臨床心理学特講8 「眠りを疎かにしている日本社会」

眠りに関する基礎知識を得たうえ で、「ヒトは寝て食べて始めて活動 できる動物である」との当然の事 実を確認し、現代日本が抱えてい る問題のかなりの部分に、我々が 動物であることの謙虚さを失い、 眠りを疎かにしたことの報いが及 んでいることを認識していただけ ればと思います。そして願わくばこ の講義が皆さんの今後の生き方 を考える際の一助になれば幸いで す。

開始は13時20分

	ידי ש שי	
1	9月30日	眠りの現状
2	10月14日	眠りを眺める
3	10月21日	眠るのは脳
4	10月28日	寝不足では・・・
5	11月 4日	眠りと年齢
6	11月11日	眠りさえすればいつ寝てもいい?
7	11月25日	眠りと物質
8	12月 2日	様々な眠り
9	12月 9日	ヒトと光
10	12月16日	睡眠関連病態
11	1月 6日	睡眠衛生の基本
12	1月13日	眠りの社会学
13	1月20日	講演会形式
14	1月27日	まとめと試験

眠りを眺める

• 眠りを眺めて感じることは?

ヒトの状態(State)

3つの要素、脳波と眼球運動と筋活動、で定義

	脳波	眼球運動	筋活動

ヒトの状態(State)

脳波、眼球運動、筋活動で定義

			脳波	眼球運動	筋活動
覚醒	活発		β波	急速・穏徐	活発
	安静		α波	急速・穏徐	活発
睡眠	レム睡眠		低振幅	急速	消失
	ノンレム睡眠	睡眠段階1	α波が50%以下	穏徐	活発
		睡眠段階2	紡錘波	なし	やや低下
		睡眠段階3	高振幅徐波が 20%以上	なし	低下
		睡眠段階4	高振幅徐波が 50%以上	なし	かなり低下

Take home message 2.

