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Psychobiological Model of Volition—Implications for Mental Disorders

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Abstract

A new psychobiological model of volitive processes and its implications for the etiopathology of mental disorders is proposed. The model is based on five elementary volitive processes. These are the volition to act; the volition to self-instrumentalize; the volition to program intentions, the volition to generate realities; and the volition to permanent existence. Imbalances in information processing in tripartite synapses and their network may be responsible for dysfunctions of self-instrumentalization. It is suggested that the volition to permanent existence unconsciously works in mental disorders, but the volition to intentional programming of realities and the volition to generate realities by communication with subjects and objects in their environment are impaired. In depression, the volition to act is constrained by hyperintentional programs that are non-feasible in the environment. In mania volitive processes are totally oriented on events in the environment without any goal-directed programming. Dysfunctions of volitive processes in schizophrenia are fundamentally caused by severe impairments of self-instrumentalization. As shown in tripartite synapses a gap between sensory information processing in the neuronal network and the inner glial networks causes the inability of schizophrenics to distinguish between the self and the other. In delusions, the destiny for communication becomes staged as pseudo-communication. Together, the study outlines a new model of volitive processes and deduces dysfunctions responsible for communication pathology and abnormal reality experiences of patients with mental disorders.

Keywords

Model of Volition, Disorders of Volition, Depression, Mania, Schizophrenia

1. Introduction

The empirical oriented framework of volition according to W. James [1] con-

fronts us with the theoretical problem of agency and authorship and mental causation. Authorship denotes that voluntary actions are basically controlled and "authored by a personal agent" either within the machinery or behind it [2]. Mental causation means that mental properties like intentions exert the power to cause physical movements of the body. Basically, a psycho-biological model of volitive processes should elucidate how the brain is organized that actions of the self and actions of another can be distinguished [3]. Brain imaging experiments indicate that different neuronal networks operate for the recognition whether the action originates from the self or not [4]. Moreover, we must not only consider the neuronal networks, but also the glial networks as distinguished structures for action generation.

Though cognition and volition are both fundamental capabilities of the mind, and must be investigated in their interactions, I will focus on volitive processes as the primary power to act enabling the reflection of their actions by cognitive operations. The psychobiological model here proposed will apply "Occams Razor" by introducing a few but elementary principles of volitive processes.

The study starts out with the description of the psychobiological model of volitive processes. Subsequently, volitive dysfunctions and impairments in depression, mania and schizophrenia are deduced from the model. In conclusion, the significance of volitive dysfunction and impairments for the understanding of communication pathology and abnormal reality experience of patients with mental disorders is discussed.

2. Architecture of Volitive Processes

Generally, volition represents a conscious decision process [3]. However, recent investigations of the processes of goal pursuit discovered that these processes also operate without conscious awareness [5]. I propose an architecture of five elementary volitive processes conceptualized as

The primordial volition to act;
The volition to self-instrumentalize;
The volition to program intentions;
The volition to generate realities;
The volition to permanent existence.

2.1. The Primordial Volition to Act

The primordial volition to act commands and controls all volitive processes. Augustinus was the first in the history of Western Philosophy who described the primordial will as absolutely free from external and internal constraints and especially independent on reason [6]. As a metaphysical conception, the primordial will is driven by the movements of the soul and can therefore not be identified in the brain. However, primary volitive processes can be described in quantum mechanics. Recently, Baer developed a theory of cognitive action cycles [7] [8]. Since action cycles rotate permanently, they function basically as volitive processes but are the prerequisite for further cognitive operations, because "volition

without cognition is blind" [9]. Importantly, action cycles are organized in a hierarchical order with an all compromising action cycle on the highest level of organization that operates as an act of self-reference.

Fundamentally, unlike the design of machines with special structures and functions, biological systems develop spontaneously and organize themselves [10]. This circularity must maintain in a living system to retain its identity through the dynamics of interaction [11]. Varela [12] formalized this circular organization as a pure act of self-reference. However, this holistic act of self-reference generated in the brain may remain a mystery and is the edge between experimental brain research and brain philosophy.

I suggest that self-reference operates on two elementary mechanisms in the brain. It represents a holistic, all integrating function, but in doing so, it must combine discontinuities or disconnections for the generation of action domains or realities. From a psychobiological point of view self-reference can be characterized as the elementary operation of narcissism [13] (see Section 2.5).

2.2. The Volition to Self-Instrumentalize

For the generation of action effects or products, the volition to act must self-instrumentalize with organs and mechanisms in the brain guaranteeing inner homeostasis and the generation of behavior for coping with the environment.

There is a lesson to be learned from neuroembryology that motility precedes reactivity in the sense of a chronological primacy of the motor over the sensory. "It is well established that the basal plate, or motor part, of the spinal cord proliferates and differentiates long before the altar plate, or dorsal part, which receives sensory input. This observation has led some to speculate on the primacy of motor function, in a way that might provoke the cognitive neuroscientists." [14]. The embryonic development allows the interpretation that the primordial volition to act starts its self-instrumentalization by movement. Typical mechanisms represent spontaneous electrical activity in the human fetal cortex [15] and spontaneous astrocytic Ca^{2+} oscillations [16]. Whereas machines are designed by humans to display special structures and functions, in living systems or the human brain structures and functions evolve spontaneously "without a priori specification or representation of a new structure" [17]. An elementary structure of self-instrumentalization for information processing in the brain represents tripartite synapses, discussed in the next section.

