Habitual 'sleep credit' is associated with greater grey matter volume of the medial prefrontal cortex, higher emotional intelligence and better mental health

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SUMMARY

In modern society, people often fail to obtain the amount of sleep that experts recommend for good health and performance. Insufficient sleep can lead to degraded cognitive performance and alterations in emotional functioning. However, most people also acknowledge that on a regular basis they obtain more sleep than they subjectively perceive they need at a minimum to stave off performance decrements, a construct we describe as subjective 'sleep credit'. Few people would contest the notion that getting more sleep is better, but data on both behavioural and neuroanatomical correlates of 'sleep credit' are surprisingly limited. We conducted a voxel-based morphometric study to assess cerebral grey matter correlates of habitually sleeping more than one's subjective requirements. We further tested whether these structural correlates are associated with perceived emotional intelligence and indices of psychopathology while controlling for age, gender, and total intracranial volume. In a sample of 55 healthy adults aged 18–45 years (28 males, 27 females), whole-brain multiple regression showed that habitual subjective 'sleep credit' was correlated positively with grey matter volume within regions of the left medial prefrontal cortex and right orbitofrontal gyrus. Volumes were extracted and regressed against self-report emotion and psychopathology indices. Only grey matter volume of the medial prefrontal cortex cluster correlated with greater emotional intelligence and lower scores on several indices of psychopathology. Findings converge with previous evidence of the role of the medial prefrontal cortex in the relationship between sleep and emotional functioning, and suggest that behaviour and brain structure vary with habitual 'sleep credit'.

INTRODUCTION

In today's society, many healthy adults often sleep less than the 7–8 h per night recommended by most experts (Ferrara, 2001; National Sleep Foundation, 2005). Both acutely and habitually insufficient sleep have been demonstrated to affect various aspects of daytime functioning adversely, such as sleep propensity, attention and alertness (Banks and Dinges, 2007; Punjabi *et al.*, 2003; Van Dongen and Maislin, 2003). Relative to being well rested, prolonged sleep debt may also increase somatic complaints and symptoms of depression, anxiety and paranoia (Kahn-Greene *et al.*, 2007). Even measures of self-perceived emotional intelligence may also be impacted negatively by sleep deprivation. More specifically, previous research indicates decreases in both global emotional intelligence, as well as in inter- and intrapersonal functioning (e.g. lower empathy towards others; reduced self-regard) and stress management (e.g. impaired impulse control) following prolonged sleep deprivation compared to rested baseline (Killgore *et al.*, 2008). Thus, there is clear and ample evidence that people do not sleep as much as is recommended physiologically, and that insufficient sleep

impairs daytime functioning. Interestingly, however, some data also suggest that approximately 40% of adults get more sleep than they think they need subjectively (National Sleep Foundation, 2005). One could argue that getting more sleep than needed subjectively would benefit behaviour and possibly even counteract the effects of physiologically insufficient sleep. Indeed, Rupp and colleagues showed in a series of studies that a brief period of 'banking sleep' by sleeping for longer than normal before a period of insufficient sleep enhances resilience to and recovery from the actual sleep loss (Rupp *et al.*, 2009a,b). One relatively unexplored aspect of the relationship between sleep and behaviour concerns whether sleeping more than needed on a habitual basis (i.e. 'sleep credit') is also associated with behavioural or emotional benefits, and whether this may be reflected in specific differences in regional brain volume.

