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Editorial

Hypothesis on two different functionalities co-existing in frontal lobe of human brains

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ABSTRACT

Human frontal lobe is a key area from where our cognition, memory and emotion display or function. In medical case study, there are patients with social dysfunctions, lack of passion or emotion as result of their frontal lobe damage caused by pathological changes, traumatic damage, and brain tumor remove operations. The syndrome of frontal lobe damage remains at large unanswered medically. From early stage of pregnancy, there exists lobe layers, nerve combine, and neurons synaptic, indicating a completion of growth of functionality inside frontal lobe. However, this completion of growth does not match the growth of human intelligence. Human infants only start and complete their cognition and memory functionality one full year after their birth which is marked by huge amount of neurons synaptic inside their frontal lobe, which is not part of a continual growth of originally developed functions. By reasoning on pathological changes of frontal lobe, a hypothesis was established that two individually functional mechanisms co-existed inside one frontal lobe. This neuron system is particularly for human beings.

Human being has a long history of wanting to understand ourselves. Socrates' quote of a saying from Delphin Temple "Knowing yourself" is nothing but an excellent example of expressing such desire. Modern neurology, throughout its research and medical practice, has accumulated many meaningful cases and data which indicate frontal lobe is one of the areas where many medical puzzles related to human social behavior, cognition, intelligence and memory exist [1,2]. Frontal lobe also is the largest tissue area of human brain. Discussions on frontal lobe usually focus on prefrontal cortex (PFC) [3]. Researches on animals also indicate frontal lobe has equally significant importance to them. Brodmann (1909/1960) has mapped in details of human brain tissues. Various observations made on frontal lobe either from developmental changes, frontal lobe damage, strokes, medical operations caused impact as well as anatomy and functional magnetic resonance imaging (FMRI) show plenty of behavior and social dysfunction from patients that are difficult to explain medically. Firstly we can take a look at traumatic pathological changes on frontal lobe from anatomy and brain growth image perspective.

Besides the cognition and memory function of frontal lobe, some researchers have already grouped certain human behaviors after patients suffered from frontal lobe damages. Luria concluded abnormal behavioral syndrome, after frontal lobe damage, include lack of mental control, no inhibitory impulsiveness, insane, sluggish, stubbornness, indifference and partial response lessness [4]. It is widely accepted that patients who suffered from frontal lobe damage tend to show insufficient social ability, lack of curiosity, becoming less communicative, losing ability to feel shamed and attached or increasingly being attracted to repetitive acts. Case report compiled by Ackerly and Benton tells patients who suffered from frontal lobe damage show tendency of doing mechanical acts, lack of imaginative and initiative ability [5]. All these data clearly tells us cases of frontal lobe damage tend to lead patients in showing various abnormal behavior and emotions.

Yet, there still lacks a general consensus among researchers as on which part of frontal lobe the damage causes such abnormal behavior or emotions. Researchers still fail to identify, inside frontal lobe, particular areas or parts to certain functionality. Researchers also found it seems individual patient has displayed different results. Such finding was widely spread among human frontal lobe research, called indefinite frontal lobe function [6]. Lab researches find frontal lobe on primates has a much more defined functionality than human's. For instance, lab research on monkeys reports some monkeys had their lateral prefrontal cortex and medial frontal cortex removed. Then they went back to their group. Though they displayed no visible physical differences to rest of the group, they showed visible inability to maintain their overall relations with rest of group. They also communicated less emotionally to rest of group. Their sexual desire reduced. So is their parental behavior. Their relations to rest of the group became more abnormal after the operation [7]. This lab research happens to be identical to the findings on human being who suffer similar frontal lobe damages.

Ackerley and Benton recorded one case in their report which has particular relevance to this paper. The patient was found, in his young age, a large part of his frontal lobe damaged already. When the patient reached adult age, another medical check found his left frontal lobe area almost hollow, his right frontal lobe has severely became smaller. Patient still had some intelligence, though clearly paranoid, not able to understand basic technical work, lack of passion to most of things, losing human skills, unable to be managed, stealing, sudden loss of his temple, restlessness and no emotional display to females [5].

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Anatomy shows human, as well as primate; start to grow its cerebral frontal lobe as soon as pregnancy is realized. Human brain has already reached its certain level of growth even at as early stage as fetus. At that stage, layers of cerebral frontal lobe, nerve combine and meylin all become fairly developed. Their growth reached the completion before birth [8]. The images of anatomy tell us frontal lobe is one of the areas where earliest growth happens. According to Rakic's researches, human frontal lobe growth started to happen and formulate through neuron migration at early pregnancy [9]. Frontal lobe neurons from axon in the form of white matter vertically rush to other areas of brain and to thalamus. This is reflected in new born baby' behavior, just like other primates.

In study on time period when synaptic formulation happens in PFC and auditory cortex using phosphotungstic acid (PTA) staining method, we find human brain develops its different parts at different times. The peak of synaptic density of temporal lobe in auditory cortex usually sits around three months after birth while the peak of synaptic density of connecting area or part of frontal lobe sits about fifteen months after birth [10,11].

