Irish Journal of Psychological Medicine (2018), **35**, 63–68. © College of Psychiatrists of Ireland 2017
doi:10.1017/ipm.2017.54

SHORT REPORT

Examining stress: an investigation of stress, mood and exercise in medical students

J. O'Flynn[1], T. G. Dinan[1,2] and J. R. Kelly[1,2,*]

[1] *Department of Psychiatry and Neurobehavioural Science, University College Cork, Cork, Ireland*
[2] *APC Microbiome Institute, University College Cork, Cork, Ireland*

Objectives. Stress is an event that threatens homoeostasis and thus causes physiological and behavioural responses to reinstate equilibrium. Excessive and/or chronic stress can be psychologically and physiologically detrimental. Examinations can represent a significant source of stress for students. The hypothalamic–pituitary–adrenal axis (HPA) is the core endocrine stress system. Investigations into the HPA response to examinations have yielded inconsistent results. The aim of this study is to further explore the relationship between examination stress, HPA axis activity, mood, sleep and exercise in students undergoing a naturalistic examination period stressor.

Methods. In total, 16 medical students participated. Students completed self-reported stress, anxiety, mood, sleep and physical activity questionnaires, and provided saliva samples during an examination-free period and an examination period 1 month later. The cortisol awakening response, representative of HPA activity, was determined from saliva samples by enzyme-linked immunosorbent assay.

Results. Anxiety levels increased ($p = 0.04$) and mood decreased ($p = 0.05$) during the examination period. There was concomitant decease in physical activity levels ($p = 0.02$). There was no significant increase in HPA activity during the examination period ($p = 0.29$). Sleep quality did not significantly worsen ($p = 0.55$) during the examination period.

Conclusions. Examination periods are associated with increased anxiety levels, lower mood and decreased physical activity. Future studies incorporating examination results and cognitive function may help to identify potential protective interventional strategies, while optimising performance.

Received 20 March 2017; Revised 15 July 2017; Accepted 31 August 2017; First published online 9 October 2017

Key words: Cortisol awakening response, exercise, hypothalamic–pituitary–adrenal axis, medical students, resilience, stress.

Introduction

College courses can be stressful, with high workloads, deadlines and examinations. Medical courses can be particularly stressful (de La Rosa-Rojas *et al.* 2015; Khan *et al.* 2016; Heinen *et al.* 2017). Examinations, provide insight into the bio-psycho-behavioural response of students to a naturalistic stressor (Bloch & Brackenridge, 1972). Sources of stress are multi-dimensional, but academic performance is a key contributing factor to stress in medical students (Beiter *et al.* 2015). The brain interprets perceived stressors to determine physiological and behavioural responses. This process can promote adaptation, but when responses are exaggerated or overused, pathology can ensue (Bradley & Dinan, 2010; Cohen *et al.* 2012; McEwen, 2017). A recent meta-analysis of mental illness in medical students found that the estimated pooled prevalence of depression or its symptoms was 27.2%, and that suicidal ideation occurred in an estimated 11.1% (Rotenstein *et al.* 2016).

In response to a stressor, the hypothalamic–pituitary–adrenal (HPA) axis initiates a hormonal cascade resulting in adrenal upregulation of cortisol, the main glucocorticoid in the body (Tsigos & Chrousos, 2002). During periods of stress, cortisol levels increase (Chida & Steptoe, 2009). Cortisol has a wide range of functions, including immune modulation and glucose control, to allow the body to cope with these stressors (McEwen & Seeman, 1999). The system works as a negative feedback loop, whereby glucocorticoids modulate their own secretion by acting at various levels of the HPA axis, such as the hippocampus, hypothalamus and pituitary.

Physiological cortisol concentration follows a specific diurnal pattern that reaches an early morning nadir, and rapidly peaks 30–45 minutes after awakening before gradually falling again until the next morning (Weitzman *et al.* 1971). The spike in cortisol concentration related to waking is called the cortisol awakening response (CAR). This has been shown to be increased in response to an acute stressor in healthy populations, though with individual variability (Pruessner *et al.* 1997; Chida & Steptoe, 2009; Kudielka *et al.* 2009).

