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Review Article

Construction of Virtual Neuron and Consolidation of Sleep and Memory Process– A Molecular Docking and Biomathematical Approach

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Abstract

Sleep plays an important role in learning and memory processing in our daily life. There is also a direct correlation to insufficient sleep increasing the risk of diabetes. As sleep can be broadly divided into SWS sleep and REM sleep within a SWS_REM cycle, this paper presents a literature review aiming to firstly summary the sequential contributions of these two kinds of sleep on declarative and procedural memory consolidation and then explain the neurobiological root of memory impairment under sleep deprivation. An attempt was made to formulate some mathematical models on neuron of sleep and memory process which are the important physiological phenomena of human beings. We find out that Nervousness, Age and Sex have great influence on sleep and memory quality, among which Nervousness has the strongest influence on sleep and memory quality. As a stepping stone in this direction, the present study is designed with a molecular docking and biomathematical approach to formulate some speculations to construct virtual neuron and consolidation of sleep and memory process.

Keywords: Virtual neuron; Two-stage model; SWS sleep; REM sleep; Memory consolidation; Sleep deprivation

Introduction

The relationship between sleep and memory never fails to fascinate human beings as we always keep curious about how the memory form and consolidate [1]. Nevertheless, recent developments in molecular genetics, neurophysiology, and the cognitive neurosciences have produced a striking body of research that provides converging evidence for an important role of sleep in learning and the reprocessing of memories. Also, sleep deprivation has become a prevalent public health epidemic with a wide range of harmful consequences, including memory and cognitive impairment [2], some scientists tend to focus attention on the neurobiological root of this universal human experience.

Previous Work

Effects of sleep stage on consolidation early study of different sleep stages in memory consolidation in rats mainly focused on REM sleep (Random Eye Movement sleep) and the consequences of REMD (REM sleep deprivation) by repeatedly waking subjects at the first signs of REM sleep. All these memory tasks were typically emotionally loaded as it has been proved that REM sleep priority to improve the consolidation of memory emotions [3,4], and it turned out that REMD is only valid for a specific period of time after learning — the so-called "REM sleep window" [5,6].

The first evidence of the causal role of SWS (Slow Wave Sleep) reactivation during memory consolidation comes from the study of human spatial location in the presence of odor [7]. Reactivation activates hippocampal memory redistribution to new cortical storage locations. On the other hand, another hypothesis, named 'sequential hypothesis', which argues that the best benefit of sleep for consolidating declarative and non-declarative memory occurs when SWS and REM sleep occur one after the other is also verified

by the cross effect of SWS/REM on declarative/procedural memory consolidation [8,9].

The Effects of Sleep/Sleep Deprivation on Memory

Sleep's promotion on memory consolidation

Sleep is characterized by the rapid occurrence of REM sleep and non-REM sleep, including slow wave sleep (SWS, Phases 3 and 4) and light sleep Phases 1 and 2 (see Figure 1, part a). In humans, the first part of the night (early sleep) is characterized by a high amount of SWS, while REM sleep is dominant in the lower half (late sleep). SWS and REM sleep are characterized by a specific pattern of electric field potential oscillations (see Figure 1, part b) (Figure 1).

In subsequent rapid eye movement (REM) sleep, the brain system operates in a "separated" mode, which is also associated with separation between long-term and temporary storage. This enables a synaptic consolidation of the local encapsulation process, which reinforces the memory characterization of system integration during previous SWS (thick line). In general, memory gets the best results from slow wave sleep and rapid eye movement sleep. However, due to their different characteristics, integrated declarative memory (which combines the characteristics of different memories in different memory systems) theoretically benefits more from SWS-related system integration, with specific and discrete procedural memory benefits more from REM sleep — associated synapse consolidation in the local brain circuit [10] (Figure 2).

