Table R1 (Northoff). *Problems and issues raised by the different commentators*

Neuroanatomy and neurophysiology of catatonia	Anterior cingulate (**Badgaiyan**), Orbitofrontal cortex (**de Oliveira-Souza et al.**), Parietal cortex (**Bearden & Monterosso**), Negative motor areas (**Marshall et al.**), GABA and inhibition **Bogerts**), Amygdala-hippocampus (**Bogerts**, **Miu & Olteanu**, **Savodnik**), Thalamus and hypersynchronous activity (**Kamal & Schiff**, **Fricchione**), Subcortical GABAergic mechanisms (**Fricchione**), Striatal dopamine-glutamate interaction (**Horvitz**), Multiple regions (**Bearden & Monterosso**, **Bogerts**, **Carroll**)
Cognitive-motor deficits in catatonia	Supervisory system and lateral inhibition (**Badgaiyan**), Cognitive deficits in catatonia (**Aleman & Kahn**), Relation between initiation and termination (**Bearden & Monterosso**), Motor neglect (Marshall et al.), Role of inhibition (**Badgaiyan**, **Bogerts**, **Marshall et al.**)
Conceptual issues	Distinction between vertical and horizontal modulation (**Horvitz**), as well as between "top-down" and "bottom-up modulation" (**Shaw**), Definition and level of "top-down modulation" (**Aleman & Kahn**), Linkage between top-down and bottom-up modulation (**Fricchione**), Anatomical structures vs. functional modulation (**Kamal & Schiff**), Definition of "lesion" (**Savodnik**), Distinction between cause and symptoms of disease (**Bogerts**, **Shaw**), "Biological" vs. "psychological" (**Hardcastle**, **Marshall et al.**, **Miu & Olteanu**, **Savodnik**)
General methodology in neuropsychiatric research	Cognitive models as a starting point (**Badgaiyan**), Description and phenomenology of symptoms (**Marshall et al.**), State vs. trait (**Bearden & Monterosso**), Distinction between cause, compensation, cooccurrence and consequences (**Savodnik**, **Shaw**), Definition of "disease" and "syndrome" (**Savodnik**, **de Oliveira-Souza et al.**), Too premature for hypothesis (**Bearden & Monterosso**, **Bogerts, Marshall et al.**, **Miu & Olteanu**)
Neurophilosophical implications	"Psychological" vs. "biological" (**Hardcastle**), Role of consciousness (**Hardcastle**), Neurobiology of self and relation to body (**Platek & Gallup**), Neurobiology of will (**de Oliveira-Souza et al.**), Monism vs. dualism (**Hardcastle**, **Marshall et al.**)

Author's Response

Neurophysiology, neuropsychiatry and neurophilosophy of catatonia

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Abstract: The excellent and highly interesting commentaries address the following concerns: (1) neuroanatomy and neurophysiology of catatonia; (2) cognitive-motor deficits in catatonia; (3) conceptual issues; (4) general methodology in neuropsychiatric research; and (5) neurophilosophical implications. The specific problems, issues, and aspects raised by the different commentators are grouped under these categories in Table R1 presented below. These five areas of concern are then discussed in the order listed in the five sections of the Response.

R1. Neuroanatomy and neurophysiology of catatonia

Badgaiyan suggests the involvement of the anterior cingulate, including its distinct motor, affective, and cognitive parts, in catatonia.

Involvement of the anterior cingulate is strongly supported by our imaging results acquired during emotional stimulation (Northoff et al. 2002a). Post-acute catatonic patients showed altered, that is, decreased signal intensity in medial orbitofrontal and ventromedial prefrontal cortex; the latter includes the subgenual and pregenual, that is, the affective part of the anterior cingulate. Moreover, abnormalities in medial prefrontal cortex, including the supragenual anterior cingulate (i.e., its motor and cognitive part) were observed.

The exact functional mechanisms and the interregulation between the different parts of the anterior cingulate, however, remain unclear. **Badgaiyan** offers an interesting explanation by hypothesizing that the motor part may be inhibited, and thus, suppressed by overactivity in the affective part. Such a hypothesis seems to be of particular interest considering the fact that akinetic mutism, which shows similar motor features, may be caused by lesions in the motor part of the anterior cingulate. However, to my knowledge, there is, so far, no direct empirical evidence for his hypothesis.

The excellent case descriptions from **de Oliveira-Souza et al.** suggest involvement of the orbitofrontal cortex especially the medial and right part. They nicely describe the behavioral anomalies which are so prominent and bizarre in these patients. They unfortunately do not describe the affective status of their patients. The medial orbitofrontal cortex might play a crucial role in catatonia as based on imaging findings and deficits in the Gambling task. These deficits might deregulate the functional balance between medial and lateral orbitofrontal cortex, which, psychologically, might be reflected in an abnormal emotional control of behavior. This remains purely speculative, however, and awaits further empirical confirmation.

