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Information Circulation in The Advanced Neural Activities of Central Neural System

Fayang Ma[1,2#]

[1]*The Pathophysiology Department, School of Basic Medical Sciences, College of Medicine, Zhengzhou University, Zhengzhou, Henan, 450000, China*

[2]*China-US (Henan) Hormel Cancer Institute, Zhengzhou, China*

[#] **Correspondence:** *E-mail: fyma@gs.zzu.edu.cn*

Abstract

Long-Term Potentiation(LTP) and Long-Term Depression(LTD) are two major forms of synaptic plasticity, which are also two well-know functional and unit activities involved in high advanced central neural system(CNS) activities, like memory. But we still know little about how the advanced CNS activities are organized in the brain and in the level of organism. Based on the current understanding and experimental evidence of neurology, we propose the term "Information Circulation" to summarize the current understandings for advanced CNS activities, and we define it as separately input neural signals finally converge in different levels of CNS and interact with each other, then neural information are circulated and processed in different levels of CNS to give out orders for next body actions. This review provides a detailed description for the functional organizations of advanced CNS activities in the term of Information Circulation. This article outlines the receiving of outside stimulation and transmission of neural information, especially transmission and procession of visual bioelectrical signals, then we described neural circuits of Information Circulation in advanced CNS activities, the corresponding specificity and dynamic properties of neural circuits, different sensation linkages, and neural synchronization for information circulation to produce consciousness in CNS. In conclusion, Information Circulation is defined as an important signature involved in advanced CNS activities.

Key words: Information Circulation; Perception; Memory; Consciousness; Neural Circuits

Abbreviations: Central Nervous System(CNS), Long-Term Potentiation(LTP), Long-Term Depression(LTD), Autonomic Nervous System(ANS), Lateral Geniculate Nucleus(LGN), Ventral Posterior Nucleus(VPN), Medial Temporal(MT), N-Methyl-D-aspartate (NMDA), α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA)

1. Introduction

Canadian psychologist Donald.O.Hebb in 1949 proposed that when two neurons interact with each other, the signals transmitted between them will be strengthened, which was defined as synaptic plasticity. LTP is a well-characterized form of synaptic plasticity, which could be induced throughout human CNS tissue (1). A large body of evidences indicate that common underlying molecular mechanisms are shared by LTP and memory, and LTP fulfills many of the criteria for a neural correlate of memory, and so synapse-based LTP may be the structural and molecular basis for memory(2-7). Synaptic depression as LTD is also a parallel mechanism for mediating memory storage(8, 9), which guarantees sufficient plastic capacity of CNS synapses and is significant for learning, forgetting and behavioral extinction(1, 10). Studies have shown that a majority of synaptic connections, some estimates run as high as 80%, are extremely weak and transmit few electrical signals, which may provide an enormous capacity for synaptic plasticity (11).

Memory, perception and consciousness are advanced CNS activities of human brain in the category of neuroscience, which are also three major subjects numberless scientists investigate in(12-14). Now we propose that Information Circulation as a common feature for these three major subjects which is described as below. First of all, multiple types of information (majorly sight, sound and feeling) are accepted by isolated biological receptors distributed throughout the body, which then are translated into certain kind of bioelectric signals, respectively. The information carried by bioelectric signals will be transmitted and processed alongside certain and segregated pathways towards the CNS, where these different types of information are “combined” together to become an integrative event, integration of different types of sensory information occurs mostly at final stations of each pathway, where coordinating linkages between heterogeneous pathways are established. Finally, decisions and actions orders are dispersed out by the efferent nervous system, to interact with outside environment, the result of the interaction turns to be a new stimulation, which makes our survival easier. The whole process could be defined as “Information Circulation”, which are continuously recalibrating our memories, perception and consciousness, and finally action. The above Information Circulation is the major one, and the minor one happens during the processing of information in CNS (Fig 1).

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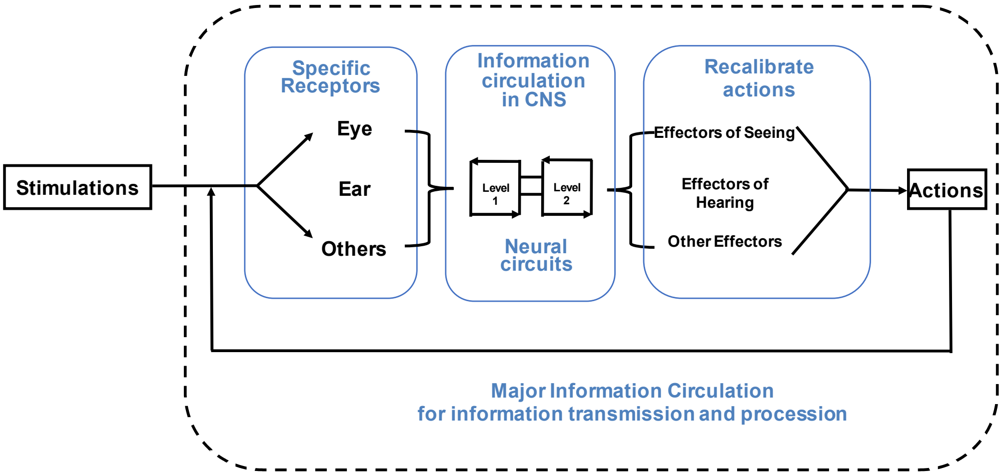

Fig 1. The model of Information Circulation during information transmission and procession
Neural bioelectrical signals circulate alongside both the major and the minor neural circuits. Firstly, stimulations from outside and self-actions are accepted by specific receptors, corresponding neural information is then being transmitted and processed in certain separated pathways, then interacted and circulated in CNS, then neural information of decisions are dispersed out in separated efferent pathways to form "actions", which combined with outside stimulations to form a new stimulation and impose on various sensational receptors again.

2. The receiving of outside stimulation and transmission of neural information

The Information Circulation originally starts from the interactions between outward stimulations and sensory receptors. Neural information being circulated and processed are firstly transformed by sensory receptors after being directly imposed by stimulations. Multiple types of sensory receptors are dispersed throughout the whole body to receive various kinds of stimulations. Several specific sensory organs only located in head to receive the information of vision, sound, odor, flavor and body posture (15).

