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Continuing Commentary

Commentary on **Ichiro Tsuda (2001). Toward an interpretation of dynamic neural activity in terms of chaotic dynamical systems. BBS 24(5):793–810.**

Abstract of the original article: Using the concepts of chaotic dynamical systems, we present an interpretation of dynamic neural activity found in cortical and subcortical areas. The discovery of *chaotic itinerancy* in high-dimensional dynamical systems with and without a noise term has motivated a new interpretation of this dynamic neural activity, cast in terms of the high-dimensional transitory dynamics among "exotic" attractors. This interpretation is quite different from the conventional one, cast in terms of simple behavior on low-dimensional attractors. Skarda and Freeman (1987) presented evidence in support of the conclusion that animals cannot memorize odor without chaotic activity of neuron populations. Following their work, we study the role of chaotic dynamics in biological information processing, perception, and memory. We propose a new coding scheme of information in chaos-driven contracting systems we refer to as *Cantor coding.* Since these systems are found in the hippocampal formation and also in the olfactory system, the proposed coding scheme should be of biological significance. Based on these intensive studies, a hypothesis regarding the formation of episodic memory is given.

Complexity is a cue to the mind

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Abstract: The relevance of chaotic itinerancy and other types of exotic dynamical behavior described by Tsuda (2001) certainly goes beyond the scope of his target article. These concepts of dynamics may offer a general framework for the understanding of complexity, which could help to restructure the analysis and conceptualization of mental states in novel ways, providing insights for the philosophy of mind.

Most philosophical treatments of the classical concepts of cognition (such as volition, consciousness, intentionality, mental content, rationality) seem to be trapped in an unsafe argument that, by and large, goes like this: There are two opposing alternatives, naturalism and anti-naturalism, both with unwanted consequences. It is generally accepted that to be a naturalist is to be a physicalist and/or functionalist and to believe in the computational mind and/or physical (computational) brain. To be a anti-naturalist is (as Dennett [1995] so aptly puts it in a somewhat different context) to play tennis with the net down. If one is a anti-naturalist, then one is free from the restrictions of science, and it is easy to justify any concept, from intrinsic intentionality to wonder tissue to qualia, without having to worry how these fit into a description of the universe as a whole. There are other points of view existing somewhere between these two extremes, such as double-aspect theories, where the physical and mental aspects of material are considered distinct, but the manner in which they are distinguished is not addressed. However, this again internalizes the naturalist/anti-naturalist dichotomy. So where do we stand? Currently, few people are completely comfortable with naturalism-cum-physicalism in a strict form; functionalism is on the defense; accepting the small wonders offered by anti-naturalism is not the approach of choice. Are there other options? Maybe there are.

The above argument goes too fast. Several hidden assumptions underlie it, which together implicitly ensure a harmony of naturalism and reductionism (or eliminativism), or a unity of naturalism and computationalism. At least two of the assumptions are easy to identify. The first is that the mind is confined to a single level of phenomena (therefore, all mental events can be treated within a single explanatory scheme). The second is that mental states are homogeneous (therefore, they are all similar, constituting a natural kind).

One kind of thing makes up the mind, and one functional theory explains how it works: cognitive science has traditionally held this. However, Ryle (1949), perhaps Haugeland (1995), and more recently van Gelder (1998) laid the groundwork for a fundamental challenge to the concept of a homogeneous mind. Their concern is with the difference between mind and cognition, but their idea that the mind is somehow not one thing is far more general. Van Gelder made a significant move when he combined this philosophical idea of ontological heterogeneity with low-level functional models to form what is known as the "dynamical hypothesis" in cognition (Port 2001). This allowed for a new kind of continuation through which the other pillar of the traditionally posited unity of the mind, that of a single functional scheme, might also be discredited.

This is the point where dynamical systems of the kind studied by Tsuda enter. We can view the dynamical hypothesis as a step in a progression towards a greater flexibility of the mind: the progression begins with the rigid symbolic systems of "classical" (Fodor-style) cognitivism, and continues through connectionism to dynamical systems, and possibly beyond.

Previous attempts to use dynamical systems as models of cognition have been unsuccessful. Ten or fifteen years ago the study of complex systems consisted of only chaos, bifurcation, and self-organization – phenomena that have a technical meaning in hydrodynamics, basic-level brain theory, and the like, but apparently are not appropriate in the description of higher cognition. They are hopelessly far from anything in which the philosopher or psychologist is interested. How can we elucidate mental content, for instance? Early-day cybernetics, based on simple dynamical systems, had nothing to offer in this regard, which is one reason that cybernetics has been ignored in cognitive science. Chaos, although representing a richer class of phenomena, is no different on this score. It appears to possess physiological relevance for the brain (see Freeman 1991, which is now a classic), but it is difficult

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to see what could be gained by assuming that mental computations are chaotic, for computations are just computations. Also, chaos is, by itself, uninteresting: in a chaotic system, most things happen on the attractor, where long-term behavior always remains qualitatively the same.

Chaotic itinerancy (CI), however, is fundamentally different, and I believe it may change our thinking about what can be achieved by dynamical systems and minds. CI is a chameleon. It can behave like one particular type of system for some time, then make a transition and behave like some other particular system, and so on, indefinitely. Although generated by a single set of equations, at a phenomenological level the process does not seem to follow any fixed rules. Yet it can be controlled, or at least this is a feasible expectation. For this reason CI may be an ideal tool for the understanding of mental processes that form and break their own rules. Part of this anticipated development is already visible in Tsuda's memory model.

The problem of conceptualizing complexity is another matter. Complex dynamical systems of the type studied by Tsuda and some other authors, including Kaneko (1998; see also Kaneko & Tsuda 2001), realize a wide spectrum of behaviors. This has far-ranging philosophical implications. Putnam (1988) may be right, after all, in his claim that every natural system can behave like any other. With Tsuda's work we start to understand that for a complex system every model can be justified. What is more, perhaps every physical system is capable of exhibiting chaotic itinerancies and other similarly rich processes. If this is true, every physical system may literally be able to realize every computational behavior. Opponents of Putnam's idea (such as Chalmers 1996) tend to assume that a system is basically something perfectly well-defined and, therefore, that the construction of a single system capable of behaving like many different systems must be seen as a dirty trick. CIs can realize such a situation quite naturally. This is a further example where the benefits of an advanced analysis made possible by the new paradigm of complex systems becomes apparent.