Bearden & Monterosso point out the crucial role of the right parietal cortex in my hypothesis, and argue that, if this

is indeed the case, catatonic patients should show similar symptoms (apraxia, hemineglect, tactile impairment) to those with lesions in this region. This is a question that occurred to me, as well. However, it should be noted that they refer to patients with exclusive lesions in the right parietal cortex, which, unlike catatonia, do not show deficits in the orbitofrontal cortex. It may therefore be hypothesized that the co-occurrent involvement of right parietal and orbitofrontal cortex may lead to a different pattern of symptoms than isolated lesions in the right parietal cortex. Moreover, a recent study demonstrated that patients with hemineglect showed lesions in the right superior temporal cortex, rather than the right posterior parietal cortex (Karnath et al. 2001). Accordingly, exact localization of lesions may differ between catatonia and hemineglect.

Marshall, **Gurd & Fink** (**Marshall et al.**) suggest the involvement of so-called negative motor areas, like the orbitofrontal cortex and the anterior cingulate. As already pointed out in both the target article and these commentaries, there is strong evidence for involvement of these regions in catatonia. Because of overlap in symptoms, it may well be imaginable that these regions could be involved in both hysterical paralysis and catatonia – although, in contrast, there is no direct evidence for alterations in active inhibition. However, there may be some indirect evidence. Behavioral inhibition may be reflected in posturing and its release by external stimuli, as, for example, when catching a ball (see Northoff et al. 1995). Physiological inhibition may be reflected in the good therapeutic efficacy of lorazepam, a GABA-A potentiator, which enhances neuronal inhibition. This is supported by abnormal (i.e., paradoxical) clinical responses to lorazepam (Northoff et al. 1999a), as well as abnormal changes in readiness potential (Northoff et al. 2000a) and orbitofrontal cortical fMRI signals (unpublished observation) after application of lorazepam in catatonic patients

Bogerts refers to the process of neuronal inhibition and potentially GABAergic mechanisms by assuming that there may be a principal deficit in neuronal inhibition underlying both schizophrenia and catatonia. His hypothesis, that deficits in neuronal inhibition are basic to the principal disease process in both schizophrenia and catatonia though manifest in different regions, is appealing, especially from a clinical point of view. As he points out, catatonia occurs often as the most severe and extreme manifestation of paranoid schizophrenia – the same underlying pathophysiological mechanisms (i.e., altered neuronal inhibition) may account for this co-occurrence.

Bogerts, **Miu & Olteanu**, and **Savodnik** argue for the potential involvement of the amygdala-hippocampal complex in the pathophysiology of catatonia. Bogerts (see also Savodnik) points out the similarity between catatonia and anxiety disorder with regard to strong and uncontrollable emotional symptoms (i.e., anxieties). Since the amygdala may be crucially involved in anxiety disorder, it should play a role in catatonia, as well. This is certainly right, and strongly supported by the existence of strong and reciprocal connections between the amygdala and the orbitofrontal cortex. Although the latter is altered in catatonia, one may assume that the former (i.e., the amygdala) is also involved. Miu & Olteanu point out the potential relevance of the hippocampus by drawing on the occurrence of catatonia in Alzheimer's disease. It is true, indeed, that many of the catatonic features, and catatonia as a whole, can be observed in dementia, Alzheimer's, and frontal lobe dementia in particular. Moreover, there is reciprocal and strong connectivity between the medial orbitofrontal cortex and the hippocampus (Barbas 2000), which makes involvement of both regions in catatonia rather likely. Finally, both schizophrenia and depression, the diseases in which catatonia most often occurs, can be characterized by abnormalities in the hippocampus (Bogerts 1997; Liotti & Mayberg 2001). Accordingly, there is some, albeit rather indirect, evidence for potential involvement of the hippocampus in catatonia.

Kamal & Schiff shift the attention to the thalamic nuclei and hypersynchronous neural activity. Rather than considering this as contradictory to my hypothesis, I would regard their comments as complementary. The cortico-subcortical loops described certainly involve the thalamic nuclei, which, in turn, may alter the neuronal pattern, consecutively leading to hypersynchronization. Hypersynchronous neural activity may account for the "deadlock" that can be observed in catatonia. However, this remains purely speculative, because there are no data at all to support such an assumption. The same remains true with regard to **Fricchione**'s suggestion of impaired desynchronization in the basal ganglia. There are no EEG data so far which have been shown any abnormalities in catatonic patients.

The problem remains, to establish a solid animal model that really mirrors catatonia as observed in humans. Although DeJong (see Northoff 1997a) claimed to have established an animal model of bulbocapnine-induced catatonia, application of the same agent led, rather, to a kind of neuroleptic-induced catalepsy, which did not react at all to GABAergic agents like lorazepam (my own unpublished observations). **Fricchione** points out the animal model by Stevens where a GABA-A antagonist was injected into the ventral tegmentum of cats and induced a catatonic-like state in cats. This may point out the relevance of subcortical GABA-ergic mechanisms which, on account of methodological reasons, have not been investigated in human catatonia so far.