For chronic sleep restriction, at least in the context of sleep disorders (e.g. chronic insomnia, obstructive sleep apnea, narcolepsy) and daytime sleepiness in otherwise healthy adults, persistently insufficient sleep and excessive daytime sleepiness have been linked to reduced grey matter volume, particularly of the ventromedial and orbitofrontal cortex (Altena *et al.*, 2010; Joo *et al.*, 2010; Killgore *et al.*, 2012b; Morrell, 2003). The mechanisms underlying this relationship remain unknown. Reduced grey matter could emerge as a function of insufficient sleep. However, animal research has not demonstrated conclusively an adverse effect of chronic sleep restriction on neuronal health (Cirelli *et al.*, 1999), although there is some recent evidence that sleep deprivation can reduce axonal sprouting in animal models of stroke (Gao *et al.*, 2010; Zunzunegui *et al.*, 2011) and may inhibit hippocampal volume and neurogenesis in laboratory animals (Mueller *et al.*, 2011; Novati *et al.*, 2011). In humans, no grey matter volume changes were observed in patients with obstructive sleep apnea following successful intervention (O'Donoghue, 2005). Thus, it may be that reduced grey matter in this region may serve as a diathesis that precedes and increases vulnerability to disordered sleep, but it is also conceivable that increased grey matter in this region may emerge as a consequence of sleeping more than the minimum needed to function without impairment. However, given the lack of empirical data on sleep and brain structure, the first step would be to demonstrate this association in humans. Therefore, the present study aimed to investigate voxel-based morphological correlates of sleeping in excess of minimal subjective requirements in a sample of healthy adults. In addition, we also attempted to test whether morphological correlates of 'sleep credit' would be associated with indices of psychopathology and emotional intelligence that have been shown previously to be sensitive to sleep loss. Based on the literature reviewed above, we hypothesized that sleeping habitually in excess of minimal subjective requirements would be associated with increased grey matter volume in ventromedial and orbitofrontal cortices. In addition, we hypothesized that grey matter volume in this brain region would be correlated negatively with lower scores on indices of psychopathology and positively with greater emotional intelligence.

METHODS

Participants

Using posted flyers and internet advertisements, we recruited 55 healthy right-handed adults (mean age 30.74 $\pm$ 8.13, range 18–45; 28 males, 27 females; mean years of education 14.96 $\pm$ 2.17, range 11–20) from the Boston metropolitan area. There was no age difference between females and males. All participants were native English speakers and underwent a detailed screening interview to determine eligibility. Based on this screening, all participants included in the study were deemed healthy (i.e. no history of neurological, psychiatric, alcohol, illicit substance use disorders or sleep disorders). Any other conditions that may influence magnetic resonance imaging (MRI; e.g. chronic pain that would not allow the subject to remain still in the scanner) and psychoactive medications (e.g. antidepressants, analgesics, anticonvulsants) were also exclusionary. Participants were compensated at a rate of $25 per hour. The McLean Hospital Institutional Review Board approved this research, which was conducted in accordance with the 1964 Declaration of Helsinki. Prior to study participation, each participant provided written informed consent.

Materials and procedures

On the day of the MRI scan, participants responded to two open-ended questions on sleep habits: (i) how much do you typically sleep on weeknights (Sunday to Thursday); and (ii) how much do you typically sleep on weekend nights (Friday to Saturday)? In addition, all participants completed the following statement on subjective sleep need: 'If I get less than __ hours of sleep, I notice an impairment in my ability to function at work'. Participant response to the first two items was used to calculate the weighted average habitual sleep (in hours). 'Sleep credit' was conceptualized as the difference between the weighted average habitual sleep and the subjectively reported minimum hours of sleep necessary until functional impairment is noticed.

In addition, participants completed the Bar-On Emotional Quotient Inventory (EQ-i; Bar-On, 2006), a self-report measure of trait emotional intelligence. The inventory contains 125 items yielding a total emotional quotient plus five composite scores (interpersonal, intrapersonal, adaptability, stress management, general mood). Individual items are answered on a five-point Likert scale ranging from 'very seldom or not true of me' to 'very often true of me or true of me'. The interpersonal composite score reflects perceived empathy and interpersonal skills, whereas the intrapersonal composite provides a measure of self-perceived awareness of personal emotions and self-regard. Adaptability reflects the perceived ability to

scrutinize challenging circumstances objectively, to resolve them and to adapt flexibly to changing situations. Stress management provides a measure of tolerance of, and perceived self-control during, taxing or challenging situations. Finally, the general mood composite reflects self-reported positive thinking and overall satisfaction with personal life. Based on previous research showing that sleep loss affects specific scales of the EQ-i (Killgore *et al.*, 2008), we restricted our analyses to the total emotional quotient and the interpersonal, intrapersonal and stress management composites.

