11% Coverage

In sum, we identified a group of children with pervasive prob-
lem behaviour in early childhood, which places these children at a heightened risk to develop problems later in life.

<Files\\Wildeboer et al. (2015) [2]> - § 1 reference coded [0,11% Coverage]

Reference 1 - 0,11% Coverage

In sum, we identified a group of children with pervasive prob-
lem behaviour in early childhood, which places these children at a heightened risk to develop problems later in life.

<Files\\Wildeboer et al. (2018)> - § 5 references coded [0,73% Coverage]

Reference 1 - 0,10% Coverage

We will focus on middle childhood, as the neurobiological correlates underlying such behavior in children are largely unknown.

Reference 2 - 0,19% Coverage

To study whether variance in donating behavior is not only situationally driven, but also has a neuroanatomical component, we examined brain morphology, more specifically cortical thickness, in relation to donating behavior in middle childhood.

Reference 3 - 0,24% Coverage

Several studies report donating behavior to be largely influenced by situational factors (e.g. Van IJzendoorn & Bakermans-Kranenburg, 2014; Van IJzendoorn et al., 2010), the current results however suggest that part of the variance in donating behavior can be explained by characteristics inherent to the child.

Reference 4 - 0,10% Coverage

This indicates that donating to a charity is not only dependent upon the specifics of the situation, but also on child characteristics.

Reference 5 - 0,11% Coverage

The pertinent effect was found in regions that have previously been associated with social norm compliance and the processing of threats of punishment.

<Files\\Willems et al. (2018)> - § 2 references coded [0,47% Coverage]

Reference 1 - 0,22% Coverage

Next, we looked at the genetic and environmental
sources of individual differences in self-control assessed with the ASCS and estimated the heritability as a function of age, informant, and sex using the classical twin design (Boomsma et al. 2002).

Reference 2 - 0,25% Coverage

This study adds to this line of research by analyzing data from a large group of same-sex and opposite sex twin pairs, collected by the The Netherlands Twin Register (NTR), providing heritability estimates for mother-, father-, teacher- and self-report of self-control, from age 7 to 16.

<Files\\Windhorst et al. (2017)> - § 8 references coded [1,56% Coverage]

Reference 1 - 0,16% Coverage

The aim of the current study is to investigate the influence of maternal harsh parenting on 6-year-old children's hair cortisol levels, a biomarker of chronic stress, and to explore whether this association is moderated by mild perinatal adversity.

Reference 2 - 0,09% Coverage

In the current study, we investigated the effects of harsh parenting
at age 3 on hair cortisol levels at age 6 in a large population-based cohort.

Reference 3 - 0,07% Coverage

In the current study we examined the effects of harsh parenting at
child age 3 on hair cortisol levels at age 6.

Reference 4 - 0,31% Coverage

Our hypothesis was that children with a history of mild perinatal adversity, defined by late prematurity or low birth weight at full term birth (as in Van der KooyHofland et al., 2012), would be more susceptible to the effects of harsh parenting and show dysregulated (either increased or decreased) cortisol levels with increasing levels of maternal harsh parenting. We expected that this association would be smaller or absent for children without a history of perinatal adversity.

Reference 5 - 0,32% Coverage

Consistent with our hypothesis, we found that the association between harsh parenting and hair cortisol levels was moderated by mild perinatal adversity. A negative association between maternal harsh parenting and cortisol levels was found, but only in children with mild perinatal adversity. These children showed lower cortisol levels when they experienced more maternal harsh parenting, and higher cortisol levels in the absence of harsh parenting, compared with children without mild perinatal adversity.

Reference 6 - 0,36% Coverage

As the results do not show main effects of either perinatal adversity or maternal harsh parenting, but do show an interaction in which susceptible individuals are influenced more by negative and positive environmental factors, it illustrates that a characteristic previously considered a risk factor (mild perinatal adversities) is a risk factors in some environments (i.e., unsupportive environments), but may at the same time predispose children with this putative “risk factor” to benefit most from positive environments (i.e., the absence of harsh discipline).

Reference 7 - 0,12% Coverage

. Our results support a differential susceptibility model with mild perinatal adversity being a susceptibility marker, increasing susceptibility to both positive and negative environments.

Reference 8 - 0,13% Coverage

The current study builds on prior evidence that mild perinatal
adversity may act as a susceptibility factor, moderating environmental effects in a “forbetterand forworse” manner (Ellis et al., 2011).