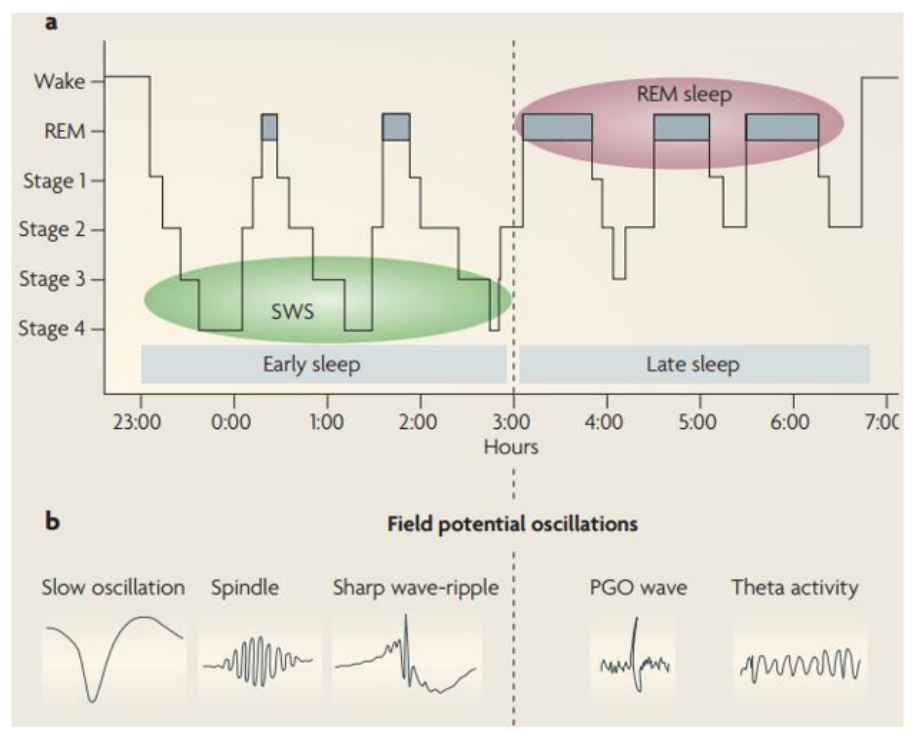

Figure 1: Effect of diameter-to-thickness ratio on stress.

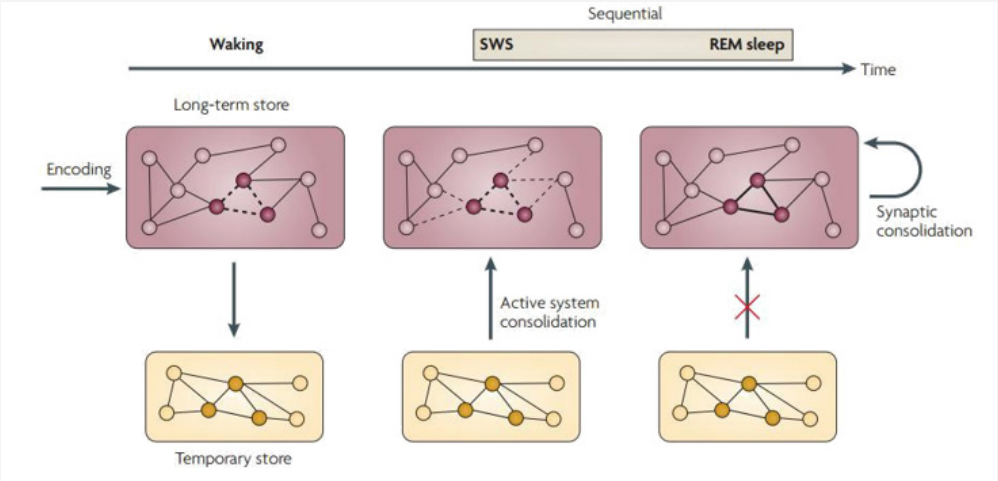

Figure 2: Sequential contributions of SWS and REM sleep on memory consolidation.

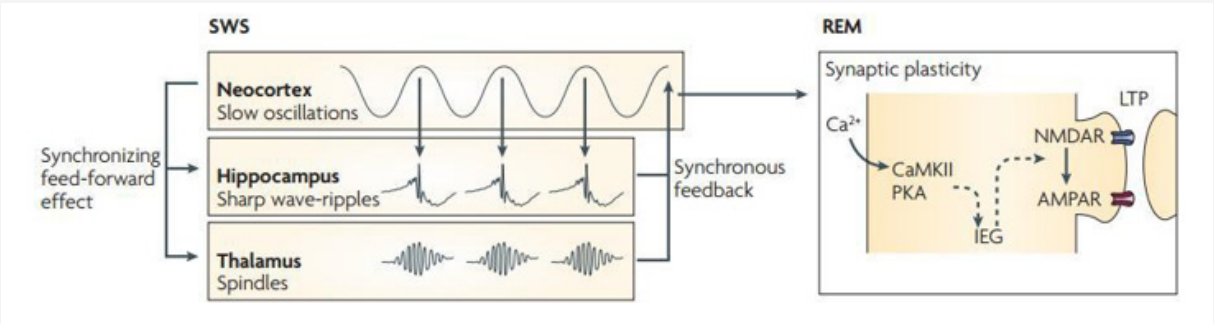

Figure 3: System consolidation hypothesis.

There are currently two hypotheses that explain the mechanism of memory consolidation during sleep. Synaptic homeostasis hypothesis [11] is a by-product of the overall synaptic reduction that occurs during sleep. The active system consolidation hypothesis suggests that the active consolidation process is caused by selective reactivation of memory in sleep [12]. The former use is to serve a global downscale of synaptic strength by the slow oscillations to achieve sustainable levels in energy and tissue volume requirements and allow for the repetitive use of synapses in future coding, and the process of the latter can be depicted [13,14]. Therefore, we have the two-stage model and sequential contributions of SWS and REM sleep on memory consolidation (Figure 3).