Then how does the neural information transmit in the nerve fibres? The bioelectric signal of *color* is processed and transmitted by three types of cone cells. Original information is accurately recorded by local membrane potentials (MPs) of sensory receptor which directly interacted with outside stimulations. Only stimulation reaches a certain threshold and in the sensing range of certain perception system, can it produce perceptions through sensory systems. Efficient temporal and spatial aggregation, totalization of MPs and postsynaptic potentials produce transmissible action potentials (APs) (16). Then, different types of neural information are integrated for perception in our CNS, and the dynamic properties of Information Circulation allow us recalibrate our actions to collect other aspects of this stimulation for memory consolidation. As long as outside stimulations are transformed into bioelectrical signals by sensory receptors, the corresponding neural information is circulated in terms of different combinational patterns of APs and transported in predicted pathways. There are several aspects of information in even one sensation like olfactory, some of information may be transmitted and processed in parallel ways, but finally these aspects will be coordinated together as an integral sensation(15).

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Homogeneous information always converges to the same neuroganglion or nuclei, finally. The information conveyed by autonomic nervous system (ANS) is relatively independent and insulated from other types of information; most evidences indicate the outward portion of afferent pathways is rarely interrupted by heterogeneous information, which accurately maintains its originality(15).

3. The process and transmission of visual information

There are always several levels and processing steps in the pathways for information transmission, each level does more than relay sensory information or stand apart as a discrete processing step from others. In the neural pathways for visual information transmission, the extensive feedback from visual cortex to the lateral geniculate nucleus (LGN) of thalamus is to optimize the thalamic contribution to segmentation and global integration(17). There are dense inhibitory and exciting reciprocal projections linking different cortical and subcortical areas inside a certain pathway which is termed as feedforward and feedback projections (15), so mutual interactions or influences exist between these levels.

Firstly, we provide insight into how is the visual information being circulated in certain neural pathways. The basic elements of visual information include color, shape, depth, movement directions, orientations and maybe some elements we don't know yet. Until now, we are not sure which of these elements are integrated to be conveyed and processed, which of them are processed independently. The information of visual world is analyzed and transmitted by a set of parallel spatio-temporal pathways(18). Electro-physiology and morphological studies indicate that information of shape, color, depth and movement are processed and transmitted in a set of relatively separate pathways, with each pathway manage one or more characters of vision. The starting point of the pathways for visual Information Circulation is the retina, which is well designed and could take meticulous visual tasks. The responses of each retinal stratum are well-ordered and make up a uniquely temporally coding mode, and this mode sustains alongside the ANS(19). Precise vertical stratification of the inner plexiform layer (IPL) makes each stratum of retina receive unique neural inputs which could be integrated to produce more than ten distinct firing outputs. These responses are ordered in the time domain (18) and the corresponding information are then delivered by dozens of classes of ganglion cells to next neural station (19).

There is one question, where do these parallel transmitted information be integrated and processed together? Each channel projects into different areas of layer four of *striate cortex*, and the information of each pathway is selectively and independently processed, then network of excitatory pyramidal axons reciprocally link pathways from multiple visual cortical areas together, these excitatory cells are glutamatergic (20-22). Finally, the segregated processed components of visual information are integrated and circulated to be processed in a larger scale of neural circuits which are connected by extensive mutual projections among multiple areas and levels, and it is the functional structure basis for the Minor Information Circulation in neural system.

3.1 The feedback system in visual Information Circulation

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For instance, blockage of neuronal activities in S1 cortex could influence response pattern of the ventral posterior nucleus(VPN) neurons, in turn feedback projections from cortex to VPN could also affect the feedforward information transmission(23).The reentry actions could facilitate the coordination of neuronal firing and visual information transmission between different levels(24), and there is a complete topological projection relations between retina and primary visual cortex, and this strict topological correspondence is essential for precise reconstruction of original vision.
The visual feedback system consists of pathways from Medial Temporal(MT) through V1 (relaying) and then to LGN, which makes possible the downward iterative interactions. For instance, larger-scale integration is carried out only at higher levels as MT, the feedback from which can inform earlier levels about the overall broad boundaries of objects, which is inaccessible by the circuits at earlier levels.

3.1.1 V1 and LGN

The feedback cells from layer 6 of V1 has sensitivity of direction and orientation, and are selective for moving stimuli, not stationary ones (25, 26)which focuses on the motion directions of objects.
The feedback from V1 could affect the spatial properties of LGN-cell receptive fields, and shift the firing patterns between burst and tonic(27-31), and enhance information transmission via LGN cells (32-36). These feedback projections mainly originate from layer 6 cells, the axon terminals of which are branched and organized retinotopically in LGN (37-40), which exert an efficient and stimuli-specific influence on the visual input from retina via the stations of LGN and layer 4 of V1(41). Exactly, the feedback from V1 could also enhances the sensitivity and surround antagonism and reduce the responses of LGN (25, 37, 42).
The asymmetric axonal arborizations of feedback projections in LGN are the structural basis for the spatial characteristics of local receptive fields. The responses of LGN cells could also be strengthened or strongly suppressed, depending on the spatial relations between the receptive fields and properties of stimuli. These actions could focus LGN ensembles on properties of the overall shape of moving stimuli extracted by MT.

3.1.2 MT and V1

MT cells are sensitive to motion direction, speed and depth of a stimulus (43), but some of these sensations in part are originally related to the properties of V1 cells (43, 44). The receptive fields of many MT cells are larger than those of V1 cells (45, 46), for neurons at higher levels sample larger areas of visual space via convergent input from earlier levels, so the responses of V1 cells could be integrated into the larger MT-cell receptive fields, including the responses of end-stopped cells of earlier level .The visual information are feedforwardly interacted with higher levels, and also being processed by feedback from MT(43, 44, 47).
When a moving stimulus entering an MT receptive field, the response of V1 cells will be affected by the feedback from MT retinotopically, with responses increase or decrease prominently in the first 10ms (48). Actually, MT send feedback projection to layer 4B and layer 6 of V1(49, 50), and contact all sets of layer 6 cells in V1 that

provides feedback to LGN, feedback signals from the broader spatial context of MT could influence the process of earlier visual information in levels of V1 and LGN (51, 52), which has a widespread effect on the response magnitude and firing patterns in a variety of LGN cells(17). And the effect of this influence follows the properties of stimulus driving MT. For instances, the perception of motion may involve a dynamic interaction between MT, V1 and LGN (41), for the signals from the cortical area of motion could meet all types of visual information relaying through LGN (53, 54) by these reciprocal connections between these cerebral levels. So the visual information circulated between different levels should be processed and modulated in the pattern of iterative interactions or Information Circulation.