<Files\\Yu et al. (2016) [1]> - § 18 references coded [4,08% Coverage]

Reference 1 - 0,15% Coverage

The current study aimed to examine whether biological characteristics (i.e., morning cortisol activity) moderated the effects of environmental context (i.e., neighbourhood density) on the development of adolescents’ externalizing problem behaviours.

Reference 2 - 0,10% Coverage

The aims of the current study were to examine personenvironment interaction effects in predicting adolescents’ development of externalizing problem behaviours.

Reference 3 - 0,17% Coverage

We first replicated previous research by estimating the main effects of neighbourhood density and CARAUCg on adolescents’ development of adolescent externalizing problem behaviours including aggression and delinquency across a period of four years from middle to late adolescence.

Reference 4 - 0,09% Coverage

Subsequently, we investigated interaction effects between CARAUCg and neighbourhood density on the development of adolescent aggression and delinquency.

Reference 5 - 0,47% Coverage

Our results indicated that neither neighbourhood density nor adolescents’ cortisol activity in general did have main effects on the initial levels and developmental changes of adolescent externalizing problem behaviours. However, there were significant interaction effects between CARAUCg and neighbourhood density in predicting adolescents’ aggression and delinquency over a period of four years. Level of CARAUCg moderated the effects of neighbourhood density on the development of adolescents’ externalizing problem behaviours. These findings provide support for person-environment perspectives and also biological sensitivity theory on the development of adolescent externalizing problem behaviours (Ellis & Boyce, 2011; Magnusson & Stattin, 2006; Susman & Ponirakis, 1997).

Reference 6 - 0,12% Coverage

The lack of main effects of neighbourhood density on aggressive
and delinquent behaviours is consistent with some prior studies (Gillis, 1974; Reijneveld et al., 2010; Wichstrom, Skogen, & Osia, 1996).

Reference 7 - 0,14% Coverage

The current study indicates that in general there were no sig-
nificant effects of CARAUCg on the development (i.e., initial levels and developmental changes) of externalizing problem behaviours across middle to late adolescence.

Reference 8 - 0,19% Coverage

We found significant interaction effects between CARAUCg

and neighbourhood density in predicting adolescent parentreported aggression and delinquency and self-reported delinquency. CARAUCg appeared to be a moderator of the association between neighbourhood density and adolescents' externalizing problem behaviours.

Reference 9 - 0,46% Coverage

The contrastive pattern suggests that high CARAUCg could be a protective factor in a low density environment but a risk factor in a high density environment. In contrast, low CARAUCg could be a protective factor in a high density environment but a vulnerable factor in a low density environment. These findings suggest that neither high nor low CARAUCg is a protective or vulnerable factor as such. Instead, it is more of the match and combination of the level of an individual's CARAUCg and the level of density of a neighbourhood environment that predicts the development of parent-reported externalizing problem behaviours. We also observed interactive effects between CARAUCg and neighbourhood density in predicting adolescent self-reported delinquent behaviours.

Reference 10 - 0,19% Coverage

These findings together provide support for general interactive perspectives and hormones-context interactions theory, claiming that individuals' developmental outcomes are dependent on interactive processes between individuals' characteristics and contextual factors (Magnusson & Stattin, 2006; Susman & Ponirakis, 1997).

Reference 11 - 0,55% Coverage

In general, the interactive patterns between neighbourhood
density and CARAUCg in predicting parental reported delinquency and aggression and self-reported delinquency suggest that high CARAUCg is a sensitivity factor which increases the negative effects of adverse environments (i.e., high density neighbourhood) but enhances the beneficial effects of positive environments (i.e., low density neighbourhood). Hence, these results support the biological sensitivity theory (Ellis & Boyce, 2011). More specifically, these findings indicate that hyper-aroused adolescents might have higher sensitivity towards not only the stresses that could be generated by a high density environment such as overcrowding, noise, impersonal space, or limited accessibility to resources but also the benefits that might be provided in a low density neighbourhood such as physical space, quietness, or availability of resources.

Reference 12 - 0,17% Coverage

In
predicting parent-reported externalizing problem
behaviours, we observed detrimental effects of a combination of a hypo-aroused biological response, namely a low cortisol awaking response, and a low density neighbourhood in predicting adolescents' externalizing problem behaviours.

Reference 13 - 0,11% Coverage

Hence, the interaction effects between CARAUCg and neighbourhood density were not likely to be confounded by any bidirectional effects between the independent and moderating variables.

Reference 14 - 0,11% Coverage

Our study showed significant main effects of SES on aggres-
sive behaviours, with adolescents from low SES families reporting higher levels of parent- and self- reported aggression.