Volitive Processes in Tripartite Synapses

In the brain model proposed the neuronal element (presynapse and postsynapse) embodies objective subjectivity and the glial part subjective subjectivity [18]. **Figure 1** shows a schematic diagram of feedforward-feedback loops between the glial network (GN) embodying subjective subjectivity (S^s) and the neuoronal network (NN) embodying objective subjectivity (S°) of the brain. First, the glial network feeds forward to the neuronal network 1). Further, the neuronal network feeds back to the glial network 2). The glial network feeds forward to the neural

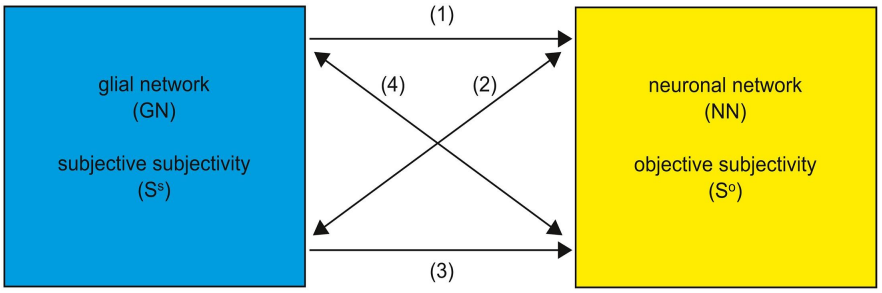

Figure 1. Feedforward-feedback loops between the glial network (subjective subjectivity) and the neuronal network (objective subjectivity).

The glial network (GN) *interpreted as subjective subjectivity* (S^s) *feeds forward to the neuronal network* (*NN*) *interpreted as objective subjectivity* (S^o) (1). S^s *feeds back to* S^s (2), S^s *feeds forward to* S^o (3) *and* S^o *feeds back to* S^o (4) *generating a loop.*

network again 3) and the neuronal network feeds back to the glial network closing the feedforward-feedback loop. Importantly, feedforward commands and controls the mental architecture [19] allowing the interpretation that the feedforward represents an elementary volitive function. We experience objective subjectivity as subjects (Thou's) in the environment and feel subjective subjectivity as Ego-consciousness on the behavioral level. For the understanding of disorders of volition the model of a tripartite synapse (consisting of the presynapse and postsynapse as the neural part and the astrocyte as the glial part) may provide a biological basis.

Figure 2 outlines the elementary mechanisms of astrocyte-synapse interactions. Contrary to the prevailing cognitive models of tripartite synapses that are primarily activated by sensory information, in the perspective of volitive processes synaptic information processing becomes first activated by spontaneous pulsations of the astrocyte. I hypothesize that these spontaneous pulsations generate gliotransmitters (GT) 1) that activate cognate receptors on the presynapse (psR) 2). This leads to the release of neurotransmitters (NT) from the presynapse that activate both postsynaptic receptors (poR) and astrocytic receptors (acR) 3). The activation of astroglial receptors enhances Ca^{2+} and Ca^{2+} waves in the astroglial network. Moreover, the spontaneous pulsations of the astrocyte not only produce gliotransmitters, but also activate astrocytic receptors in the sense of readiness configuration 5). For turning off synaptic information processing gliotransmitters occupy both presynaptic receptors and extrasynaptic receptors (esR) 6). Most importantly, in the perspective of volitive processes, the astrocyte and its network primarily activate synaptic information processing exerting a volitive feedforward mechanism on the neuronal synapse that feeds back to the astrocyte in the sense of a cognitive operation. Here we deal with a new approach to brain theory focusing on elementary volitive mechanisms as the prerequisite for cognitive operations.

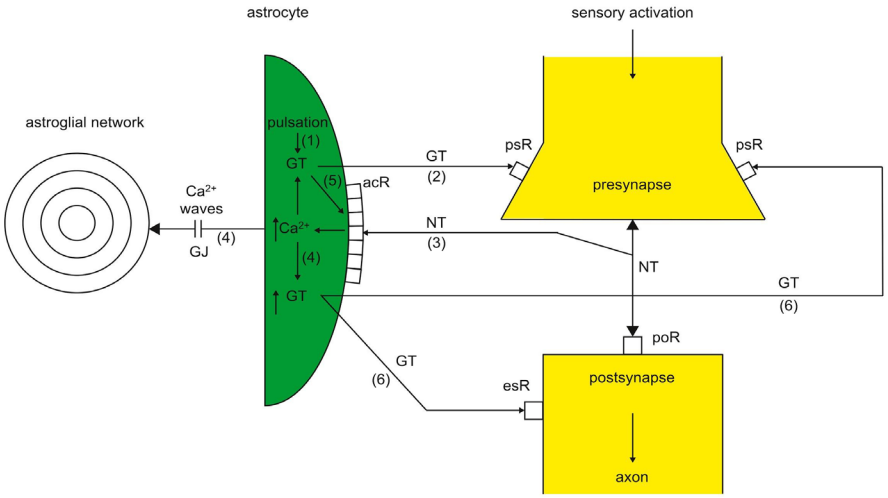

Figure 2. Elementary mechanisms of astrocyte-synapse interaction.

Spontaneous pulsation of the astrocyte generates gliotransmitters (*GT*) 1). *GTs activate presynaptic receptors* (*psR*) 2). *Sensory activation of the presynapse leads to the release of neurotransmitters that occupy both postsynaptic receptors* (*poR*) *and actrocytic receptors* (*acR*) 3). *The activation of acR by NT enhances* Ca^{2+} *concentration and* Ca^{2+} *wave propagation in the astroglial network via gap junctions* (*GJ*) 4). *GTs exert a double function activating both acR* 5) *and presynaptic and extrasynaptic receptors* 6).

2.3. Volitive Programming of Intentions

In the development of the brain, various cell types with special structures and functions become generated and are interconnected building networks. In these networks, feedback mechanisms are generated in different degrees of complexity [20] from recursion, reflective thinking, self-reflection to inter-subjective reflection [21]. These reflection processes represent not only cognitive operations, but generate also intentional programs in special networks such as in the hippocampus. Morgan and Piccinini [22] argue that cognitive Neuroscience can explain intentionality in terms of information and biological function. By identifying special neural representations the puzzling intentional properties of mental states can be explained. Basically, intentional programming has been formalized in astroglial networks [23].

