Catherine Russell is a Specialist Executive Neurodivergent Coaching Practitioner with a decade of extensive experience in Psychology, Mental Health, and Neurodiversity. Her professional journey encompasses a broad range of roles, including serving as an Assistant Psychologist, an Employment Support Neurodiversity Specialist, an Inpatient Mental Health Nursing Auxiliary, and a Therapeutic Group Facilitator for an Addictions Support Day Programme. Currently, Catherine dedicates her expertise to offering individualised neurodiverse strategy coaching.

How can Neurodiversity Strategy Coaching attenuate the adult cognitive deficits which arise from Childhood Emotional Abuse?

In this article I am going to talk about the link between emotional abuse in childhood and cognitive deficits that last into adulthood. I will also talk a bit about how these deficits occur through changes in the brain that happen as a result of the abuse and their impact on different types of cognitive ability.

First of all, here is a glossary explaining each of the cognitive functions which will come up in the article, followed by some breakdowns of the brain areas which I will refer to. I am adding these in the hope to provide an article which is accessible to anyone with experience of childhood emotional abuse who would like to understand some of the effects it can have on their adulthood neurocognitive experience and exactly how these were formed.

Cognitive Functions

- **Executive Function**

A collection of cognitive processes that support planning, monitoring and the successful execution of goals. Namely; attentional control, inhibition, working memory, and problem-solving.

- **Attention**

The ability to concentrate on a particular thought, object, action, and environment for a long time.

- **Working Memory (AKA Short Term Memory)**

A form of memory that allows an individual to temporarily hold a limited amount of information 'at the ready' for immediate mental use.

- **Processing Speed**

The time it takes an individual to do a mental task. The speed at which an individual can understand and react to information received; whether it be visual, auditory, or movement.

- **Emotional Processing**

The absorption of emotional disturbance and the time it takes to decline to the point of not interfering with new experiences and behaviour.

- **Reasoning Performance**

The ability to evaluate a situation in a logical manner and come to a conclusion using one's experience, emotions, and critical thinking abilities.

- **Visual Memory**

The ability to reconstruct visual and spatial information 20-30 minutes after initially seeing it.

As a Neurodiversity Strategy Coach, I believe it is important to identify the lasting effects of abuse with regard to cognitive differences experienced in adult life. Neurodiversity Strategy Coaching enables the client to learn

techniques for getting the best use out of their executive functions, differing attention styles, working memory, emotional regulation and more so that the client can live the life they want, whilst feeling in control, energised, and fulfilled. I believe that good trauma-informed psychotherapy of a modality that suits the individual is fundamental, followed by the addition of strategy coaching at a stage when the individual decides they are ready.

Brain Anatomy & Function

- **HPA Axis**

Hypothalamic-pituitary-adrenal axis. A complex feedback system of neurohormones that are sent between the hypothalamus, pituitary gland, and adrenal glands. The HPA Axis regulates the physiological mechanisms of stress reactions, immunity, and fertility.

- **Hippocampus**

The region of the brain that is primarily associated with memory. The hippocampus is located in the inner region of the temporal lobe and forms part of the limbic system – which is particularly important in regulating emotional responses. The hippocampus is responsible for storing long-term memories, spatial processing, and navigation.

- **Stress System (AKA Stress Response System)**

Stress is a biological and psychological response to a stressor that we do not feel we have the resources to deal with. Sudden stress typically causes an increase in heartrate and breathing alongside a loss of appetite and release of glucose from the liver for energy. If a situation is judged as being stressful the hypothalamus (at the base of the brain) is activated. The hypothalamus is in charge of the stress response and sends signals to the pituitary gland, and the adrenal medulla. This is the fight or flight response which is the short term stress response and occurs via the Sympathomedullary Pathway. Within this pathway the adrenal medulla, part of the autonomic nervous system, produces adrenalin – the cause of physiological reactions such as increased heartrate etc.

- **Long-Term Potentiation**

A long-lasting strengthening of the response of a postsynaptic nerve cell to stimulation across the synapse i.e. when neurons in the brain ‘fire

together' repeatedly and form new permanent pathways. This is related to learning and long-term memory e.g. the formation of habits after repetition for a certain period of time.

These glossaries are not exhaustive as I have refrained from elaborating on some of the neurological terms within these explanations. I would encourage you to research any terms you are curious about or feel free to contact me if you would like me to explain/discuss anything I have written in this article.

Several disorders that are associated with childhood trauma involve profound cognitive impairment e.g. Depression, PTSD, and Chronic Fatigue Syndrome. Research on animals has shown that early life stress induces structural, functional, and epigenetic alterations in brain regions involved in cognition, particularly in the hippocampus. A smaller hippocampus is associated with memory deficits in Depression and PTSD. Early life trauma is associated with the hippocampal volume loss found in depression.