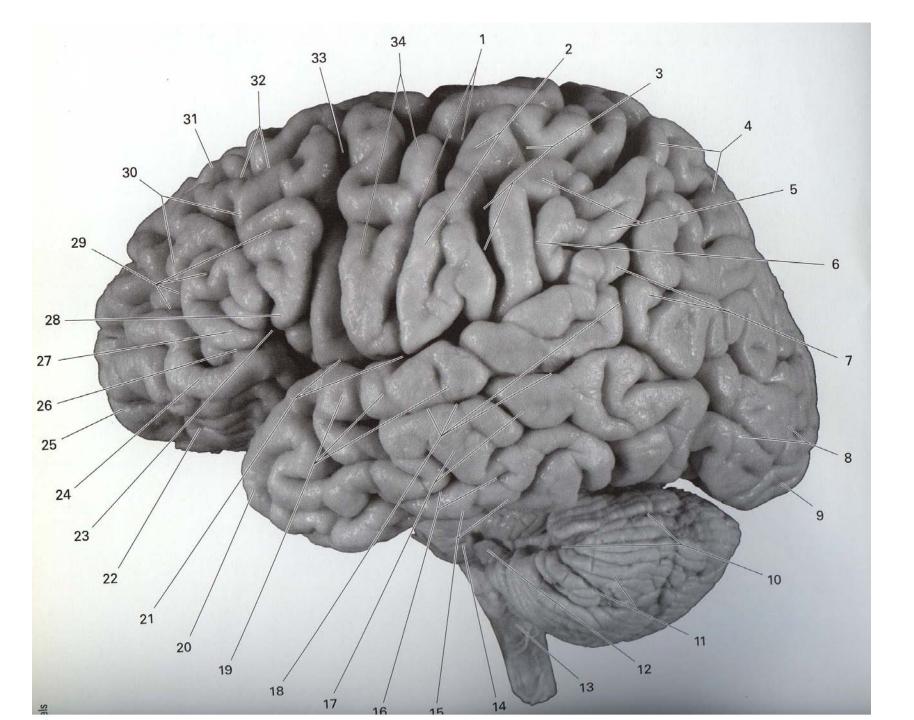
眠りを眺めるポイントは、目の動きと脳波と筋肉。

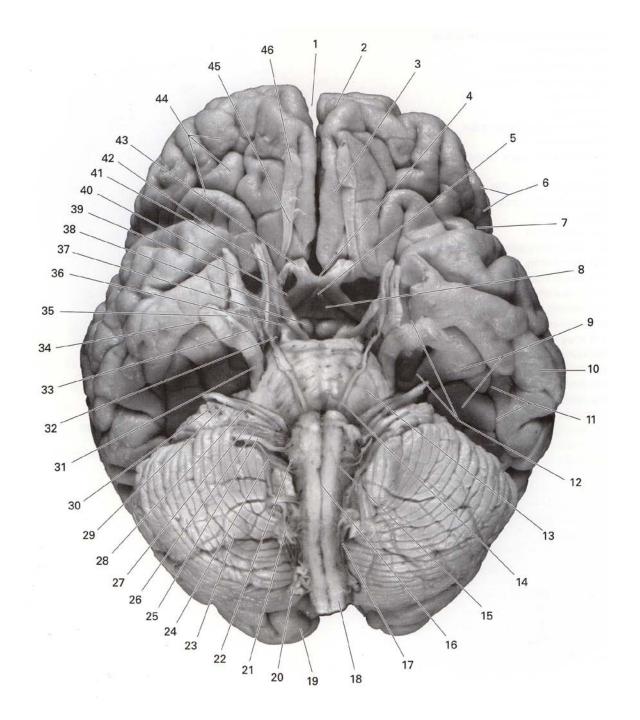
ひろふみ君のビデオ

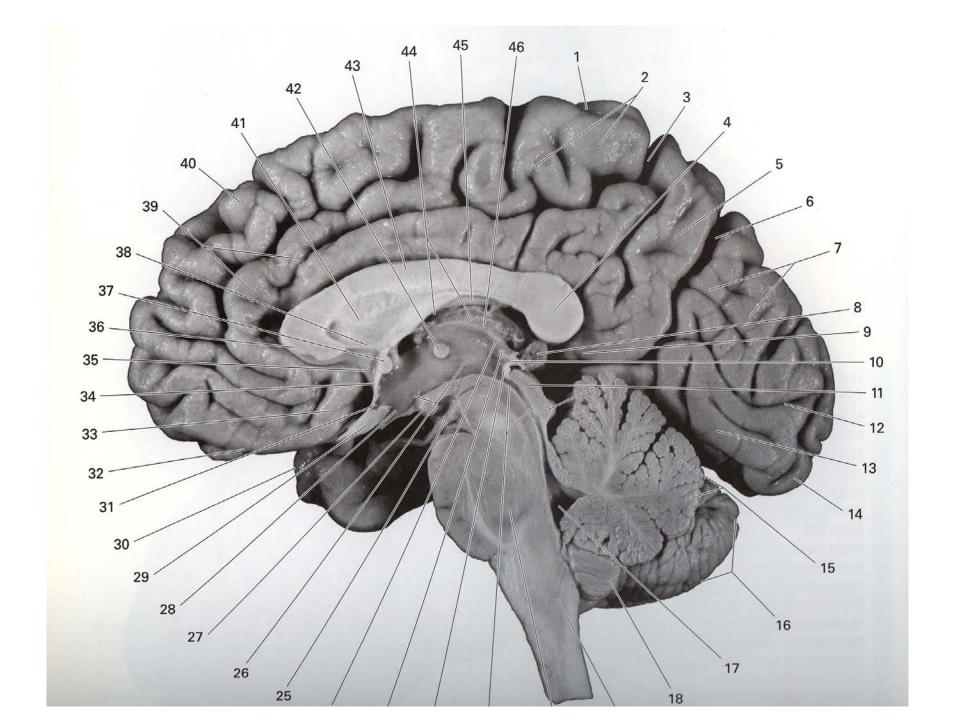
- 驚き、衝撃、イライラ、怒り、ショック、悲しい、怖い、腹がたった、唖然何に? 2歳児の行動、親の態度、子どもの夜ふかしの現状、ひきこもり予備軍、夜中に子どもを見かける、小さなおっさん、小さな体の大人、三角形が書けない5歳児、遊び場がない、無責任な周囲の大人(社会)
- 自分が親になったら 親の義務
- あらためて大切と感じたこと三食、運動、遊び、しかること、早起き早寝朝ごはん
- 知ったこと
 子どもは自然に寝るのではない、寝かしつけ、メラトニン、
 夜寝ることの重要性、眠りの影響の範囲の広さ、心と眠りは繋がっている、
 夜ふかしがしやすい仕組み(生体時計)
- 親に感謝、今後の社会への不安、今の自分の見直し(行動変容)