Figure 3 outlines memory formation and intentional programming. Six astrocytes/Ac_1...Ac_6 are interconnected via fifteen gap junctions (GJ). Astrocytes contact neuronal synapses building tripartite synapses (ts_y, only one is shown). These elementary components and conditions build an astroglial network. Frequently activated GJs (arrows) form a plaque (bold connection lines) that becomes embodied in a hierarchical loop structure. Since gap junctions are dynamic structures, they form plaques by continually adding and removing (dashed loop) channels [24]. Therefore, memory architecture does not operate immediately on actual information, but is generated by sequential phases of information [25]. Importantly, the role of gap junctions in memory formation [26] allows the interpretation that intentional programming is based on memory

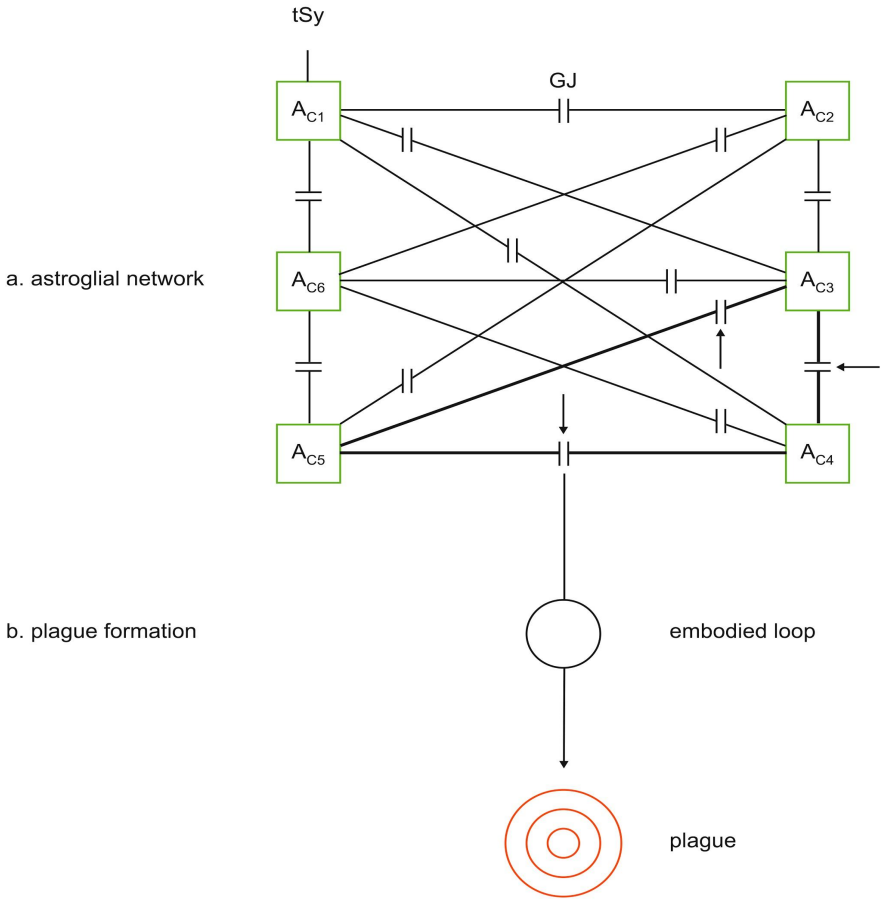

Figure 3. Formation of memory structures in the astroglial network.

Six astrocytes ($Ac_1 \cdots Ac_6$) *are completely interconnected via gap junctions* (*GJ*). *Each astrocyte* (*Ac*) *contacts a neuronal synapse* (tS_y, *only one is shown*). *Frequently activated GJs* (*arrows*) *generate a plaque. An example of plaque formation is given between an* Ac_3, Ac_4, Ac_5, Ac_3-*loop* (*bold lines*). *The loop becomes embodied in a gap junction plaque* (*cycles*) [29].

structures dynamically actualized and modified by events in the environment computed in the neuronal networks. If we assume that the astroglial networks process subjective information generating intentional programs, then they exert a feedforward mechanism to the neuronal networks in the sense of a volitive operation.

Moreover, intentional programs may control the expression of astrocytic receptors in tripartite synapses ready for occupancy with cognate neurotransmitters. Since the astrocytic receptor pattern determines the transmission of neurotransmitters, a qualitative information processing occurs in tripartite synapses [27]. Here we deal with an elementary volitive mechanism in tripartite synapses, since the astroglial network generates a kind of expectation program for appropriate environmental information.

2.4. Volitive Processes Generating Realities

Volitive processes generating realities operate on the action styles of acceptance

and rejection enabling the individual to decide which reality becomes generated selected from a repertoire of possibilities [28]. Nonetheless, the possibility to generate a reality depends on the availability of appropriate subjects and objects in the environment. Basically, an intentional program is striving for its feasibility in the environment. To guarantee individuality information must be structured by rejecting inappropriate information. Günther characterized rejection as the "index of subjectivity" [29]. Moreover, the action style of rejection is decisive in communication, since it frees us not only to reject inappropriate conditions, but also to reject appropriate possibilities. In the latter case, volitive processes create a novel reality. Normally, communication is based on an interplay between acceptance and rejection.