* Address for Correspondence: Dr J. R. Kelly, Lecturer in Clinical Psychiatry, Department of Psychiatry and Neurobehavioral Science, Rm 5.38, Biosciences Institute, University College Cork, College Road, Cork, Ireland.
(Email: johnkelly@ucc.ie)

Most studies investigating the relationship between the CAR and examination stress in students indicate an increase in cortisol output during examination periods (van Dulmen *et al.* 2007; Hewig *et al.* 2008; González-Cabrera *et al.* 2014). However, not all studies show this elevation in cortisol output during examination periods (Vedhara *et al.* 2000; Kenwright *et al.* 2011; Duan *et al.* 2013). Although, self-reported measures of stress increase during examination periods, there is no consistent and robust relationship between these psychological measures and the CAR (Weekes *et al.* 2006), and in the wider context, considerable heterogeneity exists in studies investigating the CAR (Ryan *et al.* 2016; Stalder *et al.* 2016).

Methods

Subjects

Approval of the study protocol was granted by the Cork University Hospital ethics committee and conducted in accordance with the ICH Guidelines on Good Clinical Practice, and the Declaration of Helsinki. Written informed consent was obtained from all subjects before any study procedures were conducted. Healthy fourth year medical students were recruited. Age, sex, alcohol consumption, height and weight were recorded.

Inclusion criteria were as follows: age between 20 and 35, able to provide written consent, fluent English, general good health and sitting the same examination. Exclusion criteria included were as follows: current or previous psychiatric illness, the presence of a significant acute or chronic illness, immunodeficiency, and the use of any medication that may interfere with the objectives of the study, pose a safety risk or confound the interpretation of results (including, but not limited to, anti-psychotics, anxiolytics, anti-depressants or any other serotonergic medications, or oral steroids) and tobacco use.

Design

A repeated measures design was employed (see Fig. 1*a*). The examination period chosen to represent the stressor in this study was a 3-week examination period, occurring in May 2016. This was necessary for student progression to the final year of the degree course. Multiple written examinations were performed during this period. Measurement occurred in two phases. The first phase of measurement was a non-examination period (low-intensity stress condition) in early April 2016. The second phase of measurement was the examination phase (high-intensity stress condition) in early May 2016. To reduce interference with individual examination preparation regimens, participants were given the option of completing the second phase of testing on the day before or the first day of the examination period. Participants completed questionnaires on the same morning as providing saliva samples.

Cortisol sampling and analysis

CAR was measured using saliva samples. Verbal and written instruction was provided to all participants. Participants were provided with four Salivettes (Sarstedt AG & Co., Germany) for both phases of measurement. Subjects were instructed to provide four saliva samples at, 0 minutes (upon wakening), 30, 45 and 60 minutes after waking on 1 day during each of the measurement phases. Participants were asked not to alter their usual waking time. The samples were refrigerated at −4°C until collection. Saliva samples were centrifuged at 1000 g for 5 minutes. They were aliquoted and stored at −80°C until analysis. Cortisol concentrations were determined using the Cortisol Enzyme Immunoassay Kit as per manufacturers' instruction (Enzo®; Life Sciences, New York). Assay detection limit was 0.16 nmol/l. Inter- and intra-assay percentage co-efficients of variability were 11.24% and 8.2%, respectively.

Statistical analysis

SPSS Statistics v20 (IBM) was used to analyse the data. To assess normality, Shapiro–Wilk scores were used. Parametric data were analysed using paired *t*-test and Wilcoxon matched-paired signed-rank test for non-parametric data. Pearson's correlation was used to assess for correlations between measured components.

Results

Participants

A total of 20 participants meeting inclusion and exclusion criteria were recruited. Four were lost to follow-up. In total, 16 participants completed the study. See Table 1 for demographic details.

Table 1. *Demographic data*

Component	Category	Number	Percentage of total
Age	20–25	12	75
	26–30	3	19
	31–35	1	6
Sex	F	12	75
	M	4	25
BMI	20–25	10	77
	> 25	3	23
Alcohol	0–5 units/week	8	62
	6–11 units/week	5	38

BMI, Body mass index.

https://doi.org/10.1017/ipm.2017.54 Published online by Cambridge University Press