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Author's Response

Chaotic itinerancy is a key to mental diversity

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Abstract: Kampis proposes the study of chaotic itinerancy, pointing out its significance in domains of cognitive science and philosophy. He has discovered in the concept of chaotic itinerancy the possibility for a new dynamical approach that elucidates mental states with a physical basis. This approach may therefore provide the means to go beyond the connectionist approach. In accordance with his theory, I here highlight three issues regarding chaotic itinerancy: transitory dynamics, diversity, and self-modifying system.

The main characteristics of chaotic itinerancy (CI) are summarized as follows (Kaneko & Tsuda 2001; 2003; Tsuda 2001), although not all characteristics are necessarily required:

1. There appear a relatively large number of modes possessing neutral stability, as well as definite stable and unstable modes.
2. There appears a highly ordered but irregular temporal structure, and hence the appearance of history-dependent transitions.
3. Chaotic itinerancy differs from simply chaotic behavior in the sense that these transitions can be characterized as transitory dynamics through which the system moves between low-dimensional attractor ruins and high-dimensional chaotic states.
4. The statistical convergence of physical quantities is absent or extremely weak.
5. The system does not possess tracing properties.

There is a mathematical basis for each of the above-stated characteristics. In a system that possesses the pseudo-orbit tracing property, it is guaranteed that its mathematical trajectories can be correctly traced in numerical simulations. Typical chaotic systems possess this property, but some chaotic systems do not. In such systems, it is not possible to properly trace each trajectory in simulations. This lack of the pseudo-orbit tracing property leads to another instability, which differs from the orbital instability that chaotic systems inherently possess – that is, instability with regard to computation, description, and/or observation. Therefore, contrary to the picture presented by **Kampis**, a factor for renewing a system itself exists even in conventional chaos, in principle. However, as Kampis correctly points out, this property is not enough to realize an actual renewal process. Even if such a property does not exist, it is possible to obtain precise statistical properties, which reflect the properties of an attractor as a whole. Actually, this attractor-tracing property exists even in conventional chaos. On the other hand, the statistical properties of chaotic itinerancy do not necessarily provide information about the overall attractor (Sauer 2000; 2003; Tsuda & Umemura 2003). Chaotic itinerancy therefore appears to be a new type of dynamic behavior that goes beyond the attractor concept. It is thus seen that chaotic itinerancy can be considered a form of transitory dynamics that might appear to be "non-stationary" in short-time observations.

In dynamical systems exhibiting chaotic itinerancy, there come to exist transition rules between attractor ruins. The nature of these rules is determined by chaotic itinerancy itself. The dynamical orbit is attracted to the ruins, and in this situation the number of effective degrees of freedom remains relatively small. Therefore, the system can be described with only a few modes. While the system remains in such a space, other modes become activated. As a result, the system can no longer be confined in such a space. Because there are restricted regions that can act as exits, there are a number of selected orbits that can leave (Kaneko 1998). When an orbit leaves this space, the number of effective degrees of freedom increases, and there results a large diversity of states. After some time, the orbit is attracted to another ruin, where a different set of effective modes describe the system. In such a way, such dynamical systems exhibit history-dependent activity.

In the study of dynamical behavior in a neural network model with nearest-neighbor couplings, we recently identified a mechanism involved in chaotic transitions between synchronized and de-synchronized states (Tsuda et al. 2004). Such transitions have been observed in animal and human brains (Freeman 2004; Gray et al. 1992; Lampl et

al. 1999). This behavior appears to consist of chaotic itinerancy between attractor ruins representing synchronization and de-synchronization states. It also appears that this behavior can be regarded as chaotic itinerancy between attractor ruins (which may be described by Milnor attractors), each consisting of a set of an all-synchronization state, different kinds of metachronal waves, and small chaotic orbits linking these states. Furthermore, such chaotic itinerancy accompanies the organization and reorganization of dynamic cell assemblies. There are several examples that are believed to provide a link between physical behavior and the representation of mental states using chaotic itinerancy. These examples include dynamic memory (Tsuda 1991; 1992; Tsuda et al. 1987), episodic memory formation (Tsuda & Kuroda 2001), and category formation (Tsuda 2001). The use of chaotic itinerancy to study category formation might elucidate a typical feature of diversity expressed by chaotic itinerancy, as described by **Kampis**.

Category formation can be characterized by the following two kinds of ambivalence. We call the first kind identification and discrimination ambivalence. In this case, "similar" patterns, objects, and concepts must be classified in accordance with their similarity as belonging to the same group. Here, similarity can be represented by a certain metric. Yet, it must be possible to discriminate even similar patterns, objects, and concepts. The second kind, which we call invariance and variance ambivalence, appears especially in relation to learning over periods of time that are longer than those expected in the case of the first ambivalence. For stability of cognition, the invariance of categories is required, but to allow the learning process – a variance of classification, that is – a change of categories is necessary.

Our assertion is that chaotic itinerancy can represent the types of ambivalence described above, in principle. In typical chaotic itinerancy, the attractor ruins linked by chaotic transitory orbits do not necessarily constitute all the ruins because of the dependence of statistical properties on the initial conditions. Some particular orbit linking several attractor ruins may form the largest category of series of events – an episode – with each attractor ruin representing a distinct event. This largest category consists of several sub-episodes, some of which are "similar" and some of which are different. Actually, we found such category formation in a Cantor code with chaotic itinerancy as inputs (Tsuda 2001; Tsuda & Kuroda 2001). This is possible because chaotic itinerancy contains history-dependent information of a series of events (Kaneko 1998). This scheme of coding guarantees both identification and discrimination.

From the above-described findings, it can be seen that chaotic itinerancy may be a key type of dynamic motion capable of describing ever-changing behavior with stability, which all evolving systems should exhibit. This is nothing but an evolutionary system that **Kampis** describes as a self-modifying system (Kampis 1991). In contrast to the conventional concept of a system, according to which a system consists of well-defined units with each unit assumed to possess a definite function, we consider a self-modifying system to be a life system, in which a functional unit is formed via interactions between internal states of the system. Such a system is precisely that which we have considered a "complex system" (Kaneko & Tsuda 2001).

There is sufficient evidence supporting such a specific structure of complex systems. Among others, cell differentiation and the organization of functional modules in the brain are typical examples. Every functional module in the brain, like the visual cortex, is connected to other modules in its living state, not only structurally but also functionally. In other words, functional modules are not ready-made but, rather, order-made. They are formed almost simultaneously when a total system becomes functional. This type of formation brings about a difference between the responses of organized units in the cases where these units are isolated and are embedded in the system. Furthermore, such functional modules can vary in structure and function. A similar concept was proposed as the concept of components (Rosen 1991) and as a dynamic cell assembly (Fujii et al. 1996; von der Marlsburg 1981).