Experimental findings on motile astrocyte processes may elucidate the volition mechanisms of acceptance and rejection [30]. **Figure 4** outlines a motile astrocytic process and its function in network structuring [31]. A process emanating from the astrocyte body contacts the pre-postsynaptic components of the synapse (arrow) and retracts from it (reversed arrow). The rhythmic pulsation of the astrocyte body exerts a pacemaker via the motile astrocytic processes [32]. The time scale of the rhythmic pulsation of the astrocyte body occurs in minutes, but astrocytic processes activate the synapses in shorter time scales. This mechanism is also responsible for the dynamic construction and destruction of gap junction plaques by adding (bold cycle) and removing (dashed cycle) substances (drawn in cycles) from the gap junction plaques [24]. Here we may deal with intentional programming based on dynamic construction of an embodied program. I suggest that the contacting and retracting of astrocytic processes represent the most elementary mechanism of acceptance and rejection in the brain. It exerts an action network structuring function, and allows the interpretation of a fundamental volitive mechanism.

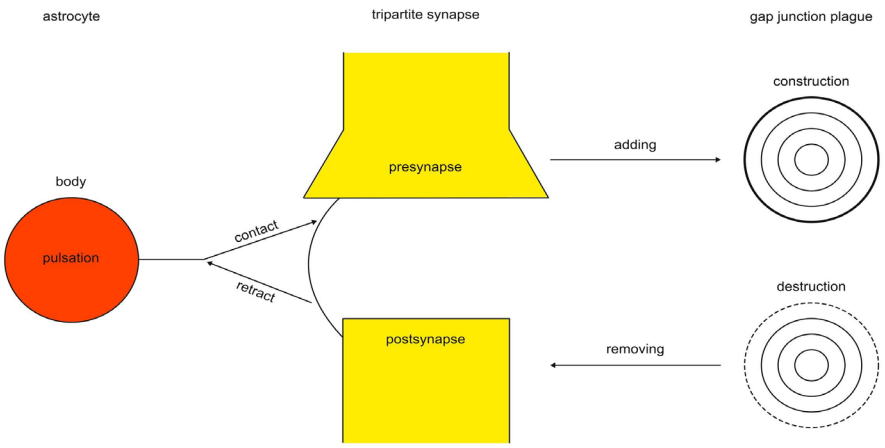

Figure 4. Motile astrocytic processes and network structuring.

From the pulsating body of the astrocyte processes emanate that either contact (*arrow*) *the pre-post synapse or retract from it* (*reversed arrow*). *This mechanism may dynamically structure the gap junction plaque formation in the astroglial network by adding* (*bold cycle*) *and removing an embodied cycle* (*dashed cycle*) [31].

On the behavioral level, intersubjective communication is based on the intentional programs of the subjective subjectivity ("J") and the objective subjectivity ("You"). The purpose of communication is to program and generate a common action domain of reality. Given that the operations of volitive processes as in the model proposed are undisturbed, communication has to cope with the situation that the intentional programs of the partners often differ in one or more aspects. Though differences arise in the dialogue, they are also unconsciously at work [5]. Notwithstanding, if conscious agreement occurs, communication is co-determined by hidden intentional programs of each partner. Therefore, a sudden break up of communication, not rationally explainable can be caused by unconscious intentional programs that reject further communication and make the generation of a common action domain impossible [33].

2.5. The Volition to Permanent Existence

Let us consider again the primordial volition to act quantum mechanically described as action cycles that permanently rotate. Biologically speaking, it is the circular organization of a living system that maintains its identity [11]. We proposed a concept of narcissism that is based on the maintenance of the circular organization of an organism maintaining its identity as an invariable logic of living systems [34]. Importantly, Freud [35] states that the narcissistic organization is never fully abandoned. I suggest that the logic of narcissism may underlie the volition to permanent existence. Masserman characterized what the essence of narcissism may be: "any human being... has at the basis of his metaphysical system a set of solipsistic formulas to this outstanding effect": "once I was absolute, transcendent and inviolate, and what I was once perhaps I have always been and shall continue to be furthermore'" [36]. Given the volition to act that works in permanent time, we may feel unconscious striving for permanent existence [33].

Admittedly, the conception of the volition to permanent existence represents the watershed where natural science ends and metaphysics begins. However, this elementary principle of volitive processes may elucidate the reality experience of patients suffering from mental disorders.

3. The Passions of Volition in Mental Disorders

The psychobiological model of volition enables the interpretation of mental disorders as disorders of volition in the same sense of passions of volition.

3.1. The Passion of Volition in Depression

The typical symptoms of major depression are depressed mood, disturbance of circadian rhythms, diminished interest, loss of pleasure, retardation or agitation, feelings of insufficiency and cognitive impairment [37]. These symptoms have the common feature of "I cannot do" connoting a dysfunction of volitive processes. Basically, I suggest that patients with depression are burdened by a hyperintentional psychobiological state, since the intentional programs are non-feasible in the en-

vironment [13] [38]. Though the volition to program intentions or realities operates in depressive episodes mostly undisturbed, the volitive process to generate realities are significantly impaired by imbalances of information processing in tripartite synapses.