Reference 15 - 0,21% Coverage

However, despite these main effects, the only significant SES by CARAUCg interaction effect was on the slope of self-reported delinquency. These findings suggest that the moderating effects of CARAUCg on the link

between neighbourhood density and externalizing problems cannot be explained by the potential overlap between neighbourhood density and SES.

Reference 16 - 0,28% Coverage

We found significant interaction effects between cortisol reac-
tivity CARAUCi neighbourhood density in predicting adolescents' self-reported delinquent and aggressive behaviours. The interaction pattern suggests that adolescents with a higher CARAUCi (i.e., a bigger change in cortisol levels post-awakening) might be more likely to be negatively influenced by a high density neighbourhood and therefore they develop higher externalizing problem behaviours.

Reference 17 - 0,19% Coverage

Finally, the focus on neighbourhood density rather than general neighbourhood deprivation provides more specific information on the contribution of neighbourhood characteristics on adolescents' externalizing problem behaviours, which is called for in a recent review (Van Vuuren, Reijneveld, Van der Wal, & Verhoeff, 2014).

Reference 18 - 0,38% Coverage

The present study revealed significant interaction effects
between neighbourhood density and CAR in predicting adolescent aggressive and delinquent behaviours. In general it indicates that individuals' biological sensitivity to environmental context plays an important role in moderating the effects of neighbourhood density on adolescents' adjustment. Findings highlight the complexities that contribute to adolescents' externalizing problem behaviours and support the theoretical idea of interactive processes between environmental context and individuals characteristics contributing to adolescents' developmental outcomes.

<Files\\Yu et al. (2016) [2]> - § 19 references coded [4,18% Coverage]

Reference 1 - 0,15% Coverage

The current study aimed to examine whether biological characteristics (i.e., morning cortisol activity) moderated the effects of environmental context (i.e., neighbourhood density) on the development of adolescents' externalizing problem behaviours.

Reference 2 - 0,17% Coverage

We first replicated previous research by estimating the main effects of neighbourhood density and CARAUCg on adolescents' development of adolescent externalizing problem behaviours including aggression and delinquency across a period of four years from middle to late adolescence.

Reference 3 - 0,09% Coverage

Subsequently, we investigated interaction effects between CARAUCg and neighbourhood density on the development of adolescent aggression and delinquency.

Reference 4 - 0,47% Coverage

Our results indicated that neither neighbourhood density nor adolescents' cortisol activity in general did have main effects on the initial levels and developmental changes of adolescent externalizing problem behaviours. However, there were significant interaction effects between CARAUCg and neighbourhood density in predicting adolescents' aggression and delinquency over a period of four years. Level of CARAUCg moderated the effects of neighbourhood density on the development of adolescents' externalizing problem behaviours. These findings provide support for person-environment perspectives and also biological sensitivity theory on the development of adolescent externalizing problem behaviours (Ellis & Boyce, 2011; Magnusson & Stattin, 2006; Susman & Ponirakis, 1997).

Reference 5 - 0,12% Coverage

The lack of main effects of neighbourhood density on aggressive
and delinquent behaviours is consistent with some prior studies (Gillis, 1974; Reijneveld et al., 2010; Wichstrom, Skogen, & Osia, 1996).

Reference 6 - 0,14% Coverage

The current study indicates that in general there were no sig-
nificant effects of CARAUCg on the development (i.e., initial levels and developmental changes) of externalizing problem behaviours across middle to late adolescence.

Reference 7 - 0,19% Coverage

We found significant interaction effects between CARAUCg
and neighbourhood density in predicting adolescent parentreported aggression and delinquency and self-reported delinquency. CARAUCg appeared to be a moderator of the association between neighbourhood density and adolescents' externalizing problem behaviours.

Reference 8 - 0,38% Coverage

The contrastive pattern suggests that high CARAUCg could be a protective factor in a low density environment but a risk factor in a high density environment. In contrast, low CARAUCg could be a protective factor in a high density environment but a vulnerable factor in a low density environment. These findings suggest that neither high nor low CARAUCg is a protective or vulnerable factor as such. Instead, it is more of the match and combination of the level of an individual's CARAUCg and the level of density of a neighbourhood environment that predicts the development of parent-reported externalizing problem behaviours.

Reference 9 - 0,19% Coverage

These findings together provide support for general interactive perspectives and hormones-context interactions theory, claiming that individuals' developmental outcomes are dependent on interactive processes between individuals' characteristics and contextual factors (Magnusson & Stattin, 2006; Susman & Ponirakis, 1997).