3.2 Visual Information Circulation

In visual system, the synaptic organization of feedforward and feedback pathways forms a trans-level circuit. So the changes of responses in V1 by the feedback from MT could further influence the activities in LGN by feedback projections from V1 to LGN, then the consequent changes in LGN circuitry will circulate in the processing of original visual input from retina. The feedback-driven modulation of neural responses at earlier levels changes the feedforward input to higher level, where the responses of feedback projection cells could be changed as a result. So the visual information could be circulated in the trans-level circuit.

But how is this feedback integrated into roles linked to the feedforward pathways? There are specific communicating mechanisms among neurons of different shape and functions (55), which causes varied conducting velocity. The transmission of visual information via magnocellular pathway to MT is potent and rapid, which triggers fast MT feedback to affect visual input relayed in a more slowly conducting pathway, as the parvocellular and konicellular dominated pathways (37, 48, 56, 57). So the visual information transmitted in different pathways are conducted in different speed, which makes the feedback influencing interactions of levels accurately and orderly.

The recalibration properties of this Information Circulation could enables predictive modulation of responses at earlier levels. Feedback from higher motion areas can influence the transfer of ascending input when, or even before, the input arrives (41). Which makes the recalibration of neural activities more accurate and in time. The extensive recalibration abilities with the reference of various short and long-term memories in the major Information Circulation distinguish humans from other species.

4. Neural circuits in Information Circulation of advanced neural activities

Neuron clusters or ensembles are functional neuronal groups and memory coding units, which could response to the same kinds of information with identical pattern of activities. There is no spatial specificity or distinguishable analogical locations for activated ensembles. But the levels of activity of the interconnected ensembles could fluctuate in temporal patterns (58, 59) and the activity patterns may represent a kind of comprehensive information. The degree of anesthesia could be reflected by the population scale of activated ensembles in sensory cortex. And the scale of a neuronal assemble is determined by how many neurons could be involved and activated

instantaneously at the same time (55).
The outside stimulation flows in and rapidly being processed by the reflex arc of gill-withdrawal reflex, just for its linear and simple organizations of neurons. In humans, how are the neural ensembles being organized in Information Circulation to produce perceptual consciousness? The large amount of simultaneously activated ensembles may be structural basis for advanced neural activities as perceptual consciousness, and activity patterns of neural ensemble from different cerebral areas could be synchronized and integrated into the flow of Information Circulation (24). By this way, different types of information could be circulated in larger neural circuits, and the information of stimulus could be represented by the engrams of simultaneously activated ensemble groups in the Information Circulation, which encodes from common features to specific properties(60).

4.1 The specificity of the neural circuits for the Information Circulation

First we introduce the specificity of the pathways for memory process and storage, memory should not be stored at other sites which is isolated from neuronal networks for information processing. The biological basis for the modulations of sensory-to-motor circuitry synapses is the same site and mechanism for procedural memory storage(10). The changes of synaptic plasticity at the connections of presynaptic and postsynaptic cells serve to store the long-term memory, and then memory should be stored or distributed in the countless neural circuits, the same pathway for information transmission. As the visual information are distributed in the whole neuronal pathway, not only represent in higher cortical levels.
The engrams of memory should be stored in the synapses of neuronal circuits that are originally activated (61-63). So the information of memory is circulated trans-synaptically. The amount of activated neurons involving in previous memory processing increases when imposed by stimulations resembling the initial ones, and memory retrieval relies on reinvoking patterns of activity that occurred at the time of encoding, both of which may account for that the viewing of original stimulations could help recalling(64, 65). The same neural ensembles activated in the acquisition of learned fear are reactivated during the remembrance of the memories(66). To some extent, the downward feedback from higher visual cortex to lower levels regulates the activities of neurons with same or similar function(55), which follow the specificity of the Information Circulation. In conclusion, the pathways and neural circuits for Information Circulation are stimulus specific.

4.2 Dynamic properties of the neural circuits for the Information Circulation

The dynamic properties of neurotransmitter and corresponding receptors, synapses and related intracellular messenger molecules, neurons and connected fibers are the structural and molecular basis for the never ending Information Circulation.
The dynamically activated and reactivated N-Methyl-D-aspartate (NMDA) postsynaptic receptors are closely related with LTP and formation of spatial memory(63, 67). α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) is another glutamate receptor, the dynamic internalization and externalization of which is also

significant for synaptic plasticity, and the activation of NMDA receptors is induced by the activation of AMPA(22). The blockage of NMDA in amygdala are linked with forget of fear, which is the result of interruption of dynamic properties of synaptic components(68).
The neural circuits for memory storage should be actively sustained to maintain the dynamic flowing of Information Circulation, and the process of which are related with the activation of synaptic components as PCK-ζ and cytoplasmic polyadenylation element binding protein(CPEB), the blockage of which would interfere with normal circulation of memory information (69-72). It is PKA which tranlocates into nuclei initiating protein synthesis and transforming short-term memory to long-term memory (62, 73), and this transformation could consolidate the pathways for memory Information Circulation. The information of behavioral modification circulates not only through the changes of synaptic electrical properties, but also via the changes of the number of synaptic connections distributed in the neural circuits (74).
The growth of new axonal fibers and neurogenesis would interrupt the previous engrams of memory, for the integration of new components in the existing neural circuits would damage the specificity of the pathways for Information Circulation, and which may be the cause for the forget of the first three years of life (64, 75, 76).
Improved stepping behavior after training spinal-injured animals shows that axonal projections of younger rats are of greater plasticity, which is demonstrated anatomically with denser VLF (ipsilateral ventrolateral funiculus) projections to hindlimb motoneurons after neonatal injury (77).
Most fibers within the cerebral white matter tract are bidirectional, which reciprocally associate different cerebral areas(21), and certain properties of these fibers could be changed as the information circulate through them. Myelination of fibers could increase the speed of information transmission and strengthen multi-regional connections, the degree of myelination and signal intensity in axons are positively correlated, and neural information will leak out and disappear if without myelin sheath (75, 78). The myelin sheath becomes larger and thicker when we learn difficult skills, the process of which is also accompanied with gradually decreased synaptic plasticity. Once myelination finished, axon will lose its plasticity, which may be for original engram consolidation(75). Only the forebrain white matter will be developed and flourished by study of playing piano after puberty (76), which is the only place where myelination of fibers has not finished yet after puberty. Until about age of twenty-five the myelination process in prefrontal lobe will be almost finished. As people are getting old, the locus in cortex for myelination becomes more and more limited, but it does occur sometimes. Violent changes of environment could influence intelligence development in the key period of myelination. The abnormal cerebral whiter matter may come from abnormal myelination of unstable axons and may result adolescent schizophrenia. Nogo-A which is myelin-associated inhibitor of axonal growth, is majorly present in oligodendroglia cell of the adult and developing nervous system(79), the decrease expressing of which could result behavior alterations in laboratory rodents, possibly and particularly corresponding with human schizophrenia(80). And in adults, Nogo-A is able to modulate the neuronal and synaptic plasticity through Nogo receptor (NgR) (81, 82).