Every participant also completed the computerized Personality Assessment Inventory (PAI; Morey, 1991) as an index of several dimensions of psychopathology. The PAI contains 344 statements that are rated using one of four response options ('false, not at all true', 'slightly true', 'mainly true', 'very true'). It yields 11 clinical subscales (somatic complaints, anxiety, anxiety-related disorders, depression, mania, paranoia, schizophrenia, borderline features, antisocial features, drug-related problems, alcohol-related problems). Based on previous findings from the literature showing that sleep deprivation affects specific scales on the PAI (Kahn-Greene *et al.*, 2007), we restricted our primary analyses to four clinical scales (i.e. somatic complaints, anxiety, depression, paranoia). Raw scores for each scale and subscale used in this study were converted into T scores based on the normative data provided with the scoring programme and in the test manual (Morey, 1991).

MRI parameters

We acquired structural magnetic resonance images at 3.0 Tesla using a 12-channel head coil (Siemens Tim Trio, Erlangen, Germany) and a T1-weighted three-dimensional MPRAGE sequence (TR/TE/flip angle: 2.1s/2.25 ms^{-1}/12°; 128 sagittal slices; 256 × 256 matrix; in-plane resolution: 1 × 1 × 1 mm; slice thickness: 1.33 mm).

Voxel-based morphometry

The VBM8 toolbox in SPM8 was used for preprocessing of structural images (Wellcome Department of Imaging Neuroscience Group, London, UK; http://www.fil.ion.ucl.ac.uk/spm/; http://dbm.neuro.uni-jena.de/vbm.html). The modulated voxel-based morphometry (i.e. grey matter volume was corrected for total brain volume) applied default settings. That is, each structural image was first DARTEL-normalized to match the Montreal Neurological Institute template. Then, a fully automated algorithm within SPM8 segmented each image into grey matter, white matter and cerebrospinal fluid. Finally, normalized grey matter images were smoothed with an 8-mm full-width at half-maximum isotropic Gaussian kernel.

Statistical analysis

The statistical analysis involved three nested steps. The first step tested the association between the amount of 'sleep credit' and voxelwise grey matter volume. Thus, normalized smoothed grey matter images were entered into a whole-brain general linear model in SPM8 using a threshold of $P < 0.001$, uncorrected, with a cluster extent $k \geq 90$, which was determined statistically as the expected number of voxels per cluster that would be expected by chance based on the theory of Gaussian random fields applied to this analysis (as provided in the standard VBM8 output). Age and gender were included as nuisance covariates. The second step tested whether the grey matter volume clusters from the first step were associated with scores on the emotional intelligence and psychopathology indices. Here, eigenvariates, as extracted from each significant cluster, were entered stepwise as regressors into a series of multiple regression models in IBM SPSS Statistics for Macintosh version 20 (IBM Corp., Armonk, NY, USA). To limit type I error, we ran one primary analysis for the total score of the EQ-i and PAI, respectively (i.e. five models: EQ-i total; PAI somatization; PAI anxiety; PAI depression; PAI paranoia) using a Bonferroni-corrected significance threshold $P < 0.01$ for each model. The third analysis step examined associations between grey matter volume and composite scores of the two tests used (i.e. EQ-i: interpersonal, intrapersonal, stress management; PAI: somatization conversion/somatization/health concerns; anxiety cognitive/affective/physiological; depression cognitive/affective/physiological; paranoia hypervigilance/persecution/resentment). Follow-up analyses were conducted only for significant primary analyses and significant regressors. Normal distribution of all dependent variables was tested with the Shapiro–Wilk test in SPSS. For normally distributed data (i.e. all EQ-i composites), we derived Pearson correlations, whereas Spearman correlations were computed for the non-normally distributed composite scores (i.e. all PAI composites). Again, per inventory, we applied a Bonferroni-corrected significance threshold $P < 0.017$ ($\alpha = 0.05$ divided by three subscores).