Reference 10 - 0,55% Coverage

In general, the interactive patterns between neighbourhood
density and CARAUCg in predicting parental reported delinquency and aggression and self-reported delinquency suggest that high CARAUCg is a sensitivity factor which increases the negative effects of adverse environments (i.e., high density neighbourhood) but enhances the beneficial effects of positive environments (i.e., low density neighbourhood). Hence, these results support the biological sensitivity theory (Ellis & Boyce, 2011). More specifically, these findings indicate that hyper-aroused adolescents might have higher sensitivity towards not only the stresses that could be generated by a high density environment such as overcrowding, noise, impersonal space, or limited accessibility to resources but also the benefits that might be provided in a low density neighbourhood such as physical space, quietness, or availability of resources.

Reference 11 - 0,11% Coverage

In sum, findings corroborate a conceptualization of stress activity as biological sensitivity to context by showing that high CARAUCg can both hinder and promote adaptive functioning.

Reference 12 - 0,17% Coverage

In
predicting parent-reported externalizing problem

behaviours, we observed detrimental effects of a combination of a hypo-aroused biological response, namely a low cortisol awaking response, and a low density neighbourhood in predicting adolescents' externalizing problem behaviours.

Reference 13 - 0,17% Coverage

There was no significant association between CARAUCg and neighbourhood density in this study, thus it is rather unlikely that there would be a bidirectional process whereby certain adolescents develop higher CARAUCg as a result of living in high density neighbourhoods, or vice versa.

Reference 14 - 0,11% Coverage

Hence, the interaction effects between CARAUCg and neighbourhood density were not likely to be confounded by any bidirectional effects between the independent and moderating variables.

Reference 15 - 0,11% Coverage

Our study showed significant main effects of SES on aggres-
sive behaviours, with adolescents from low SES families reporting higher levels of parent- and self- reported aggression.

Reference 16 - 0,21% Coverage

However, despite these main effects, the only significant SES by CARAUCg interaction effect was on the slope of self-reported delinquency. These findings suggest that the moderating effects of CARAUCg on the link between neighbourhood density and externalizing problems cannot be explained by the potential overlap between neighbourhood density and SES.

Reference 17 - 0,28% Coverage

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tivity CARAUCi neighbourhood density in predicting adolescents' self-reported delinquent and aggressive behaviours. The interaction pattern suggests that adolescents with a higher CARAUCi (i.e., a bigger change in cortisol levels post-awakening) might be more likely to be negatively influenced by a high density neighbourhood and therefore they develop higher externalizing problem behaviours.

Reference 18 - 0,19% Coverage

Finally, the focus on neighbourhood density rather than general neighbourhood deprivation provides more specific information on the contribution of neighbourhood characteristics on adolescents' externalizing problem behaviours, which is called for in a recent review (Van Vuuren, Reijneveld, Van der Wal, & Verhoeff, 2014).

Reference 19 - 0,38% Coverage

The present study revealed significant interaction effects
between neighbourhood density and CAR in predicting adolescent aggressive and delinquent behaviours. In general it indicates that individuals' biological sensitivity to environmental context plays an important role in moderating the effects of neighbourhood density on adolescents' adjustment. Findings highlight the complexities that contribute to adolescents' externalizing problem behaviours and support the theoretical idea of interactive processes between environmental context and individuals characteristics contributing to adolescents' developmental outcomes.

<Files\\Yu et al. (2017)> - § 6 references coded [1,63% Coverage]

Reference 1 - 0,09% Coverage

If a longitudinal association between adolescent depression and violence is

Reference 2 - 0,41% Coverage

identified, prevention efforts could further be shifted to adolescents, especially because there is clear evidence of effectiveness of treatment in this developmental period.15 Therefore, we have analysed data from three longitudinal
studies to investigate links between depression and violence using both community and clinical samples of adolescents and young adults.

Reference 3 - 0,20% Coverage

The finding that depression in adolescents and young
adults is associated with long-term violent outcomes has some potentially important implications for intervention and prevention.

Reference 4 - 0,31% Coverage

Overall, this suggests, of course, that the timely treatment
of depression is likely to decrease the subsequent risk of violent behaviors, and there needs to be more effort to this end. Thus, early identification and treatment of depression will have wide public health benefits.