As described in Section 2.2.1 both the neuronal component and the glial component (astrocyte) of a tripartite synapse are endowed with receptors for occupancy by neurotransmitter substances. In normal function neurotransmission between the neuronal synapse and the astrocyte is balanced, since all receptors are activated with an appropriate amount of transmitter substances. **Figure 5** outlines imbalances in tripartite synapses responsible for the pathophysiology of major depression. Gap junctions (GJ) are overexpressed in the astroglial network (red cycle) causing an overexpression of astrocytic receptors (acR ↑) 1). The occupancy of acR with neurotransmitters (NT) is incomplete, since not all receptors are activated by NT in real time 2). The underactivation of acR causes a diminished Ca^{2+} concentration and production of gliotransmitters (GT) (down arrows) 3). This leads to a protracted activation of postsynaptic receptors (psR), extrasynaptic receptors (esR), and a protracted negative feedback on presynaptic receptors (psR) 4) delaying neurotransmission 5). Note that the intentional programs (drawn as cycles) are "frozen" (red cycle), since the diminished Ca^{2+} concentration cannot activate the astroglial network in real time (dashed arrowed

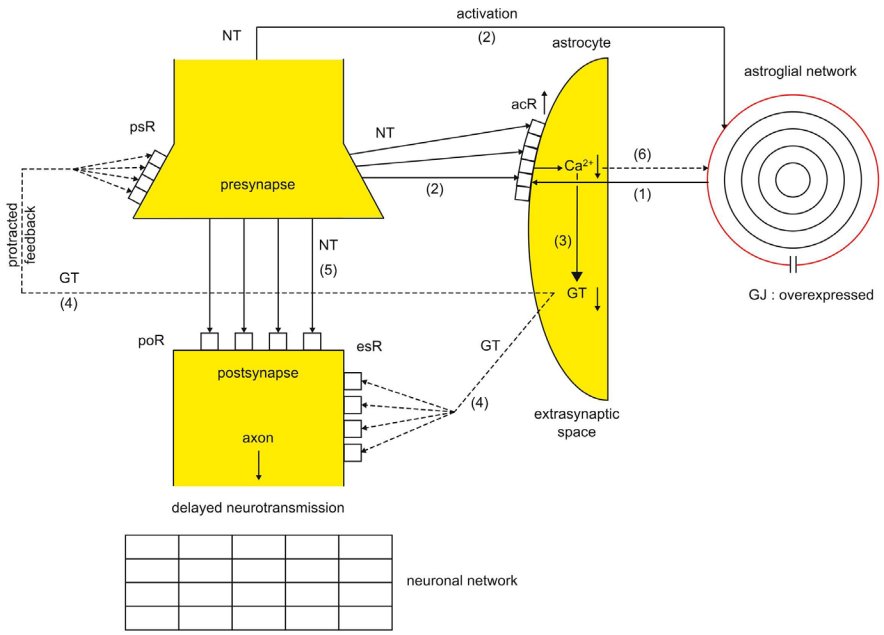

Figure 5. Imbalanced tripartite synapse in depression.

In the astroglial network the gap junctions (*GJ*) *are overexpressed* (*red cycle*) *causing an overexpression of astrocytic receptors* (*acR*, *upwards arrow*) 1). *Neurotransmitters* (*NT*) *released from the presynapse activate postsynaptic receptors* (*poR*), *acR and the astroglial network* 2). *Since acR are overexpressed*, *NT cannot completely activate acR* 3). *This leads to diminished* Ca^{2+} *concentration and production of gliotransmitters* (*downward arrows*). *The underproduction of GT leads to the protracted activation of presynaptic receptors* (*psR*) *and extrasynaptic receptors* (*esR*) 4) *delaying neurotransmission* 5).

line) 6). The prolonged synaptic information processing is explanatory for retardation of depressive behavior and the slowing down time experience felt as "time stands still". Fundamentally, the overexpression of gap junctions, genetically and epigenetically-educationally determined (red cycle) may be experienced by the patient as "high aspirations" [39] like a mountain too high to climb. Importantly, Kalis and colleagues presented a model of the weakness of will in depression focusing on option generation as a neglected topic in decision making [40].

Considering the action styles of acceptance and rejection, we observe a typical behavior in depression. Usually, patients feel forced to accept their burdening existential state unable to adapt intentional programs to the environmental situation. However, some patients are able to radically reject the unbearable pain of depression by comitting suicide. We read in suicide notes that suicides are often convinced that their lives will continue in "paradise" [41]. According to Plato [42], the will to permanent existence and immortality is a fundamental desire of human beings. However, the road to eternity is very painful in depression, since disordered volitive processes inhibit the generation of constructive realities, even in everyday life. Though a chronic course of illness with significant cognitive impairment can occur, after the remission of a depressive episode the volition to create novel realities can successfully work again. We know from the history of culture and science that geniuses are indeed inclined to depression, but regain their creativity after the remission of a depressive episode [43].

3.2. The Passion of Volition in Mania

The core symptoms of mania are inflated self-esteem or grandiosity, pressure to keep talking, decreased need for sleep, flight of ideas, distractibility, increase of activity and exercise, and involvement in pleasurable activities [37]. I propose that a disorder of volitive processes may underlie the abnormal manic behavior. Basically, the volition to act and the volition to generate realities are totally event-oriented or work spontaneously without any goal directed programming. Moreover, the volition to permanent existence and immorality is experienced by the patient as grandiosity. However, the generation of realities is incomplete and mostly destructive, since self-instrumentalization is impaired. As outlined in depression imbalances in tripartite synapses may also be responsible for the pathophysiology of mania.

Figure 6 depicts the core pathophysiological mechanisms of mania caused by imbalances in tripartite synapses. The underexpression of gap junctions (GJ ↓) in the astroglial network causes also an underexpression of astrocytic receptors (acR ↓) 1). Neurotransmitters (NT) released from the presynapse overactivate astrocytic receptors 2). This leads to advanced Ca^{2+} concentration and production of gliotransmitters (GT ↑) 3). Therefore, gliotransmitters overactivate extrasynaptic receptors (esR) and presynaptic receptors (psR) leading to a shortened feedback on the presynapse 4), and a flooding of postsynaptic receptors (poR) with neurotransmitters 5). Note that intentional programs (cycles) in

