The stress connection: Neuroimaging studies of emotion circuits in social stress, personality, and stress-related psychopathology

Veer, I.M.

CHAPTER 9

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DUTCH SUMMARY

Ieder organisme is uitgerust met een aangeboren systeem dat adaptief om moet kunnen gaan met situaties die onze fysieke en psychologische gesteldheid bedreigen. Zulke situaties worden ook wel stressoren genoemd. Wanneer we geconfronteerd worden met een stressor, zet ons brein een reeks neuro-endocriene reacties in gang die zowel lichaam als geest in staat stellen een gepaste reactie op de stressvolle situatie te geven. Overleving van het organisme staat hierbij steeds centraal. Nadat het hoofd is geboden aan de stressor is het echter ook van belang weer terug te keren naar een rusttoestand, ook wel bekend als *homeostase*. Een flexibele interactie tussen het activeren en het remmen van het stresssysteem is onontbeerlijk voor onze fysieke en geestelijke gezondheid.

De amygdala, een kleine en evolutionair oude hersenkern die in beide hersenhelften verborgen ligt onder de neocortex, is van groot belang voor het initiëren van stressresponsen. De kernfunctie van de amygdala is dan ook het brein te alarmeren wanneer de omgeving ons saillante informatie verschaft. Dat wil zeggen, informatie die ons helpt onze overlevingskansen in algemene zin te vergroten, bijvoorbeeld in het geval van dreigend gevaar, maar ook bij potentiële beloningen. De amygdala heeft sterke verbindingen met kernen in de hersenstam die het autonome zenuwstelsel aansturen, die op hun beurt weer basale functies als ademhaling en hartslag beïnvloeden. Via deze route wordt het organisme fysiek en geestelijk in staat gesteld snel op een stressor te reageren. Deze eerste reactie wordt met name gemedieerd door het hormoon (nor)adrenaline.

Tegelijkertijd wordt de trager opererende hypothalamus-hypofyse-bijnierschors (Engelse afkorting: HPA) as geactiveerd, met als belangrijkste hormonale eindproduct cortisol. Waar (nor)adrenaline een nagenoeg direct effect heeft, piekt cortisol typisch pas 10 tot 20 minuten na aanvang van de stressor. Een van de functies van dit hormoon is dan ook het ondersteunen van het bereiken van homeostase. Terwijl cortisol in het lichaam onder andere de energiehuishouding reguleert, zorgt het hormoon in het brein voor een belangrijke terugkoppeling op de HPA-as. Hiermee wordt het beëindigen van de stressrespons gefaciliteerd en de verdere aanmaak van

cortisol gestopt.

De studies beschreven in dit proefschrift hadden tot doel om de neurale mechanismen te identificeren die een persoon in staat stellen om adequaat op een stressor te reageren en daarvan te herstellen, en om na te gaan welke rol cortisol hierin speelt. Ook werd onderzocht hoe deze regulerende circuits in het brein onder druk staan bij mensen met een verhoogde kwetsbaarheid voor een stress-gerelateerde psychische stoornis en bij mensen met een depressie of posttraumatische stress. Hierbij is gebruik gemaakt van magnetische resonantie imaging (MRI), waarmee zowel structuur als functie van het brein gemeten kan worden. In de meeste studies is een specifieke MRI methode toegepast, waarmee bekeken kon worden hoe verschillende hersengebieden met elkaar communiceren (ook wel functionele connectiviteit genoemd) bij het initieren en weer afremmen van een stressrespons.

In **hoofdstuk 2** worden de effecten van acute sociale stress beschreven op het vermogen irrelevante afleidende stimuli te negeren tijdens het uitvoeren van een werkgeheugentaak. Gezonde deelnemers moesten gedurende anderhalve seconde een aantal letters onthouden, waarbij op hetzelfde moment een neutraal of emotioneel negatief plaatje werd getoond. Dit plaatje was irrelevant voor het correct uitvoeren van de taak en moest dan ook genegeerd worden. Vervolgens kregen de deelnemers een reeks letters te zien en moesten zij aangeven een van de onthouden letters voorkwam in deze reeks. De werkgeheugen prestatie, gemeten aan de hand van de reactietijden op de tweede reeks letters, was langzamer wanneer negatieve plaatjes werden getoond dan wanneer neutrale plaatjes werden getoond, met name voor deelnemers die van tevoren een praatje hadden moeten geven voor een beoordelingscommissie bestaande uit drie voor de proefpersoon onbekende leden (sociale stress) in vergelijking met een controlegroep zonder stress. In het brein werd een zelfde patroon gezien: ventrale hersengebieden betrokken bij verwerking van emotionele stimuli (zoals de amygdala) waren actiever bij proefpersonen na sociale stress, terwijl activatie in dorsale gebieden belangrijk voor het uitvoeren van een cognitieve taak (zoals de dorsolaterale prefrontale cortex) juist verminderd was wanneer de afleidende plaatjes werden getoond. Tot slot bleek dat minder interferentie van de afleidende plaatjes en een verminderde activiteit van de ventrale hersengebieden beide gerelateerd waren aan een hogere cor-