眠るのは脳

• :課題:あなたが脳を感じるとき。







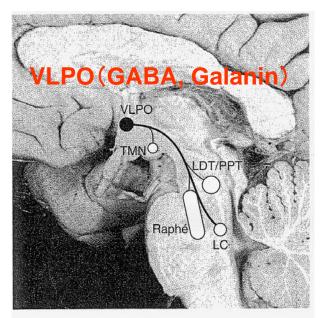


図 32 腹側外側視索前野 (ventrolateral preoptic area: VLPO) からの投射をヒト脳の正中矢状断面に示す

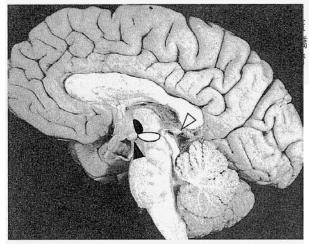
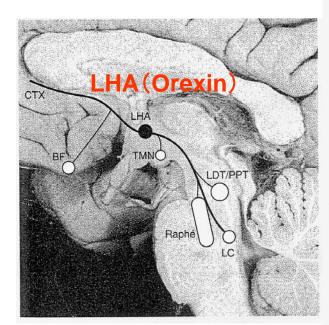
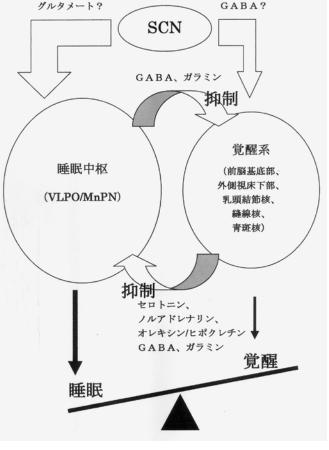


図 31 Economo の睡眠調節中枢を示すヒト脳の正中 矢状断面

白抜きの部分の病変が傾眠をもたらし、黒塗りの部分 の病変が不眠をもたらす。白抜きの矢頭は松果体を、 黒塗りの矢頭は乳頭結節を示す。





黒塗り病変で不眠 →

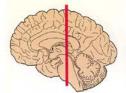
睡眠中枢(視床下部前部)

VLPO(GABA, Galanin)

白塗り病変で傾眠 →

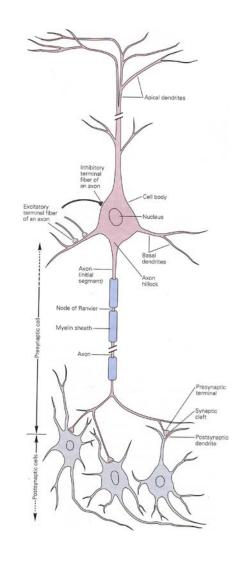
覚醒中枢(視床下部後部)

TMN(Histamine), LHA(Orexin)