These are characteristics of ever-changing systems, which are typically described by chaotic itinerancy and may well be related to the flexible change of mental states.

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The letter "r" before author's initials stands for Author's Response to CC references

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Commentary on **Bernhard Hommel, Jochen Müsseler, Gisa Aschersleben, & Wolfgang Prinz (2001). The Theory of Event Coding (TEC): A framework for perception and action planning. BBS 24(5):849–878.**

Abstract of the original article: Traditional approaches to human information processing tend to deal with perception and action planning in isolation, so that an adequate account of the perception-action interface is still missing. On the perceptual side, the dominant cognitive view largely underestimates, and thus fails to account for, the impact of action-related processes on both the processing of perceptual information and on perceptual learning. On the action side, most approaches available conceive of action planning as a mere continuation of stimulus processing, thus failing to account for the goal-directedness of even the simplest reaction in an experimental task. We propose a new framework for a more adequate theoretical treatment of perception and action planning, a theory postulating that perceptual contents and action plans are coded in a common representational medium by feature codes with distal reference. Accordingly, perceived events (perceptions) and to-be-produced events (actions) are equally represented by integrated, task-tuned networks of feature codes – cognitive structures we call event codes. We give an overview of evidence from a wide variety of empirical domains, such as spatial stimulus-response compatibility, sensorimotor synchronization, or ideomotor action, showing that our main assumptions are well supported by the data.

Does TEC explain the emergence of distal representations?

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Abstract: Hommel et al. (2001) try to explain the emergence of distal representations by an evolutionary account which includes their theory of event coding. A closer look at the way the terms "distal representations" and "representations of events" are defined reveals, however, that their hypothesis of a common code for perceived and to-be-produced events is in fact superfluous. Moreover, it shows that they mix up empirical facts with conceptual/definitional facts in the second assumption of their explanation.

In the "Epilogue" to their target article (sect. 5), Hommel et al. (2001) ask: "How is it possible that our mental experience refers to events in the outer world [. . .], given the fact that it derives from events in our bodies and our minds?" They try to answer that question by an evolutionary account which includes their theory of event coding. First, at some point in evolution, a common representational system for both perceived and to-be-produced events evolved as a means to plan action. Second, the most efficient way of implementing such a system is by creating distal representations. Put together, "the invention of identical codes for perceived and to-be-produced events implies the construction of reality" (■■■).

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Obviously, the emphasis here is on "identical." Hommel et al.'s main topic, the hypothesis of a common code, holds the central position in this explanation. A closer look, however, reveals that recourse to commonality is in fact superfluous. If an agent has representations for events, he already possesses distal representations, regardless of whether they are couched in a common representational medium.

My argument is quite simple. Question 1: What is a distal representation? Answer: By calling a representation "distal," we say something about its extension. A distal representation is a representation of a distal entity; it is a representation that does not refer to something inside the agent but to entities in the outer world. For example, a representation of spikes in Susan's brain does not belong to her distal representations because its extension does not consist of things in her environment.

Question 2: What is a representation of a perceived or to-be-produced event? Part 1 of the answer is as before: By calling something a "representation *of*," we say something about its extension. A representation of a perceived or to-be-produced event is a representation that refers to such an event. Part 2 of the answer: In the context of Hommel et al.'s article, "event" does not mean "event inside the agent" but, rather, "outer event"; therefore, a representation of an event is a representation whose referent is an occurrence in the person's environment. For example, a representation of electrical activity in Susan's visual cortex is not among her representations of a perceived or to-be-produced event, in the relevant sense of "event."

With these definitional facts in mind, it is clear why recourse to a common code plays no role in Hommel et al.'s explanation. A representation of a perceived or to-be-produced event is, by definition, a distal representation because it refers to a distal entity. Hence, if, at some point in evolution, a species acquires the ability to represent perceived events, or the ability to represent to-be-produced events, or both, then by definition this is sufficient for having distal representations. It does not matter at all whether those different kinds of events are coded within a common format. Even if the structure of the representations standing for perceived events differs widely from the structure of the representations standing for to-be-produced events, they are nonetheless distal representations because being such a representation means nothing else than representing entities in the outer world. It is not "the invention of identical [!] codes for perceived and to-be-produced events" that "implies the construction of reality," but just the invention of – whatever – codes for events.

Hommel et al.'s explanation therefore boils down to: We have distal codes because evolution has provided us with codes for perceived and to-be-produced events as a means to plan action. Here, their second assumption plays no role anymore. In light of my remarks, this should not come as a great surprise because the assumption sounds somewhat odd anyway. Hommel et al. claim that

the most efficient way of producing a common system of representations referring to perceived and to-be-produced events is by creating codes with distal reference. I would rather say that this is, again by definition, the only way of implementing any system of representations referring to events, whether or not they are coded within a common medium.

Remember that "event" means here "event in the outer world." On that ground, the creation of event codes implies the creation of distal codes because representations that refer to events in the environment are, necessarily, distal representations. Hence, creating representations of distal entities is not a more or less efficient way of implementing representations of events. It is, rather, a trivial consequence of the way in which the terms "distal representations" and "representations of events" are defined. Claiming that it is the most efficient way is like claiming that the most efficient way for a married man to become a bachelor is by getting unmarried. If this were the most efficient way, there would be other, less efficient, ways. But there are none. Therefore, it is true that producing event codes requires creating distal codes – but note, first, that this is not an empirical but a conceptual truth; and, second, it has nothing to do with the common representational system for perception and action advocated by Hommel et al.