Memory impairment after sleep deprivation

Marion and his colleagues once used electrophysiological, molecular and behavioural indices to non-invasively study LTP-like plasticity in humans after sleep and sleep deprivation. The outcome shows that LTP-like plasticity decreases after sleep deprivation verified by declined MEP amplitudes, lower potential modulator level and less correctly recalled word-pairs during the declarative memory task [15]. For further studying, Tudor et al extended that 5 hours of sleep loss attenuated both mTORC1-mediated phosphorylation of 4EBP2 and the interaction between eukaryotic initiation factor 4E (eIF4E) and eIF4G in the hippocampi of sleep-deprived mice using an in vivo protein translation assay. What is more, by increasing the abundance of 4EBP2 in hippocampal excitatory neurons before sleep deprivation the abundance of phosphorylated 4EBP2 increased, and the amount of eIF4E-eIF4G interaction restored and hippocampal protein synthesis to that seen in mice that were not sleep-deprived and prevented the hippocampus-dependent memory deficits associated with sleep loss [16]. Effects of sleep deprivation on protein synthesis procedure is also verified [17] (Figure 4).

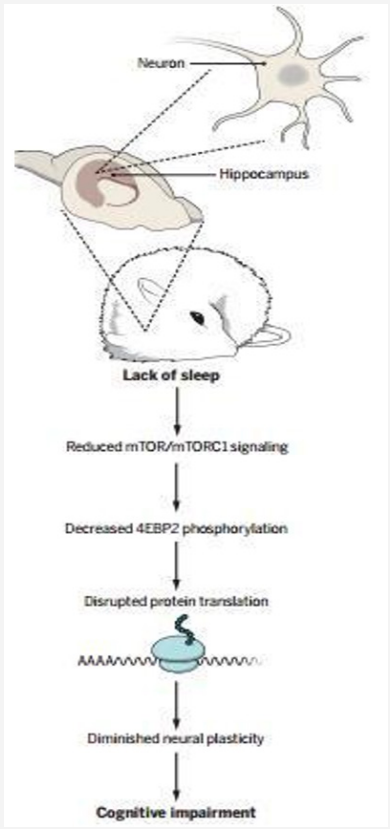

Figure 4: Effects of sleep deprivation on protein synthesis procedure.

And the molecular neurobiology mechanism of sleep on protein synthesis procedure is also verified [18]. Next, we consider the neuronal functions in sleep and memory consolidation process by the following fractional-order differential equations:

$$\begin{cases} \dfrac{d^{\alpha_1}x_1}{dt^{\alpha_1}} = \alpha\left(x_2 - x_1\right) + x_4 \\ \dfrac{d^{\alpha_2}x_2}{dt^{\alpha_2}} = hx_1 - x_1x_3 + cx_2 \\ \dfrac{d^{\alpha_3}x_3}{dt^{\alpha_3}} = x_1x_2 - bx_3 \\ \dfrac{d^{\alpha_3}x_3}{dt^{\alpha_3}} = x_2x_3 + rx_4 \end{cases}$$

where 0<i<1, (i =1,2,3,4) is a parameter describing the order of the system in sleep and memory consolidation process, x_i (i=1,2,3,4) is the anti-synchronization function of the time t. If a=35, b=3, c=12, h=7, 0.085 ≤r ≤0.798, then the system is in a chaotic state.

Suppose that some ractional-order differential equations are response systems of the neuronal functions in sleep and memory consolidation process:

$$\begin{cases} u_1(t) = -D_t^{\beta_1}x_1 - D_t^{\alpha_1}x_1 - \left(\hat{\alpha}\left(y_2 - y_1\right) + y_4\right) + \hat{\alpha}\left(x_2 - x_1\right) + x_4 - k_1e_1 \\ u_2(t) = -D_t^{\beta_2}x_2 - D_t^{\alpha_2}x_2 - \left(\hat{h}\left(y_1 - y_1y_3 + \hat{c}y_2\right)\right) + \hat{h}x_1 - x_1x_3 + \hat{c}x_2 - k_2e_2 \\ u_3(t) = -D_t^{\beta_3}x_2 - D_t^{\alpha_3}x_2 - \left(y_1y_2 - \hat{b}y_3\right) + x_1x_2 + \hat{b}x_3 - k_3e_3 \\ u_4(t) = -D_t^{\beta_4}x_2 - D_t^{\alpha_4}x_2 - \left(y_2y_3 - \hat{r}y_4\right) + x_2x_3 + \hat{r}x_3 - k_4e_4 \end{cases}$$

where $\hat{a},\hat{b},\hat{c},\hat{h},\hat{r}$ are the estimated values of the parameters a, b, c, h, r on the system (3.1) respectively; and $0<\beta_i<1$,(i= 1,2,3, 4) is a parameter describing the order of the system (3.2); $d^q/dt^q = D_t^q$, q=α_i, β_i, (i =1, 2,3,4) are the sleeping time in the Caputo sense, and $x=(x_1(t), x_2(t), x_3(t), x_4(t))^T$, $y=(y_1(t), y_2(t), y_3(t), y_4(t))^T$ are the status vectors of system (3.1) and system (3.2) respectively. U (t, x, y) =$((u_1(t), u_2(t), u_3(t), u_4(t))^T$ is the controller.