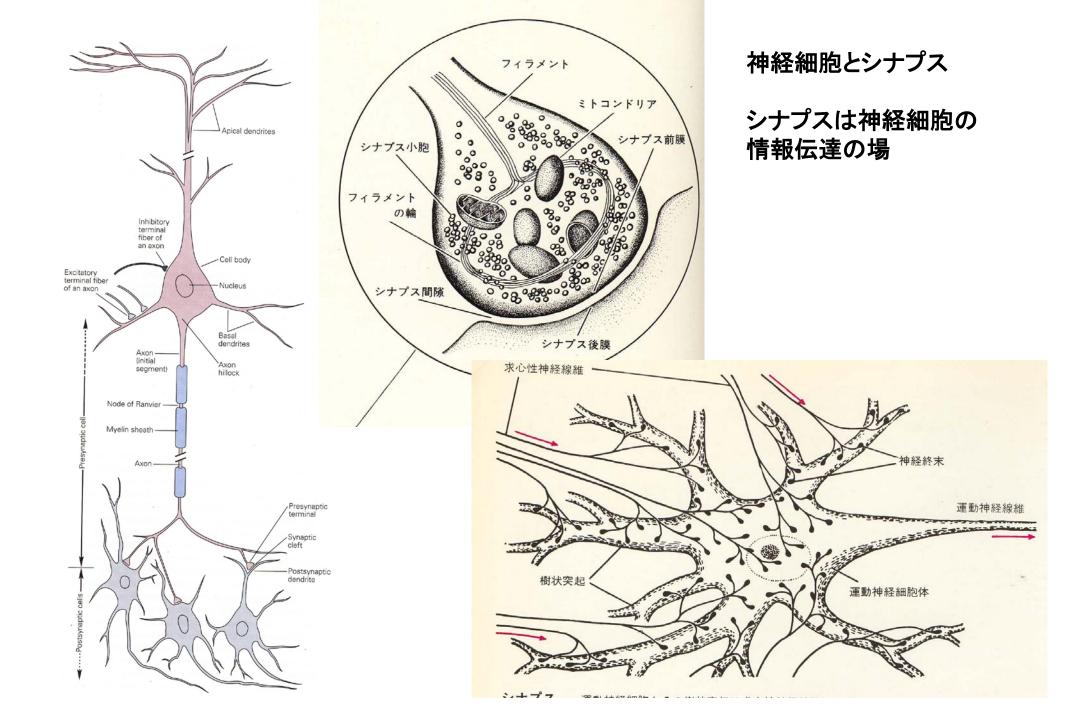


Coronal Section Through Posterior Commissure (1X) with Vessel Territories (0.7X)





大脳の表層は灰白質(神経細胞) 大脳の深層は白質(神経線維)

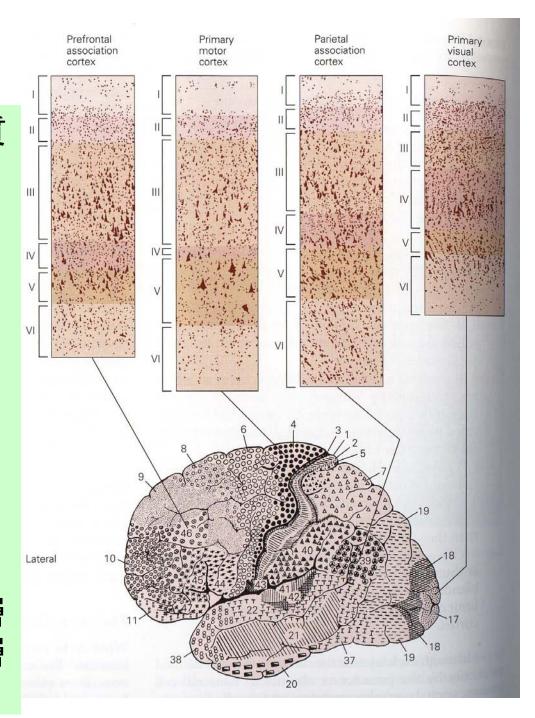


大脳表層の様子

大脳皮質(Cortex):大脳表面の皮質は約140億個の神経細胞から構成され、それらの神経細胞がシナプスによって複雑に結びつき神経回路網を形成しています。神経細胞の周囲にはグリア細胞があり、神経細胞に栄養を与えたり、脳の構造を保つのに役立っています。