The same problem arises if one wants to investigate the exact interaction between cortical glutamatergic projections and dopaminergic nigrostriatal neurons in the striatums. As claimed by **Horvitz**, the interaction between both kinds of neurons may be altered in Parkinson's disease, which consecutively may account for muscle rigidity accompanying akinesia. It is one of the most distinguished features of akinesia in catatonia, in that it is not accompanied by an increase in muscle tone, which, in contrast, may be either on a normal or even lower (i.e., decreased) level. One may subsequently assume that the glutamatergic-dopaminergic interaction in the striatum may be different in catatonia from Parkinson's disease.

Finally, it should be pointed out that several authors (**Bearden & Monterosso**, **Bogerts**, **Carroll**) support the assumption of diffuse and multiple lesions in catatonia. This was the reason why I put the emphasis on a network model, involving several regions and circuits, rather than on a single and particular anatomical location.

R2. Cognitive-motor deficits in catatonia

Badgaiyan makes the interesting assumption that the supervisory system and lateral inhibition may be disturbed in

catatonia. The selection of action from among competing action sequences may be disturbed. Clinically, the inability to select appropriate action from among different kinds of actions may be reflected in both hypokinetic and hyperkinetic symptoms. In hypokinetic symptoms, no further action can be selected, whereas in hyperkinesias the switch between different actions is disturbed. Accordingly, there is clinical evidence for a deficit in the selection of action. Whether this is caused by alteration in lateral inhibition, however, remains unclear. Nevertheless, as already pointed out, assumption of altered inhibition (i.e., lateral inhibition) seems rather likely. Neuroanatomically, this is supported by potential involvement of the ventrolateral prefrontal cortex, which may be related to inhibitory functions.

Neuropsychological results show a cognitive deficit in decision-making in catatonia as investigated with the Gambling task (own data, not published yet). This answers the question for cognitive deficits raised by **Aleman & Kahn**. However, the exact relationship of these cognitive deficits to catatonic symptoms remains unclear. One may speculate that the behavioral symptoms in particular may be related with these deficits in decision-making.

Bearden & Monterosso raise the issue of the relation between initiation and termination of movements in catatonia. There is, apparently, a deficit in the termination of movements, because otherwise, patients would be able to complete their movements. However, initiation and termination are closely linked with each other. For example, terminating a movement presupposes an initiation for termination. Accordingly, initiation and termination cannot really be separated from each other. Because of their close linkage, catatonic patients show deficits in the internal initiation of movements, as observed in my ball experiments (Northoff et al. 1995). However, unlike patients with Parkinson's disease, catatonic patients also show deficits in the termination of movements, resulting in posturing. These deficits were also observed in the ball study and were described as a deficit in the "voluntary generation of movements." Accordingly, the present assumption of alteration in termination is not contradictory to my earlier statement of deficits in the initiation of movements.

Marshall et al. raise the comparison between motor anosognosia in catatonia and motor neglect. They are certainly right in doing so, and support their claim by neuroanatomical evidence. I fully agree with them. However, as also pointed out by them, motor neglect does not lead to posturing. Accordingly, motor neglect may account for the lack of awareness of posturing, rather than for posturing itself. Instead of equating catatonic symptoms with motor neglect, I would suggest that catatonia might reflect a higher (i.e., cognitive) form of motor neglect. However, at present, this claim remains purely speculative. It is certainly true, as they state, that further phenomenological and psychological information is necessary in order to elucidate the exact nature of the motor deficits.

Badgaiyan, **Bogerts**, and **Marshall et al.** point out the crucial role of inhibition in catatonia. It should be noted, however, that the exact meaning and level of inhibition should be defined: Do they mean behavioral inhibition? Psychological inhibition? Physiological inhibition, as it might be reflected in GABA-ergic mechanisms? All of these different levels of inhibition might dissociate from each other. For example, behavioral inhibition might be subserved by physiological (i.e., neuronal) excitation. Accordingly, the meaning of the term "inhibition" should be specified and discussed in full detail. With regard to catatonia, the exact relationship between the different kinds of inhibition remains unclear and can only be speculated about.