If α_i = 0.98, (i =1, 2,3, 4), a = 35, b = 3, c= 12, h= 7, r= 0.5, then the diagram of the attractors of system (3.1) can be seen in (Figure 5).

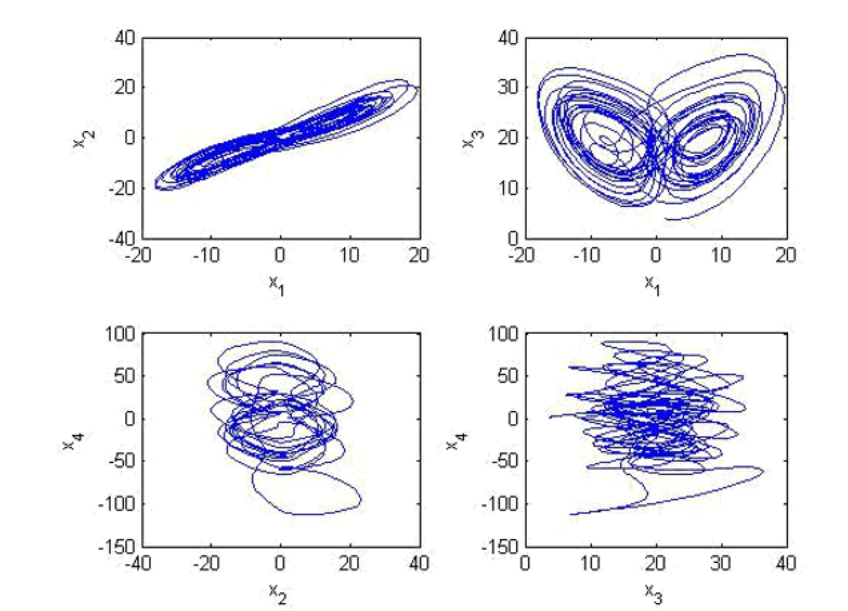

Figure 5: Diagram of the attractors indicating memory.

Neurotransmitters signals Transportation

Neurotransmitters are chemical agents secreted at the end of axons of nerve cells that diffuse across the synaptic gap and transmit information to adjoining cells such as neurons, muscle cells, and glands, by altering their electrical state or activity. There are many neurotransmitters with a variety of structures and functions.

In order to identify the neuronal functions in sleep and memory consolidation process shown above, the sleeping time step is taken as h= 0.0025s, and

T=30s, α_i=0.98, i= (1, 2, 3, 4), β_i=0.96, i= (5, -------9), a=35, b=3, c=12, h=7, r=0.5,

The time taken for the simulation, and the order parameters, with the initial state value of system (3.1) is

taking system, to select the system x (0) = (2, 0,1,1), the initial state value of system (3.2) is y(0)=(-4,-2,1,-5). Therefore, the sleeping process between system (3.1) and system (3.2) is shown in the following (Figure 6).

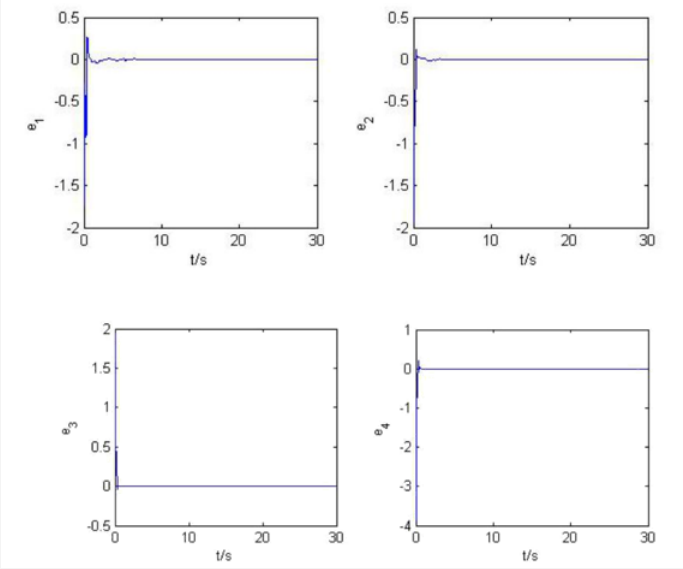

Figure 6: Modifiable structures error e_1, e_2, e_3, e_4 curve between system (3.1) and system (3.2).

According to the definition of neurotransmitters signals transportation, suppose that the neurotransmitters signals transportation is e= x+ y. If for any x (0), y(0) satisfy the condition $\lim_{t\to\infty}\|e\| = \lim_{t\to\infty}\|x(t)+y(t)\| = 0$, then we say that system (3.1) and system (3.2) achieve modifiable structures.