Reference 5 - 0,42% Coverage

In addition, the findings that both depressive symptoms and clinical diagnosis were associated with future violence indicate that prevention of violence should be undertaken at both the population level in young persons with depressive symptoms who are not in contact with health care services, and in a targeted approach to a clinical population with specialist medical contact.

Reference 6 - 0,19% Coverage

These findings highlight the need for improved treatment of depression in young people, clarification of mechanisms, and, if further validated, review of clinical guidelines.

<Files\\Zondervan-Zwijnenburg et al. (2019) [1]> - § 8 references coded [1,27% Coverage]

Reference 1 - 0,22% Coverage

We investigated parental age effects on offspring
externalizing and internalizing problems around age 10–13 years in four Dutch population-based cohorts: Generation R (Gen-R), the Netherlands Twin Register (NTR), the Research on Adolescent Development and Relationships—Young cohort (RADAR-Y), and the TRacking Adolescents' Individual Lives Survey (TRAILS) (see Table 1)

Reference 2 - 0,01% Coverage

Predictors

Reference 3 - 0,04% Coverage

so the favorable effect of parental age is not solely due to SES.

Reference 4 - 0,50% Coverage

Parental age seemed unrelated to child internaliz-

ing problem behavior, especially when accounting for SES. Tentatively, older parenthood might be associated with both high and low vulnerability to develop internalizing problems. On the one hand, older parents may have a lower probability of internalizing problems because they are less likely to have a background characterized by deprivation and social instability (Robson & Pevalin, 2007), known to be related to internalizing problems such as anxiety and depression. On the other hand, internalizing problems can increase the probability of older parenthood, by hampering engagement in and consolidation of romantic relationships (Manning, Trella, Lyons, & Toit, 2010; Sandberg-Thoma & Kam Dusch, 2014). Possibly, both processes play a role, and their joint influence results in a lack of net result.

Reference 5 - 0,14% Coverage

The relatively consistent beneficial effect of
advanced parenthood for childhood externalizing problems may seem unexpected, given mixed findings from earlier research on more common mental health problems (De Kluiver, Buizer-Voskamp,

Reference 6 - 0,05% Coverage

The beneficial effect of advanced parental age could have more than one explanation.

Reference 7 - 0,13% Coverage

The observation that older parents have offspring with fewer externalizing problems, tended to disappear when SES was taken into account. This shows that demographic factors can indeed compensate for the biological disadvantages.

Reference 8 - 0,18% Coverage

The analytic strategy applied to large cohorts
showed us a beneficial association between advanced parental age and externalizing problem behavior, whereas for internalizing problem behavior there was no beneficial association with parental age. We found no evidence for a harmful effect of advanced parenthood.

<Files\\Zondervan-Zwijnenburg et al. (2019) [2]> - § 10 references coded [1,17% Coverage]

Reference 1 - 0,22% Coverage

We investigated parental age effects on offspring
externalizing and internalizing problems around age 10–13 years in four Dutch population-based cohorts: Generation R (Gen-R), the Netherlands Twin Register (NTR), the Research on Adolescent Development and Relationships—Young cohort (RADAR-Y), and the TRacking Adolescents' Individual Lives Survey (TRAILS) (see Table 1)

Reference 2 - 0,14% Coverage

We tested effects with and without adjusting for child gender and socioeconomic status (SES). SES was included as a covariate to get an impression of the relative importance of socioeconomic factors in explaining parental age effects.

Reference 3 - 0,01% Coverage

Predictors

Reference 4 - 0,04% Coverage

so the favorable effect of parental age is not solely due to SES.

Reference 5 - 0,07% Coverage

Parental age seemed unrelated to child internaliz-
ing problem behavior, especially when accounting for SES.

Reference 6 - 0,14% Coverage

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Reference 7 - 0,05% Coverage

The beneficial effect of advanced parental age could have more than one explanation.

Reference 8 - 0,20% Coverage

The finding that the negative relation of parental age and externalizing problems became weaker when SES was taken into account, indicates that the relatively high SES of older parents, or SES-related selection effects (Robson & Pevalin, 2007) at least partly explained why their children have a decreased probability of externalizing problems.

Reference 9 - 0,13% Coverage

The observation that older parents have offspring with fewer externalizing problems, tended to disappear when SES was taken into account. This shows that demographic factors can indeed compensate for the biological disadvantages.

Reference 10 - 0,18% Coverage

The analytic strategy applied to large cohorts
showed us a beneficial association between advanced parental age and externalizing problem behavior, whereas for internalizing problem behavior there was no beneficial association with parental age. We found no evidence for a harmful effect of advanced parenthood.