R3. Conceptual issues

The first conceptual issue concerns the distinction between vertical and horizontal modulation. **Horvitz** raises two questions: first, the exact relation between a particular kind of modulation (i.e., vertical or horizontal) and symptoms; and second, the relationship between anatomical structures and functional modulation. There is certainly no exclusive relationship between particular symptoms and a specific kind of modulation (i.e., horizontal and vertical). Catatonia, for example, may eventually involve vertical modulation as well, with top-down modulation of subcortical nuclei involved in affective regulation (locus coeruleus, raphe nuclei). Parkinson's disease, on the other hand, may involve horizontal modulation, as, for example, dysregulation of prefrontal cortical areas, accounting for emotional processing by motor/premotor cortical areas. Accordingly, it is not a matter of "All-or-Nothing," but rather a matter of "More or less," with regard to the kind of modulation involved. The same remains true for the distinction between "bottom-up" and "top-down" modulation, which, rather than being absolute, must be considered as "relative," as pointed out by **Shaw**. Because of the widespread, and often strong and reciprocal, cortical-subcortical and cortico-cortical connectivity, a sharp and exclusive distinction between the distinct kinds of modulation remains impossible. This is probably reflected in relative, rather than absolute, differences between clinical symptoms, like, for example, akinesia. Catatonia seems to be dominated by alterations in horizontal modulation, whereas Parkinson's disease may rather be characterized by predominant changes in vertical modulation.

Aleman & Kahn raise the question for the definition of "top-down modulation." They contrast the anatomo-connectional cortico-subcortical definition with a rather psychological definition by cognitive-sensory interaction. They are right in emphasizing the distinction, since both cases do not necessarily fall together. This, for example, is the case in visual attention, where prefrontal cortical areas top-down modulate sensory cortical regions. This cortico-cortical modulation might be subsumed under the term "horizontal modulation" in the anatomo-connectional sense. There is apparently some confusion and rather unclear definition of the various kinds of modulation. To clarify these issues must be considered an important task which might contribute substantially to a better understanding of the pathophysiological mechanisms in psychiatric disorders. Aleman & Kahn have pointed out hallucinations and affective-behavioral alteration as other examples where altered top-down modulation may be crucial. As they describe, cortico-cortical and cortico-subcortical modulation might go together, resulting, neuropsychologically, in top-down modulation. Consequently, top-down modulation and horizontal modulation might be regarded as equally important and should be seen to be complementary rather than exclusive, because they describe different levels of operation – that is, anatomo-connectional as well as neuropsychological.

My emphasis was on pointing out these distinct kinds of modulations and the different levels they were operating on. It is not that I forgot the loops and circuitry by Alexander et al., as is suggested by **Fricchione**. Rather, my concept of the distinct kinds of modulation, which point out the functional level rather than the structural anatomy, must be regarded as complementary. Fricchione is certainly right in noting the neglect of subcortical regions – the basal ganglia, in particular – which resulted in a lack of discussion of the neuromedical origin of catatonia. My focus was concentrated on the cortical-cortical interactions and the distinct kinds of modulations as these are questioned in the consideration of psychogenic catatonia. However, these kinds of modulation do not necessarily exclude subcortical-cortical modulation, that is, bottom-up modulation. Fricchione's suggestion, for linking top-down and bottom-up modulation in order to account for both psychogenic and organic catatonia, might therefore be considered as a good model for further investigation.

This leads us to the second question, the relation between functional modulation and anatomical structures. I would claim that the clinical symptoms themselves, in both disorders, cannot be directly related to particular deficits in specific anatomical structures, but rather, are related to particular alterations in functional modulations (i.e., circuits and loops). For example, the nigrostriatal dopaminergic deficit is the cause of the dysregulation in the "motor loop" in Parkinson's disease, which then accounts for the motor symptoms. Accordingly, anatomical structures can be regarded as a necessary, but not sufficient, condition for generation of clinical symptoms. For example, a particular anatomo-structural lesion may predispose and increase vulnerability to a certain dysregulation in functional modulation, as pointed out by **Kamal & Schiff**. However, there may also be anatomo-structural lesions without dysregulation in functional modulation, which may be reflected in an absence of clinical symptoms. Functional modulation, which operates on and across different anatomical structures, may therefore be regarded as a sufficient condition. This is, for example, reflected in psychogenic disorders. Despite the absence of a particular anatomo-structural lesion, psychogenic disorders show alterations in functional modulation, whereas their clinical symptoms resemble the diseases having lesions in those anatomical structures on which the loops and circuits operate. Accordingly, the relations between anatomical structures and functional modulation can be manifold. Different constellations can be possible and may account for major and minor differences in clinical symptoms.

Closely related to the difference between structure and function, is the concern raised by **Savodnik**, regarding the definition of a "lesion." He argues that catatonia cannot be characterized by lesions in the Virchowian sense, because no anatomo-cellular correlate has been detected so far. However, within the present framework, the concept and definition of a "lesion" should be extended to include not only anatomo-structural lesions, but also alterations in functional modulation (i.e., loops and circuits). These may, for example, concern alterations in vertical and horizontal modulation, as is the case in catatonia. Moreover, this extended definition of "lesion" could then also account for psychogenic disorders, and would therefore bridge the "old" gap between the structural and functional level, and thus between "organic" and "psychogenic" disorders. Presupposing this definition of a "lesion," catatonia, too, can be regarded as a "disease," which makes its characterization as a "social construct," as suggested by Savodnik, superfluous.