The neurons in the pathways for Information Circulation could be refreshed as well. There are thousands of newborn neurons appeared everyday in dentate gyrus, and the survival of which could be promoted by environmental enrichment and some kind of learning, then the survived newly born neurons could integrate into hippocampal networks and information could be circulated through these new pathways(64, 83-86). And there is a critical period after the neurogenesis during which special stimulations is significant for survival(65), this time period is consistent with the pivotal period for the growth of axons and dendrite of new generated neurons (87), and the survival rate correlates with amount and difficulty degree of exercise, not with learning speed (75). The newborn neurons in hippocampus have greater responsiveness than mature granule cells and may contribute to engrams by representing the previous experiences (88, 89), which is essential for difficult task learning (90).

But the dynamic properties may originally be caused by ceaseless changing stimuli. For instance, the effect of surround antagonism which is essential for the transmission of visual information is only limited to moving stimuli other than stationary subjects (25). Functions of orientation column located in striate cortex would be impaired if the visual environment are deprived or abnormal (91, 92). Transcranial direct-current stimulation (tDCS) may be useful for consciousness recovering in vegetative patients(93). And consistent stimulations of locomotor training could promote the signal circulation of local circuitry and improve the stepping performance of rat after the injury of spinal cord (77). Which all means the significance of the outside dynamic stimulations to the integrity of pathways for Information Circulation.

The intervention of dynamic property and disorders of neural connectivity would result the malfunction of Information Circulation. And abnormal Information Circulation could lead to loss of memory, which is closely related with many psychiatric disorders as anxiety, schizophrenia and depression(10, 11, 94).

The electro-physiological activities of neural circuits are dynamic, so does the information they are representing. We can assume that neural information is always in the state of dynamic flowing, or the circulation mode of neural information.

5. Different sensation linkages

The above passages review the structural basis and properties of the visual Information Circulation in the corresponding neural circuits. Now we are beginning to explain the applications of Information Circulation in advanced functional neural activities, as in different sensation linkage, perception and consciousness. The presumption is that any two regions with a consistently high correlation are linked, perhaps by an actual bundle of nerve fibres, but certainly by working together in some way (94).

Neural circuits distributed in different cerebral areas are linked by feedforward and feedback projections between them to form a larger scale of neural circuits. The cerebral areas consist of different levels and various functional differing regions exist within each level. As mentioned above, the processing of visual Information Circulation should be operated by mutual interactions between higher and lower visual levels, and by the ones between different areas within one level, especially within primary visual cortex. The response patterns of neurons which locate in the larger circuits for

Information Circulation could be inter-affected.

The Information Circulation through multi-cortical areas of different functions may connect and integrate a variety of sensory information together to produce combinational perceptions of consciousness, which should be the ultimate goal of the transmission and procession of individual sensory information(24). The structural basis for multi-sensation integration are these trans-regional axons which bundle together and comprise much of the fiber tracts termed as brain white matter (95). The effect of horizontal mutual projections among cortical area is smooth, medium and slow, but have widespread and extensive influences. Contrarily, the concentrated projections between different cerebral levels are less, but the excitatory effect of which is intensive and immediate.

The storage of declarative memories requires the involvement of hippocampus (76, 96, 97), and John O'Keefe found the memory of surrounding environment could be registered in hippocampus in a pattern of integrated information (98), and this process may rely on the coordination of multi-sensory systems. Vision of the movement of a speaker's mouth could affect the listening content (99). For these auditory memory may be initially coded together with visual information. The perceptual effect observes that speech motor learning could affect perceptual classification of speech sounds (99). The structural basis is the somatosensory inputs to cochlear nucleus and bidirectional interactions between somatosensory and auditory cortical areas (100-106). Besides that, the responses of primary motor cortex could be facilitated by the perception of speech movement and speech sounds(107, 108). Speech perception could also be affected by the stimulation to premotor cortex(109), which means the perceptual change and motor learning are associated and interacted.

The visual information of speech movement and auditory information of sound are flowing in and are being perceived and processed coordinately together, then the information of the movements of speech flows out, and being perceived again as a new stimulus information. This kind of information flowing are circulated, with every steps being bidirectionally interacted with each other. In conclusion, the Information Circulation is not restricted to one single sensory being processed segregated from others, but individual sensory information are always finally being integrated together as cross-modal sensory information and being processed in the Information Circulation in a larger scale (Fig 2).

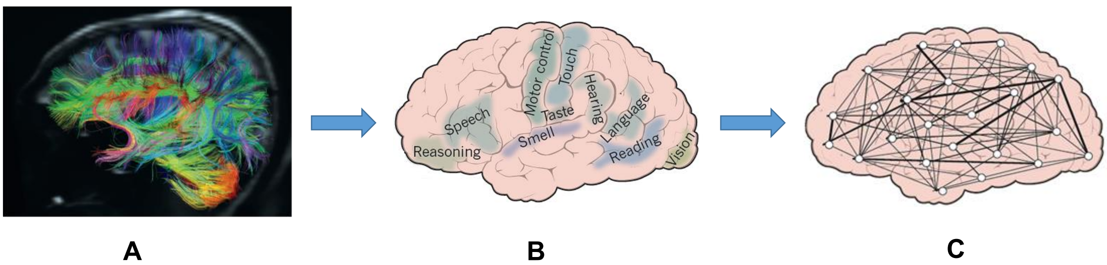

Fig 2. Different brain regions are being connected by neuronal fibers forming a complex connectome. (A) is the result of the brain's neuronal fibres detected by the technology of Diffusion Spectrum Imaging. The directions of fibres are represented by different colors. If the related and

interconnected neural structures with specific functions (B) are all combined and integrated together, there should be a very complicated neuronal network (C) linked by these fibres. Then if multi-cerebral areas of different functions were connected with each other, then how does the neural information being transmitted and processed in such a network? The organization pattern of Information Circulation is proposed as the viewpoint.