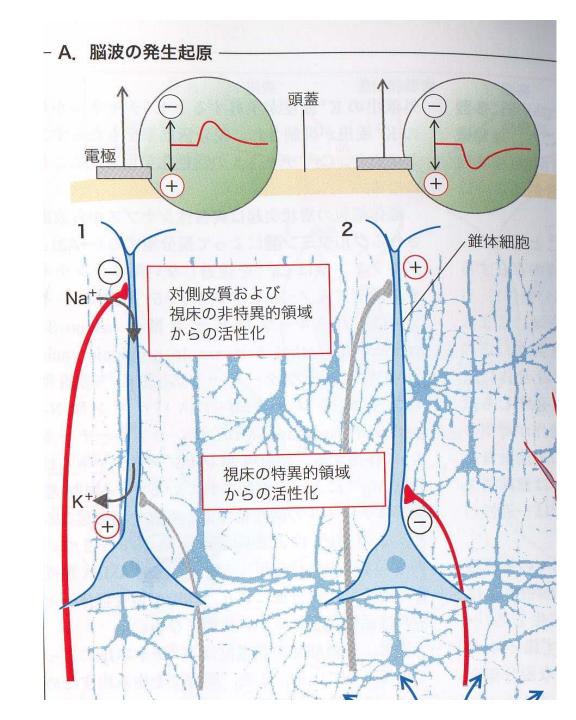
皮質の構造:

(1)6層構造: ヒトの大脳皮質には 6層構造を持つ部分が見られます。 これらは(1)叢状層(2)小錐体細胞 層(3)中錐体細胞層(4)星状細胞層 (5)大錐体細胞層(6)紡錐体細胞層 の6層からなっています。



脳波とは?

大脳表層の錐体細胞の 樹状突起に発生するシナプ ス後電位の総和を反映。

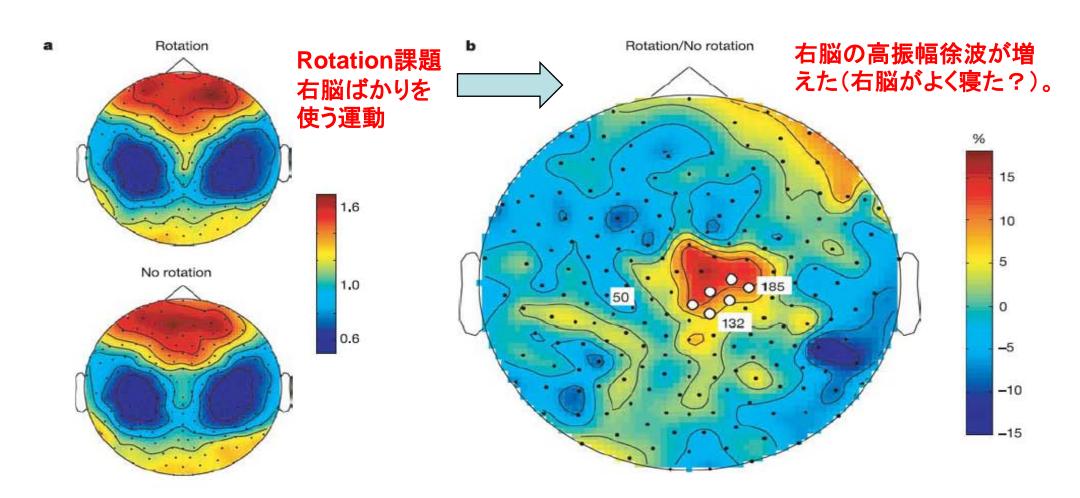


Local sleep and learning

Reto Huber¹, M. Felice Ghilardi², Marcello Massimini¹ & Giulio Tononi¹

特定の脳領域のみを利用する学習課題を 課すことで、その領域での徐波睡眠活動 量が局所的に増大した。

さらに学習の後に起こるSWAの局所的増大は、睡眠後の課題成績の増進と相関していることもわかった。



¹Department of Psychiatry, University of Wisconsin, Madison, Wisconsin 53719, USA