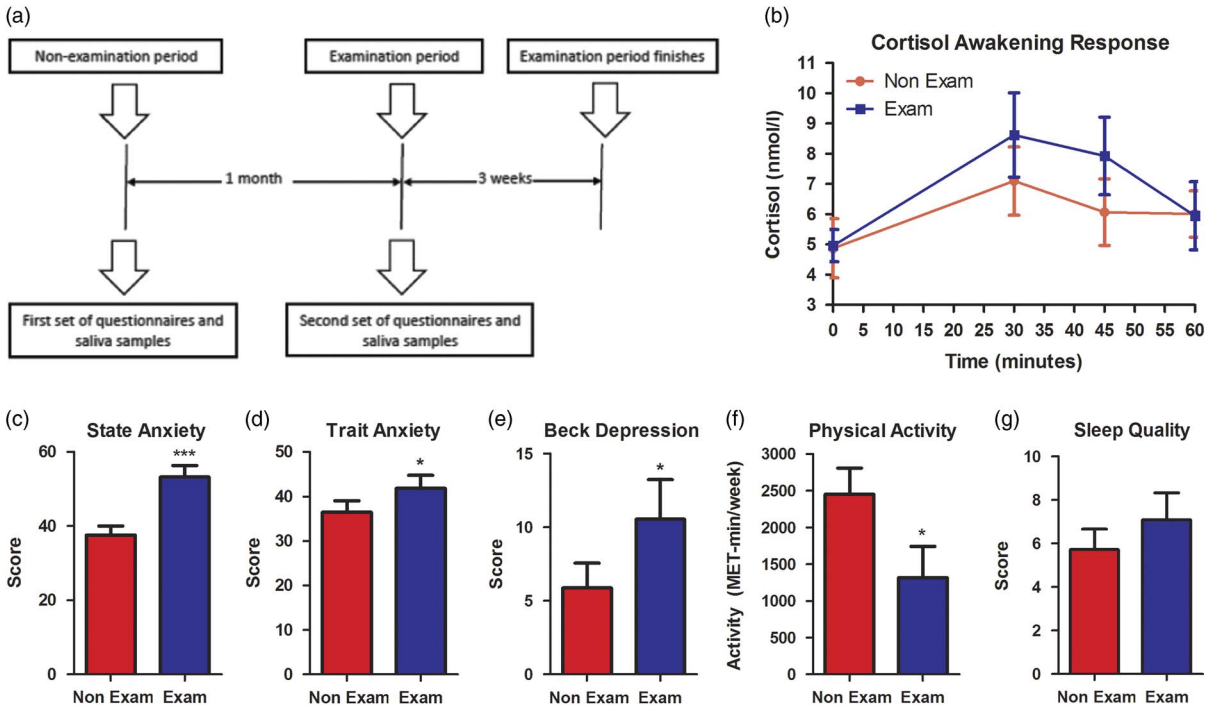

Fig. 1. Bio-psycho-behavioural response to examination period. A repeated measures design was employed (*a*). In total, 16 students completed self-reported stress, anxiety, mood and physical activity questionnaires, and saliva samples during a non-examination (low-intensity stress condition) and 4 weeks later during an examination (high-intensity stress condition). There was no significant difference in the cortisol awakening response (*b*). Anxiety levels increased during the examination period as measured by the State-Trait Anxiety Inventory (*c*,*d*). Depression scores measured using the Beck Depression Scale increased during the examination period (*e*). Physical activity questionnaires was decreased during the examination period (*f*) and although sleep quality worsened during the examination period, it did not reach significance (*g*).

Bio-psycho-behavioural response to examination period

There was no significant difference in the CAR (F (1.82, 14.02) = 0.91, p = 0.42) (Fig. 1*b*). The area under the curve with respect to ground, representative of the total cortisol secretion between timepoints (t (11) = −0.97, p = 0.35), the change in total cortisol secretion, known as delta (t (11) = 1.17, p = 0.27) and the area under the curve with respect to increase (t (11) = 1.10, p = 0.29) was not significantly altered (data not shown). The State (t (12) = −5.05, p < 0.001) (Fig. 1*c*) and Trait anxiety (t (11) = −2.38, p = 0.04) (Fig. 1*d*) component of the State-Trait Anxiety Inventory increased during the examination period. Depression scores measured using the Beck Depression Scale (BDI) increased during the examination period (t (12) = −2.15, p = 0.05) (Fig. 1*e*). Physical activity (decreased during the examination period (t (12) = 2.51, p = 0.03) (Fig. 1*f*). Although sleep quality worsened during the examination period, it did not reach significance (t (10) = −0.61, p = 0.55) (Fig. 1*g*).