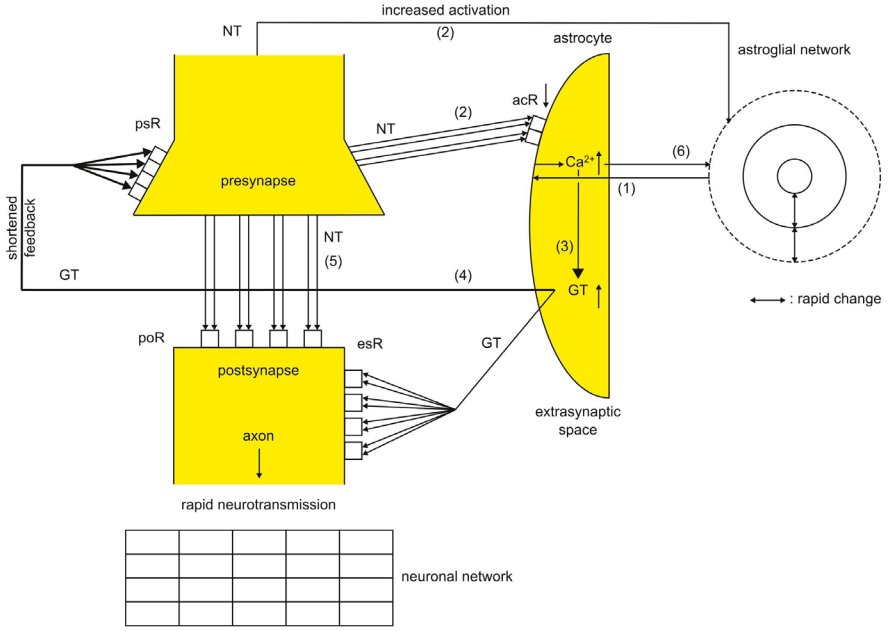

Figure 6. Imbalanced tripartite synapse in mania.

Underexpressed gap junctions (*GJ*) *in the astroglial network* (*dashed cycle*) *cause an underexpression of astroglial receptors* (*acR*, *downwards arrow*) 1). *Neurotransmitters* (*NT*) *released from the presynapse overactivate acR and the astroglial network* 2). *The overactivation of asR leads to enhanced* Ca^{2+} *concentration and the production of gliotransmitters* (*GT*, *upwards arrow*) 3). *The overproduction of GTs overactivates presynaptic receptors* (*psR*) *and extrasynaptic receptors* (*esR*) *causing a shortened feedback on the presynapse* 4) *and a flooding of postsynaptic receptors* (*poR*) *leading to rapid neurotransmission* 5). *The underexpression of GJ may cause a rapid change of the structure of the astroglial network* (*double headed arrows*).

the astroglial network rapidly change (double headed arrows), since the enhanced Ca^{2+} concentration overactivates the astroglial network (bold arrow) 6).

Though experiments with patients in acute mania are difficult to conduct, Öngür and colleagues [44] found that the excitatory neurotransmitter glutamate is overactivated in patients with acute mania. Interestingly, re-entry may be faster in mania [45]. Re-entry is a process of ongoing parallel and recursive signaling between separate neuronal groups. Given that tripartite synapses are organized in feedforward-feedback loops, the fastening of feedback loops in mania is in some aspects comparable to the hypothesis of dysfunction of re-entry in neuronal networks [46]. Basically, rapid cycles in information processing in tripartite synapses may underlie manic distractibility, flight of ideas, overactivity and circadian disturbances, especially insomnia.

Considering the model of volition proposed, the volition to self-instrumentalize cannot complete actions or thoughts, because of accelerated synaptic information processing leading to a rush of activities and confusion typical for manic behavior. Though patients in acute mania are convinced that they can do everything they want, the behavior is not well-programmed, since the actions are totally environment-dependent and not event-oriented controlled. Hence, the

manic reality experience I characterize as "pseudo-omnipotence" [46]. Whereas in depression patients perceive time too slowly, in mania time runs too fast [47]. According to Moskalewicz and Schwartz mania is "a rebellion against the limiting facticity of existence. A leap towards the future is ultimately a leap beyond temponality, one that results in quasi-eternal or timeless experience" [48]. This manic time experience may fundamentally be caused by the volition to permanent existence and its vain coping attempt with the unfeasible.

Given that the volition to generate realities operates on acceptance and rejection of environmental information, three elementary communication styles of manic patients we are observed. Some patients impress with their serene mood accepting the fate of treatment. This manic communication style, I characterize as acceptance mania. Other patients behave highly irritable, angry and spontaneously aggressive. Here the communication style of rejection prevails characterized as "rejection mania". Moreover, we also observe a rapid change between the acceptance and rejection type of manic behavior [46].

3.3. The Passion of Volition in Schizophrenia

The typical symptoms of schizophrenia are delusions and hallucinations (positive symptoms) and thought disorder, catatonic symptoms and affective flattening (negative symptoms) [37]. Positive symptoms reflect cognitive confusion caused by the loss of boundaries between the self and others, since the patient is unable to distinguish between his/her own thoughts and perceptions of the environment [49]. Though we know that schizophrenia has a multifactorial etiopathology, it is of significance that already Kraepelin [50] and Bleuler [51] highlighted the basic role of avolition in the phenomenology and the course of schizophrenia. Currently, it is suggested that avolition underlies the negative symptoms of schizophrenia [52]. Avolition is investigated by psychological experiments and associated with dysfunctions in various brain areas [53]. Importantly, Frith states that "passivity experiences involve a failure to recognize who is the agent of an action. Poverty of will results from a failure to generate actions that are appropriate in the social context" [54]. Basically, avolition determines the functional outcomes in schizophrenia and directly influence neurocognitive dysfunctions [52].

Let me focus on elementary dysfunctions of volitive processes in paranoid schizophrenia deduced from the psychobiological model of volition proposed. Given the various dysfunctions and impairments in brains with schizophrenia, self-instrumentalization is severely affected. The underlying pathophysiological mechanisms can be shown on imbalances in tripartite synapses.