RESULTS

On average, self-reported habitual sleep was 7.46 h [standard deviation (SD) = 0.80, range 5.5–9.0]. The minimum hours of sleep needed subjectively before functional impairment was noticeable to the individual varied greatly from 2 to 10 h (mean = 5.65, SD = 1.35). Therefore, on average, participants 'banked' 1.8 h of 'sleep credit' on a habitual basis (SD = 1.49, range −3 to 5.5). The mean total EQ-i was 100.60 (SD = 13.89, range 69–126; interpersonal: mean = 99.98, SD = 14.76, range 59–125; intrapersonal: mean = 100.55, SD = 14.88, range 59–125; stress management: mean = 102.89, SD = 12.21, range 76–125). All mean PAI scale and subscale scores were within normal ranges (somatic complaints: mean = 46.07, SD = 6.88, range 39–89; anxiety: mean = 47.47, SD = 9.39, range 35–73; depression: mean = 47.20, SD = 10.82, range 36–101; paranoia: mean = 51.53, SD = 10.46, range 32–74).

As evident in Fig. 1, greater 'sleep credit' correlated with greater grey matter volume in two clusters: (i) left gyrus

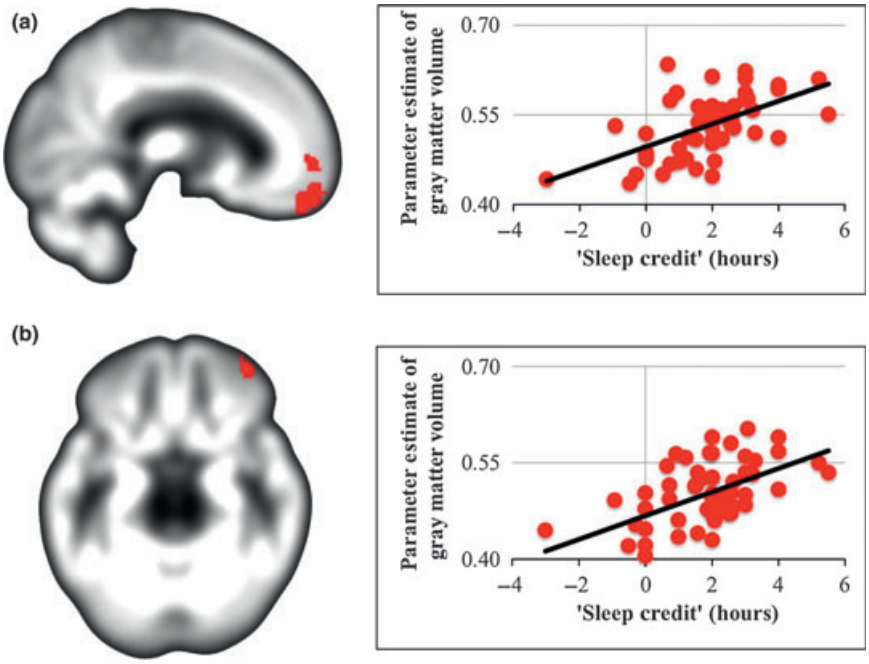

Figure 1. (a) Sagittal view of the left gyrus rectus/orbitofrontal gyrus cluster (overlaid on sample-specific T1 mean image) that was correlated positively with sleeping in excess of subjective need and the corresponding scatterplot showing the relationship between sleeping more than needed subjectively (in hours) and average grey matter volume for the cluster located at Montreal Neurological Institute (MNI) coordinates $x = -6$, $y = 52$, $z = -21$. (b) Axial view of the right middle orbitofrontal gyrus cluster (overlaid on sample-specific T1 mean image) that was correlated positively with sleeping in excess of subjective need and the corresponding scatterplot showing the relationship between 'sleep credit' (i.e. sleeping more than needed subjectively) and average grey matter volume for the cluster located at MNI coordinates $x = 39$, $y = 51$, $z = -18$.

