Research Article/ Popularised GENRE to IMRAD. ARCHEOLOGICAL EXPLORATION

1 Dark puffy eyes, a feeling of deep exhaustion, and a foul mood to match – we've all experienced the side effects of a lack of sleep. It's no wonder that sleep-deprivation has been used as a method of torture.	12 The volunteers were put through two rounds of tests while their brains were scanned, both the day after a good night's sleep and after being awake for 24 hours. In one test, volunteers were asked to give the direction in which yellow dots moved on a screen.
11 Moreover, a region of the brain called the amygdala, which is known to play a part in emotion, fired up in response only to emotional images when the volunteers had had a good night's sleep. But when they were sleep-deprived, it reacted to neutral images in the same way as emotional ones	2 Together, the experiments suggest that when we're sleep-deprived we tend to see normal, everyday situations as particularly worthy of our attention, says Hendler. "You lose neutrality," she says. "The ability of the brain to tell what's important is compromised – it's as if everything is important."
3 Sleep plays a key role in protecting our emotional well-being. A single night of sleep deprivation (SD) is known to trigger emotional difficulties (Pilcher and Huffcutt, 1996; Anderson and Platten, 2011), leading to increased subjective stress and anxiety (Minkel et al., 2012) and enhanced sympathetic reactions to unpleasant stimuli (Zhong et al., 2005; Franzen et al., 2009).	10 Despite emerging links between sleep loss and emotional reactivity, it remains unclear how sleep modulates cognitive control of emotion and its resulting emotional profile. Given previous indications of prefrontal and limbic vulnerability to sleep loss, we aimed to directly examine the effect of sleep on cognitive control of emotion, using two complimentary cognitive–emotional tasks (recorded using fMRI and EEG), while manipulating sleep in a within-subject design.

9 *Participants*. Eighteen adults (age range, 23–32 years; mean, 26.8 _ 3 years; 10 females) completed a repeated-measures crossover design. Participants were healthy with no history of sleep, neurologic, or psychiatric disorders (assessed using a detailed medical history questionnaire). Normal sleep–wake patterns were further validated using actigraphy and subjective sleep logs, as detailed below.	4 *fMRI data acquisition and preprocessing.* Imaging was performed on a 3T GE Horizon echo speed scanner with a resonant gradient echoplanar imaging system (GE Healthcare). All images were acquired using a standard head coil. The scanning session included functional T2*-weighted images (FOV, 200 mm; matrix size, 96 _ 96; voxel size, 3 _ 3 _ 4; TR, 3000 ms; TE, 35 ms; slice thickness, 4 mm; 32–39 slices without gap, oriented according to the fourth ventricle; flip angle, 90°) and a three dimensional anatomical scan using T1 spoiled gradient-recalled acquisition in a steady state sequence (1 _ 1 _ 1 mm).
5 After SD, amygdala activity no longer differentiated between distractors, responding equally to both neutral and negative distractors (t(16)_0.1; for the right amygdala, t(16)__0.38, both p_0.7).	7 Furthermore, the change in ACC–amygdala connectivity was significantly correlated with the decrease in accuracy scores after SD (r_0.63, p_0.007). These results are depicted in Figure 6.
8 These findings may further suggest that neutral stimuli could be regarded as emotionally ambiguous (Cooney et al., 2006), with their processing dependent on intact cognitive control of emotion. If so, processing of neutral stimuli could shift toward emotional saliency as the threshold for limbic activation is altered by sleep loss.	6 Such an encompassing effect of SD on the threshold for emotional activation, expressed in both limbic and task-related regions, could imply the involvement of PFC dysregulation. As mentioned above, the PFC is particularly vulnerable to sleep loss, and its medial regions are known to play a key role in emotion regulation via top-down modulation of limbic regions, such as the amygdala (Davidson, 2002; Sotres-Bayon et al., 2004).