Reference

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Commentary on **Zenon W. Pylyshyn (2002). Mental imagery? In search of a theory. BBS 25(2):157–182.**

Abstract of the original article: It is generally accepted that there is something special about reasoning by using mental images. The question of how it is special, however, has never been satisfactorily spelled out, despite more than thirty years of research in the post-behaviorist tradition. This article considers some of the general motivation for the assumption that entertaining mental images involves inspecting a picture-like object. It sets out a distinction between phenomena attributable to the nature of mind to what is called the cognitive architecture, and ones that are attributable to tacit knowledge used to simulate what would happen in a visual situation. With this distinction in mind the paper then considers in detail the widely held assumption that in some important sense images are *spatially displayed* or are *depictive,* and that examining images uses the same mechanisms that are deployed in visual perception. I argue that the assumption of the spatial or depictive nature of images is only explanatory if taken literally, as a claim about how images are physically instantiated in the brain, and that the literal view fails for a number of empirical reasons – for example, because of the cognitive penetrability of the phenomena cited in its favor. Similarly, while it is arguably the case that imagery and vision involve some of the same mechanisms, this tells us very little about the nature of mental imagery and does not support claims about the pictorial nature of mental images. Finally, I consider whether recent neuroscience evidence clarifies the debate over the nature of mental images. I claim that when such questions as whether images are depictive or spatial are formulated more clearly, the evidence does not provide support for the picture-theory over a symbol structure theory of mental imagery. Even if all the empirical claims were true, they do not warrant the conclusion that many people have drawn from them; that mental images are depictive or are displayed in some (possibly cortical) space. Such a conclusion is incompatible with what is known about how images function in thought. We are then left with the provisional counterintuitive conclusion that the available evidence does not support rejection of what I call the "null hypothesis"; namely, that reasoning with mental images involves the same form of representation and the same processes as that of reasoning in general, except that the content or subject matter of thoughts experienced as images includes information about how things would look.

Dr. Pylyshyn: Should this "*in* what is called the cognitive architecture"? Or perhaps "*as* to what . . . "?

Spatial inference: No difference between mental images and mental models

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Abstract: In contrast to Pylyshyn's view, there is no such thing as "reasoning in general." Different types of reasoning tasks are solved with different reasoning strategies. A more specific null hypothesis is that *spatial inference* with mental *images* involves the same representational formalism as that of spatial inference with mental *models.* There is no evidence that this hypothesis must be rejected.

Pylyshyn's line of argumentation starts with a "null hypothesis" and ends, after a scholarly review of empirical work, with the conclusion that the null hypothesis cannot be refuted. His hypothesis reads: "reasoning with mental images involves the same form of representation and the same processes as that of reasoning in general" (Pylyshyn 2002, p. 157). Unfortunately, the way in which the hypothesis is stated prevents it from being refuted. It has the form *X involves the same Y as Z,* with an *X* whose ontological status is disputed and a non-existing Z. There is no such thing as "reasoning in general" because different types of reasoning tasks are solved with different reasoning strategies. Consider, for instance, the following inference problem:

The hammer is to the right of the pliers
The screwdriver is to the left of the pliers
The wrench is in front of the screwdriver
The saw is in front of the hammer
Which spatial relation holds between the wrench and the saw?

In principle, such tasks could be solved by a reasoning strategy that consists of applying inference rules as suggested by Rips (1994). But this account has an important consequence: the premises must be kept separately in mind throughout the whole reasoning process. No integrated representation of the problem information is constructed. Empirical evidence suggests that a different type of strategy is adopted by most subjects: An integrated representation, or mental model, is constructed in working memory and constitutes the information on which the reasoning process operates (see, e.g., Johnson-Laird & Byrne 1991). A mental model for a spatial inference task encodes a specific spatial configuration satisfying all the premises; for example:

screwdriver pliers hammer
wrench saw

What is specific (and spatial) in this integrated representation? First, we need to clarify what is meant by properties of a

representation. Discussing the functional space, Pylyshyn argues that a matrix considered as a data structure without the computational processes operating on it does not embody any intrinsic representational constraints. Although this is a common view in cognitive science (Anderson 1978), it is surprising from a computational perspective, because representation without processing is a void concept. All representational constraints on images are extrinsic in Pylyshyn's sense because they are resulting from the interaction between data structures and processing (Schlieder 1998). Therefore, it seems that the distinction between intrinsic and extrinsic constraints of data structures should be abandoned. In contrast, properties (e.g., intrinsic vs. extrinsic) of representational formalisms should be considered that encompass both data and processes. In the case of spatial inferences, the representational formalism consists of a data structure, the mental model, and processes that construct, inspect, and modify this model.

Does mental imagery play a role in this representational system as reports of subjective experiences suggest (cf. Kosslyn 1994, p. 324)? Or does spatial inference, as mental model theory suggests, rely on a representational formalism that is spatial in the sense that it represents the relative position of objects to other objects, but not necessarily visual features such as shape, size, distance, and so on? In response to these questions, we propose to reformulate Pylyshyn's null hypothesis: Spatial inference with mental *images* involves the same form of representation and the same processes as spatial inference with mental *models*. Obviously, this hypothesis is more specific as it refers not to reasoning in general but to a particular reasoning strategy (X = spatial inference with images; Y = representational formalism; Z = spatial inference with mental models). At the same time, it is more amenable to empirical testing because it asks for the involvement of cognitive systems devoted either to visual or to spatial processing. There are several groups of experiments that help to answer our questions. For instance, if reasoning involves visual images that differ from models, then the "imageability" of the materials should affect reasoning performance. Johnson-Laird et al. (1989) failed to find any differences between problems that were easy or difficult to visualize. And Knauff and Johnson-Laird (2002) found that if the content of an inference problem helps to construct a spatial mental model, then comprehension and reasoning proceed smoothly. But, if the content gives rise to visual images, reasoning is impeded by irrelevant visual details. Klauer et al. (1997) showed that spatial inference interferes with the preoccupation of visual and spatial working memory, whereas phonological secondary tasks do not impair reasoning performance (cf. also Vandierendonck & de Vooght 1997). However, if the visual and the spatial components in the secondary tasks are varied separately, processes devoted to purely visual features of objects turn out to be irrelevant (Knauff et al. 2004). Experiments using functional brain imaging measured cortical activity during spatial inference in parietal areas but not in early visual areas (e.g., Goel & Dolan 2001, Knauff, et al. 2002). The parietal cortex is thought to play a key role in spatial processing and working memory (e.g., Baker et al. 1996) and in the integration of sensory information from all modalities into egocentric spatial representations (Burgess et al. 2001; Xing & Andersen 2000).

The details of the reported studies are not as important as the general points: (1) In the case of spatial inferences, it seems reasonable that mental models together with the processes that construct, inspect, and modify these models encode the spatial properties described in the premises of the inference problem. Hence, the representational formalism (i.e., representations and processes) is intrinsically spatial. (2) The abstract spatial nature of the mental models corresponds to activity in areas of the brain that are involved in the processing of spatial information from different modalities. (3) The mental models do not necessarily represent visual features (shape, size, etc.) and the subjective experience of visual imagery is probably an irrelevant side effect. Overall, based on this evidence we cannot reject the specific null hypothesis that spatial inference with mental *images* involves the same representational formalism as that of spatial inference with mental *models*. Whether this conclusion generalizes to all sorts of reasoning, as claimed by Pylyshyn, definitely needs further investigation.