6. Neural Synchronization for Information Circulation to Produce Consciousness in CNS.

Human brain can not only encode specific information to a certain event, but could extract commonality and abstract conception from present life experience and then generalize it to others(60). It also has the ability of associative thinking and creativity, the structure basis of which may be hyper-connectivity; neural ensembles of independent memories which rarely have contact with each other are connected to form new combination patterns of information of neuronal ensembles (11).

As what we have discussed above, the production of perceptual consciousness needs the participation of simultaneously synchronized ensemble activities into the Information Circulation. Now we review the mechanism of synchronizations and reentry, which are based on functional organization of ensembles. The reentry is the strong and instantaneous reactivation of the initial neural ensembles distributed in multiple brain areas, which will repeat for a while with diminished amplitude after being imposed by stimulus(60) and the experience of consciousness needs effective and accurate reentry mechanism, which is interactions of various cortical areas in a short time range(24). Electro-synapses make the synchronization of neural activities more feasible. And the structure for trans-regional synchronization is the reciprocal projections between ensembles from multi-cerebral areas, the activities of which also sustain the activation state of assembles (21, 55). The response patterns and synchronization of neuron assemblies are stimulus-specific and with temporal and spacial features. Feedback impulses from higher levels to lower ones may cause synchronization of activities in different levels of visual nervous system, which is to integrate a variety of visual information together and ensure precisely and temporally coding of information. For instances, the large feedback projections from the 6th layer of primary visual cortex to LGN could regulate and optimize the response patterns of LGN neurons (41), then responses of LGN ensembles could be furtherly synchronized. In conclusion, the consciousness of visual perception may be achieved by dozens of visual areas and visual related areas, in which the ensemble activities with tempo-spatio features are circulated and synchronized to perceive extensive views.

Forwardly, projections from brainstem and thalamus to cortex are of certain conduction velocity and could spontaneously send impulses to modulate the global level of brain arousal by neuronal synchronization throughout the brain, so the state of consciousness and alert could be sustained (110-112). Consciousness keeps forming and dismissing transiently, and an individual consciousness can not exist all along (55), which corresponds with the dynamic property of neural activities. For stimulus changes all the time and so does the activated pathways for Information Circulation which specific to stimulus (55). The inhibitory and excitatory mechanism between neurons could timely end the activation of neural ensembles and replace it with new ones, which makes the possibility of prompt alteration of multi-signal transmission(113-115), and results in large-scale transient construction, dismiss and reconstruction of pathways for Information Circulation, which may be the structural basis for perceptual consciousness (116).

Different types of neurons activated in different patterns could reflect a variety of information accumulated in life(55). An integrated consciousness should result from synchronization of neuron ensembles dispersed at posterior cortex (for sensory representation) and frontal lobe (for schedule, memory and language) (55). The activities of hundreds of millions neurons are synchronized to produce the experience of consciousness in a range of ~100ms and disappeared in less than one second(55), just like the never ending fluctuating tide.

7. Conclusions

In conclusion, the Information Circulation is a pattern for information processing, the structural basis is the neuronal circuits distributed in different cerebral areas connected by reciprocal projections, to form a larger-scale neural circuit, which is a functional integrity. What's more, the neural circuits for Information Circulation are characterized by specificity property and dynamic property.

Conflict of interest

The author has no conflict of interest to declare.

References

1. Cooke SF, Bliss TV. Plasticity in the human central nervous system. Brain. 2006;129(Pt 7):1659-73.
2. Abel T, Martin KC, Bartsch D, Kandel ER. Memory suppressor genes: inhibitory constraints on the storage of long-term memory. Science. 1998;279(5349):338-41.
3. Abel T, Nguyen PV, Barad M, Deuel TA, Kandel ER, Bourtchouladze R. Genetic demonstration of a role for PKA in the late phase of LTP and in hippocampus-based long-term memory. Cell. 1997;88(5):615-26.
4. Bourtchuladze R, Frenguelli B, Blendy J, Cioffi D, Schutz G, Silva AJ. Deficient long-term memory in mice with a targeted mutation of the cAMP-responsive element-binding protein. Cell. 1994;79(1):59-68.
5. Huang YY, Kandel ER, Varshavsky L, Brandon EP, Qi M, Idzerda RL, et al. A genetic test of the effects of mutations in PKA on mossy fiber LTP and its relation to spatial and contextual learning. Cell. 1995;83(7):1211-22.
6. Silva AJ, Paylor R, Wehner JM, Tonegawa S. Impaired spatial learning in alpha-calcium-calmodulin kinase II mutant mice. Science. 1992;257(5067):206-11.
7. Silva AJ, Stevens CF, Tonegawa S, Wang Y. Deficient hippocampal long-term potentiation in alpha-calcium-calmodulin kinase II mutant mice. Science. 1992;257(5067):201-6.
8. Boyden ES, Katoh A, Pyle JL, Chatila TA, Tsien RW, Raymond JL. Selective engagement of plasticity mechanisms for motor memory storage. Neuron. 2006;51(6):823-34.