Moreover, the distinction between the cause of a disease and the symptoms of a disease should be considered. The present hypothesis aims at pointing out the pathophysiological correlates underlying the different kinds of symptoms in catatonia. Although, in contrast, it does not say much about the pathophysiology related to the cause of these changes. Because the hypothesis focuses predominantly on the pathophysiological correlates of symptoms, it rather neglects the dynamic nature and course of catatonia, as has been noted by **Bogerts** and **Shaw**. Both these commentators are certainly right that, in order to obtain a full and complete pathophysiological account of catatonia, its dynamic nature and course should be taken into account. However, focus on the symptoms themselves, with neglect of the dynamic course, does not make the comparison with Parkinson's disease worthless (see Shaw's commentary in this regard), as long as it does not claim to be a comparison between both diseases (but rather, between their symptoms). Shaw is certainly right, however, in pointing out the necessity of giving the exact stage of the disease (early or late) to which the motor symptoms in Parkinson's refer.

The difference between pathophysiological correlates of the disease cause and the disease symptoms is nicely reflected in Parkinson's. The nigrostriatal dopaminergic deficit may somehow be regarded as the correlate of the disease cause (although the cause for the degeneration of these neurons remains unclear), whereas the changes in the "motor loop" are instead the correlate of the motor symptoms. As pointed out by **Bogerts**, the disease cause remains unclear in catatonia, and it may be of anatomo-structural nature. Accordingly, the distinction between disease cause and disease symptoms may reflect the distinction between the anatomo-structural and functional level. Although – as, for example, in psychogenic disorders – this is not necessarily the case.

The term "cause" of particular symptoms may be further specified and may refer either to a particular disease or a syndrome. **Bogerts** remarks that there is a lack of clear specification as to whether catatonia is a syndrome, or a disease by itself. As pointed out in my studies, I regard catatonia as being a syndrome (see also **Carroll**). As a result, catatonia can be associated with a variety of different diseases, from which it may turn out to be a "common functional final pathway." For example, fever can be associated with a variety of different diseases. Nevertheless, there is a specific pathophysiological correlate of fever that remains absent in patients with the same disease, but without fever. Fever may therefore be regarded as an analogous model syndrome for catatonia. Considering fever, which is the extreme manifestation of an underlying disease, catatonia may indeed be regarded as the "extreme end" of certain neuropsychiatric diseases, such as, for example, affective and schizophrenic disorders. In contrast to Bogerts' implicit assumption, the syndrome character of catatonia and its characterization as an "extreme end" are not mutually exclusive.

As pointed out by **Bogerts**, **Miu & Olteanu**, **Carroll**, and **Bearden & Monterosso**, catatonia can apparently be related, not to one particular anatomical structure, but rather, to multiple and different ones. It therefore defies strict localizationism. However, this does not mean that

catatonia can be related to the whole brain, as presupposed in holism. The present concept of description of loops and circuits, which operate across several but specific anatomical structures, defies and undermines the exclusive and opposite distinction between localizationism and holism. The term "up- and down regulation" does, therefore, refer primarily to specific circuits, rather than to transmitters as suggested by **Shaw**. However, transmitters should not be neglected entirely, because the functional output of the circuits may essentially depend on the kinds of transmitters. Instead, the present hypothesis can be regarded as an attempt to provide the groundwork for a more dynamic approach and to move beyond or undermine the classical distinction between localizationist and holistic approaches, which are still quite prevalent in neuropsychiatry, either explicitly or implicitly.

Finally, the distinction between "biological" and "psychological," which reflects the distinction between "psychogenic" and "organic" catatonia, is raised by several commentators, either implicitly or explicitly (**Hardcastle**, **Marshall et al.**, **Miu & Olteanu**, **Savodnik**). I do not intend to say that "psychogenic" disorders are cortical and "organic" disorders are subcortical. Instead of making an "absolute" difference, I would rather call for a "relative" distinction with matters of degree. The loops and circuits cross the boundary between cortical and subcortical regions and, therefore, "relativize" this distinction. **Hardcastle** is subsequently right in claiming that this "division" is "too simple." However, it may reappear in a "relativized" form within the terms "top-down" and "bottom-up" modulation, and thus in the direction of the modulation within one particular cortical-subcortical loop/circuit. It is this characterization of the different directions of the modulation within the same loops/circuits that may account for the subtle and minor differences in otherwise almost similar clinical symptoms of organic and psychogenic disorders. For example, detailed and exact clinical observation reveals subtle differences between hysterical paralysis and organic paralysis. The regions, pointed out by **Marshall et al.** (orbitofrontal, anterior cingulate) in hysterical paralysis, are usually not affected in the case of organic paralysis. However, they may lead to abnormal top-down modulation of those regions usually affected and lesioned in organic paralysis. Hysterical paralysis can thus neither be "localized" in cortical regions nor in subcortical areas, whereas the direction of modulation may be specified in this regard.