Discussion

Using a bio-psycho-behavioural appraisal of a naturalistic stressor, we found that examination periods are associated with significantly increased anxiety levels and decreased mood in students. Furthermore, there was a decreased level of self-reported physical activity. This increase in self-reported stress and anxiety measures during examination periods is consistent with previous studies (Weekes *et al.* 2008; González-Cabrera *et al.* 2014). Both state and trait anxiety levels increased during the examination period in our study (Figs 1*c*,*d*). Although, the increase in Perceived Stress Scale (PSS) was not significant (data not shown), the scores during both the non-examination and examination period indicate a moderate level of stress (Cohen & Janicki-Deverts, 2012). Other studies have demonstrated an increase in the PSS (Vedhara *et al.* 2000; Duan *et al.* 2013) and we note that the PSS scores in our study were lower than those identified in a group of science students by Vedhara *et al.* (2000).

The Yerkes–Dodson law suggests an optimal amount of stress is required for the optimal performance of a difficult task, such as an examination (Yerkes & Dodson, 1908; Heins *et al.* 1983). Excess stress may overwhelm coping mechanisms (Stowell *et al.* 2008) and impact cognition (Lupien *et al.* 2007). Indeed, a large population-based study in adults showed that elevated cortisol was associated with poorer cognitive function (Lee *et al.* 2007). As mentioned, the relationship

https://doi.org/10.1017/ipm.2017.54 Published online by Cambridge University Press

between CAR and psychological stress measures is inconsistent (van Eck *et al.* 1996; Gaab *et al.* 2006; Weekes *et al.* 2006; Duan *et al.* 2013; González-Cabrera *et al.* 2014) and our study also failed to show a clear relationship between any of the cortisol measures and the psychological measures, suggesting a more complex relationship between psychological measures and the CAR. A study by Preuss *et al.* (2010) found a stronger HPA response to oral presentations compared to written examinations.

It is well established that mood and anxiety are interlinked. Using the BDI we show that mood significantly worsened during the examination period (Fig. 1*e*). However, it is important to note that the BDI scores remained within the minimal range, and did not reach clinically relevant levels. This may not be surprising considering we excluded students with current or previous history of mental health-related disorders, but future studies following students throughout the year would provide insight into potential evolving mood-related problems. As mentioned above, depression-related symptoms in medical students are high (Rotenstein *et al.* 2016), and highlight the need to identify strategies for preventing the risk of developing these disorders in this population.

Stress-related disorders, such as depression and anxiety, are associated with an impaired regulation of stress hormones (Holsboer & Ising, 2010), with adverse implications for cognition (Hinkelmann *et al.* 2009; Hinkelmann *et al.* 2013). Although, two meta-analyses concluded that cortisol levels in patients with depression were increased (Knorr *et al.* 2010; Stetler & Miller, 2011), the association between clinical subtypes and their biological substrates is inconsistent and variable at the individual level (Lamers *et al.* 2013). The CAR has been useful in understanding the psychobiology of stress, but in order to enhance this understanding and provide the means to assess novel therapeutic agents, multiple readouts are necessary (Allen *et al.* 2014).

Consistent with previous studies (Steptoe *et al.* 1996) we show that examinations interrupt normal physical activity of students (Fig. 1*f*). Considering the positive impact of exercise on anxiety, mood and cognition, this study highlights the importance of maintaining regular exercise both in non-examination and examination periods. Exercise can also improve sleep quality (Loprinzi & Cardinal, 2011). The precise neurobiology of sleep is not completely understood, though the link between stress and sleep quality is more defined (Hirotsu *et al.* 2015). A recent preclinical study, showed that a core function of sleep is to renormalise overall synaptic strength (de Vivo *et al.* 2017). Student sleep quality worsened slightly during the examination period, but did not reach significance (Fig. 1*g*). The 'good' quality sleep range for the Pittsburgh Sleep Quality Index (PSQI) is less than five (Buysse *et al.* 1989). With mean PSQI scores greater than five both before and during examinations, and a positive association between perceived stress and poor sleep quality during the non-examination period, our findings are consistent with the literature suggesting that students may have poor sleep quality (Tsai & Li, 2004). Both acute and chronic sleep disturbance can negatively impact mood and cognitive performance in students (Curcio *et al.* 2006; Hershner & Chervin, 2014).