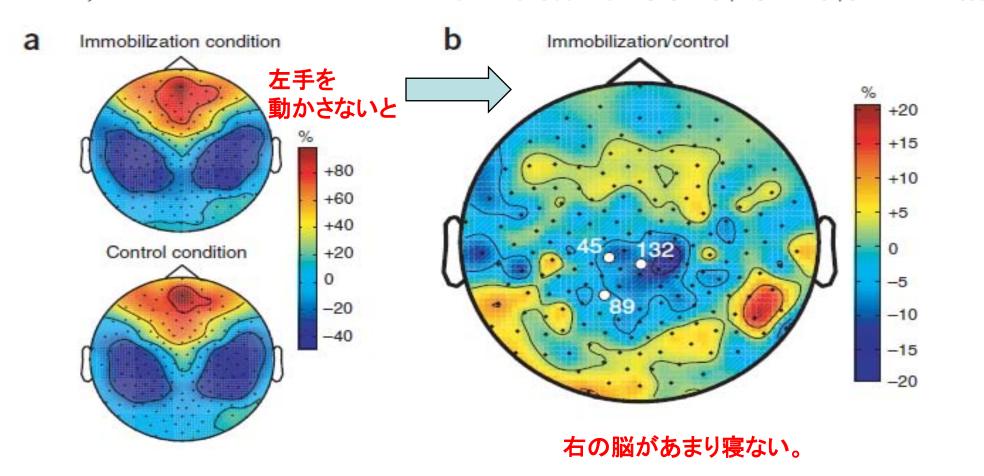
²Center for Neurobiology and Behavior, Columbia College of Physicians and Surgeons, New York, New York 10032, USA NATURE | VOL 430 | 1 JULY 2004 |

Arm immobilization causes cortical plastic changes and locally decreases sleep slow wave activity

Reto Huber¹, M Felice Ghilardi², Marcello Massimini¹, Fabio Ferrarelli¹, Brady A Riedner³,

Michael J Peterson¹ & Giulio Tononi¹

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たとえば手を握ろう。そう考えると、脳のなかの手を握ろうという意思を司る神経細胞が活動します。神経細胞が活動するということは、活動電位が生じることで、この活動電位は神経細胞から伸びるひも一軸策一を伝わり、途中いくつかの神経細胞を経由して、最終的には大脳の表層の灰白質のなかの運動野と呼ばれる場所にある手の運動を司る神経細胞との接続部分であるシナプスに到達します。

シナプスというのは神経細胞から伸びる軸策の先っぽと、別の細胞、今の場合は運動野にある手の運動を司る神経細胞ですが、その細胞との間にあるごく狭い隙間のことです。軸策の先っぽに活動電位が到着すると、軸策の先っぽから神経伝達物質が放出されます。シナプスに出た神経伝達物質は、シナプスに面した神経細胞の表面にある受容体にくっつきます。すると神経伝達物質の種類等に応じて神経細胞が影響を受けます。この場合は運動野にある手の運動を司る神経細胞に活動電位が惹起され、その活動電位が、今度は大脳の運動野の神経細胞から伸びる軸策が延々と脊髄の中を伝わって脊髄の前のほうの部分にある運動細胞とのシナプスにまで到達します。

運動細胞から伸びる軸策は手を握ることに関わるいくつかの筋肉に向かって伸びています。ですから運動細胞で発生した活動電位は筋肉に伝わり、「手を握る、という」運動となるのです。

Sleeping to Reset Overstimulated Synapses

The purpose of sleep is one of the toughest puzzles in biology. Some scientists think animals slumber primarily to save energy. Others have proposed that sleep has special relevance for learning and memory. A newer hypothesis borrows from both ideas, suggesting that sleep dials down synapses that have been cranked up by a day's worth of neural activity. Because stronger synapses use more energy and take up more space, the thinking goes, this synaptic cooldown helps conserve both energy and precious real estate in the brain. It also ensures that synapses don't max out and lose the ability to grow stronger if

they're called upon to encode some new experience into memory the next day.