rectus/superior and medial orbital frontal gyrus (OFG; 892 voxels, $T = 4.81$, peak-level Montreal Neurological Institute (MNI) coordinates: $x = -6$, $y = 52$, $z = -21$) and (ii) right middle OFG (149 voxels, $T = 4.43$, peak-level MNI coordinates: $x = 39$, $y = 51$, $z = -18$).

Table 1 presents results of the multiple regression analysis on the total EQ-i. Only grey matter volume of the cluster comprising the left gyrus rectus, superior and medial OFG correlated with total EQ-i. This means that greater grey matter in this medial PFC cluster was associated with greater global perceived emotional intelligence. Follow-up Pearson correlations with Bonferroni correction to $P < 0.017$ showed positive associations between this cluster's grey matter volume and the interpersonal subscale only [interpersonal: $r = 0.38$, $P < 0.017$; intrapersonal: $r = 0.28$, not significant (NS); stress management: $r = 0.23$, NS].

Table 2 presents results of the multiple regression analyses on the PAI clinical scales somatization, depression, anxiety and paranoia. Similar to the findings for the EQ-i, only grey matter volume of left gyrus rectus/medial and superior OFG correlated with PAI psychopathology. Overall, greater grey matter volume in this cluster in the medial PFC correlated with lower scores on indices of psychopathology, but this was true only for somatic complaints, depression and paranoia, but not anxiety. For PAI somatization, none of the follow-up Spearman correlations survived Bonferroni correction (conversion: $r = -0.29$, $P = 0.03$; somatization: $r = -0.15$, $P = 0.29$; health concerns: $r = -0.29$, $P = 0.03$). For PAI depression, follow-up Spearman correlations showed negative associations between grey matter volume of this cluster in the medial PFC and cognitive and physiological, but not affective, symptoms of depression (cognitive: $r = -0.34$, $P < 0.017$; affective: $r = -0.29$, $P = 0.03$; physiological: $r = -0.36$, $P < 0.017$). For PAI paranoia, follow-up Spearman correlations also showed negative associations between grey matter volume of this

Table 1 Stepwise linear regression of grey matter volume on emotional intelligence

Total EQ-i	*B*	*SE B*	ΔR^2	β
Step 1				
Constant	47.84	18.81		
L. gyrus rectus/superior and medial OFG	99.31	35.26	0.13	0.36*
Step 2				
Constant	44.95	20.77		
L. gyrus rectus/superior and medial OFG	92.30	41.00	0.13	0.34
R. middle OFG	13.22	38.55	0.01	0.05

EQ-i, emotional quotient; OFG, orbitofrontal gyrus; SE, standard error.
*$P < 0.01$.

Table 2 Stepwise linear regression of grey matter volume on hypothesized PAI psychopathology scales