$$\begin{cases} u_1(t) = -D_t^{\beta_1}x_1 - D_t^{\alpha_1}x_1 - \left(\hat{\alpha}(y_2-y_1)+y_4\right) + \hat{\alpha}(x_2-x_1) + x_4 - k_1e_1 \\ u_2(t) = -D_t^{\beta_2}x_2 - D_t^{\alpha_2}x_2 - \left(\hat{h}(y_1 - y_1y_3 + \hat{c}y_2)\right) + \hat{h}x_1 - x_1x_3 + \hat{c}x_2 - k_2e_2 \\ u_3(t) = -D_t^{\beta_3}x_2 - D_t^{\alpha_3}x_2 - \left(y_1y_2 - \hat{b}y_3\right) + x_1x_2 + \hat{b}x_3 - k_3e_3 \\ u_4(t) = -D_t^{\beta_4}x_2 - D_t^{\alpha_4}x_2 - \left(y_2y_3 - \hat{r}y_4\right) + x_2x_3 + \hat{r}x_3 - k_4e_4 \end{cases}$$

On the basis of adaptive control methods, we can give the virtual neurotransmitter by serotonin and acetylcholine systems in central nervous system involving sleep and memory:

where $e_1 = x_1 + y_1$, $e_2 = x_2 + y_2$, $e_3 = x_3 + y_3$, $e_4 = x_4 + y_4$, $k_i > 0$, (i=1, 2,3, 4). If $t \to \infty$, then $\|e\| = 0$, and system (3.1) and system (3.2) achieve modifiable structures indicating memory process.

If we put (4.1) and system (3.1) to system (3.2), then the following error equations can be obtained between the groups for some fractional differential equations:

$$\begin{cases} D_t^{\beta_1}e_1 = -e_1(x_2 - x_1) - k_1e_1 \\ D_t^{\beta_2}e_2 = -e_dx_1 - e_cx_2 - k_2e_2 \\ D_t^{\beta_3}e_3 = e_bx_3 - k_3e_3 \\ D_t^{\beta_4}e_4 = -e_rx_4 - k_4e_4 \end{cases}$$

where $e_a = a - \hat{a}, e_b = b - \hat{b}, e_c = c - \hat{c}, e_h = h - \hat{h}, e_r = r - \hat{r}$ are the parameter estimation errors.

Next, according to (4.2), we design the adaptive update law for each parameter estimation error:

$$\begin{cases} D_t^{\beta_5}e_a = (x_1 - x_2)e_1 \\ D_t^{\beta_6}e_b = -x_3e_3 \\ D_t^{\beta_7}e_c = x_2e_2 \\ D_t^{\beta_8}e_h = x_1e_2 \\ D_t^{\beta_9}e_r = x_4e_4 \end{cases}$$

where 0<i<1, (i = 5,6,7,8,9).

According to $e_a = a - \hat{a}, e_b = b - \hat{b}, e_c = c - \hat{c}, e_h = h - \hat{h}, e_r = r - \hat{r}$ and (4.3), we can get the parameters of the adaptive control law:

$$\begin{cases} D_t^{\beta_5}e_a = (x_1 - x_2)e_1 \\ D_t^{\beta_6}e_b = -x_3e_3 \\ D_t^{\beta_7}e_c = x_2e_2 \\ D_t^{\beta_8}e_h = x_1e_2 \\ D_t^{\beta_9}e_r = x_4e_4 \end{cases}$$

According to (4.2) and (4.3), we get the total error of the system:

$D_t^{\beta}E$

Where

$$D_t^{\beta}E = (D_t^{\beta_1}e_1, D_t^{\beta_2}e_2, D_t^{\beta_3}e_3, D_t^{\beta_4}e_4, D_t^{\beta_5}e_a, D_t^{\beta_6}e_b, D_t^{\beta_7}e_c, D_t^{\beta_8}e_h, D_t^{\beta_9}e_r)^T$$

$E = (e_1, e_2, e_3, e_4, e_5, e_a, e_b, e_h, e_r)^T$, $0<\beta_i<1$,(i=----------9).

Then we consider Eq. (4.5), and expand the formula, we obtain:

$$D_t^{\beta}\begin{pmatrix} e_1 \\ e_2 \\ e_3 \\ e_4 \\ e_a \\ e_b \\ e_c \\ e_h \\ e_r \end{pmatrix} = AE = \begin{pmatrix} -k_1 & 0 & 0 & 0 & -(x_2-x_1) & 0 & 0 & 0 & 0 \\ 0 & -k_2 & 0 & 0 & 0 & 0 & -x_2 & -x_1 & 0 \\ 0 & 0 & -k_3 & 0 & 0 & x_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k_4 & 0 & 0 & 0 & 0 & -x_4 \\ x_2-x_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -x_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & x_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & x_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & x_4 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

Setting P = E_9. Then we obtain the following result:

$AP=PA^T$

$=A+A^T= -Q$

$$\begin{pmatrix} -2k_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -2k_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -2k_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -2k_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

where $k_i > 0$, (i=1,2,3,4) Q=diag $2k_1$, $2k_2$, $2k_3$, $2k_4$, 0, 0, 0, 0, 0).

It is easy to see that Q=diag $2k_1$, $2k_2$, $2k_3$, $2k_4$, 0, 0, 0, 0, 0 is a semi-positive definite matrix. Then, the state variable of (4.5) $E = (e_1, e_2, e_3, e_4, e_5, e_a, e_b, e_h, e_r)^T$ is asymptotically stable, that is, $e_1, e_2, e_3, e_4, e_5, e_a, e_b, e_h, e_r$ approach zero asymptotically with time.