R4. General methodology in neuropsychiatric research

Badgaiyan emphasizes the need for consideration of cognitive models as a starting point for psychiatric research by "delineating the underlying deficits of cognitive information processing," which should replace the focus on symptoms. The commentator replaces symptoms with cognitive models, because the same symptoms, as for example akinesia, may show different underlying neurocognitive disturbances. Drawing parallels between symptoms may therefore be problematic. However, in addition to similarities, we pointed out subtle differences between catatonic and Parkinsonic akinesia, which concerned not only subtle motor features (muscle tone), but also the predominantly subjective experience of akinesia. Total replacement of symptoms by cognitive models as a starting point, as implicitly suggested by Badgaiyan, should therefore be rejected, because then the subjective experience would be neglected. Especially in psychiatric disturbances, the role of subjective experience (i.e., phenomenology) is often neglected and regarded as superfluous in the search for a neurobiological substrate.

The present hypothesis of catatonia, in contrast, aims to demonstrate the necessity of considering subjective experience as a starting point for the generation of a neurobiological hypothesis (see Northoff et al. 1998; 2002b). Differences and/or special features of subjective experience must have a specific underlying physiological substrate. Accordingly, subjective experience and phenomenology may serve as a starting point for the generation of a neurobiological hypothesis. Cognitive models may thereby serve as an intermediate step, which may bridge the gap between subjective experience and symptoms, on the one hand, and physiological and anatomical substrates, on the other. In the present case of akinesia in catatonia, this intermediate position is supposed to be filled by reference to the model of Miall and Wolpert (1996).

In addition to their subjective experience in the first-person perspective, the symptoms themselves should be described objectively as accurately as possible from a third-person perspective. This point is raised by **Marshall et al.** Their question of recognition of other postures in other persons by catatonic patients is an interesting one and probably aims at the function of the observation system. Is there a specific dissociation between observation and awareness of one's own and other's movements in catatonia? Unfortunately, no data have been reported yet. Are catatonic patients "living statues," holding strange postures like the artists in Paris? Yes, they are "living statues," but they are not like these artists. These artists probably do show increased muscle tone and muscle strength to hold their postures. This is not the case in catatonic patients, who often show either normal or even decreased muscle tone. Moreover, they do not show abnormal muscle strength. Finally, moreover, unlike those artists in Paris, catatonic patients are not able to deliberately and voluntarily start and stop their postures, because they remain unaware of them. Accordingly, it seems rather unlikely that the artists in Paris, as observed by Marshall et al., may be "hidden and nondetected" catatonic patients that need lorazepam.

Moreover, complementing subjective experience and objective symptoms, the exact characterization of their occurrence should be considered. Are the symptoms state or trait markers? This point is raised by **Bearden & Monterosso**, and they are completely right in emphasizing it. As a result of the fact that imaging of patients in an acute catatonic state remains (practically and ethically) almost impossible, most pathophysiological findings concern the post-acute state, and therefore may be considered to be "trait markers" rather than "state markers." One may therefore concede that dysfunction in the reported regions may predispose a person for the development of catatonic symptoms, whereas they may not be considered as the anatomofunctional substrate of the symptoms themselves. Total dissociation between "state- and trait markers" with regard to their respective pathophysiological substrates subsequently cannot be excluded. The best way to generate a pathopysiological hypothesis about the symptoms themselves (i.e., "state markers") probably would be the development of an

animal model. This also would allow for a distinction between the cause, compensatory mechanisms, co-occurrence, and consequences of the disease, as emphasized by **Savodnik** and **Shaw**, which, due to lack of available knowledge, is rather underemphasized or neglected in my hypothesis. Moreover, the meaning of the term "syndrome," as raised by **de Oliveira-Souza et al.** should be considered. I fully agree with their definition of catatonia as a syndrome as analogous to other syndromes in medicine such as fever or coma. Catatonia as a psychomotor syndrome may consequently be regarded as the "common final functional pathway" of various different causes which reflect the different (psychogenic and organic) origins of catatonia.

These considerations lead us to two more basic questions, the first one regarding the definition of a disease, and the second one regarding the time point, or timing, of a neuropsychiatric hypothesis.

The question regarding the definition of disease is raised by **Savodnik** and has long been debated in psychiatry. Can behavioral symptoms, as observed in psychiatry, be defined as a disease in the absence of a pathophysiological substrate providing the unifying umbrella? Or are they mere social constructs, as suggested by Savodnik? How shall the search for pathophysiological substrates proceed methodologically? Or does a neurobiology of psychiatry remain impossible altogether? Considering the recent advances in our understanding of higher brain function, the last question can almost certainly be denied. My own position on this issue is that an accurate and detailed account of both subjective experience and objective symptoms may serve as the starting point for the development of a pathophysiological hypothesis as intermediated by cognitive models (see also above). Such an approach presupposes a so-called first-person neuroscience (see Lutz et al. 2002; Northoff 2003), where first-person perspective data from subjective experience are directly included in analysis of third-person perspective data about physiological processes.

