

PATHWAYS TO YAWNING: MAKING SENSE OF THE THOMPSON CORTISOL HYPOTHESIS

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Abstract

Yawning apparatus and the exact location of the yawn reflex remains controversial. Yet yawning is a significant behavioural response that may potentially be a new diagnostic marker of neurological disease such as multiple sclerosis. Evidence of brain cooling following yawning supports the Thompson Cortisol Hypothesis which postulates the association between cortisol, electrical nerve activity and yawning within the known stress-response system, the Hypothalamus-Pituitary- Adrenal-axis. Changes in cortisol levels are important because they present as a new potential diagnostic tool in the early diagnosis of neurological symptoms.

Keywords: *Biomarker; Cortisol; Diagnosis; Neurological disorder; Pathways; Thompson Cortisol Hypothesis; Yawning*

1. INTRODUCTION

1.1 *Myths about yawning*

Yawning manifests itself in many different ways making it difficult to research because its origin has historically and apparently become so allusive to researchers. Yawning is accessible to many of us and has been anecdotally associated with sleepiness; lack of oxygen; headaches; excessive fatigue (Catli *et al.* 2014); anxiety; after taking antidepressants; and as a contagious form, after seeing animals and humans yawning (Prasad, 2008; Sarnecki, 2008; Provine, 2012).

Association between yawning and brain temperature regulation has been proposed because of the link between excessive yawning and brain temperature fluctuations in people with multiple sclerosis (MS) (Gallup & Gallup, 2007; Thompson, 2010). Circadian rhythm and the control of brain temperature is the responsibility of the hypothalamus which is linked to the pituitary gland, and the adrenal glands. Normally, the hypothalamus-pituitary-adrenal (HPA) axis produces sufficient hormones to protect against stress and to provide the body with readiness for physical exertion (Thompson, 2014a). Cortisol is the naturally produced hormone that protects the body against being stressed physically and psychologically, and recently has been reported in association with yawning in a number of experimentally-controlled trials (Thompson & Bishop, 2012; Thompson, 2014a; Thompson, Frankham & Bishop, 2014; Thompson, Rose & Richer, 2014).

1.2 *Contagious yawning*

Mental Attribution Theory (Platek *et al.* 2003; Norscia & Palagi, 2011) has been presented as the reason for

contagiously yawning when we are empathic to others who yawn, and even to animals that we care about, who yawn in our presence. Yawning happens in the womb (Reissland *et al.* 2012) and is more frequent in new-born babies than toddlers since sleep deprivation increases the chances of us yawning which may make us more vulnerable to the effects of stress and fatigue (Giganti *et al.* 2007).

2. CORTISOL AND YAWNING

2.1 *Cortisol as a stress hormone*

It is understood that cortisol acts to protect our body against stress, both physical and psychological stress loadings. It also regulates the other hormones released within the HPA-axis. It is suggested that, as part of its stress-protection and stress-response, cortisol elicits yawning by increasing the electrical activity of the nerves in the muscles around the jaw line, giving rise to yawning (Thompson & Zisa, 2011).

2.2 *Thompson Cortisol Hypothesis*

The Thompson Cortisol Hypothesis (Thompson, 2010; Thompson & Bishop, 2012; Thompson, Frankham & Bishop, 2014; Thompson, Rose & Richer, 2014) is the first evidence-based report that links cortisol with yawning, and shows that cortisol rises when we yawn. It is suggested that the rise in cortisol level triggers our yawning response. Implications of this research are that yawning is the mechanism for controlling hormone regulation and hypothalamus temperature regulation.

Reports of brain-stem ischaemic stroke patients have indicated that spontaneous yawning, gives rise to parakinesia brachialis oscitans in which the paralyzed arm may rise on yawning

(Wimalaratna & Capildeo, 1988; Thompson, 2010; Walusinski, Neau & Bousslavsky, 2010). Recently, swallow reflex and yawning have also been postulated to be temporally related (Kimiko *et al.* 2014).

2.3 Yawning and the motor cortex

From the Functional Magnetic Resonance Imaging studies reported, yawning seems to involve the frontal and parietal lobes, insula and amygdala (Krestel *et al.* 2013) though it is probable that there is a threshold level of cortisol to be reached following fatigue, empathy, or sleep deprivation, to elicit yawning. Electrical Myographical (EMG) nerve activity in the jaw muscles is increased with yawning, and is also associated with rises in cortisol levels (Thompson, 2013; Thompson, 2014b). EMG feedback as well as hormone level changes within the HPA-axis continually regulate cortisol and adrenaline production within this feedback closed loop.

The motor cortex is likely to be involved in this feedback loop since it controls movement including jaw-line muscle activation. Other influences in this mechanism may be the amygdala (Norcia & Palagi, 2011), especially during empathic (or contagious) yawning where psychologically we are influenced by our mood and the sense of belonging to a particular social grouping. Hence, we respond by yawning because others (or animals) are yawning in our presence (Guggisberg *et al.* 2010).