tisolrespons in de stress groep. Deze resultaten lijken erop te wijzen dat het brein de verwerking van belangrijke informatie uit de omgeving voorrang geeft ten koste van een verminderde cognitieve prestatie in nasleep van acute stress, waarbij cortisol mogelijk een modulerende rol speelt.

Hoofdstuk 3 beschrijft de late effecten van sociale stress op functionele connectiviteit van de amygdala tijdens een scan waarbij de proefpersoon niet bezig is met het uitvoeren van een specifieke taak (*resting-state*). Een uur na de stress werd in de stressgroep, vergeleken met de controlegroep, sterkere connectiviteit gevonden met de precuneus, posterieure cingulaire cortex, en de ventromediale prefrontale cortex. Deze gebieden die in de mediale lengteas van het brein liggen en behoren tot de kerncentra van het default mode network, spelen een belangrijke rol in geheugen, emotie regulatie en sociale cognitie. In tegenstelling tot de gevonden relatie bij de werkgeheugentaak, waren verschillen in cortisolrespons niet gerelateerd aan de sterkte van de connectiviteit in de stressgroep. De gevonden stresseffecten op functionele connectiviteit van de amygdala zouden wel eens, ook al is het voorlopig speculatief, gerelateerd kunnen zijn aan het bereiken van (gedragsmatige) homeostase na stress, wat langdurig kan aanhouden na de initiële stressrespons.

In **hoofdstuk 4** werd bekeken in hoeverre functionele connectiviteit van de amygdala geassocieerd is met individuele verschillen in endogene cortisol fluctuaties, ditmaal bij proefpersonen die de stressmanipulatie niet hadden ondergaan. Het bleek dat een sterkere cortisol afname gedurende het experiment samenhing met een sterkere negatieve connectiviteit van de amygdala met de mediale prefrontale cortex, met name het gedeelte dat de perigenuale anterieure cingulaire cortex wordt genoemd. Deze resultaten zouden indicatief kunnen zijn voor een door cortisol gemedieerd regulerend netwerk dat zorgt voor een adaptieve regulering van stress- en, in meer algemeen zin, emotionele reactiviteit.

Verschillen in functionele connectiviteit tussen proefpersonen met depressie en gezonde controles staan centraal in **hoofdstuk 5**. Hiertoe werden verscheidene hersennetwerken bekeken tijdens een resting-state scan. Een ventraal netwerk, bestaande uit hersengebieden die van belang zijn voor de verwerking van emotionele stimuli, liet verminderde integratie van de bilaterale amygdala zien in de depressie-

groep vergeleken met gezonde controle proefpersonen. Ook werd verminderde negatieve connectiviteit met de linker frontale pool gevonden in het taak-positieve netwerk (geassocieerd met aandachtsprocessen en uitvoering van diverse cognitieve taken), en zwakkere connectiviteit met de linguale gyrus in een primair visueel netwerk. Geen van de gevonden verschillen was gerelateerd aan de ernst van de depressie, wat suggereert dat deze verschillen meer een algemeen kenmerk van het ziektebeeld zijn dan een afspiegeling van de huidige toestand van de depressie. Deze bevindingen kunnen wijzen op een minder adaptieve verwerking van emotionele informatie in ventrale affectieve hersengebieden en een verstoorde werkzaamheid van dorsale cognitieve gebieden, twee processen die de kern vormen van huidige netwerkmodellen van depressie.