ACKNOWLEDGMENTS

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Author's Response

From reifying mental pictures to reifying spatial models

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Abstract: Assuming that the vehicle of imaginal thought is a spatial model may not be quite as egregious an error as assuming it is a two-dimensional picture, but it represents no less a reification error. Because the model is not a literal physical layout, one is still owed an explanation for *why* spatial properties hold in the model – whether because of architectural constraints or by stipulation. The difference is like the difference between explaining behavior from a principle and predicting it by looking it up in a list. In the latter case no purpose is being served by calling it a mental model.

There is much in the **Knauff & Schlieder** commentary with which I agree. For example, I agree that spatial representations are not images in the sense of being perceivable pictures and I agree that representations are to be viewed in conjunction with the processes that operate over them. What I disagree with is the conclusion these commentators draw from their premises. In particular they appear to make exactly the same reification error that pervades the entire imagery literature, only they reify spatial layouts rather than pictorial objects.

Interestingly, the example they offer is the very one I have discussed elsewhere (Pylyshyn 2003, pp. 451–55). It concerns the location of a collection of objects. Discussion of such examples invariably involves exhibiting a table on which the objects are laid out in a fixed pattern. **Knauff & Schlieder** refer to this as an "integrated representation . . . constructed in working memory" and claim that it is spatial "in the sense that it represents the relative position of objects to other objects." They clearly do not wish to assume there is a table in the head, so it is a puzzle why the "integrated" characterization is not equally true of a representation such as the one they give in the problem statement, augmented by some preparatory inferences such as those concerning the relative locations of pliers and hammer, screwdriver and saw, and so on – exactly the sorts of inferences that have been shown to occur in the course of understanding a problem statement (e.g., Bransford & Johnson 1973). Why is this not an "integrated representation"?

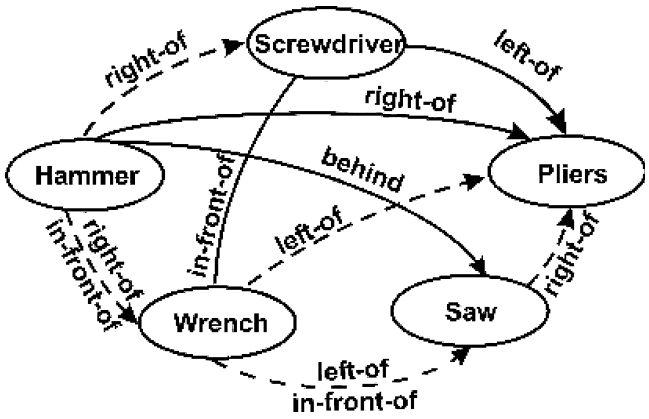

Figure R1 (Pylyshyn). A network implementation of the mental model example cited by Knauff & Schlieder. Solid lines are part of the problem statement. Dotted lines indicate inferred relations inserted in the course of understanding the problem statement.

When it is expressed as a network such as in Figure R1, is that integrated enough? It allows some inferences to be "read off" the data-structure quickly, just as in the mental model.

It is clear that **Knauff & Schlieder** do not want such a data structure to qualify as a "mental model" although they may be hard put to say why. In fact it is at this point that talk of mental models invariably lapses into the same reification error as does talk of mental pictures: It assumes that the objects in the mental model conform to physical principles such as rigidity, group movement, invariance of relative locations with changes to some parts of the model, and so on, and that objects stay put and are available for inspection as attention is applied to it. The "frame problem" in Artificial Intelligence (Pylyshyn 1987) arises precisely because one is not entitled to make such assumptions in general. To the extent that any such assumptions hold in a particular problem, they have to be explicitly stated. If they hold for every problem in a certain general domain (e.g., all problems involving spatial layouts), there is some prospect that they might be built in to the computational architecture. But spatial properties of the sort that are appealed to in the example are clearly contingent – the problem could have been one in which the objects were floating in a turbulent liquid or attached to balloons. Though this would still be a problem involving spatial locations, nobody would reason about it by examining the sort of spatial model that Knauff & Schieder advocate. Why not? Clearly because in this case the assumptions about rigid configuration would not hold. But this just shows that the constraints are part of the particular problem statement (they are cognitively penetrable) and therefore need to be expressed in "frame axioms" rather than in the fixed architecture.

This has nothing to do with whether the properties are in the data structures or in the algorithm. Rather, it has to do with whether the properties are being modeled as part of the mental architecture or as part of the problem understanding. It does no good to point to the computer model and say that the array *together with its access process* is functionally like a table, because it could be functionally like anything one chooses since the function it can compute is unconstrained (beyond being a recursive function). An explanation has to have fewer degrees of freedom than the data it explains – it has to say why *this* pattern rather than *that* pattern occurs when either could have been programmed.

In order for the program to serve an explanatory role, the theorist must say how it maps onto the mental process. In particular, the theorist has to make a commitment as to whether the array-plus-algorithm is presented as a model of the cognitive process or as an emulation of the medium of representation (i.e., the mental architecture; see Pylyshyn 1984). To abandon the distinction between architecture and process is to abandon the distinction between explaining and describing (or asserting). It is the difference between deriving a prediction from an assumption about the nature of the computational architecture and reading it off a list in which one has stored what will happen under various circumstances. Both may be equally predictive, but the list is not explanatory because it is unconstrained.

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The letter "r" before author's initials stand for Author's Response to CC references

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Commentary on **William A. Phillips & Steven M. Silverstein (2003). Convergence of biological and psychological perspectives on cognitive coordination in schizophrenia. BBS 26(1):65–82.**

Abstract of the original article: The concept of locally specialized functions dominates research on higher brain function and its disorders. Locally specialized functions must be complemented by processes that coordinate those functions, however, and impairment of coordinating processes may be central to some psychotic conditions. Evidence for processes that coordinate activity is provided by neurobiological and psychological studies of contextual disambiguation and dynamic grouping. Mechanisms by which this important class of cognitive functions could be achieved include those long-range connections within and between cortical regions that activate synaptic channels via NMDA-receptors, and which control gain through their voltage-dependent mode of operation. An impairment of these mechanisms is central to PCP-psychosis, and cognitive capabilities that they could provide are impaired in some forms of schizophrenia. We conclude that impaired cognitive coordination due to reduced ion flow through NMDA-channels is involved in schizophrenia, and we suggest that it may also be involved in other disorders. This perspective suggests several ways in which further research could enhance our understanding of cognitive coordination, its neural basis, and its relevance to psychopathology.