Limitations

This was a pilot study, and as such, our sample size was small. The phase of female participant menstrual cycle was not recorded during this study. The sympathoadrenal medullary system, intensity or duration of examination preparation and student coping style were not recorded.

Conclusions

We have characterised, using a naturalistic stressor, the bio-psycho-behavioural response to examination stress in students. We have shown that examination periods increase anxiety, decrease mood. In parallel, there was a decrease in physical activity levels. Taken together, our results highlight the importance of an awareness of the impact of stress on students, and paves the way to inform and refine interventions that may reduce the negative impact of stress, while optimising performance. Larger studies, investigating oral examinations, or objective structured clinical examinations to further develop our understanding of the bio-psycho-behavioural components, correlated with examination performance and cognition could assist with the identification of vulnerability factors in the student population.

Acknowledgements

The authors would like to thank the University College Cork Medical School, for providing the funding for this study via the Research Supplement Award.

Financial Support

This work was supported by the University College Cork Medical Research Supplement Award.

Conflicts of Interest

J.O.F. and J.R.K. report no conflicts of interest. T.G.D. reported that his research is supported by SFI (grant number SFI/12/RC/2273) and by the Health Research Board (HRB) through Health Research Awards (grants numbers HRA_POR/2011/23, HRA_POR/2012/32

and HRA-POR-2-14-647) and through EU GRANT 613979 (MYNEWGUT FP7-KBBE-2013-7). T.G.D. has been an invited speaker at meetings organised by Servier, Lundbeck, Janssen and AstraZeneca, and has ongoing research collaborations with Suntory Wellness, Danone Nutritia, Mead Johnson Nutrition, Cremo and 4D-Pharma.

Ethical Standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committee on human experimentation with the Helsinki Declaration of 1975, as revised in 2008. The authors assert that ethical approval for publication of this study has been provided by the Cork University Hospital ethics committee.

References

Allen AP, Kennedy PJ, Cryan JF, Dinan TG, Clarke G (2014). Biological and psychological markers of stress in humans: focus on the Trier Social Stress Test. *Neuroscience and Biobehavioral Reviews* **38**, 94–124.

Beiter R, Nash R, McCrady M, Rhoades D, Linscomb M, Clarahan M, Sammut S (2015). The prevalence and correlates of depression, anxiety, and stress in a sample of college students. *Journal of Affective Disorders* **173**, 90–96.

Bloch S, Brackenridge CJ (1972). Psychological, performance and biochemical factors in medical-students under examination stress. *Journal of Psychosomatic Research* **16**, 25–33.

Bradley AJ, Dinan TG (2010). A systematic review of hypothalamic-pituitary-adrenal axis function in schizophrenia: implications for mortality. *Journal of Psychopharmacology* **24**, 91–118.

Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ (1989). The Pittsburgh Sleep Quality Index – a new instrument for psychiatric practice and research. *Psychiatry Research* **28**, 193–213.

Chida Y, Steptoe A (2009). Cortisol awakening response and psychosocial factors: a systematic review and meta-analysis. *Biological Psychology* **80**, 265–278.

Cohen S, Janicki-Deverts D (2012). Who's stressed? Distributions of psychological stress in the United States in probability samples from 1983, 2006, and 2009. *Journal of Applied Social Psychology* **42**, 1320–1334.

Cohen S, Janicki-Deverts D, Doyle WJ, Miller GE, Frank E, Rabin BS, Turner RB (2012). Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proceedings of the National Academy of Sciences of the United States of America* **109**, 5995–5999.

Curcio G, Ferrara M, De Gennaro L (2006). Sleep loss, learning capacity and academic performance. *Sleep Medicine Reviews* **10**, 323–337.

de La Rosa-Rojas G, Chang-Grozo S, Delgado-Flores L, Oliveros-Lijap L, Murillo-Perez D, Ortiz-Lozada R, Vela-Ulloa G, Carreazo NY (2015). Level of stress and coping strategy in medical students compared with students of other careers. *Gaceta Medica De Mexico* **151**, 443–449.

de Vivo L, Bellesi M, Marshall W, Bushong EA, Ellisman MH, Tononi G, Cirelli C (2017). Ultrastructural evidence for synaptic scaling across the wake/sleep cycle. *Science* **355**, 507–510.