Research Article/ Popularised GENRE to IMRAD. CORRECTION

Dark puffy eyes, a feeling of deep exhaustion, and a foul mood to match – we've all experienced the side effects of a lack of sleep. It's no wonder that sleep-deprivation has been used as a method of torture. New Scientist	The volunteers were put through two rounds of tests while their brains were scanned, both the day after a good night's sleep and after being awake for 24 hours. In one test, volunteers were asked to give the direction in which yellow dots moved on a screen. New Scientist
Moreover, a region of the brain called the amygdala, which is known to play a part in emotion, fired up in response only to emotional images when the volunteers had had a good night's sleep. But when they were sleep-deprived, it reacted to neutral images in the same way as emotional ones New Scientist	Together, the experiments suggest that when we're sleep-deprived we tend to see normal, everyday situations as particularly worthy of our attention, says Hendler. "You lose neutrality," she says. "The ability of the brain to tell what's important is compromised – it's as if everything is important." New Scientist
Introduction Sleep plays a key role in protecting our emotional well-being. A single night of sleep deprivation (SD) is known to trigger emotional difficulties (Pilcher and Huffcutt, 1996; Anderson and Platten, 2011), leading to increased subjective stress and anxiety (Minkel et al., 2012) and enhanced sympathetic reactions to unpleasant stimuli (Zhong et al., 2005; Franzen et al., 2009).	**Introduction** Despite emerging links between sleep loss and emotional reactivity, it remains unclear how sleep modulates cognitive control of emotion and its resulting emotional profile. Given previous indications of prefrontal and limbic vulnerability to sleep loss, **we** aimed to directly examine the effect of sleep on cognitive control of emotion, using two complimentary cognitive–emotional tasks (recorded using fMRI and EEG), while manipulating sleep in a within-subject design.

Materials and methods *Participants*. Eighteen adults (age range, 23–32 years; mean, 26.8 _ 3 years; 10 females) completed a repeated-measures crossover design. Participants were healthy with no history of sleep, neurologic, or psychiatric disorders (assessed using a detailed medical history questionnaire). Normal sleep–wake patterns were further validated using actigraphy and subjective sleep logs, as detailed below.	Materials and methods *fMRI data acquisition and preprocessing.* Imaging was performed on a 3T GE Horizon echo speed scanner with a resonant gradient echoplanar imaging system (GE Healthcare). All images were acquired using a standard head coil. The scanning session included functional T2*-weighted images (FOV, 200 mm; matrix size, 96 _ 96; voxel size, 3 _ 3 _ 4; TR, 3000 ms; TE, 35 ms; slice thickness, 4 mm; 32–39 slices without gap, oriented according to the fourth ventricle; flip angle, 90°) and a three dimensional anatomical scan using T1 spoiled gradient-recalled acquisition in a steady state sequence (1 _ 1 _ 1 mm).
Results After SD, amygdala activity no longer differentiated between distractors, responding equally to both neutral and negative distractors (t(16)_0.1; for the right amygdala, t(16)__0.38, both p_0.7).	Results Furthermore, the change in ACC–amygdala connectivity was significantly correlated with the decrease in accuracy scores after SD (r_0.63, p_0.007). These results are depicted in Figure 6.
These findings may further suggest that neutral stimuli could be regarded as emotionally ambiguous (Cooney et al., 2006), with their processing dependent on intact cognitive control of emotion. If so, processing of neutral stimuli could shift toward emotional saliency as the threshold for limbic activation is altered by sleep loss.	Such an encompassing effect of SD on the threshold for emotional activation, expressed in both limbic and task-related regions, could imply the involvement of PFC dysregulation. As mentioned above, the PFC is particularly vulnerable to sleep loss, and its medial regions are known to play a key role in emotion regulation via top-down modulation of limbic regions, such as the amygdala (Davidson, 2002; Sotres-Bayon et al., 2004).

Sleep deprivation has been shown recently to alter emotional processing possibly associated with reduced frontal regulation. Such impairments can ultimately fail adaptive attempts to regulate emotional processing (also known as cognitive control of emotion), although this hypothesis has not been examined directly. Therefore, we explored the influence of sleep deprivation on the human brain using two different cognitive–emotional tasks, recorded using fMRI and EEG. Both tasks involved irrelevant emotional and neutral distractors presented during a competing cognitive challenge, thus creating a continuous demand for regulating emotional processing.