Autism and schizophrenia: Similar perceptual consequence, different neurobiological etiology?

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Abstract: Phillips & Silverstein (P&S, 2003) propose that NMDA-receptor dysfunction may be the fundamental neurobiological mechanism underlying and associating impaired holistic perception and cognitive coordination with schizophrenic psychopathology. We discuss how the P&S hypothesis shares different aspects of the weak central coherence account of autism from both theoretical and experimental perspectives. Specifically, we believe that neither those persons with autism nor those with schizophrenia integrate visuo-perceptual information efficiently, resulting in incongruous internal representations of their external world. However, although NMDA-hypofunction may be responsible for perceptual impairments in schizophrenia and possibly autism, we suggest that it is highly unlikely that NMDA-hypofunction is specifically responsible for the autistic behavioral symptomology, as described by P&S in their target article.

Autism and schizophrenia are heterogeneous and complex neurobiological disorders defined by a continuum of subtypes that are differentiated by cognitive and behavioral manifestations. In line with the Phillips & Silverstein (P&S, 2003) statement that "[the] fragmentation of mental functions is the prima facie evidence of impaired cognitive function" (■■■), insight regarding the nature of cognitive dysfunction in these two conditions may be derived from an evaluation of visuo-perceptual capabilities necessitating different levels of neural information processing. The motive for such assessment and subsequent interpretation originates from the fact that persons affected by these disorders share a common perceptual manifestation, namely, impaired perceptual organization reflected by abnormal performance on tasks requiring Gestalt-like or holistic visual analysis. It is therefore not surprising that interest regarding perceptual processing in autism has increased significantly since the introduction of neurobehavioral theories suggesting that a portion of abnormal autistic cognition and behavior may be explained in terms of the inefficient integration of visuo-perceptual information (e.g., Frith 1989; Mottron & Belleville 1993). Such theories share the notion that persons with autism do not integrate visuo-perceptual information efficiently into coherent percepts, characterized by a predominantly *local* approach to visual processing to the detriment of holistic information analysis. The *weak central coherence* (WCC) account of autism (Frith 1989) is arguably the most flexible of these theories because it offers a theoretical framework describing inefficient integration of information at different levels, including at a perceptual level (i.e., *perceptual coherence;* Happé 1999). These theories encapsulate anecdotal accounts of the fragmented visual world described by persons with autism (see, e.g., Gerland 1997; Gradin 1996).

Interestingly, accounts of "perceptual and apperceptual fragmentation" have also been described in schizophrenia studies (Arieti 1966) and have provided experimental evidence of inefficient perceptual grouping in this disorder. Such impairments have been exemplified by demonstrations of impaired performance necessitating the integration or grouping of complex static and dynamic visual information into meaningful percepts. As it has in autism, such evidence has led to notions of "spatio-temporal disintegration" of visual perception (Izawa & Yamamoto 2002) and anomalies regarding perceptual grouping schizophrenia (Place & Gilmore 1980; Silverstein et al. 2000).

How then, can one evaluate the integrity of early or preattentive neuro-integrative mechanisms mediating perceptual grouping? One method is to investigate the complex motion analysis capabilities of persons with autism and schizophrenia. Considered to be a form of dynamic grouping (Watt & Phillips 2000), complex motion analysis exemplifies early neuro-integrative processing since local motion information must be integrated across space and time before a global or coherent motion direction can be discriminated. Results from recent psychophysical studies directly assessing complex motion analysis mediated by extrastriate motion-sensitive mechanisms (i.e., V2/V3, MT) have demonstrated a decreased sensitivity to complex motion in autism (see, e.g., Bertone et al. 2003; Blake et al 2003; Gepner & Mestre 2002; Milne et al. 2002; Spencer et al. 2000). Results from these studies have been for the most part interpreted as a dysfunction of the dorsal stream processing or as a localized neural impairment of motion-sensitive areas in autism (Blake et al. 2003; Gepner & Mestre 2002; Milne et al. 2002; Spencer et al. 2000). In the only study investigating *both* simple and complex motion perception (Bertone et al. 2003), decreased sensitivity was evidenced for *only* complex motion types necessitating increased neural circuitry and integration to be resolved. For this reason, these findings were interpreted as a decreased capacity to integrate complex perceptual information rather than specific motion processing impairment per se (Bertone et al. 2003). The results from this study are very similar to those of Chen et al. (2003), who also demonstrated a decrease for complex but not simple (or local) motion in schizophrenia. Although the Chen et al. interpretation is more congruent with local neural dysfunction (i.e., dysfunction implicating motion-sensitive areas), when considered along with those of Bertone et al. (2003) they provide clear evidence of impaired dynamic Gestalt organization in both schizophrenia and autism. As mentioned by P&S, these analogous results can be interpreted as exemplary evidence of impaired cognitive coordination or, analogously, weak central coherence, in either condition.

Persons with autism and schizophrenia therefore share the following perceptual consequences: predominant local analysis of visual information and inefficient neuro-integrative perceptual processing, as well as anecdotal accounts of a fragmented perceptual

Jonathan, please check quote against P&S T.A. original in BBS 26(1), 2003 [printed version] and insert section and paragraph numbers here, as per BBS style – section abbreviated to sect., paragraph to para. – S.M.

world. The logically ensuing question is whether such common perceptual manifestations are the consequences of similar neurobiological etiology, specifically NMDA-hypofunction. If one interprets inefficient complex motion analysis as manifestation of impaired cognitive coordination, then the tentative answer is yes. Since such analysis involves gestalt-like integration over time and space that is believed to be mediated by NMDA-receptor activity, it is possible that autism and schizophrenia share impaired analysis of complex information at a *perceptual* level due to NMDA-hypofunction. However, impaired complex motion analysis has been demonstrated and interpreted differently for a variety of conditions defined by different behavioral manifestations. Such conditions include normal aging (Habak & Faubert 2000), dementia of the Alzheimer's type (Gilmore et al. 1994; Trick & Silverman 1991), dyslexia (Cornelissen et al. 1998; Talcott et al. 2000), and Parkinson's disease (Trick et al. 1994). Therefore, a consistent association between perceptual dysfunction defined by impaired complex motion analysis and clinical symptomology is not evidenced.