Duan H, Yuan Y, Zhang L, Qin S, Zhang K, Buchanan TW, Wu J (2013). Chronic stress exposure decreases the cortisol awakening response in healthy young men. *Stress* **16**, 630–637.

Gaab J, Sonderegger L, Scherrer S, Ehlert U (2006). Psychoneuroendocrine effects of cognitive-behavioral stress management in a naturalistic setting – a randomized controlled trial. *Psychoneuroendocrinology* **31**, 428–38.

González-Cabrera J, Fernández-Prada M, Iribar-Ibabe C, Peinado JM (2014). Acute and chronic stress increase salivary cortisol: a study in the real-life setting of a national examination undertaken by medical graduates. *Stress: The International Journal on the Biology of Stress* **17**, 149–156.

Heinen I, Bullinger M, Kocalevent RD (2017). Perceived stress in first year medical students – associations with personal resources and emotional distress. *BMC Medical Education* **17**, 4.

Heins M, Fahey SN, Henderson RC (1983). Law students and medical-students – a comparison of perceived stress. *Journal of Legal Education* **33**, 511–525.

Hershner SD, Chervin RD (2014). Causes and consequences of sleepiness among college students. *Nature and Science of Sleep* **6**, 73–84.

Hewig J, Schlotz W, Gerhards F, Breitenstein C, Lurken A, Naumann E (2008). Associations of the cortisol awakening response (CAR) with cortical activation asymmetry during the course of an exam stress period. *Psychoneuroendocrinology* **33**, 83–91.

Hinkelmann K, Moritz S, Botzenhardt J, Riedesel K, Wiedemann K, Kellner M, Otte C (2009). Cognitive impairment in major depression: association with salivary cortisol. *Biological Psychiatry* **66**, 879–885.

Hinkelmann K, Muhtz C, Dettenborn L, Agorastos A, Moritz S, Wingenfeld K, Spitzer C, Gold SM, Wiedemann K, Otte C (2013). Association between cortisol awakening response and memory function in major depression. *Psychological Medicine* **43**, 2255–2263.

Hirotsu C, Tufik S, Andersen ML (2015). Interactions between sleep, stress, and metabolism: from physiological to pathological conditions. *Sleep Science* **8**, 143–152.

Holsboer F, Ising M (2010). Stress hormone regulation: biological role and translation into therapy. *Annual Review of Psychology* **61**, 81–109, c1-11.

Kenwright K, Liddell PW, Bloom L, Zucker-Levin A, Nolen AH, Faulkner LW, Batorski RE (2011). Salivary cortisol levels in students challenged with a testing stressor. *Clinical Laboratory Science: Journal of the American Society for Medical Technology* **24**, 221–226.

Khan M, Fatima A, Shanawaz M, Fathima M, Mantri A (2016). Comparative study of stress and stress related factors in medical and engineering colleges of a south Indian city. *Journal of Evolution of Medical and Dental Sciences* **5**, 3153–3156.

https://doi.org/10.1017/ipm.2017.54 Published online by Cambridge University Press

Knorr U, Vinberg M, Kessing LV, Wetterslev J (2010). Salivary cortisol in depressed patients versus control persons: a systematic review and meta-analysis. *Psychoneuroendocrinology* **35**, 1275–1286.

Kudielka BM, Hellhammer DH, Wust S (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology* **34**, 2–18.

Lamers F, Vogelzangs N, Merikangas KR, de Jonge P, Beekman AT, Penninx BW (2013). Evidence for a differential role of HPA-axis function, inflammation and metabolic syndrome in melancholic versus atypical depression. *Molecular Psychiatry* **18**, 692–699.

Lee BK, Glass TA, McAtee MJ, Wand GS, Bandeen-Roche K, Bolla KI, Schwartz BS (2007). Associations of salivary cortisol with cognitive function in the Baltimore memory study. *Archives of General Psychiatry* **64**, 810–818.

Loprinzi PD, Cardinal BJ (2011). Association between objectively-measured physical activity and sleep, NHANES 2005–2006. *Mental Health and Physical Activity* **4**, 65–69.