Therefore, we achieve the neuronal functions in sleep and memory consolidation process by a number of adaptive robust set of fractional differential equations anti-synchronization indicating memory process.

Chemical and Physicael Propertis

In this part, mainly through the mathematical method, the preliminary screening of the data in Table 1 for the chemical and physicael propertis of neuron, and respectively by MATLAB and SPSS 22 software to the data provided by principal component analysis, that can only find that the influence factors on the quality of sleep and memory process standard, but can't find out what factors. Then we use the binary logistic regression model to process the data (Table 1&2).

First, we extract the data from the above Table 2 to make the 12*7 Table, and then use the principal component analysis method in MATLAB software (The code is shown in the last Annex 1 of this

article). We find out that the most influential factors are Formal Charge, Heavy Atom Count, Defined Atom Stereocenter Count. Finally, we analyze the data in Table 2, draw the broken line diagram (The code is shown in the last Annex 2 of this article), which is the following Figure 7, and we find out that the fifth neurotransmitters GABA is the most prominent one [19-62] (Figure 7).

Table 1: Chemical and physical properties of the neuron.

Property Name	Property Value						
	L-Adrenaline	Nor Adrenaline (Norepinephrine)	Dopamine	Serotonin	GABA	Acetylcholine	Glutamate
Molecular Weight	183.207 g/mol	169.18 g/mol	153.181 g/mol	176.219 g/mol	103.121 g/mol	146.21 g/mol	147.13 g/mol
Hydrogen Bond Donor	4	4	3	3	2	0	3
Hydrogen Bond Acceptor Count	4	4	3	2	3	2	5
Rotatable Bond Count	3	2	2	2	3	4	4
Complexity	154	142	119	174	62.7	115	145
Topological Polar Surface Area	72.7 A^2	86.7 A^2	66.5 A^2	62 A^2	63.3 A^2	26.3 A^2	101 A^2
Monoisotopic Mass	183.09 g/mol	169.074 g/mol	153.079 g/mol	176.095 g/mol	103.063 g/mol	146.118 g/mol	147.053 g/mol
Exact Mass	183.09 g/mol	169.074 g/mol	153.079 g/mol	176.095 g/mol	103.063 g/mol	146.118 g/mol	147.053 g/mol
XLogP3	-1.4	-1.2	-1	0.2	-3.2	0.2	-3.7
Compound Is Canonicalized	TRUE	TRUE	TRUE	TRUE	TRUE	TRUE	TRUE
Formal Charge	0	0	0	0	0	1	0
Heavy Atom Count	13	12	11	13	7	10	10
Defined Atom Stereocenter Count	1	1	0	0	0	0	1
Undefined Atom Stereocenter Count	0	0	0	0	0	0	0
Defined Bond Stereocenter Count	0	0	0	0	0	0	0
Undefined Bond Stereocenter Count	0	0	0	0	0	0	0
Isotope Atom Count	0	0	0	0	0	0	0
Covalently-Bonded Unit Count	1	1	1	1	1	1	1

Table 2: The compression of the chemical and physicael propertis of the neuron.

Property Name	L-Adrenaline	Nor Adrenaline (Norepinephrine)	Dopamine	Serotonin	GABA	Acetylcholine	Glutamate
Molecular Weight	183.207	169.18	153.181	176.219	103.121	146.21	147.13
Hydrogen Bond Donor Count	4	4	3	3	2	0	3
Hydrogen Bond Acceptor Count	4	4	3	2	3	2	5
Rotatable Bond Count	3	2	2	2	3	4	4
Complexity	154	142	119	174	62.7	115	145
Topological Polar Surface Area	72.7	86.7	66.5	62	63.3	26.3	101
Monoisotopic Mass	183.09	169.074	153.079	176.095	103.063	146.118	147.053

Exact Mass	183.09	169.074	153.079	176.095	103.063	146.118	147.053
XLogP3	-1.4	-1.2	-1	0.2	-3.2	0.2	-3.7
Formal Charge	0	0	0	0	0	1	0
Heavy Atom Count	13	12	11	13	7	10	10
Defined Atom Stereocenter Count	1	1	0	0	0	0	1

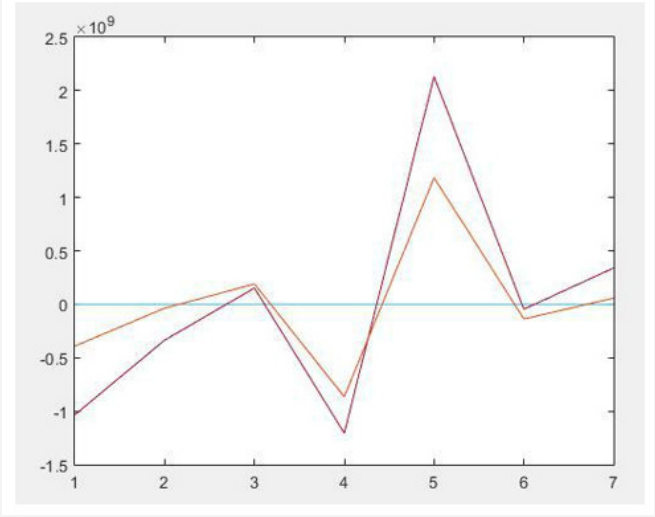

Figure 7: Broken line diagram of (Table 2).