P&S's argument for associating NMDA-receptor hypofunction with perceptual, cognitive, and behavioral manifestations in schizophrenia is based in part on the schizomimetic effects of NMDA-antagonists. Blocking NMDA-receptor channels in non-schizophrenic persons results in schizophrenia-like symptomology (referred to as PCP-psychosis), which according to P&S is congruous with symptoms of "cognitive disorganization" (see Table 1 in the target article). Interestingly, Carlsson (1998) has used a similar argument to explain autistic perceptual and behavioral symptomology, adding that, like schizophrenia, abnormal glutamatergic interactions with other neurotransmitter systems (i.e., dopaminergic or serotonergic) may at least in part be responsible for the described autistic symptomology. Given the implication of NMDA-receptor activity in long-term potentiation (LTP), it can be argued that meaningful internal neural representations of their physical environment based on the efficient integration of perceptual information is compromised in persons with schizophrenia and in persons with autism. Consequently, appropriate behavior based on these representations would be abnormal and interpreted as being as part of the characteristic symptomology of either condition.

It can be argued that persons with autism and schizophrenia share (1) similar subjectively described and objectively measured manifestations of impaired Gestalt-like perception, probably the result of the inefficient integration of perceptual information, and (2) respective mimetic effects of NMDA-receptor antagonists. Given these similarities, can autism be considered to be a hypoglutamatergic disorder at a behavioral level if viewed within the context of P&S's working hypothesis? Probably the most important discrepancy between schizophrenia and autism regarding the possible implication of NMDA-hypofunction in their respective psychopathology concerns the onset of clinical symptomology. Although both conditions are considered to be congenital, their clinical symptoms are initially evidenced at different ages: between adolescence and young adulthood in the case of schizophrenia and around the age of three for autism. Taking this into account, even if the perceptual consequences of both disorders implicate NMDA-hypofunction, the effects of these consequences on symptomology is less evident. For example, it can be argued that the nature of schizophrenic hallucinations and delusions, which are not typically manifested in autism, are based on previously constructed percepts that have some associated affective value. In most cases, persons with schizophrenia associate a predominantly adverse affect (i.e., terror or confusion) to their abnormal perceptual experience, much like what is experienced during a drug-induced psychosis. In the case of autism, it can be argued that such constructs are never fully developed and, therefore, associations between perceptions and affects are never fully developed. Furthermore, persons with autism *grow up* with an abnormal perception of the world and, therefore, although maladaptive, their characteristic visually-related autistic behavior is usually void of negative affect (i.e., the pleasurable feeling experienced when fascinated with a specific part of an object). Consequently, one can argue that persons with schizophrenia and autism have different affective reactions to incongruent representations of their visual environment.

Finally, one must take into account that at the onset of autistic symptomology, the neural development of the autistic perceptual system is incomplete (i.e., neural pruning) compared to that of persons with schizophrenia, making the behavioral link between NMDA-hypofunction and clinical manifestations in these two disorders that much more complicated. In conclusion, although there is the possibility that NMDA-hypofunction may underlie the *perceptual* consequence manifested in schizophrenia and autism, it is much less probable that NMDA-hypofunction is selectively responsible for *behavioral* symptomology, a general association made by P&S regarding schizophrenia.

A common link between aging, schizophrenia, and autism?

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Abstract: Phillips & Silverstein (P&S, 2003) have proposed that NMDA-receptor hypofunction is the central reason for impaired cognitive coordination and abnormal gestalt-like perceptual processing in schizophrenia. We suggest that this model may also be applicable to non-pathological (or normal) aging given the compelling evidence of NMDA-receptor involvement during the aging process that results in age-related change in higher-level perceptual performance. Given that such deficits are present in other neurological disorders such as autism, an argument for a systematic assessment of perceptual functioning in these conditions may be posited.

Phillips & Silverstein (P&S, 2003) propose that there are reduced numbers of NMDA-receptors in schizophrenia that result in abnormal gestalt-like perceptual grouping. Fundamental to their argument is the implication of NMDA-receptor activity in long-term potentiation (LTP) which allows local events to be integrated into more global (or higher-level) perceptual constructs. As is the case for schizophrenia, there is abundant evidence of reduced LTP caused by NMDA-receptor dysfunction in the non-pathological (or normal) aging process that is accompanied by a decline in cognitive functioning (Gazzaley et al. 1996; Wenk et al. 1991; see also Segovia et al. 2001 and Rosenzweig & Barnes 2003 for reviews). We suggest that there exists a parallel and selective decline in higher-level perceptual information processing in normal aging, supporting P&S's notion that NMDA-receptor activity is involved in perceptual organization. This suggestion is based on results from a series of recent studies demonstrating that the normal aging process has a much greater impact on mid- to high-level perceptual functioning, which requires increased neural integration, than on lower-level perceptual processes (Faubert 2002). The differential efficacy of low-level and higher-level perceptual processing may also have consequences within certain types of cognitive functioning in aging, such as performance on visual working memory tasks.

As P&S have pointed out, perception is not a monolithic process, for it involves context integration and complexity constructs based on an infinite number of neural events. Recent studies have examined this exact process in normally aging observers in a number of visual modalities including motion and orientation (Habak & Faubert 2000), texture (Herbert et al. 2002), and form perception (Faubert & Bellefeuille 2002; Sara & Faubert 2000). Findings from these studies have demonstrated that as the amount of neural processing required to generate complex perceptual constructs increases, so does age-related loss in performance (Fau-

bert 2002). The goal of this commentary is to bring to light the similarities between normal aging and schizophrenia regarding inefficient higher-level integrative or gestalt-like perception. In addition, we propose that P&S's hypothesis implicating NMDA-receptor hypofunctioning in abnormal higher-level perception in schizophrenia may also be applicable to normal aging given the comparable evidence of increasing NMDA-receptor dysfunction during the aging process.

Perceptual complexity can be exemplified by the differential amount of neural integration needed to resolve first- and second-order visual information (Cavanagh & Mather 1989), the latter of which necessitates the activity of larger neural networks to be perceived (e.g., Bertone & Faubert 2003; Chubb et al. 2001; Nishida et al. 1997; Wilson et al. 1992). Habak and Faubert (2000) demonstrated a larger age-related increase in motion and orientation discrimination thresholds when the stimuli are defined by second-order attributes (e.g., texture). These results suggest that the age-related loss in performance is due to perceptual complexity of the second-order information and is not specific for visual attribute (motion or orientation). Because second-order image resolution implicates additional neural processing regardless of the type of information, it can be argued that decreased NMDA-mediated LTP may be the neurobiological mechanism responsible for the decrease in perceptual performance.