Hoofdstuk 6 beschrijft een studie naar hippocampus- en amygdala (mediale temporale kwab) volumes van vrouwen met posttraumatische stress stoornis (PTSS) en een geschiedenis van interpersoonlijk trauma gedurende hun jeugd. Een kleiner volume van de rechter amygdala werd gevonden in de PTSS groep vergeleken met een groep vrouwen zonder stoornis. De linker amygdala en bilaterale hippocampus verschilden niet tussen de twee groepen. De volumevermindering bleek specifiek voor de basolaterale en centromediale nuclei groepen van de rechter amygdala. Tot slot was een kleinere rechter amygdala volume geassocieerd met een zwaardere geschiedenis van seksueel misbruik in de jeugd. Deze resultaten kunnen wijzen op een verstoring van het normale ontwikkelingstraject van de amygdala door een sterk traumatiserende ervaring, waardoor iemand kwetsbaarder wordt voor het ontwikkelen van een affectieve stoornis later in het leven.

Tot slot beschrijft **hoofdstuk 7** in hoeverre functionele connectiviteit van de amygdala geassocieerd is met individuele verschillen in neuroticisme en extraversie, persoonlijkheidsfactoren die in verband worden gebracht met respectievelijk kwetsbaarheid voor en weerbaarheid tegen affectieve stoornissen. Een hogere mate van neuroticisme was geassocieerd met sterkere amygdala connectiviteit met de precuneus en verminderde amygdala connectiviteit met de temporale pool, insula, en superieure temporale gyrus. Deze resultaten kunnen wijzen op een minder adaptieve perceptie en verwerking van zelfrelevante en sociaal-emotionele informatie in meer

neurotische personen. Extraversie, aan de andere kant, was geassocieerd met een sterkere amygdala connectiviteit met de putamen, temporale pool, en insula. Mogelijk weerspiegelt deze bevinding de verhoogde gevoeligheid voor beloningen en een beter sociaal-emotioneel functioneren, wat vaak wordt gevonden bij meer extraverte mensen. De voor deze persoonlijkheidsfactoren specifieke connectiviteitspatronen bieden mogelijk inzichten over de neurale processen die ten grondslag liggen aan een verhoogde kwetsbaarheid voor, of juist weerbaarheid tegen affectieve stoornissen.

Samenvattend, is in dit proefschrift een reeks studies beschreven, waarvan de resultaten laten zien hoe stress informatieverwerking kan beïnvloeden en veranderingen kan veroorzaken in de communicatie tussen hersengebieden, ook nadat de stressvolle gebeurtenis al lang voorbij is. Verder is een hersencircuit gevonden waarmee cortisol mogelijk stressresponsen moduleert, en zijn persoonlijkheidsfactoren die geassocieerd zijn met kwetsbaarheid voor of weerbaarheid tegen affectieve stoornissen in verband gebracht met veranderingen in hersennetwerken die betrokken zijn bij het verwerken en reguleren van emoties. Tot slot zijn kleinere volumes van specifieke subkernen van de amygdala gerapporteerd, welke een verband kunnen hebben met specifieke symptomen van posttraumatische stress, en is verminderde integriteit van affectieve en regulerende hersennetwerken gevonden in depressie. De resultaten uit dit proefschrift vergroten onze kennis over de effecten van stress en stresshormonen op het brein en bieden belangrijke nieuwe aanknopingspunten voor toekomstig onderzoek.

Chapter 9

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Ilya Veer
Berlin, December 2014

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CURRICULUM VITAE

Ilya Veer, born in Amsterdam on June 15th 1981, attended high school at the *Barlaeus Gymnasium* in Amsterdam from 1993 till 1999. In 2005 he received his MSc in clinical neuropsychology and biological psychology from the *University of Amsterdam* (cum laude). Ilya started his PhD at the *Leiden Institute for Brain and Cognition* (LIBC) and the *Leiden University Medical Center* (LUMC) in October 2006, supervised by prof. dr. Serge Rombouts and prof. dr. Mark van Buchem. Between June 2011 and April 2013 he worked as a postdoctoral researcher at the LIBC, during which he was involved in setting up the research-dedicated LIBC scanner facility, and provided training and scientific support for the institute's researchers. As of April 2013, Ilya works as a postdoctoral researcher at the department of Psychiatry and Psychotherapy of the *Charité University Hospital* (Berlin, Germany). In the research group of prof. dr. Henrik Walter, he continues to study the effects of stress on emotion regulation and the brain.

Chapter 9

LIST OF PUBLICATIONS

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