Chugani in his positron emission tomography (PET) on live brain development by studying glucose metabolism, found different charts of glucose metabolism representing different areas or parts of brain's development period [12]. In other words, it is one specific brain part which grows with its particular glucose metabolism chart at a specific time. For instance, relatively speaking, frontal lobe is the latest that develops (six to twelve months after birth), about ten months later than other parts like parietal lobe, temporal lobe, primary visual cortex, basal ganglia, and cerebellar hemisphere [12].

Above findings clearly indicate frontal lobe in human brains grows at very early stage. Neverthless, this still does not answer another question which is why, one full year after birth, human infant brain reaches another peak of synaptic density in frontal lobe connecting area despite the fact frontal lobe functionality remains under a normal working condition. Do infants, after twelve months of their birth, display any frontal lobe functionality that does not seem normal? If we look at data from observations made by Luria on different behaviors by patients with frontal lobe damage, the answer clearly is a no from pregnancy to one full year old infant. We can also say one full year old infant or younger, his intelligence capacity is more or less the same or very similar to a chimpanzee in same age group. Diamond also believes human infants at one full year old or younger are not able to engage any intelligent activities [13].

Whether the way how human frontal lobe neurons connect is different from that of the animals

From clinic frontal lobe syndrome to behaviors display by frontal lobe patients, it seems CLF has its highly sophisticated system, if we combine points above. Human infants, in one year after their birth or less, have their frontal lobe functionality fully grown. This growing process could even complete when it still remains fetus. Twelve months after birth, infants face another synaptic density peak in their frontal lobe connecting area. That is not a part of continual growth of frontal lobe's initial development, but rather a brand new growth of a brand new functionality. Even though this new growth happens within the frontal lobe that already has originally developed functionalities. The two functionalities are not co-related. Neurons from both functionalities grow right next to each other in the same area or part. Hypothesis human beings, in one year after its birth, start to show two individually different functionalities co-exist inside frontal lobe, that is Original Frontal Lobe Location Function (OFLF) and new Cognition and Memory Functionality Networks (CMFN). Growth of CMFN is the start of human intelligence. The peak of synaptic density in frontal lobe connecting area matches the time of intelligence development of human being.

The initial growth for the purpose of connecting different parts of brain in the form that frontal lobe neurons vertically rush into other parts of brain in white matters happens at infant stage [8,21], similar to radial unit hypothesis [9]. PFC contains massive amount of networks, responsible for connecting different functionalities, activities, cognitions and other remote areas [14]. But the development of CMFN happens inside the space of cortex which was originaly developed and formulated. And it formulates a networks or tend to become a networks of that kind, which inconsistently connects synapse horizontally. The formation of this inconsistently connected networks of synapse and its tendency to link or eat each other up into a larger one eventually grows into a networks, which is similar to the closed loop circuit (CLC). Partial damage would have no impact on the overall operation, this is one of the characteristics of CLC. This new process tends to bring pressure and squeeze to the space occupied by originally grown functionalities. This process is possibly responsible for the creation of individual indefinite frontal lobe function as stated by [6]. It is also possible that CMFN could extend to orbitofrontal cortex (OFC), Supeior Temporal Sulcus and the front cortex of Superior Temporal Gyrus.

CMFN has simpler formality in comparison to original OFLF. It only has storage function of massive amount of memory and processed information. It is different from episodic memory which is not part of discussion here. Memorized information in CMFN can been taken out of it or put back in it anytime without any special tasks serving as activations so to speak (see discussion on intrinsic activity of brain in this paper). CMFN has its own independent nerve circular connection which grows by itself.

In neurological anatomy, in relation to nerve circular connection, there are 2 theories that happen to share with my hypothesis. One of them says there is no direct connection between PFC and hippocampus. Information being transmitted to hippocampus happens mainly through cingulate gyrus, parahippocampal gyrus, entorhinal cortex [15]. Another theory suggests there are nerve fibers in PFC which directly connect to hippocampus and thalamus [16,17]. Croxsen et al. (2005) believes the spread of nervous tracks in cerebral cortex connected to prefrontal lobe has its irregularity [18]. In anatomy the circular connections between PFC and hippocampus do commonly exit. There also exist some evidences that there are various connections between lateral prefrontal cortex, medial frontal cortex, orbit frontal lobe and BA6 behind thalamus in Brodmann area. There are also other evidences showing nerve connections between frontal lobe and lower thalamus. Above argument suggests multiple nerve connections between cortex and thalamus. Thus, newly formed CMFN starts to function on its own in the following year. It will take a long time to reach its full growth if one observes on myelinization process.

Whether human frontal lobe damage and autistic-spectrum disorder would support this hypothesis.

Why do we assume CMF grows to become an independent networks? The complicated pathologic changes of frontal lobe lead us to this hypothesis. The evolution of human being's physical conditions up till today has brought us more questions than answers in regard to our brain. Human infants reach one year old age in their growth of synaptic networks formulation. If there is local over connectivity in the process of increasing density of synaptic cell, certain syndrome will show up. The frontal lobe of human brain is one of areas in modern neurology that have most unanswered questions. These unanswered questions include, though not necessarily restrict to, pathologic changes in growth, traumatic changes of frontal lobe, social function disability after accidents and