9. Lisberger SG, Morris EJ, Tychsen L. Visual motion processing and sensory-motor integration for smooth pursuit eye movements. Annu Rev Neurosci. 1987;10:97-129.
10. Kandel ER. The biology of memory: a forty-year perspective. J Neurosci. 2009;29(41):12748-56.
11. Lehrer J. Neuroscience: Making connections. Nature. 2009;457(7229):524-7.
12. Lisman J, Cooper K, Sehgal M, Silva AJ. Memory formation depends on both synapse-specific modifications of synaptic strength and cell-specific increases in excitability. Nat Neurosci. 2018;21(3):309-14.
13. Heuer K, Toro R. Role of mechanical morphogenesis in the development and evolution of the neocortex. Phys Life Rev. 2019.
14. Berlucchi G, Marzi CA. Neuropsychology of Consciousness: Some History and a Few New Trends. Front Psychol. 2019;10:50.
15. Spors H, Albeanu DF, Murthy VN, Rinberg D, Uchida N, Wachowiak M, et al. Illuminating vertebrate olfactory processing. J Neurosci. 2012;32(41):14102-8.
16. Giuliodori MJ, Zuccolilli G. Postsynaptic potential summation and action potential initiation: function following form. Adv Physiol Educ. 2004;28(1-4):79-80.
17. Sillito AM, Jones HE. Corticothalamic interactions in the transfer of visual information. Philos Trans R Soc Lond B Biol Sci. 2002;357(1428):1739-52.
18. Roska B, Werblin F. Vertical interactions across ten parallel, stacked representations in the mammalian retina. Nature. 2001;410(6828):583-7.
19. Werblin F, Roska B. The movies in our eyes. Scientific American. 2007;296(4):72-9.
20. Arimatsu Y, Ishida M, Kaneko T, Ichinose S, Omori A. Organization and development of corticocortical associative neurons expressing the orphan nuclear receptor Nurr1. J Comp Neurol. 2003;466(2):180-96.
21. Markov NT, Ercsey-Ravasz MM, Ribeiro Gomes AR, Lamy C, Magrou L, Vezoli J, et al. A weighted and directed interareal connectivity matrix for macaque cerebral cortex. Cereb Cortex. 2014;24(1):17-36.
22. Snyder SH. Neurotransmitters, receptors, and second messengers galore in 40 years. J Neurosci. 2009;29(41):12717-21.
23. Nicolelis MA, Ribeiro S. Seeking the neural code. Scientific American. 2006;295(6):70-7.
24. Edelman GM, Gally JA. Reentry: a key mechanism for integration of brain function. Front Integr Neurosci. 2013;7:63.
25. Murphy PC, Sillito AM. Corticofugal feedback influences the generation of length tuning in the visual pathway. Nature. 1987;329(6141):727-9.
26. Xiao DK, Marcar VL, Raiguel SE, Orban GA. Selectivity of macaque MT/V5 neurons for surface orientation in depth specified by motion. Eur J Neurosci. 1997;9(5):956-64.
27. Kirkland KL, Sillito AM, Jones HE, West DC, Gerstein GL. Oscillations and long-lasting correlations in a model of the lateral geniculate nucleus and visual cortex. J Neurophysiol. 2000;84(4):1863-8.
28. McClurkin JW, Optican LM, Richmond BJ. Cortical feedback increases visual information transmitted by monkey parvocellular lateral geniculate nucleus neurons. Vis Neurosci. 1994;11(3):601-17.
29. Sherman SM. Tonic and burst firing: dual modes of thalamocortical relay. Trends Neurosci. 2001;24(2):122-6.

30. Sillito AM, Jones HE, Gerstein GL, West DC. Feature-linked synchronization of thalamic relay cell firing induced by feedback from the visual cortex. Nature. 1994;369(6480):479-82.
31. Worgotter F, Nelle E, Li B, Funke K. The influence of corticofugal feedback on the temporal structure of visual responses of cat thalamic relay cells. J Physiol. 1998;509 (Pt 3):797-815.
32. Larkum ME, Zhu JJ, Sakmann B. A new cellular mechanism for coupling inputs arriving at different cortical layers. Nature. 1999;398(6725):338-41.
33. Miller KD. A model for the development of simple cell receptive fields and the ordered arrangement of orientation columns through activity-dependent competition between ON- and OFF-center inputs. J Neurosci. 1994;14(1):409-41.
34. Pinto DJ, Hartings JA, Brumberg JC, Simons DJ. Cortical damping: analysis of thalamocortical response transformations in rodent barrel cortex. Cereb Cortex. 2003;13(1):33-44.
35. Roy SA, Alloway KD. Coincidence detection or temporal integration? What the neurons in somatosensory cortex are doing. J Neurosci. 2001;21(7):2462-73.
36. Usrey WM. The role of spike timing for thalamocortical processing. Curr Opin Neurobiol. 2002;12(4):411-7.
37. Jones HE, Wang W, Sillito AM. Spatial organization and magnitude of orientation contrast interactions in primate V1. J Neurophysiol. 2002;88(5):2796-808.
38. Murphy PC, Duckett SG, Sillito AM. Feedback connections to the lateral geniculate nucleus and cortical response properties. Science. 1999;286(5444):1552-4.
39. Murphy PC, Duckett SG, Sillito AM. Comparison of the laminar distribution of input from areas 17 and 18 of the visual cortex to the lateral geniculate nucleus of the cat. J Neurosci. 2000;20(2):845-53.
40. Murphy PC, Sillito AM. Functional morphology of the feedback pathway from area 17 of the cat visual cortex to the lateral geniculate nucleus. J Neurosci. 1996;16(3):1180-92.
41. Sillito AM, Cudeiro J, Jones HE. Always returning: feedback and sensory processing in visual cortex and thalamus. Trends Neurosci. 2006;29(6):307-16.
42. Jones HE, Sillito AM. The length-response properties of cells in the feline dorsal lateral geniculate nucleus. J Physiol. 1991;444:329-48.
43. Born RT, Bradley DC. Structure and function of visual area MT. Annu Rev Neurosci. 2005;28:157-89.
44. Pack CC, Conway BR, Born RT, Livingstone MS. Spatiotemporal structure of nonlinear subunits in macaque visual cortex. J Neurosci. 2006;26(3):893-907.
45. Albright TD, Desimone R. Local precision of visuotopic organization in the middle temporal area (MT) of the macaque. Exp Brain Res. 1987;65(3):582-92.
46. Maunsell JH, Newsome WT. Visual processing in monkey extrastriate cortex. Annu Rev Neurosci. 1987;10:363-401.
47. Pack CC, Livingstone MS, Duffy KR, Born RT. End-stopping and the aperture problem: two-dimensional motion signals in macaque V1. Neuron. 2003;39(4):671-80.
48. Hupe JM, James AC, Girard P, Lomber SG, Payne BR, Bullier J. Feedback connections act on the early part of the responses in monkey visual cortex. J Neurophysiol. 2001;85(1):134-45.
49. Rockland KS, Knutson T. Feedback connections from area MT of the squirrel monkey to areas V1 and V2. J Comp Neurol. 2000;425(3):345-68.
50. Shipp S, Zeki S. The Organization of Connections between Areas V5 and V2 in Macaque Monkey Visual Cortex. Eur J Neurosci. 1989;1(4):333-54.