	B	*SE B*	ΔR^2	β
PAI somatization				
Step 1				
Constant	47.84	18.81		
L. gyrus rectus/superior and medial OFG	99.31	35.26	0.13	0.36*
Step 2				
Constant	44.95	20.77		
L. gyrus rectus/superior and medial OFG	92.30	41.00	0.13	0.34
R. middle OFG	13.22	38.55	0.01	0.05
PAI anxiety				
Step 1				
Constant	76.16	13.05		
L. gyrus rectus/superior and medial OFG	−53.99	24.46	0.08	−0.29
Step 2				
Constant	72.64	14.38		
L. gyrus rectus/superior and medial OFG	−62.49	28.38	0.08	−0.34
R. middle OFG	16.03	26.68	0.01	0.09
PAI depression				
Step 1				
Constant	96.64	14.15		
L. gyrus rectus/superior and medial OFG	−93.07	26.53	0.19	−0.43*
Step 2				
Constant	103.90	15.45		
L. gyrus rectus/superior and medial OFG	−75.52	30.50	0.19	−0.35
R. middle OFG	−33.13	28.67	0.02	−0.16
PAI paranoia				
Step 1				
Constant	106.14	13.20		
L. gyrus rectus/superior and medial OFG	−102.80	24.74	0.25	−0.40*
Step 2				
Constant	114.63	14.30		
L. gyrus rectus/superior and medial OFG	−82.29	28.23	0.25	−0.40*
R. middle OFG	−38.72	26.54	0.03	−0.20

PAI, personality assessment inventory; OFG, orbital frontal gyrus; SE, standard error.
*Bonferroni-corrected $P < 0.01$.

medial PFC cluster and all three subscores of the PAI paranoia scale (hypervigilance: $r = -0.44$, $P < 0.017$; persecution: $r = -0.40$, $P < 0.017$; resentment: $r = -0.37$, $P < 0.005$).

Finally, it was conceivable that even subclinical psychopathology or level of education could have affected the subjective estimates of how much sleep was needed before experiencing impairments. Therefore, to address this possibility, we correlated scores on each of the hypothesized PAI scales and education (in years) with the minimum amount of sleep reported before noticeable impairment. Sleep need was not related significantly to depression ($r = 0.22$, $P = 0.11$), somatization ($r = 0.11$, $P = 0.43$) or anxiety ($r = 0.17$, $P = 0.21$), but was related to level of paranoia ($r = 0.34$, $P = 0.01$). Subjective minimum sleep need before the emergence of noticeable work impairment was correlated negatively with years of education ($r = -0.30$, $P = 0.02$). This suggests that higher education level was associated generally with a lower perceived sleep need.

DISCUSSION

Habitual sleep in excess of perceived minimal need to avoid impairment, defined here as 'sleep credit', was associated with greater grey matter volume of the medial frontal and orbitofrontal cortex, regions important to emotional perception and affective regulation. Furthermore, individual variation in grey matter volume of the medial prefrontal cortex cluster was associated with global self-perceived emotional intelligence, in particular capacities involving interpersonal skills that contribute to the ability to understand and relate well with others. Similarly, greater grey matter volume of the same cluster was correlated with reduced severity on several indices of psychopathology, particularly in terms of overall somatic complaints, depression and paranoia. In short, sleeping more than the minimum required to sustain performance was associated with increased grey matter volume in cortical areas critical to emotional regulation, and larger volume of these areas was associated with better emotional and psychological health.

Our data offer additional support to a line of converging empirical studies showing that function, structure and connectivity of medial prefrontal and orbitofrontal cortices are vital to the understanding of sleep and its relationship to behaviour. For example, decreased metabolic activity following one night of sleep deprivation was not restricted to, but predominant within, this brain region, particularly within the bilateral gyrus rectus (Thomas *et al.*, 2000). Furthermore, attentional lapses appear to be linked to diminished activity in the medial prefrontal cortex (Chee *et al.*, 2008). Volumetric data also point to an important connection between the medial prefrontal cortex and sleep-related problems. For instance, grey matter volume in the context of both sleep disorders and increased daytime sleepiness in healthy adults tends to be reduced in this region in association with greater sleep-related pathology (Altena *et al.*, 2010; Joo *et al.*, 2009; Killgore *et al.*, 2012b; Morrell, 2003). Lastly, functional connectivity of the medial prefrontal cortex with other emotional and socially relevant brain regions has been shown to be correlated inversely with sleep duration (Killgore *et al.*, 2012a) or is particularly weakened following a full night of sleep deprivation (Yoo *et al.*, 2007). Our data complement these findings by showing that grey matter structure within the medial prefrontal cortex varies systematically with habitual 'sleep credit'.