In this week's issue, two studies with fruit flies provide what some researchers say is the most compelling evidence to date for this provocative hypothesis. One finds that levels of several synaptic proteins increase during wakefulness and decline during sleep; the other finds a similar rise and fall in synapse number. "Together, these findings very clearly demonstrate that one major function of sleep is to reduce, on a structural level, synaptic connectivity in the brain," says Jan Born, a neuroscientist who studies sleep at the University of Lübeck in Germany and was not involved with either study.

The so-called synaptic homeostasis hypothesis was first protransmitter release, declined after flies had a chance to sleep. This pattern held up even when flies slept at odd hours, confirming that the proteins fluctuate with the sleep-wake cycle, not the time of day.

The second paper, on page 105, describes changes in synapse number during sleep. But the experiments weren't conceived as a direct test of the synaptic homeostasis hypothesis, says senior author Paul Shaw of Washington University in St. Louis, Missouri. Instead, the goal was to investigate how daytime activities influence subsequent sleep. Shaw's lab had previously found that flies sleep

Sleepless synapses. After 16 hours without sleep (bottom panel), synaptic protein levels increase (indicated by warm colors) in the fruit fly brain.

enough to restore increased sleep after social enrichment.

These findings provide an intriguing link between two major regulators of sleep, Cirelli says. The circadian clock tells animals when to sleep, she explains, but the duration of sleep depends on how long they've been awake and what they've done during that time. The new findings suggest that some of the same cells and genes involved in regulating the circadian clock may also be involved in sensing sleep need.

In the same paper, Donlea and colleagues also report findings that seem to support the

> synaptic homeostasis hypothesis: They found that the same social experiences that increase the need for sleep also increase the number of synapses between lateral ventral neurons and their partners in the brainstem. After sleep, synapse numbers had declined.

Together, the two papers provide compelling evidence for synaptic downscaling during sleep, says Robert Stickgold, a neuroscientist at Harvard University who was initially skeptical of Tononi and Cirelli's hypothesis. Even so, Stickgold thinks it's unlikely that downscaling happens only during sleep or that synaptic strengthening is limited to waking hours. Human and rodent studies have suggested, for example, that sleep may be important for consolidating newly formed memories (Science, 9 March 2007, p. 1360), a process

Sleeping to Reset Overstimulated Synapses

The purpose of sleep is one of the toughest transmitter release declined after this had a grouph to rectangle

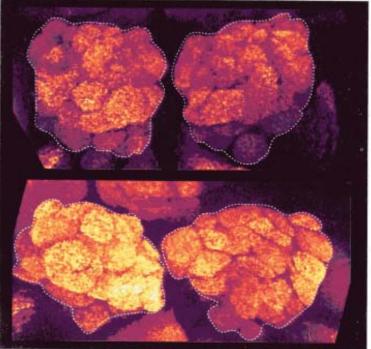
ショウジョウバエでは、複数の神経連結 (連絡)蛋白質の発現が覚醒後に亢進し、 睡眠後に低下する。

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Sleep is of the brain, by the brain and for the brain

J. Allan Hobson¹

Sleep is a widespread biological phenomenon, and its scientific study is proceeding at multiple levels at the same time. Marked progress is being made in answering three fundamental questions: what is sleep, what are its mechanisms and what are its functions? The most salient answers to these questions have resulted from applying new techniques from basic and applied neuroscience research. The study of sleep is also shedding light on our understanding of consciousness, which undergoes alteration in parallel with sleep-induced changes in the brain.

"government of the people, by the people, for the people"は「人民から構成する、人民による、人民のための行政」という意味。1380年にイギリスで出版された<u>旧約聖書にジョン・ウィクリフが</u>序文として書き込んだ文章であり、<u>牧師のセオドア・パーカー</u>が著書で紹介したのを引用したものと思われる。ゲティスバーグ演説

Take home message 2.