51. Silvanto J, Cowey A, Lavie N, Walsh V. Striate cortex (V1) activity gates awareness of motion. Nat Neurosci. 2005;8(2):143-4.
52. Silvanto J, Lavie N, Walsh V. Double dissociation of V1 and V5/MT activity in visual awareness. Cereb Cortex. 2005;15(11):1736-41.
53. Casagrande VA. A third parallel visual pathway to primate area V1. Trends Neurosci. 1994;17(7):305-10.
54. Schiller PH, Logothetis NK, Charles ER. Functions of the colour-opponent and broad-band channels of the visual system. Nature. 1990;343(6253):68-70.
55. Koch C, Greenfield S. How does consciousness happen? Scientific American. 2007;297(4):76-83.
56. Raiguel SE, Lagae L, Gulyas B, Orban GA. Response latencies of visual cells in macaque areas V1, V2 and V5. Brain Res. 1989;493(1):155-9.
57. Schmolesky MT, Wang Y, Hanes DP, Thompson KG, Leutgeb S, Schall JD, et al. Signal timing across the macaque visual system. J Neurophysiol. 1998;79(6):3272-8.
58. Friedman-Hill S, Maldonado PE, Gray CM. Dynamics of striate cortical activity in the alert macaque: I. Incidence and stimulus-dependence of gamma-band neuronal oscillations. Cereb Cortex. 2000;10(11):1105-16.
59. Shu Y, Hasenstaub A, Badoual M, Bal T, McCormick DA. Barrages of synaptic activity control the gain and sensitivity of cortical neurons. J Neurosci. 2003;23(32):10388-401.
60. Tsien JZ. The memory code. Researchers are closing in on the rules that the brain uses to lay down memories. Discovery of this memory code could lead to the design of smarter computers and robots and even to new ways to peer into the human mind. Scientific American. 2007;297(1):52-9.
61. Frey U, Huang YY, Kandel ER. Effects of cAMP simulate a late stage of LTP in hippocampal CA1 neurons. Science. 1993;260(5114):1661-4.
62. Martin KC, Casadio A, Zhu H, Yaping E, Rose JC, Chen M, et al. Synapse-specific, long-term facilitation of aplysia sensory to motor synapses: a function for local protein synthesis in memory storage. Cell. 1997;91(7):927-38.
63. Morris RG, Garrud P, Rawlins JN, O'Keefe J. Place navigation impaired in rats with hippocampal lesions. Nature. 1982;297(5868):681-3.
64. Frankland PW, Kohler S, Josselyn SA. Hippocampal neurogenesis and forgetting. Trends Neurosci. 2013;36(9):497-503.
65. Tashiro A, Makino H, Gage FH. Experience-specific functional modification of the dentate gyrus through adult neurogenesis: a critical period during an immature stage. J Neurosci. 2007;27(12):3252-9.
66. Reijmers LG, Perkins BL, Matsuo N, Mayford M. Localization of a stable neural correlate of associative memory. Science. 2007;317(5842):1230-3.
67. Bliss TV, Collingridge GL. A synaptic model of memory: long-term potentiation in the hippocampus. Nature. 1993;361(6407):31-9.
68. Ressler KJ, Rothbaum BO, Tannenbaum L, Anderson P, Graap K, Zimand E, et al. Cognitive enhancers as adjuncts to psychotherapy: use of D-cycloserine in phobic individuals to facilitate extinction of fear. Arch Gen Psychiatry. 2004;61(11):1136-44.
69. Serrano P, Friedman EL, Kenney J, Taubenfeld SM, Zimmerman JM, Hanna J, et al. PKMzeta maintains spatial, instrumental, and classically conditioned long-term memories. PLoS Biol. 2008;6(12):2698-706.

70. Si K, Giustetto M, Etkin A, Hsu R, Janisiewicz AM, Miniaci MC, et al. A neuronal isoform of CPEB regulates local protein synthesis and stabilizes synapse-specific long-term facilitation in aplysia. Cell. 2003;115(7):893-904.
71. Si K, Lindquist S, Kandel ER. A neuronal isoform of the aplysia CPEB has prion-like properties. Cell. 2003;115(7):879-91.
72. Theis M, Si K, Kandel ER. Two previously undescribed members of the mouse CPEB family of genes and their inducible expression in the principal cell layers of the hippocampus. Proceedings of the National Academy of Sciences of the United States of America. 2003;100(16):9602-7.
73. Hegde AN, Inokuchi K, Pei W, Casadio A, Ghirardi M, Chain DG, et al. Ubiquitin C-terminal hydrolase is an immediate-early gene essential for long-term facilitation in Aplysia. Cell. 1997;89(1):115-26.
74. Bailey CH, Chen M. Long-term memory in Aplysia modulates the total number of varicosities of single identified sensory neurons. Proceedings of the National Academy of Sciences of the United States of America. 1988;85(7):2373-7.
75. Fields RD. White matter matters. Scientific American. 2008;298(3):42-9.
76. Sneed A. Why babies forget. Scientific American. 2014;311(2):28.
77. Petruska JC, Ichiyama RM, Jindrich DL, Crown ED, Tansey KE, Roy RR, et al. Changes in motoneuron properties and synaptic inputs related to step training after spinal cord transection in rats. J Neurosci. 2007;27(16):4460-71.
78. Carp JS, Tennissen AM, Wolpaw JR. Conduction velocity is inversely related to action potential threshold in rat motoneuron axons. Exp Brain Res. 2003;150(4):497-505.
79. Enkel T, Berger SM, Schonig K, Tews B, Bartsch D. Reduced expression of nogo-a leads to motivational deficits in rats. Front Behav Neurosci. 2014;8:10.
80. Kristofikova Z, Vrajova M, Sirova J, Vales K, Petrasek T, Schonig K, et al. N-Methyl-d-Aspartate Receptor - Nitric Oxide Synthase Pathway in the Cortex of Nogo-A-Deficient Rats in Relation to Brain Laterality and Schizophrenia. Front Behav Neurosci. 2013;7:90.
81. Petrasek T, Prokopova I, Sladek M, Weissova K, Vojtechova I, Bahnik S, et al. Nogo-A-deficient Transgenic Rats Show Deficits in Higher Cognitive Functions, Decreased Anxiety, and Altered Circadian Activity Patterns. Front Behav Neurosci. 2014;8:90.
82. Pourabdolhossein F, Mozafari S, Morvan-Dubois G, Mirnajafi-Zadeh J, Lopez-Juarez A, Pierre-Simons J, et al. Nogo receptor inhibition enhances functional recovery following lysolecithin-induced demyelination in mouse optic chiasm. PloS one. 2014;9(9):e106378.
83. Ge S, Goh EL, Sailor KA, Kitabatake Y, Ming GL, Song H. GABA regulates synaptic integration of newly generated neurons in the adult brain. Nature. 2006;439(7076):589-93.
84. Kempermann G, Kuhn HG, Gage FH. More hippocampal neurons in adult mice living in an enriched environment. Nature. 1997;386(6624):493-5.
85. Leuner B, Gould E, Shors TJ. Is there a link between adult neurogenesis and learning? Hippocampus. 2006;16(3):216-24.
86. Leuner B, Mendolia-Loffredo S, Kozorovitskiy Y, Samburg D, Gould E, Shors TJ. Learning enhances the survival of new neurons beyond the time when the hippocampus is required for memory. J Neurosci. 2004;24(34):7477-81.
87. Hastings NB, Gould E. Rapid extension of axons into the CA3 region by adult-generated granule cells. J Comp Neurol. 1999;413(1):146-54.