Major & Nater (2010)

A meta-analysis by Pollock et al. (2022) on Early Life Adversity (ELA) found that the right hippocampus and amygdala are indicated in adult neurostructure. This reflects Grey Matter Volume (GMV) reductions in people without psychopathology. According to the Stress Accelleration hypothesis, the hippocampus and amygdala mature earlier as a response to adverse environments in order to increase the chance of survival and procreation. This premature neurostructural development may increase the individual's vulnerability to psychopathology. In comparison to non-ELA individuals, the window of neuroplasticity is shortened as the process of maturation would end before that of non-ELA exposed individuals.

Studies have demonstrated that individuals diagnosed with PTSD have impairments in their intellectual functioning, attention, working memory, processing speed, learning and executive function. A study performed by Gould et al. (2012) involving relatively healthy adults living in the community who experienced early life stressors suggests an association between emotional abuse/neglect and executive function, emotional processing, and visual memory deficits. A stronger association was found between neglect and emotional processing and processing speed than abuse. Major and Nater (2010) found a connection between emotional abuse in childhood and impaired spatial working memory as an adult. The

study was performed with healthy adults who had no diagnosed psychiatric illness and found that exposure to emotional neglect in particular was associated with cognitive underperformance in tasks involving the hippocampus. Findings demonstrated that the cognitive deficits were associated directly with the childhood trauma itself as opposed psychiatric illness or substance / alcohol abuse occurring after the trauma.

Dahman et al. (2018) found that early life adversity is linked to dysfunctional neurodevelopment especially in the hippocampus, and dysfunction of the stress system including the HPA axis. Findings showed that early life adversity is associated with an altered relationship between hippocampus volume and HPA axis activity. These physical neurological alterations persisted even after the children had lived in a stable and caring environment for years. This suggests that the visual memory deficits unveiled by Gould et al. (2012) have occurred due to this function relying on long-term potentiation within the hippocampus.

More studies on humans are required to further examine the cognitive performance of people exposed to childhood trauma as thus far most experimentation has occurred on animals. Future practice should involve assessing cognitive functions separately to any psychiatric assessments. Neuropsychological training should be administered in the case of deficits identified. If early interventions are made this may prevent the individual from incurring long-term deficits in memory, the main cognitive function that is suggested to be associated with childhood trauma. (Major & Nater, 2010).

Delayed development has been demonstrated in the emotion circuitry of individuals with ELA. Under/over-estimating threat-level are indicated as a result of amygdala alterations, which can lead to emotional dysregulation – a symptom present in many psychiatric disorders. The hippocampus is also involved in stress responses and reduction in its volume has been associated with cognitive deficits such as impaired memory consolidation. ELA has been shown to reduce volume of the left inferior frontal gyrus (IFG), which is associated with top-down thinking and affective control. Reduction in IFG volume are related to impaired emotional regulation and development of psychiatric disorders such as depression.

As a result of these metanalytic findings we are able to identify targets of intervention for the impacts that ELA have on the brain, such as

neurofeedback training which has shown promising results when training patients to purposefully regulate brain activity. It would also be worth looking at emotion-regulation strategies / CBT to see if they are also capable of similar regulatory effects on brain structures involved and further still, the possibility of long term morphological alterations.

Pollok et al. (2022)

References

Dahmen, B., Puetz, V. B., Scharke, W., von Polier, G. G., Herpertz-Dahlmann, B., & Konrad, K. (2017). Effects of Early-Life Adversity on Hippocampal Structures and Associated HPA Axis Functions. *Developmental Neuroscience*, *40*(1), 13–22. https://doi.org/10.1159/000484238

Gould, F., Clarke, J., Heim, C., Harvey, P. D., Majer, M., & Nemeroff, C. B. (2012). The effects of child abuse and neglect on cognitive functioning in adulthood. *Journal of psychiatric research*, *46*(4), 500–506. https://doi.org/10.1016/j.jpsychires.2012.01.005

Majer, M., Nater, U. M., Lin, J. M. S., Capuron, L., & Reeves, W. C. (2010). Association of childhood trauma with cognitive function in healthy adults: a pilot study. *BMC Neurology*, *10*(1). https://doi.org/10.1186/1471-2377-10-61

Pollok, T. M., Kaiser, A., Kraaijenvanger, E. J., Monninger, M., Brandeis, D., Banaschewski, T.,Eickhoff, S. B., & Holz, N. E. (2022). Neurostructural traces of early life adversities: A meta-analysis exploring age- and adversity-specific effects. *Neuroscience & Biobehavioral Reviews*, *135*, 104589. https://doi.org/10.1016/j.neubiorev.2022.104589