Sleep is of the brain, by the brain, and for the brain.

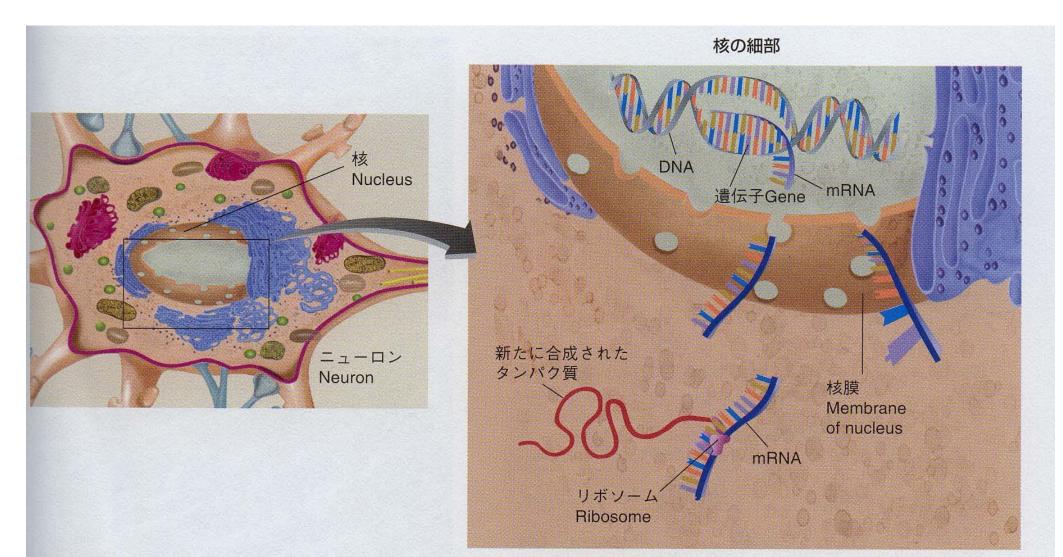
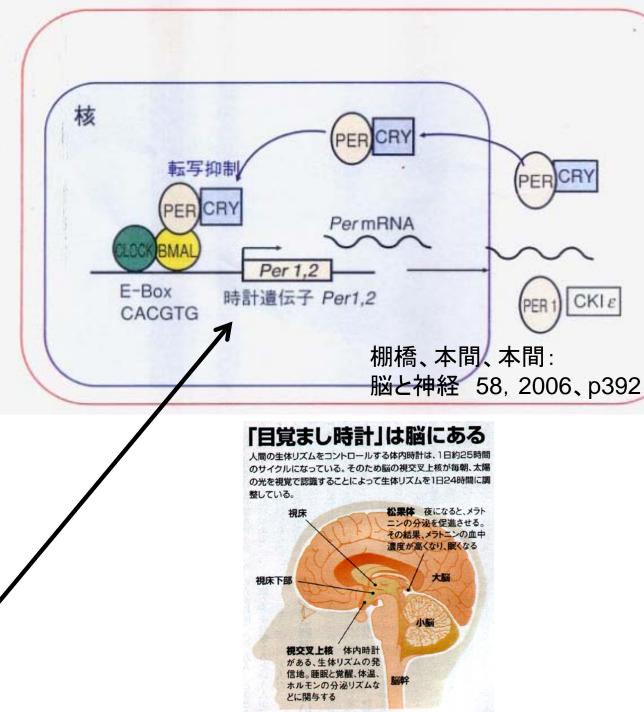


図 2.6

タンパク質の合成. 遺伝子が活性化すると、メッセンジャー RNA 分子に情報のコピーがつくられる. メッセンジャー RNA は核を離れ、リボソームに付いて、そこでタンパク質が合成される.





視交叉上核への刺激の時刻が 時計遺伝子産物レベルに与える影響



PER1 転写レベル

Rosenwasser & Turek Principles and Practice of Sleep Medicine 2005, 355