The present study also builds upon and extends previous research into the beneficial effects of excess (Anderson *et al.*, 2009) or 'banked' sleep (Rupp *et al.*, 2009a,b). Previously, it was shown that 'banking sleep' for 1 week was associated with greater resilience to, and better recovery

from, a period of insufficient sleep (Rupp *et al*., 2009a,b). In these studies, 'banking sleep' was conceptualized as getting more than habitual sleep (e.g. 10 h time in bed instead of an individual's typical 8 h). The authors argued that sleep history needs careful consideration in experimental sleep deprivation studies due to its potential moderating effect, and that one night of baseline sleep might not provide a sufficient baseline measure. Our data suggest habitual 'sleep credit' - that is, sleeping more than needed subjectively (e.g. getting 8 h if one perceives 5 h to be the point at which an impairment would be noticed) - as an additional putative moderator of the sleep–behaviour relationship. We showed that sleeping more than needed subjectively was associated with greater grey matter volume within the medial prefrontal cortex, a brain region that has been implicated strongly in sleep, sleep deprivation and their behavioural correlates. While the cross-sectional nature of our data do not allow us to draw conclusions regarding the direction of this effect, it allows us to pose the question of whether individuals who habitually sleep more than their subjective need might also be more resistant to acute sleep deprivation – possibly because of an advantageous neuronal substrate. Future studies employing longitudinal assessment, neuroimaging and acute sleep deprivation may be able to address this possibility.

Probably very few would contest that surplus sleep provides a range of benefits. Our data showed that sleeping in excess of the amount needed subjectively to avert degraded performance was linked to greater grey matter volume in a cortical region critical to both cognition and emotion (Fuster, 2008). Thus, in additional to highlighting the neurostructural benefits associated with 'sleep credit', our data replicated previous findings of behavioural deficits associated with insufficient sleep, particularly in terms of complex cognition such as emotional intelligence and psychological health (Kahn-Greene *et al*., 2007; Killgore *et al*., 2008). In those previous studies, prolonged sleep deprivation led to a degradation of both global and specific aspects of emotional intelligence, whereas psychopathological symptoms, including somatization, anxiety, depression and paranoia, increased without sleep. Our data showed that most of these same symptom dimensions, including somatization, depression and paranoia, were correlated with grey matter volume of the medial prefrontal cortex cluster, the very region that was implicated in habitually sleeping longer than needed subjectively. Longitudinal data are needed to establish whether habitual 'sleep credit' in fact induces grey matter changes in this region or whether these cortical volume differences reflect a biological substrate permitting decreased sleep need.

One previous study investigated whether the difference between habitual sleep duration and perceived sleep need was associated with a variety of sleep and trait measures such as sleep propensity, subjective daytime sleepiness, psychomotor vigilance, anxiety and personality (Anderson *et al*., 2009). Interestingly, no difference emerged in these putative behavioural correlates of 'sleep credit' (i.e. getting more sleep than needed), sleep deficit (i.e. getting less sleep than needed) and getting as much sleep as needed. However, neurostructural and higher-cognitive correlates such as emotional intelligence were not investigated, and the accumulated 'sleep credit' in our sample was, on average, greater than in the Anderson *et al.* (2009) study, suggesting limited comparability between studies. Future research should investigate whether 'sleep credit' contributes to elementary cognitive functions such as psychomotor vigilance, as well as higher-order cognitive processes such as emotional intelligence to resolve this apparent discrepancy.