For $X \neq 0$ and natural number n, taking the logarithmically differential into consideration yields

$$\left[f_{\alpha,\beta}(x)\right]^{(n)} = \frac{1}{x^{n+1}}\left[\sum_{k=0}^{n}(-1)^k \frac{n!x^{n-k}\varphi^{(n-k-1)}(x+y)}{(n-k)!} - (-1)^n n!\ln\Gamma(y) - \alpha(-1)^{(n-1)}(n-1)!x\right]$$

where $\psi^{(-1)}(x+y)$ and $\psi^{(0)}(x+y)$ stand for $\ln\Gamma$(x+y) and $\psi(x+y)$ respectively.

Furthermore, differentiating $x^{n+1}\left[f_{\alpha,\beta}(x)\right]^{(n)}$ directly gives

$$\left\{x^{n+1}\left[f_{\alpha,\beta}(x)\right]^{(n)}\right\} = (-1)^{n-1}x^n\left[(-1)^{n+1}\varphi^{(n)}(x+y) - \frac{\alpha(n-1)!}{x^n}\right]$$

Making use of (2.11) and (2.13) shows that for all $n \in N$ and any fixed y >0, the double inequality

$$\int_0^\infty t^{x-1}e^{-xt}\left[e^{-yt}\left(1+\frac{1}{2}\right)^t - \alpha\right]dt = \frac{n-1}{(x+y)^n} - \frac{1}{2}\frac{n!}{(x+y)^{n+1}} - \frac{\alpha(n-1)!}{x^n} \le \frac{(-1)^{n-1}}{x^n} = \left\{x^{n+1}\left[f_{\alpha,\beta}(x)\right]^{(n)}\right\} \le \frac{(n-1)!}{(x+y)^n} + \frac{n!}{(x+y)^{n+1}} - \frac{\alpha(n-1)!}{x^n} = \int_0^\infty t^{x-1}e^{-xt}\left[e^{-y}(1+t) - \alpha\right]dt$$

holds for all $x \in (-y,\infty)\setminus\{0\}$ and $\alpha \in (-\infty,\infty)$

For any fixed $y \in (0,\infty)$, let u(t) and v(t) be defined on $(-\infty,\infty)$ by $u(t) = e^{-yt}\left(1+\frac{1}{2}\right)^t$ and v(t)= e^{-yt} (1+t)respectively.

Differentiating u(t) and v(t) directly, we obtain

$$u(t) = e^{-yt}\left(\frac{1}{2} - y - \frac{1}{2}yt\right).$$

$$v(t) = e^{-yt}\left(1 - y - yt\right).$$

Therefore, for given $y \in (-\infty,\infty)$, we have

$$u(t) = \begin{cases} > 0, t < 1/y - 2 \\ < 0, t > 1/y - 2 \end{cases},$$

and

$$v(t) = \begin{cases} > 0, t < 1/y - 2 \\ < 0, t > 1/y - 2 \end{cases},$$

From (5.6) and (5.7), we conclude that for all t > 0 we obtain u (t)> 0,
and

$$v(t) = \begin{cases} < 1, y \ge 2 \\ \le e^{-(1-y)}/y, 0 < y < 1 \end{cases}$$

From (5.3) and (5.8) -(5.9), it is easy to see that

$$\frac{(-1)^{n+1}}{x^n}\left\{x^{n+1}\left[f_{\alpha,\beta}(x)\right]^{(n)}\right\} = \begin{cases} > 0, if\ \alpha \le 0\ for fixed y > 0 \\ < 0, f\ \alpha \ge 0\ for fixed y \ge 1 \\ < 0, f\ \alpha \ge e^{-(1-y)}/y for fixed o < y < 1 \end{cases}$$

For all $n \in N$ $x \in (-y,\infty)\setminus\{0\}$

On the one hand, if $x \in (0,\infty)$ then the inequalities (5.10) can be equivalently changed into

$$x^{2k+1}\left[f_{\alpha,\beta}(x)\right]^{(2k-1)} = \begin{cases} > 0, if\ \alpha \le 0\ for fixed y > 0 \\ < 0, f\ \alpha \ge 0\ for fixed y \ge 1 \\ < 0, f\ \alpha \ge e^{-(1-y)}/y for fixed o < y < 1 \end{cases}$$

and

$$x^{2k}\left[f_{\alpha,\beta}(x)\right]^{(2k-1)} = \begin{cases} > 0, if\ \alpha \le 0\ for fixed y > 0 \\ < 0, f\ \alpha \ge 0\ for fixed y \ge 1 \\ < 0, f\ \alpha \ge e^{-(1-y)}/y for fixed o < y < 1 \end{cases}$$