Instead of being considered as a "unified theory," the present hypothesis about catatonia rather may be regarded from a heuristic point of view, which may guide and focus neurobiological investigation. One may subsequently start with an often observed and preliminarily defined constellation and co-occurrence of specific symptoms and subjective experiences. Although the definition of a disease can be put on hold, this, however, does not prevent neurobiological research. Once pathophysiological data are obtained, the definition of these symptoms as a disease entity may ultimately be decided.

The question of the timing of the present hypothesis about catatonia has been raised by several authors: Isn't it too premature to develop a hypothesis about catatonia? **Bearden & Monterosso** mention the complexity of catatonia; **Bogerts** raises the problem of the lacking pathohistological correlate; **Marshall et al.** bring up the lack of detailed clinico-phenomenological knowledge; and **Miu & Olteanu** note the possibility of too many alternative explanations as obstacles to a hypothesis or theory of catatonia. Therefore, they argue, it is premature to develop such a hypothesis.

I agree with all commentators with regard to the points they raise, as already discussed above. However, I think that they may potentially presuppose a different and much stronger meaning of the term "hypothesis" than I originally intended. "Hypothesis" in the present sense points out a preliminary character, rather than a fixed and definitive character as, for example, in a "unified theory." Moreover, hypothesis in the present sense remains very much open to modification in the process of acquisition of further data. The hypothesis in the present sense can subsequently be regarded only as a starting point rather than an end point. As such, it serves as a coherent conceptualization of present and available data, which then may guide, focus, and restrict further neurophenomenological and neurobiological investigation, the results of which, in turn, may make modification of the initial hypothesis necessary. Accordingly, the present hypothesis may not be regarded as a "unified theory," which can be either verified or falsified. Instead, it may rather be modified, specified, and complemented in the course of further investigation.

The complexity of catatonia, as demonstrated nicely by **Bearden & Monterosso**, makes the development of a hypothesis, in this sense, necessary, because otherwise, the lack of any kind of conceptualization of the complexity of catatonia could make any further neurobiological approach doomed to failure. Moreover, the hypothesis may serve as a guide for restricting and limiting the focus of the search for a pathohistological correlate, as emphazised by **Bogerts**. In addition, the hypothesis may serve to raise novel clinico-phenomenological questions, as pointed out by **Marshall et al.**, which may provide us with a "new look" on "old and well known" clinical symptoms. Finally, the hypothesis attempts to reduce the number of alternative explanations, although, because of its preliminary character (being a starting point rather than an end point, see above), it remains unable to reduce them down to the possibility of either verification or falsification, as implicitly suggested by **Miu & Olteanu**. Accordingly, it may be too early and premature to formulate a "unified theory" of catatonia with consecutive verification and falsification. However, it may not be premature or too early to generate a hypothesis for focusing and guiding further and future neurobiological research into catatonia.

R5. Neurophilosophical implications

Hardcastle points out the importance of consciousness in the distinction between "psychological" and "biological," which, according to her, cannot be related to the distinction between cortical/top-down and subcortical/bottom-up. I certainly agree that consciousness (i.e., conscious experience) may be crucial to the distinction between "biological" and "psychological," at least at present. However, she neglects two other factors. First, conscious experience changes with dependence on the respective environment, and thus, on our state of knowledge. For example, diseases nowadays classified as "biological" (e.g., epilepsy) were regarded as "psychological" before their underlying neurobiological substrate had been revealed. Accordingly, the distinction between "biological" and "psychological" does not depend only on our conscious experience, but also on our environment.

Second, "experience" includes not only conscious experiences but also unconscious ones. There may be much more unconscious experience than conscious experience, that is, the latter may be only the "tip of the iceberg" (see also Northoff 2003). This is reflected in the relevance of psychodynamics as a method for the description and reve-

lation of these unconscious experiences, which may be manifest in a variety of different clinical symptoms like, for example, hysterical paralysis. If these unconscious processes are so abundant and may even determine conscious experience, the search for their underlying neurobiological substrate may be at least as important as the one for conscious experience. One may consequently speak of an attribution of "overimportance" to consciousness as compared to unconsciousness (Northoff 2003). This "overimportance" may be reflected in the focus of both neuroscience and philosophy on consciousness, which, in part, may be a result of the methodological difficulty of getting access to unconsciousness. Any "theory of consciousness" should consequently be accompanied by a "theory of unconsciousness" with respect to both the underlying neurobiological substrate and the philosophical implications.

