Past and lower in females (hypo-secretion)

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Past research has indeed found a strong link between peer maltreatment and increased psychotic symptoms (Wolke et al., 2014). Whilst this relationship has already been clearly established, by multiple sources, the specific underlying mechanisms which drive this link, are less clear. Explanations of a biological nature have been proposed, regarding the dysregulation of the HPA axis (van Dam et al., 2012) and changes in Leukocyte Telomere length (Simon et al., 2006). Additionally, cognitive explanations have also been supported, suggesting that irrational and biased perceptions of reality, can mediate the development of psychotic symptoms (Garety et al., 2001).

A biological mechanism proposed to explain the relationship between peer maltreatment and the subsequent increase in psychotic symptoms, includes the dysregulation of the Hypothalamic-Pituitary-Adrenal Axis Theory (HPA) (van Dam et al., 2012). The HPA axis is one of the two systems involved in controlling stress responses, specifically in the release of glucocorticoids e. g. cortisol (Gunnar & Quevedo, 2007).

Peer maltreatment is often of an upsetting and stressful nature, and can lead to the activation of the flight or fight response. When activated, adrenocorticotropic hormone is released from the pituitary gland, causing cortisol levels to peak (Vaillancourt et al., 2008). Frequent and intense exposure to abuse and maltreatment can lead to an unnatural imbalance of cortisol, where bloodstream cortisol levels tend to be higher in males (hyper-secretion) and lower in females (hypo- secretion) (Schreier et al.

, 2009). This imbalance can often have an effect on receptor sensitivity (Vaillancourt, et al., 2008). Baseline levels of cortisol do not match the body's natural circadian rhythm patterns, which could lead to inappropriate stress responses (over exaggerated or degraded responses) (Vaillancourt et al., 2008). As a result of this, maltreated individuals might lack the ability to successfully monitor and control their biological responses towards stressful events. Not only this, but higher levels of cortisol have also been associated with hippocampal damage, where volume reductions are usually observed (Bremner & Vermetten, 2001).

Therefore, increased prevalence of psychotic symptoms could be due to a combination of chemical imbalances and morphological changes which help mediate inappropriate stress responses and behaviour. The biological mechanisms associating peer maltreatment with psychotic outcomes, are not strictly limited to one explanation. Additionally, researchers have successfully investigated the links between Leukocyte Telomere shortening and development of psychotic symptoms (O'Donovan et al., 2011). Telomeres are nucleoproteins which are attached to the ends of chromosomes and have the function of preventing ' end-to-end recombination' (Simon et al., 2006). Telomeres can act as biological age, mortality and health markers due to the changes in structure length over time (O'Donovan et al., 2011).

Shorter telomeres are usually associated with increases in age, however, prolonged and intense exposure to stress can speed up the shortening process (Simon et al., 2006). The severe psychological stress, often created as a result of maltreatment and abuse, can cause telomeres to shorten in

length at a rate which is 10 years ahead of normal development (Simon et al., 2006). Shorter telomeres are of significance and relevance because they are usually linked to ‘ old’ age and greater health problems e.

g. cardiovascular disease, bipolar, cancer in animals etc. (Wong & DePinho, 2003). Nevertheless, ‘ young’ individuals experiencing heightened and/or chronic stress due to maltreatment, can also experience ‘ wear and tear’ to the body’s biological and cognitive systems (Chrousos, 1998).

The biological abnormalities e. g. accelerated telomere shortening, could mediate an increase in psychotic symptoms (amongst those who have suffered abuse), as they ‘ mimic’ aged and damaged cells. Whilst the true causal direction of this relationship remains to be further clarified, an established link between psychotic disorders and premature biological ageing (shorter telomeres) has indeed been established (Lindqvist et al., 2015). Alternative non-biological mechanisms have also been put forward to explain this phenomenon.

Existing research has equally supported the use of cognitive explanations (Garety et al., 2001). The increased likelihood of experiencing psychotic symptoms, after maltreatment, could be due to biased and self-destructive perceptions of reality (Fisher et al., 2013). Some individuals carry a genetic disposition for certain psychotic-like symptoms, which makes them extremely reactive and emotionally vulnerable (Freeman et al., 2002). Stressful events in the environment can act as a precipitator, which triggers the onset of symptoms (Freeman et al.

, 2002). Following stressful events, individuals have a natural tendency to seek meaning and explanation in order to help find closure (Garety et al., 2001). Those with high genetic vulnerability, will not succeed in this process due to cognitive impairments, emotional changes, the decay of perception and judgements abilities (Garety et al., 2001). High emotional distress, created from maltreatment, can lead to distorted beliefs about one's self and the world. More specifically, low self-esteem, depression, anxiety can motivate negative schemas, which casts a pessimistic and unfavorable view on life (Fisher et al.

, 2013). Individuals are more likely to undergo Biased Conscious Appraisal Processes, where biased judgements are formed regarding the ' anomalous' stressful experiences (Garety et al., 2001).

Irrational external attributions are made in order to explain the anomalous experiences, leading to distorted views. The more maltreatment experienced, the greater the chances of irrational biased psychotic explanations being used, rather than more realistic ones (White et al., 2000).

Psychotic symptoms can therefore arise, due to biased, incorrect and negative perceptions of stressful events e. g. maltreatment. The errors in cognition could create inner-outer cognitive confusion which could further manifest into symptoms such as delusions, hallucinations, disorganized speech etc.

(Emsley et al., 2003). In summary, differing views on the underlying mechanisms linking peer maltreatment and increased psychotic symptoms have led to multiple plausible explanations.

Biological explanations tend to be more objective and comparable, however, this should not detract from equally reasonable cognitive perspectives. Since no single mechanism has been defined, it is worth noting that perhaps one single framework is not adequate enough to globally explain an increase in psychotic symptoms. Symptoms of psychosis include a wide range of behaviours e. g.

hallucinations, catatonia, affective flattening etc. making the discovery of a single underlying mechanism an extremely difficult or impossible task (Emsley et al., 2003).