88. Kee N, Teixeira CM, Wang AH, Frankland PW. Preferential incorporation of adult-generated granule cells into spatial memory networks in the dentate gyrus. Nat Neurosci. 2007;10(3):355-62.
89. Ramirez-Amaya V, Marrone DF, Gage FH, Worley PF, Barnes CA. Integration of new neurons into functional neural networks. J Neurosci. 2006;26(47):12237-41.
90. Leuner B, Glasper ER, Mirescu C. A critical time for new neurons in the adult hippocampus. J Neurosci. 2007;27(22):5845-6; discussion
91. Blakemore C, Cooper GF. Development of the brain depends on the visual environment. Nature. 1970;228(5270):477-8.
92. Wiesel TN, Hubel DH. Ordered arrangement of orientation columns in monkeys lacking visual experience. J Comp Neurol. 1974;158(3):307-18.
93. Civitarese G. Transformations in hallucinosis and the receptivity of the analyst. The International journal of psycho-analysis. 2015;96(4):1091-116.
94. Bardin J. Neuroscience: Making connections. Nature. 2012;483(7390):394-6.
95. Wedeen VJ, Rosene DL, Wang R, Dai G, Mortazavi F, Hagmann P, et al. The geometric structure of the brain fiber pathways. Science. 2012;335(6076):1628-34.
96. Scoville WB, Milner B. Loss of recent memory after bilateral hippocampal lesions. J Neurol Neurosurg Psychiatry. 1957;20(1):11-21.
97. Squire LR. Memory and the hippocampus: a synthesis from findings with rats, monkeys, and humans. Psychol Rev. 1992;99(2):195-231.
98. O'Keefe J, Dostrovsky J. The hippocampus as a spatial map. Preliminary evidence from unit activity in the freely-moving rat. Brain Res. 1971;34(1):171-5.
99. Nasir SM, Ostry DJ. Auditory plasticity and speech motor learning. Proceedings of the National Academy of Sciences of the United States of America. 2009;106(48):20470-5.
100. Jousmaki V, Hari R. Parchment-skin illusion: sound-biased touch. Curr Biol. 1998;8(6):R190.
101. Foxe JJ, Wylie GR, Martinez A, Schroeder CE, Javitt DC, Guilfoyle D, et al. Auditory-somatosensory multisensory processing in auditory association cortex: an fMRI study. J Neurophysiol. 2002;88(1):540-3.
102. Fu KM, Johnston TA, Shah AS, Arnold L, Smiley J, Hackett TA, et al. Auditory cortical neurons respond to somatosensory stimulation. J Neurosci. 2003;23(20):7510-5.
103. Kayser C, Petkov CI, Augath M, Logothetis NK. Integration of touch and sound in auditory cortex. Neuron. 2005;48(2):373-84.
104. Murray MM, Molholm S, Michel CM, Heslenfeld DJ, Ritter W, Javitt DC, et al. Grabbing your ear: rapid auditory-somatosensory multisensory interactions in low-level sensory cortices are not constrained by stimulus alignment. Cereb Cortex. 2005;15(7):963-74.
105. Schurmann M, Caetano G, Hlushchuk Y, Jousmaki V, Hari R. Touch activates human auditory cortex. Neuroimage. 2006;30(4):1325-31.
106. Shore SE, Zhou J. Somatosensory influence on the cochlear nucleus and beyond. Hear Res. 2006;216-217:90-9.
107. Fadiga L, Craighero L, Buccino G, Rizzolatti G. Speech listening specifically modulates the excitability of tongue muscles: a TMS study. Eur J Neurosci. 2002;15(2):399-402.
108. Watkins KE, Strafella AP, Paus T. Seeing and hearing speech excites the motor system involved in speech production. Neuropsychologia. 2003;41(8):989-94.
109. Meister IG, Wilson SM, Deblieck C, Wu AD, Iacoboni M. The essential role of premotor cortex in speech perception. Curr Biol. 2007;17(19):1692-6.

110. Gollo LL, Mirasso C, Villa AE. Dynamic control for synchronization of separated cortical areas through thalamic relay. Neuroimage. 2010;52(3):947-55.
111. Boly M, Moran R, Murphy M, Boveroux P, Bruno MA, Noirhomme Q, et al. Connectivity changes underlying spectral EEG changes during propofol-induced loss of consciousness. J Neurosci. 2012;32(20):7082-90.
112. Tahvildari B, Wolfel M, Duque A, McCormick DA. Selective functional interactions between excitatory and inhibitory cortical neurons and differential contribution to persistent activity of the slow oscillation. J Neurosci. 2012;32(35):12165-79.
113. Doesburg SM, Green JJ, McDonald JJ, Ward LM. From local inhibition to long-range integration: a functional dissociation of alpha-band synchronization across cortical scales in visuospatial attention. Brain Res. 2009;1303:97-110.
114. Edelman GM, Gally JA, Baars BJ. Biology of consciousness. Front Psychol. 2011;2:4.
115. Buzsaki G, Wang XJ. Mechanisms of gamma oscillations. Annu Rev Neurosci. 2012;35:203-25.
116. Hipp JF, Engel AK, Siegel M. Oscillatory synchronization in large-scale cortical networks predicts perception. Neuron. 2011;69(2):387-96.