Figure 7 outlines unbalanced tripartite synapses responsible for the pathophysiology of paranoid schizophrenia. Non-functional astrocytic receptors (acR, crosses) cannot be activated by neurotransmitters (NT). Since the Ca^{2+} concentration and the production of gliotransmitters (GT) does not become activated, an unconstrained neurotransmission occurs, and gliotransmitters cannot exert a feedback on extrasynaptic receptors (esR) and presynaptic receptors (psR). This

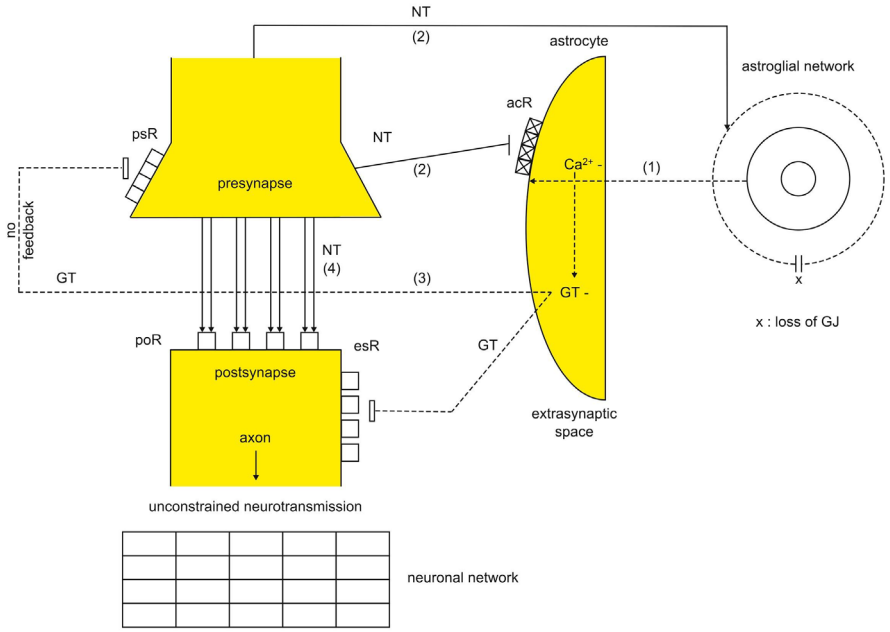

Figure 7. Unbalanced tripartite synapse in paranoid schizophrenia.

In parallel with the loss of gap junctions (*GJ*) *in the astroglial network, astrocytic receptors* (*acR*) *become non-functional* (*crosses*) 1). *Therefore, neurotransmitters* (*NT*) *cannot activate acR* (*line with bar*), *but the activation of the astroglial network continues* 2). *This leads to minimal concentration of* Ca^{2+} *and production of gliotransmitters* (*GT*). *Since GTs exert no feedback on the presynaptic receptors* (*psR*) (*dashed line, bar*) 3), *an unconstrained neurotransmission occurs* 4).

leads to a flooding of postsynaptic receptors (poR) by neurotransmitters. In the state of paranoid schizophrenia intentional programs still work, but the progressive loss of gap junctions reduces the domain of intentions (dashed cycle). Since the flux of neurotransmission is unmodulated by the astrocyte, a gap between the sensory information processing in the neuronal network and the "inner" astroglial network, responsible for incoherent thoughts, delusions and affective flattening, exists [55] [56].

In spite of the fact that schizophrenia is a chronic devastating process basically caused by the degeneration of astroglia [57], in the first stage of illness delusions and hallucinations prevail. In this stage, the neuronal network may not be severely affected and memory structures may still work in the astroglial networks. If we assume that normally the volition to program intentions is dependent on both memory and environmental information, in schizophrenia the memory structures in the astroglial networks may also function as intentional programs, because they cannot be modified by information from the environment. Consequently, schizophrenics are incapable of communicating their intentions to others. Decisively, this is not amotivation or avolition, but basically a disorder of communication.

Keromes and colleagues suggest that self-other recognition impairments might be a possible endophenotypic trait of schizophrenia [58]. The causal role of voli-

tive dysfunctions in the reality experience of patients with paranoid schizophrenia may shed some light on the conception of this endophenotypic trait. Basically, the "concealed" intentions that staged in delusion and hallucinations cannot be tested in the environment, but are experienced as real. Crucially, the volition to generate realities is at work, but the patient cannot qualify goals, since the self-boundaries are dissolved. The loss of self-boundaries may be caused by the loss of astroglial boundary setting function in tripartite synapses leading to a generalization of information processing as outlined in **Figure 8**. Since the astrocytes (Ac_i; Ac_j) of compartment x and compartment y have nonfunctional receptors (asterisks), they cannot influence synaptic information processing. This leads to a compartmentless neuronal network and generalization of information processing. **Figure 8** depicts a group of eight neurons completely interconnected by 28 lines (according to the formula $^{n/2}(n-1)$) [59]. Such a brain is incapable of qualitatively structuring environmental information. One may argue that the neuronal network per se is compartmentalized but there is a qualitative difference between neuronal compartments and the glial determined compartments. In addition to sensory information processing in neuronal compartments, glial neuronal compartments may exert an information structuring function so that the brain is able to recognize qualitative differences between subjects and objects in the environment [28].