for $k \in N$

From (5.1), then simple computation shows that

$$\lim_{x\to 0} x^{n+1}\left[f_{\alpha,\beta}(x)\right]^{(n)}$$

for all $n \in N$ and any given $y \in (0,\infty)$. As a result,

$$x^{2k+1}\left[f_{\alpha,\beta}(x)\right]^{(2k)} = \begin{cases} > 0, if\ \alpha \le 0\ for fixed y > 0 \\ < 0, f\ \alpha \ge 0\ for fixed y \ge 1 \\ < 0, f\ \alpha \ge e^{-(1-y)}/y for fixed o < y < 1 \end{cases}$$

and

$$x^{2k}\left[f_{\alpha,\beta}(x)\right]^{(2k+1)}=\begin{cases}>0, if\ \alpha\le 0\ for fixed y>0\\ <0, f\ \alpha\ge 0\ for fixed y\ge 1\\ <0, f\ \alpha\ge e^{-(1-y)}\ /\ y for fixed o<y<1\end{cases}$$

for all $k\in N$ and all x > 0.

Therefore, (5.14) and (5.15) imply

$$x^{2k+1}\left[f_{\alpha,\beta}(x)\right]^{(2k-1)}=\begin{cases}>0, if\ \alpha\le 0\ for fixed y>0\\ <0, f\ \alpha\ge 0\ for fixed y\ge 1\\ <0, f\ \alpha\ge e^{-(1-y)}\ /\ y for fixed o<y<1\end{cases}$$

$$(-1)^n\left[f_{\alpha,\beta}(x)\right]^{(n)}=\begin{cases}>0, if\ \alpha\le 0\ for fixed y>0\\ <0, f\ \alpha\ge 0\ for fixed y\ge 1\\ <0, f\ \alpha\ge e^{-(1-y)}\ /\ y for fixed o<y<1\end{cases}$$

for all $n\in N$ and all x > 0.

Hence, if either $\alpha\ge e^{-(1-y)}/y$ for given 0 <y <1 or $\alpha\ge 1$ for given $y\ge 1$, the function (1.13) is strictly

logarithmically completely monotonic with respect to x on

$$\left\{x^{n+1}\left[f_{\alpha,\beta}(x)^n\right]\right\}=\begin{cases}>0, if\ \alpha\le 0\ for fixed y>0\\ <0, f\ \alpha\ge 0\ for fixed y\ge 1\\ <0, f\ \alpha\ge e^{-(1-y)}\ /\ y for fixed o<y<1\end{cases}$$

for $n\in N$

In view of (5.13), we can conclude that

$$\left\{(x+y)^{n+1}\left[f_{\alpha,\beta}(x)^n\right]\right\}=\begin{cases}>0, if\ \alpha\le 0\ for fixed y>0\\ <0, f\ \alpha\ge 0\ for fixed y\ge 1\\ <0, f\ \alpha\ge e^{-(1-y)}\ /\ y for fixed o<y<1\end{cases}$$

for $n\in N$ It is obvious that (5.18) is equivalent to that (5.14) and (5.15) hold for any given y> 0

and $x\in(-y,0)$ .Therefore, it is easy to prove similarly that (5.16) is also valid on $x\in(-y,0)$ for any given

y>0 and all $n\in N$

As a result, we can conclude that nervousness has the strongest influence on sleep and memory quality.

Conclusion

SWS and REM sleep complement each other to optimize memory integration. In the SWS process in which slow oscillations induce a broad synchronization of neuronal activity, active system consolidation combines newly encoded memory with pre-existing long-term memory to induce conformational changes in their respective expressions. System integration (priority affects explicit coding of behavior-related information) synergizes with global synaptic downscaling, primarily to rule out saturation of synaptic networks. Subsequent REM sleep, characterized by desynchronization of the neural network, may reflect the detachment of the memory system—possibly stabilizing the transformed memory by consolidating undisturbed synapses.

Sleep deprivation suppressed activation of the kinase complex mTORC1 which leads to a decrease in phosphorylation of 4EBP2, leading to disruption of protein translation, reduced neuroplasticity, and ultimately cognitive impairment. Restoring protein synthesis by increasing the amount of phosphorylated 4EBP2 protein in the hippocampus, a function normally performed by mTORC1 can compensate the memory impairment caused by sleep deprivation. Also, some alternative mechanisms may actuate as well in the protein synthesis reduction which needs a further studying.

The development of neurotransmitters and its complex functions during sleep and memory process are influenced by numerous factors. In this study, some mathematical speculations have been proposed on the basis of structural and functional characteristics of virtual neuron (especially the physiological phenomena of human beings) with a molecular docking and biomathematical approach to formulate some speculations to the consolidation of sleep and memory process. This could pave a way to formulate more mathematical speculations related with neuron, and finally these data and approaches will be useful for constructing virtual neuron with the help of biomathematics.

Conflict of Interest

We have no conflict of interests to disclose and the manuscript has been read and approved by all named authors.

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