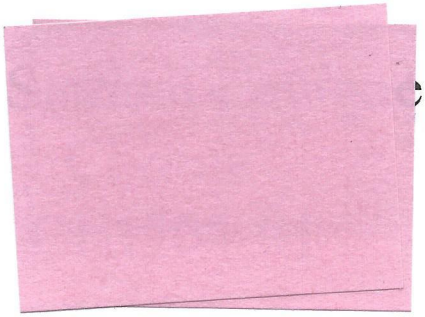
# Projet : L'article de presse scientifique erroné ou pas ?

Travail présenté à Robin St-Pierre



École secondaire des Patriotes-de-Beauharnois

Le 29 mai 2017

Article





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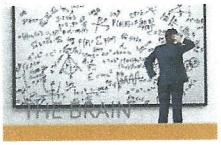

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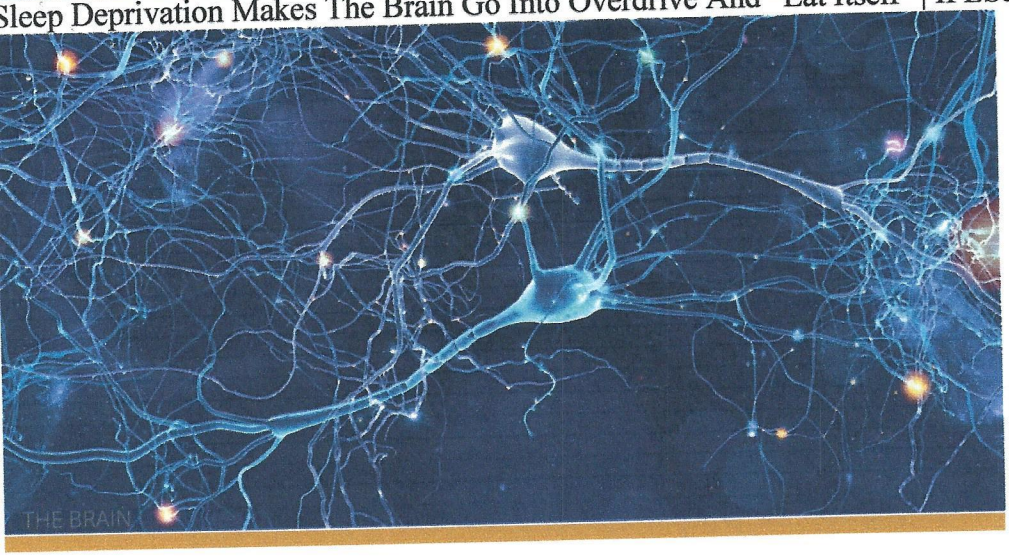


GET SOME SLEEP! SHUTTERSTOCK/WHITEHOUNE



The never-ending quest for the secrets of sleep have taken us to a point where the brain explores its very self. Now, a new study suggests that chronic sleep deprivation can make the brain "eat itself", as the cells that digest cellular debris go into overdrive.

**By Kristy Hamilton**
26/05/2017, 17:33

As we know, sleep is crucial to the proper functioning of the brain. While we slumber, toxic byproducts from the day are cleared out – a bit of neural housekeeping, if you will, to keep things in working order.

Part of this hardworking team is microglia cells, whose job it is to ingest waste products from the nervous system, gobbling up the cellular debris of worn-out and dead cells. Another worker, astrocytes, are the multi-taskers of the brain, performing a range of functions and duties – one of which includes pruning unnecessary synapses to help rewire the brain (and you thought your job was important).

For the study, published in the Journal of Neuroscience, the team tested four groups of mice: The first were allowed to sleep for as long as they wanted, the second were periodically woken up, the third stayed awake for an extra eight hours, and the fourth were sleep-deprived for five days in a row.

In the well-rested mice, astrocytes were active in 6 percent of the synapses, whereas the eight-hour group showed 8 percent astrocyte

eaten by astrocytes because of sleep loss," neuroscientist Michele Bellesi from the Marche Polytechnic University in Italy told New Scientist. However, the team didn't specify whether this activity is detrimental or helpful during dire sleep loss.

The discovery that was more intriguing, worrying, and in need of follow-up was the activity of the microglia. They too were more active, but only after chronic sleep loss – a sustained activation previously linked to Alzheimer's and other forms of neurodegeneration.

As the authors write: "Chronic sleep loss activates microglia cells and promotes their phagocytic [digesting waste] activity, apparently without overt signs of neuroinflammation, suggesting that extended sleep disruption may prime microglia and perhaps predispose the brain to other forms of insult."

The most complex organ of all, the brain is constantly creating, strengthening, and deconstructing an intricate labyrinth of passages in our brain. It is a never-ending project where some biological structures get constructed and others get broken. Further work will help clarify whether this astrocyte activity and microglia activation protects or hinders the brain in times of distress. But just in case, get more sleep!