For the motor cortex to be involved in yawning, it is probably stimulated by cortisol production. For this to occur, the motor cortex needs to be in communication with the hypothalamus which is triggered by cortisol and regulates brain temperature. Since brain temperature may be lowered following yawning, it is postulated that the change in brain temperature is communicated, via the hypothalamus (within the HPA-axis), to the motor cortex so that yawning can be ceased [Figure 1].

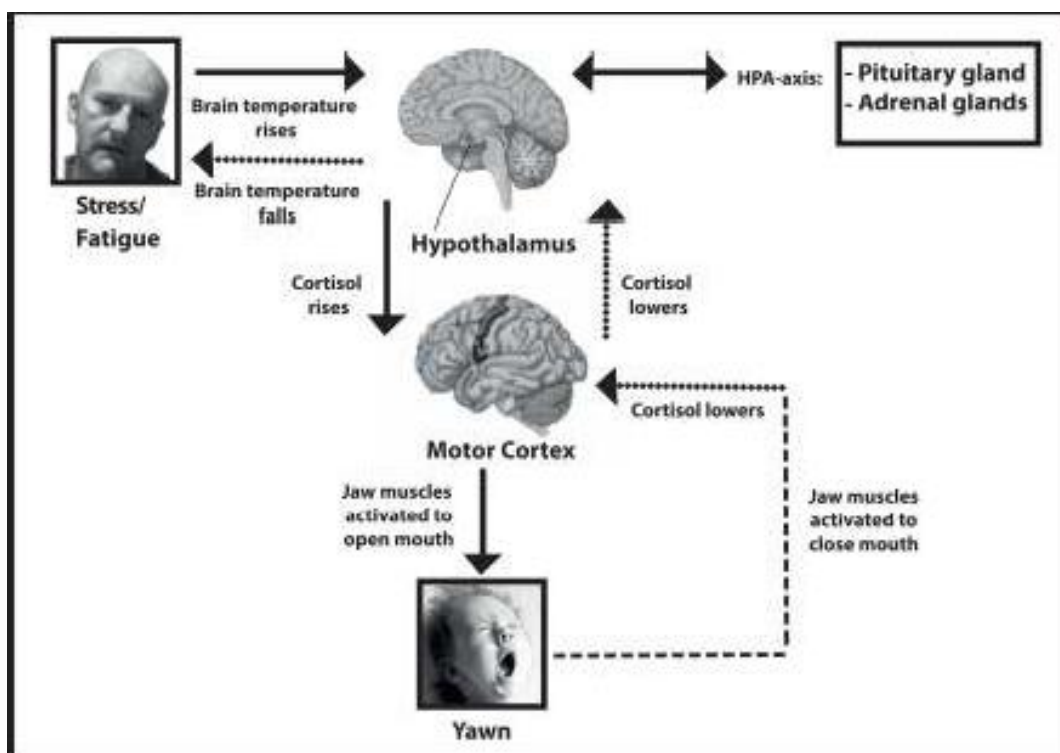


Figure 1. Jaw Muscle Activation and Yawning due to Cortisol Rises

Symptom relief such as in lowering brain temperature, in people with multiple sclerosis, has been evidenced by Gallup and Gallup (2007). Excessive yawning is a common symptom of multiple sclerosis due to excessive fatigue (Fleming & Pollak, 2005).

2.4. Yawning and the brain-stem

The brain-stem is an evolutionary structure known to be of importance for our vital functions. Ischaemia in this region clearly affects these functions, such as in stroke, and tracts to the motor cortex are the likely method of

communicating status of movement but also feedback about yawning responses. This is probable because of the unusual yawning and involuntary responses of the upper paralyzed limb of brain-stem ischaemic stroke patients (Walusinski, *et al.* 2010), but also because jaw-line muscle movement is governed by the motor cortex (Thompson, 2011;2013).

It is theorised that cortisol plays an important role in regulating, indirectly, the movement of limbs (through musculature) via feedback to the motor cortex, or more precisely, via the hypothalamus that communicates with the motor cortex [Figure2].