I hypothesize that in paranoid schizophrenia the volition to program realities still operates in the glial networks, but the volition to generate the programmed (intended) realities lacks the goals in the environment, since the patient cannot

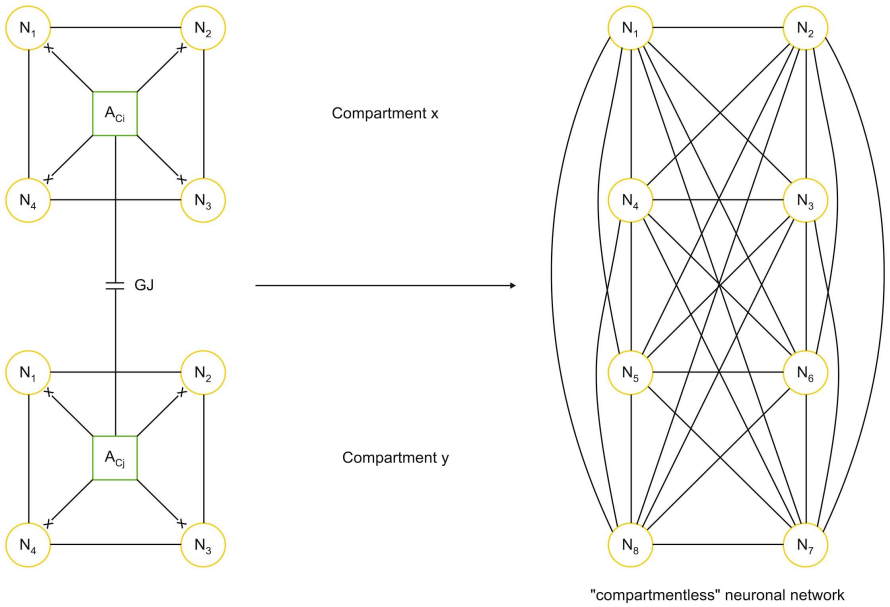

Figure 8. Loss of the glial boundary-setting function and generalization of information processing in paranoid schizophrenia.

Astrocytes (Ac_i; Ac_j) *of compartment x and compartment y have non-functional receptors* (*asterisks*) *that cannot influence synaptic information processing. This leads to a compartmentless neuronal network and the generalization of information processing* [59].

distinguish between the inner and outer realities. Importantly, despite the interaction between the neuronal synapse and the astrocyte being interrupted, the activation of the astroglial network by neurotransmitters and ions continues [60]. In this fashion memory structures or intentional programs may be still at work. Especially, in scenic delusions the patient generates an inner reality in the sense of a volitive process that is primarily not caused by cognitive impairment. Have schizophrenics an unconscious desire to communicate with others? Importantly, Frith suggests that delusions of control involve a failure in action attribution. This could be related to dysfunctions in the intentional binding mechanism and implies the reduced capability of understanding the intentions of others [54]. However and most importantly, patients with paranoia are basically unable to find goals for the realization of their intentions in the environment, since they experience themselves as the "universe". There is no space for intersubjective communication. The failure to understand the intentions of the others is therefore the consequence.

Moreover, we often observe delusions or hallucinations with a metaphysical content and grandiosity. For example, a patient is absolutely convinced that he is Caesar, Napoleon, Churchill and "Urbi" (neologism). Most interestingly, a patient said: "I am the universe and I am eternity. Do you know how much I am suffering?" Here we deal with a total generalization of concepts caused by the loss of self-boundaries. At least the contents of these delusions may indicate that the volition to permanent existence is at work deep in the personality, experienced as the "eternal now" [61].

4. Conclusions

The significance of the model of volition presented lies in a psycho-biological description of elementary volitive processes that may determine conscious volitive actions on the behavioral level. These are: the primordial volition to act; the volition to self-instrumentalize; the volition to program intentions; the volition to generate realities, and the volition to permanent existence. This model enables a new interpretation of mental disorders as disorders of volition.

I suggest that the volition to act and the volition to permanent existence basically operate in mental disorders, but the volitive processes of self-instrumentalization, programming of intentions (realities) and generating realities are typically impaired in depression, mania and schizophrenia. In depression the patient is burdened by a hyperintentional psychobiological state, since the intentional programs are non-feasible in the environment. The volition to act is constrained by "frozen" intentional programs unconsciously felt as a striving for permanent existence. In mania, the volition to act and the volition to generate realities is totally oriented on events in the environment or works spontaneously without any goal-directed programming. The volition to permanent existence and immortality is experienced as ideas of grandiosity.

Dysfunction of volitive processes in schizophrenia is fundamentally caused by severe impairments of self-instrumentalization. Since glia loses their bounda-

ry-setting function in synaptic information processing, a gap between the sensory information processing in the neuronal networks and the "inner" astroglial network arises so that the patient is unable to distinguish between the self and the others. Though schizophrenia is a chronic devastating process, in paranoid schizophrenia patients are still able to program intentions (realities), but cannot communicate their intentions. Note, this is not a-volition in the sense of a-motivation, but the incapacity to realize intentions, called dysintentionality [62]. In other words, in delusions the destiny for communication becomes stayed as pseudo-communication. Moreover, delusions with metaphysical content (God, Devil, eternity) may indicate that the volition to permanent existence and immortality is unconsciously working in the personality of patients with schizophrenia.

One may argue that the introduction of the volition to permanent existence is pure speculation and cannot be scientifically founded. However, this concept may elucidate the reality experience of patients suffering from mental disorders in the sense of a passion of volition. Current psychiatric research focuses on cognitive impairment [63], but cognitive impairments may be basically caused by dysfunctions of volitive processes. Though abnormal decision processes play a central role in mental disorders [64], we still lack a comprehensive theory of volition. The present study outlines a new psychobiological model of volitive processes and deduces dysfunctions responsible for the communication pathology and abnormal reality experience of patients with mental disorders.

Finally, It must be mentioned that the model of volition here proposed focuses on tripartite synapses and the astroglial networks, but have to be elaborated within a broader framework of neuron-glial interactions. This basically concerns the controlling functions of astrocytes of homeostasis in neuronal tissue functions [65] and the role of calcium waves in the action of feelings [66] [67].

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Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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