studies, including white noise, exposure to cold temperature, loud noises and anticipation of stressful situations such as giving a speech. Overall, these stressors result in acute stressful situations. Other studies screened for subjects who experience a prolonged stressful situation such as being primary caregiver to a terminal loved one.

It has been shown that subjects who have been exposed to prolonged life stressors are more likely to complain of symptoms of GERD. One study demonstrated a correlation between discussion of emotionally charged topics and nonpropulsive activity in the esophagus. Another study assessed gastric acid output in relation to personality traits. It was found that subjects who were considered to have a higher level of impulsivity and expressed emotions more freely were more likely to react with an increase in gastric acid output when subjected to stress simulated by a problem-solving session than patients with low level of impulsivity. In fact, subjects with low level of impulsivity reacted to this stress with a decrease in gastric acid secretion. Increased gastric acid secretion has been seen in subjects with a higher tendency towards emotional lability. One study evaluated the relationship among stress, psychological traits associated with chronic anxiety, acid reflux parameters and perceptions of reflux symptoms. The researchers found that stress tasks did not influence objective measurements of acid reflux (total acid exposure, number of acid reflux events and duration of longest acid reflux event). Another significant finding was that reflux patients who were chronically anxious and exposed to prolonged stressful stimuli may be more likely to perceive low-intensity esophageal stimuli as painful reflux symptoms. Therefore, even normal esophageal acid exposure could trigger complaints of GERD symptoms. Also, it is not a specific psychiatric disorder that may be responsible for gastrointestinal distress but the presence of psychological distress that predisposed a patient to have clinical manifestations of GERD.

A study by Naliboff et al. found that 'vital exhaustion', which is a measure of sustained stress symptoms, was most closely correlated with symptoms of heartburn. Fass et al. have shown that acute auditory stress can exacerbate heartburn symptoms in GERD patients by enhancing perceptive response to intra-oesophageal acid exposure. This greater perceptual response is associated with greater emotional responses to the stressor.

EFFECTS OF STRESS ON BEHAVIOUR AND SLEEP

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Key words: stress, glucocorticoid receptor, cortisol, memory, cognition

Psychologists Yerkes and Dodson described already in 1908 the inverted-U shaped relationship between arousal and performance. When the level of arousal heightens, behavioural performance increases but only to a certain point. When the level of arousal, produced by stress, becomes too high, adequacy of behaviour decreases while sleep is affected. Physical and mental performance generally follows the level of circulating stress hormones, in particular the glucocorticoid hormone cortisol. The secretion of cortisol in response to a stressful event triggers a chain of events, ultimately leading to energy for fight-or-flight behaviour. Under non-stressed basal conditions, the level of cortisol follows a circadian pattern: a maximum in the morning, necessary for daily activities, with slowly declining levels during the day, and a trough during sleep. Cortisol binds to two glucocorticoid receptor subtypes: Type I with a high affinity and Type II with a lower affinity for cortisol. There are also differences in brain location between the types. The differences between the two subtypes results in a discrepancy of receptor occupation. During the nocturnal sleep trough all Type I receptors are occupied by the endogenous hormone, while during the morning wake peak Type 1 receptors are fully saturated and Type 2 receptors come into action. The mix of Type I and Type II occupation is also the situation by stressful events. The differential qualities

of both receptors have created a new hypothesis about the cortisol effects on behaviour and sleep (De Kloet et al, 1999; Lupien et al, 2007). Elevated levels of cortisol due to stress have commonly detrimental effects on performance, such as on memory, but it is more than once reported that cortisol could have positive effects on cognition. The Type I/Type II ratio hypothesis suggests that performance by cortisol can be enhanced when Type I receptors are activated. However, when both Type I and Type II receptors are saturated, shifting the ratio towards Type II occupancy, performance and sleep are affected. It is in this way that the double function of cortisol as a sleep/wake–hormone as well as a stress-hormone, can be understood. The hypothesis is now that the inverted-U shaped relationship between arousal and stress at one side and behavioural and cognitive performance at the other, might be explained by the presence of two different types of glucocorticoid receptors.

THE NEUROSCIENTIFIC BASIS OF EVIDENCE-BASED TREATMENTS FOR PTSD – A SELECTIVE REVIEW

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Key words: posttraumatic Stress Disorder , selective serotonin reuptake inhibitors, memory, cognitive behavioral therapies

Posttraumatic Stress Disorder (PTSD) includes a) exposure to a significant traumatic event, b) intrusive recollections, c) avoidant symptoms d) increased physiological arousal or amnesia for the traumatic event). While up to 90% of the US adult civilian population has a lifetime exposure to at least one significant traumatic event, only 8-10% develop PTSD. While PTSD can result in significant morbidity and dysfunction, evidence-based treatments can now be matched with an emerging understanding of underlying neurobiology.

Meta-analysis demonstrates that certain selective serotonin reuptake inhibitors (SSRIs) and serotonin and norepinephrine reuptake inhibitors (SNRIs) are superior to placebo in attenuating PTSD symptoms. Unfortunately, the effect size is small (0.23), side-effects are common and these drugs may be less efficacious when PTSD results from combat trauma rather than from civilian trauma.

Recurring nightmares of the triggering event contribute to the sleep disturbances that affect some 70% of patients with PTSD. Although available data unequivocally support the efficacy (effect size ~ 1.0) of prazosin, an α1-adrenergic receptor antagonist, it remains underutilized in most clinical settings.

Patients with PTSD are more sensitive to the anxiogenic effects of an intravenously administered 5HT2C agonist or an adrenergic α2 receptor antagonist. This suggests that SSRIs and SNRIs may act by downregulation hypersensitive 5HT2C receptors. Analogously, since presynaptic α2 receptors are inhibitory to efflux of noradrenaline, symptoms of PTSD could be mediated by excessive stimulation of postsynaptic α1 receptors. Prazosin may act to attenuate this stimulation.

Brain regions involved in modulation of emotion, such as the dorsal and rostral anterior cingulate cortices, as well as the ventromedial prefrontal cortex show decreased activity in PTSD, resulting in excessive input from the amygdala, an evolutionarily older brain region dominant in threat responses. Thus, PTSD can be conceptualized as a dysregulation of brain circuits that integrate historical information of a traumatic event (memory) and autonomic responses.

Fortunately, memory storage is not a onetime event but a process repeated with each use of that memory. Retrieval of a memory renders it temporarily available for modification at the cellular and systems level. This principle is thought to underlie several independently developed psychotherapeutic approaches. The observation that recurring, disturbing thoughts of PTSD could be permanently abolished, if the subject's eyes were automatically moving in a multi-saccadic manner while the disturbing thought was