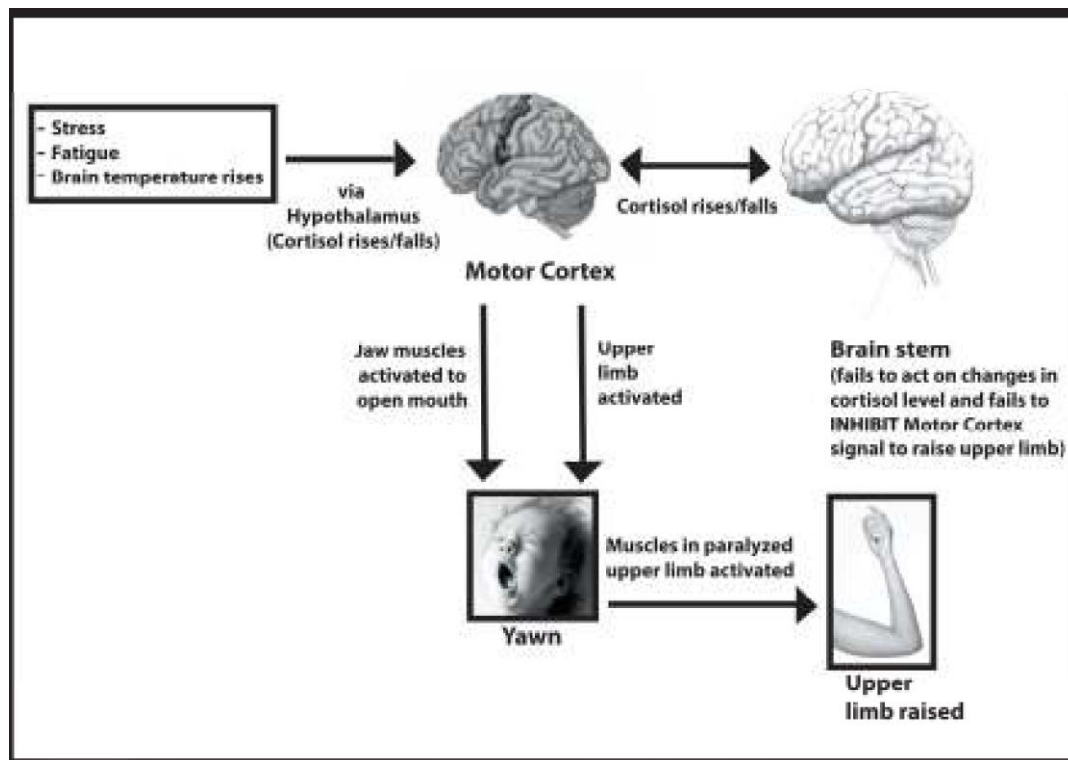


Figure 2. Paralyzed Upper Limb Rise in Ischaemic Stroke

In this instance, it is suggested that the brain stem fails to act on the change in levels of cortisol and allows upper limb musculature (signalled by the motor cortex because of rises in cortisol) to contract, and raise the upper limb. In the stroke patient, the cortisol levels are inadequately detected thus resulting in the movement in the paralyzed limb (Wimalaratna &

Capildeo, 1988; Walusinski, 2009). In contrast, on the unaffected side of the body, movement is inhibited, because the signal (rise in cortisol) is recognised within the brain stem structure.

Comparison between pathways illustrated (Figs. 1 & 2), allows postulation that the brain-stem is involved inhibition of movement. Reacting to

cortisol levels detected by the hypothalamus, the brain-stem acts to inhibit movement of the upper limb when it is not required to move. Failure of this inhibition may be evidenced in parakinesia brachialis oscitans (Walusinski, 2006; 2007) following brain-stem ischaemic stroke.

Of course, hormones do not act singularly and it is acknowledged that this scenario may be a simplification because the hypothalamus, in particular, contributes to the HPA-axis which also governs secretion of adrenaline. However, it is highly plausible that in brain stem ischaemia, it is the loss of neuronal detection systems that would normally communicate with the motor cortex, that are responsible for the strange movement in the paralyzed upper limbs of stroke patients. It is plausible that the detection system relies on the monitoring of cortisol levels, which when faulty, result in yawning without the recognition of falling cortisol levels after yawning.

2.5 Implications for MS

In MS, it is the incomplete innervation and loss of the myelin sheath around nerves that is largely responsible for the uncoordinated movements seen in people with the disorder. Since the cause of fatigue in MS is not well understood, an evaluation of the attentional network during intrinsic and phasic alerting tasks has been performed (Périn *et al.* 2010).

Importantly, the rise in temperature seen in MS patients is coexistent with fatigue together with excessive yawning (Gallup & Gallup, 2008; 2010). It is postulated, therefore, that cortisol levels rise when yawning occurs in people with MS, just as it is evidenced in healthy people (Thompson & Bishop, 2012), but at higher levels

than seen in the healthy population because of the presence of excessive fatigue which is frequently seen as a common symptom of MS (Gallup & Gallup, 2008).

Brain scans performed before and after induced fatigue and yawning may well be the conclusive answer to theories suggesting communication between motor cortex, HPA- axis, and brain-stem regions, and potentially supported by cortisol assay. An on-going multi-centre study conducted at Bournemouth University by the author together with French neuroscientists at Université Paris X Ouest Nanterre La Défense, Hôpital Universitaire Amiens, and Jules Verne Université de Picardie, France (Thompson *et al.* 2014) may help towards illuminating such theories.

3. CONCLUSION

Yawning and cortisol is of continued interest to neuroscientists, clinical practitioners, neurologists and theorists. Excitement has been re-kindled with the suggestion that cortisol, and yawning, may present as the next new potential diagnostic biomarker for neurological disease detection. Clearly, further research is indicated but it is important in pursuing this possibility if it means that there may be a potential use of one of our oldest observed behaviour, yawning, that for centuries, philosophers such as Hippocrates (Vigier, 1620) has upheld for inclusion in their list of “useful natures”.

4. ACKNOWLEDGEMENT

Thanks to Natalie Thompson, Photo Editor of Pynq.net for providing professional artwork; and Pynq Thompson for providing her image.

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