Source initial

JNeurosci
THE JOURNAL OF NEUROSCIENCE
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Research Article | Research Articles, Cellular/Molecular

# Sleep Loss Promotes Astrocytic Phagocytosis and Microglial Activation in Mouse Cerebral Cortex

Michele Bellesi, Luisa de Vivo, Mattia Chini, Francesca Gilli, Giulio Tononi, and Chiara Cirelli

Journal of Neuroscience 24 May 2017, 37 (21) 5263-5273; DOI: https://doi.org/10.1523/JNEUROSCI.3981-16.2017



**Abstract**

We previously found that *Mertk* and its ligand *Gas6*, astrocytic genes involved in phagocytosis, are upregulated after acute sleep deprivation. These results suggested that astrocytes may engage in phagocytic activity during extended wake, but direct evidence was lacking. Studies in humans and rodents also found that sleep loss increases peripheral markers of inflammation, but whether these changes are associated with neuroinflammation and/or activation of microglia, the brain's resident innate immune cells, was unknown. Here we used serial block-face scanning electron microscopy to obtain 3D volume measurements of synapses and surrounding astrocytic processes in mouse frontal cortex after 6–8 h of sleep, spontaneous wake, or sleep deprivation (SD) and after chronic (~5 d) sleep restriction (CSR). Astrocytic phagocytosis, mainly of presynaptic components of large synapses, increased after both acute and

chronic sleep loss relative to sleep and wake. MERTK expression and lipid peroxidation in synaptoneurosomes also increased to a similar extent after short and long sleep loss, suggesting that astrocytic phagocytosis may represent the brain's response to the increase in synaptic activity associated with prolonged wake, clearing worn components of heavily used synapses. Using confocal microscopy, we then found that CSR but not SD mice show morphological signs of microglial activation and enhanced microglial phagocytosis of synaptic elements, without obvious signs of neuroinflammation in the CSF. Because low-level sustained microglia activation can lead to abnormal responses to a secondary insult, these results suggest that chronic sleep loss, through microglia priming, may predispose the brain to further damage.

**SIGNIFICANCE STATEMENT** We find that astrocytic phagocytosis of synaptic elements, mostly of presynaptic origin and in large synapses, is upregulated already after a few hours of sleep deprivation and shows a further significant increase after prolonged and severe sleep loss, suggesting that it may promote the housekeeping of heavily used and strong synapses in response to the increased neuronal activity of extended wake. By contrast, chronic sleep restriction but not acute sleep loss activates microglia, promotes their phagocytic activity, and does so in the absence of overt signs of neuroinflammation, suggesting that like many other stressors, extended sleep disruption may lead to a state of sustained microglia activation, perhaps increasing the brain's susceptibility to other forms of damage.

astrocyte cortex microglia mouse sleep sleep deprivation

Historique: Facebook acceuil le 26 mai 2017 à 18:30h-I Fucking Love Science le 26 mai mai 2017 à 17:33h -The Journal of neuroscience le 24 mai 2017

Crédibilité de la source : Pour vérifier la crédibilité de la source j'ai fait une recherche à l'aide de Schoolar sur les principaux auteurs de l'articles orginals et ceux-ci ont des exploits scientifiques renomeée et l'expérience du sommeil restreint y étais compris parmis certains.

Protagonistes : Les scientifiques oeuvrant dans le domaine de la neuroscience Michele Belesi,Luisa de Vivo,Mattia Chini,Francesca Gilli,Giulio Tononi, Chiara Cirelli tous de l'universitée du Wisconsin et quel que rat de laboratoire.

Faits:L'étude fait, démontre que le manque de sommeil intense augmente les marqueurs périphériques de l'inflammation et ces changements seraient liées à la capacité a l'homme de produire des cellules immunitaires dans le cerveau et donc cette surproduction de cellule immunitaire ce qui prédisposerait le cerveau a ce faire mal lui-même.L'article n'a été rejetté par

aucune communauté scientifique et été qualifé comme vrais.

Citation :
Comment ont-ils procédés?:Here we used serial block-face scanning electron microscopy to obtain 3D volume measurements of synapses and surrounding astrocytic processes in mouse frontal cortex after 6–8 h of sleep, spontaneous wake, or sleep deprivation (SD) and after chronic (~5 d) sleep restriction (CSR).

Qu'ont-ils trouvés exactement ?: Using confocal microscopy, we then found that CSR but not SD mice show morphological signs of microglial activation and enhanced microglial phagocytosis of synaptic elements, without obvious signs of neuroinflammation in the CSF. Because low-level sustained microglia activation can lead to abnormal responses to a secondary insult, these results suggest that chronic sleep loss, through microglia priming, may predispose the brain to further damage.

Conclusion:Mon avis sur l'article en me basant sur les critères 2 à 6 de la liste d'étape est que je n'ai rien trouvé qui m'empècherait de croire que cette article n'est pas erroné,en d'autres mots cette article es selon moi parfaitement vrais et en plus sur une base scientifique il n'est pas nouveau qu'il y a une corrélation entres des problèmes de santé et le manque de sommeil.