Platek & Gallup point out the implications of the present hypothesis for the neurobiology of the self. There are indeed disturbances of the self in catatonic patients, which we investigated in a separate study. We used the Repertory-Grid test, which asks for characterization and description of the person (i.e., self) and then allows for semiquantitative analysis (see Northoff et al. 2002b). The catatonic patients indeed showed severe alterations in their "self-constructs" as compared to noncatatonic depressive, manic, and schizoaffective patients, which, in addition, correlated significantly with alterations in orbitofrontal cortical signal intensity during emotional stimulation. These results, therefore, lend strong support to the assumption of alterations of the "self" in catatonic patients. Moreover, they point out the relevance of both the body and the right orbitofrontal cortex for the self. Platek and Gallup cite additional support for involvement of the right orbitofrontal cortex in the self by referring to studies of self-face recognition by Keenan et al. (2000). Moreover, they emphasize the role of the body and a kinaesthetic model and relate this to an orbitofrontal-parietal loop. There is further strong support for involvement of the body in the generation of the self. Disturbances in the body image may also lead to disturbances in the self (see Northoff 2001b; 2003). The body may consequently be regarded as a constitutive and necessary (though not sufficient) condition for the self of a person. It is this crucial role of the body for the self that may shed new perspectives on both the neurobiology and the philosophy of the self. Neurobiologically, it may guide further studies in the search for a correlate of the self. Catatonia, with its apparent alteration in the orbitofrontal-parietal connections, may serve here as a paradigmatic example for the close linkage between body and self. Although, philosophically, it may counter-argue models of the self, which, as derived from Descartes, are purely mental and consequently non-bodily. As a result, empirically more realistic and plausible models of the self may be developed in philosophy.

de Oliveira-Souza et al. suggest that the behavioral anomalies in catatonia reflect disturbance of the will in these patients – the symptoms of passivity and negativism oscillating between the two extremes of free or nonfree will. This is a very interesting suggestion and might shed some light on the neurobiological mechanisms underlying the will. Considering the findings in catatonia, the orbitofrontal cortex might play a crucial role in generating behavioral choices and alternatives which, on a phenomenal level, may be related with the will. One may distinguish the possibility of behavioral choices from the subjective experience or feeling of having a choice. The possibility of the latter is raised by the findings from Libet. Both components may not necessarily be subserved by the same neural correlates. Oliveira-Souza et al.'s suggestion might concern the behavioral choices rather than the subjective experience itself, but it might nevertheless be regarded as a good starting point into the neurobiological exploration of our will. I consequently fully agree with them that "catatonia opens a window into this as yet obscure landscape of the human mind."

Finally, the old issue of monism versus dualism is raised by **Marshall et al.** and **Hardcastle**. Marshall et al. speak of a "rejection of two-substance dualism," with the consequence being that all diseases display both physical and psychological symptoms; while Hardcastle rejects the distinction between "biological" and "psychological," because in the end, everything will be "housing in the brain" and thus be "biological" anyway. Aren't these two positions rather contradictory?

Before arguing in further detail, I would like to introduce a distinction that is often rather neglected in the current discussion. One should distinguish between the ontological, the epistemic, and the empirical level, which do not necessarily have to be in full accordance with each other; that is, they may dissociate from each other (see also Northoff 1999c; 2003). For example, **Marshall et al.** reject ontological dualism, though on an empirical level they still maintain some sort of dualism by claiming the co-occurrence of physical and psychological symptoms. In contrast, **Hardcastle** refers exclusively to the ontological level when she speaks of "biological" versus "psychological." Accordingly, both positions are not incompatible, because they both refer to different levels (i.e., ontological and empirical) while ostensibly rejecting any form of ontological dualism.

I agree with the rejection of ontological dualism, but I also accept empirical dualism. This empirical dualism may potentially be traced back to some sort of epistemic dualism. I already mentioned above that both first- and third-person perspective accounts should be considered in the exploration of neuropsychiatric diseases. At this point, I want to go even one step further by claiming that first- and third-person perspectives may have distinct (although potentially overlapping) neurobiological substrates (see Northoff 2003, for further details). Epistemic dualism leads subsequently to empirical dualism. However, since both perspectives may be related to the anatomo-functional (and nonmental) substrates of the brain, both epistemic and empirical dualism remain compatible with ontological monism (see Northoff 2000b; 2001a; 2003).

The conjunction between ontological monism, on the one hand, and epistemic and empirical dualism, on the other, may be well reflected in catatonia as a psychomotor syndrome (see also Northoff 1999). The most strange and bizarre forms of objective behavior in the third-person perspective can be related to the brain, and to the corresponding subjective experience in the first-person perspective which also may be related to the brain, but through different loops and circuits (i.e., lateral orbitofrontal-parietal circuit, medial orbitofrontal-striatal-pallidal circuit). Epistemic dualism between first- and third-person perspectives may thus be reflected in empirical dualism. However, both behavior and experience can be related principally to the same underlying ontological substrate (i.e., the physical stuff of the brain), which implies ontological monism. Catatonia may consequently be regarded as a paradigmatic example of

the mind-brain relation, empirically, epistemically, and ontologically (see Northoff 1997b; 1999c).

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Letters "a" and "r" appearing before authors' initials refer to target article and response, respectively.

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