Finally, although our primary hypotheses were restricted to four PAI subscales (i.e. somatization, depression, anxiety, paranoia), as previous research has shown sleep deprivation to affect these scales specifically (Kahn-Greene *et al*., 2007), we also conducted additional exploratory analyses. For comprehensiveness and to obviate any bias in reporting, we conducted multiple regressions with the remaining non-hypothesized PAI subscales (i.e. anxiety-related disorders, mania, schizophrenia, borderline features, antisocial features) at a Bonferroni-corrected threshold of $P < 0.05$. Interestingly, the specificity of the association between sleep deprivation and PAI psychopathology demonstrated by Kahn-Greene *et al.* (2007) was also observed in the present study of 'sleep credit'. Specifically, after correction for multiple comparisons, grey matter volume did not relate to any of these scales except for schizophrenia, which showed a negative association with grey matter volume within the left gyrus rectus, superior and medial OFG only (Table S1). We also found that formal education level was correlated negatively with the perceived amount of sleep necessary before functional work impairments become noticeable. While not hypothesized, this negative relationship suggests that individuals with higher educational attainment perceive themselves as able to function with less sleep before experiencing impaired performance. There are several possible explanations for this relationship, including greater cognitive ability among those with higher educational attainment, which might confer greater cognitive reserve, or individuals with lower education may have been less reliable in reporting their sleep need and impairment levels. These questions were not addressed directly in the present study and will require additional research.

The study is not without limitations and our results need to be replicated, preferably in a larger independent sample with prospective objective measurement of actual sleep via actigraphy or some method of ambulatory monitoring (Anderson *et al*., 2009). Despite including 55 healthy adults, our sample size was still at the lower end of the ideal number of subjects to be included in a multiple regression analysis with two predictors. Also, whereas most regression diagnostics (i.e. standardized residuals, Durban–Watson test statistic, collinearity statistic, Cook's distance, Mahalanobis distance, covariance ratio) did not reveal any violation of multiple regression assumptions for any of the variables of interest, hat values exceeded common thresholds on PAI scales of

somatization, anxiety and depression for a few selected subjects, suggesting possible excess leverage effects. We therefore cannot readily generalize results regarding these three variables beyond our sample. Additionally, our definition of 'sleep credit' is not unassailable. Here, we defined 'sleep credit' as the amount of sleep that an individual obtains habitually relative to that person's own subjectively perceived sleep need, defined as the threshold of sleep necessary before daytime impairment is noted. Alternatively, 'sleep credit' could be defined in relation to subjective sleep necessary for optimal performance in daily life. While conceptually similar and potentially related, 'lack of impairment' and 'optimal functioning' are clearly distinct constructs that may be affected differentially by sleep loss. It is likely that the behavioural and brain structural correlates of excess sleep beyond an 'optimal functioning' threshold would be different to those explored here examining excess sleep beyond a 'no impairment' threshold. Teasing apart the role of sleep and brain structure on each of these deserves further study. It remains to be shown whether and how differences in operationalization of 'sleep credit' might influence findings of grey matter correlates and their association with emotional intelligence and mental health. Future investigations should also include a more detailed assessment of the type of impairment (e.g. worse memory, difficulties in concentration, changes in emotional response and expression) that is noticed when sleep is less than subjective need, including daytime sleepiness (Killgore *et al*., 2012b). Also of interest might be an assessment of each participant's specific work situation and job requirements to determine whether the effects of 'sleep credit' might depend upon such work-setting factors. Furthermore, it may be useful to explore the reasons why some participants habitually obtain the sleep they need subjectively while others do not. Lastly, readers should bear in mind that these data are correlational, and therefore causality cannot be inferred.

In conclusion, this is the first voxel-based morphometric study to show that sleeping in excess of subjective minimal need is associated significantly with greater grey matter volume within the medial prefrontal cortex, a region critical to the monitoring and control of affective processes. Notably, the volume of this same region was correlated independently with greater emotional intelligence and lower scores on indices of psychopathology in the same participants. These data support the notion that sleeping beyond the minimal subjective requirements for adequate performance may affect brain structure and relevant emotional capacities.

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CONFLICTS OF INTEREST

No conflicts of interests declared.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Table S1. Stepwise linear regression of grey matter volume correlates of non-hypothesized PAI psychopathology.