Lupien SJ, Maheu F, Tu M, Fiocco A, Schramek TE (2007). The effects of stress and stress hormones on human cognition: implications for the field of brain and cognition. *Brain and Cognition* **65**, 209–237.

McEwen BS (2017). Allostasis and the epigenetics of brain and body health over the life course: the brain on stress. *JAMA Psychiatry* **74**, 551–552.

McEwen BS, Seeman T (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences* **896**, 30–47.

Preuss D, Schoofs D, Schlotz W, Wolf OT (2010). The stressed student: influence of written examinations and oral presentations on salivary cortisol concentrations in university students. *Stress* **13**, 221–229.

Pruessner JC, Wolf OT, Hellhammer DH, Buske-Kirschbaum A, von Auer K, Jobst S, Kaspers F, Kirschbaum C (1997). Free cortisol levels after awakening: a reliable biological marker for the assessment of adrenocortical activity. *Life Sciences* **61**, 2539–2549.

Rotenstein LS, Ramos MA, Torre M, Segal JB, Peluso MJ, Guille C, Sen S, Mata DA (2016). Prevalence of depression, depressive symptoms, and suicidal ideation among medical students a systematic review and meta-analysis. *JAMA-Journal of the American Medical Association* **316**, 2214–2236.

Ryan R, Booth S, Spathis A, Mollart S, Clow A (2016). Use of salivary diurnal cortisol as an outcome measure in randomised controlled trials: a systematic review. *Annals of Behavioral Medicine* **50**, 210–236.

Stalder T, Kirschbaum C, Kudielka BM, Adam EK, Pruessner JC, Wust S, Dockray S, Smyth N, Evans P, Hellhammer DH, Miller R, Wetherell MA, Lupien SJ, Clow A (2016). Assessment of the cortisol awakening response: expert consensus guidelines. *Psychoneuroendocrinology* **63**, 414–432.

Steptoe A, Wardle J, Pollard TM, Canaan L, Davies GJ (1996). Stress, social support and health-related behavior: a study of smoking, alcohol consumption and physical exercise. *Journal of Psychosomatic Research* **41**, 171–180.

Stetler C, Miller GE (2011). Depression and hypothalamic-pituitary-adrenal activation: a quantitative summary of four decades of research. *Psychosomatic Medicine* **73**, 114–126.

Stowell JR, Tumminaro T, Attarwala M (2008). Moderating effects of coping on the relationship between test anxiety and negative mood. *Stress and Health: Journal of the International Society for the Investigation of Stress* **24**, 313–321.

Tsai LL, Li SP (2004). Sleep patterns in college students – gender and grade differences. *Journal of Psychosomatic Research* **56**, 231–237.

Tsigos C, Chrousos GP (2002). Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research* **53**, 865–871.

van Dulmen S, Tromp F, Grosfeld F, ten Cate O, Bensing J (2007). The impact of assessing simulated bad news consultations on medical students' stress response and communication performance. *Psychoneuroendocrinology* **32**, 943–950.

van Eck M, Berkhof H, Nicolson N, Sulon J (1996). The effects of perceived stress, traits, mood states, and stressful daily events on salivary cortisol. *Psychosomatic Medicine* **58**, 447–458.

Vedhara K, Hyde J, Gilchrist ID, Tytherleigh M, Plummer S (2000). Acute stress, memory, attention and cortisol. *Psychoneuroendocrinology* **25**, 535–549.

Weekes N, Lewis R, Patel F, Garrison-Jakel J, Berger DE, Lupien SJ (2006). Examination stress as an ecological inducer of cortisol and psychological responses to stress in undergraduate students. *Stress* **9**, 199–206.

Weekes NY, Lewis RS, Goto SG, Garrison-Jakel J, Patel F, Lupien S (2008). The effect of an environmental stressor on gender differences on the awakening cortisol response. *Psychoneuroendocrinology* **33**, 766–772.

Weitzman ED, Fukushima D, Nogeire C, Roffwarg H, Gallagher TF, Hellman L (1971). 24 hour pattern of episodic secretion of cortisol in normal subjects. *Journal of Clinical Endocrinology & Metabolism* **33**, 14–22.

Yerkes RM, Dodson JD (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology* **18**, 459–482.

https://doi.org/10.1017/ipm.2017.54 Published online by Cambridge University Press