Gestalt-like perceptual grouping, as described by P&S, is exemplified by symmetry perception; it involves the spatial organization (e.g., symmetrical) of local stimulus elements (e.g., dots) into meaningful percepts. Therefore, perceiving symmetry involves the integration of local elements across the putative axis. Given the evidence of NMDA-receptor hypofunction in aging, we would expect an age-related loss in this type of spatial grouping task. In effect, a clear age-related deficit regarding the detection of bilateral symmetry detection has been demonstrated (Herbert et al. 2002).

The P&S NMDA-perception hypothesis predicts that long-range perceptual processes are affected in normal aging because NMDA depletion results in reduced LTP. Therefore, task performance based on the processing of information within a specific image attribute would be less affected by aging when compared to performance necessitating the processing and integration of information from two separate image attributes. A recent study by Faubert and Bellefeuille (2002) demonstrated that spatial frequency discriminations performed within an attribute condition (e.g., luminance vs. luminance or color vs. color) are less affected by age than are intra-attribute discriminations (e.g., luminance vs. color) when compared with younger observers. Similar age-related deficits in long-range processing have been demonstrated for tasks necessitating the simultaneous integration of information within a large spatial area prior to efficient perceptual decision making (e.g., size discrimination) compared to tasks for which the information is presented in sequence (temporal forced choice in same location; Sara & Faubert 2000). Taken together, these findings suggest a selective age-related loss for tasks soliciting long-range perceptual processing, as proposed with regard to schizophrenia by P&S. Again, these age-related decreases in performance are compatible with abnormal LTP.

As mentioned, the differential efficacy of low-level and higher-level perceptual functioning in aging may also have consequences affecting performance on visual working memory tasks (Faubert 2002). Recent studies have clearly demonstrated that normal aging has little effect on the capacity to retain either spatial frequency or size information defined by low-level perceptual information (Faubert & Bellefeuille 2002; Sara & Faubert 2000). This has led Faubert (2002) to conclude that perception and visual working memory are affected in similar ways during aging, in that processing of low-level information that does not require long-range processing (or complex networks, as termed by Faubert) is minimally affected. However, perceptual or working memory processes that require more sophisticated neural network structures will show age-related decline. Faubert (2002) suggested that this is the result of the Simultaneous Access Network Deficit (SAND) hypothesis of aging, which appears to us to be comparable to the NMDA-perception hypothesis offered by P&S in both a functional and possibly a neurobiological perspective.

In conclusion, we believe there is evidence in the normal aging process of the impaired gestalt-like visuo-perceptual grouping and accompanying neurobiological mechanism proposed by P&S which is comparable to that in schizophrenia and, possibly, other neurological disorders such as autism (cf. the preceding commentary by Bertone et al. [2004] in this issue). This suggestion is based on the fact that NMDA hypofunction and its relation to cognitive deficits appear to be relatively elaborated and specific to the aging literature, particularly with respect to currently available animal and human models demonstrating reversibility of some age-related effects (e.g., Baxter et al. 1994; Held et al. 2002). Furthermore, if impaired cognitive coordination, as reflected by abnormal gestalt-like perceptual performance, is the consequence of reduced glutamatergic neurotransmission, then the aging model is more suitable than schizophrenia for unidirectional hypotheses testing because glutaminergic NMDA receptor density decreases progressively with age. If NMDA hypofunction and gestalt-like information processing are functionally related, then one could predict not only a decline in higher-level perceptual information processing with increasing age but also the rate at which such a decline in performance would occur (Trick & Silverman 1991). These suggestions do not in any way detract from the proposal posited by P&S implicating NMDA hypofunction to abnormal holistic perceptual processing in schizophrenia. It simply states that if a link does exist between NMDA hypofunction, perceptual organization, and higher-order cognitive processing, non-pathological aging seems to be just as compatible a neuro-behavioral model for the P&S hypothesis as schizophrenia.

Authors' Response

Unity and diversity in disorders of cognitive coordination

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Abstract: Studies of aging and autism as outlined by **Bertone, Mottron, & Faubert** (**Bertone et al.**) and by **Faubert & Bertone** suggest that disorders of cognitive coordination involving impairments of dynamic gestalt grouping and context-sensitivity may be common to several different disorders. We agree that such studies may shed light on these processes and their neuronal bases. However, we also emphasize that dynamic grouping and context-sensitivity can fail in various ways, and that, although the underlying pathophysiology may often involve NMDA-receptor malfunction, many different malfunctions are possible, and each of these may result from any one of a number of different etiologies.

R1. Disorders of cognitive coordination in aging and autism

The commentators suggest that cognitive coordination is impaired in both cognitive aging and autism. They empha-

size disorders of perception, but do not imply that other domains of cognition are of lesser consequence. The target article also emphasized perception, though it made clear that coordinating interactions are of crucial importance to all cognitive domains. Here we discuss aging in section R2 and autism in section R3. In each section we discuss psychological and then neurobiological issues. Section R4 raises the issue of individual differences within each of the different disorders, and section R5 presents a simple conclusion.

R2. Age, perception, and NMDA-receptor malfunction

Faubert & Bertone note that many cognitive capacities tend to decline with age, and suggest that several of these may involve processes which we include under the broad heading of *cognitive coordination.* Their central suggestion is that, in accord with our general conceptual framework, these changes in cognition may be the result of an age-related decline in NMDA-receptor activity. Within our conceptual framework coordinating processes can be divided into those concerned with dynamic grouping and those concerned with contextual disambiguation. They are most obviously related to perception but, as coordination is relevant to all cognitive domains, an analogous discussion in relation to other domains such as memory, language, and motor control would be worthwhile.

Different cognitive functions decline with age to different extents. Decline is greatest in tests of "fluid intelligence," those that require novel solutions to novel problems, and is least in tests of "crystallized intelligence," those that depend predominantly on the use of well-learned bodies of knowledge such as vocabulary (Rabbitt 1993; Robbins et al. 1998). This is consistent with the view that cognitive coordination is particularly sensitive to the effects of aging because tests of fluid intelligence are likely to rely strongly on processes of dynamic organization and contextual modulation. However, we know of no evidence that normal cognitive aging involves anything analogous to formal thought disorder or phenomenal experiences of self-fragmentation, so a close analogy with schizophrenia seems unlikely. Nevertheless, **Faubert & Bertone**'s formulation recalls the similarity between aging and schizophrenia as conceptualized by Kraepelin, whose term for the illness was *dementia praecox* (early dementia). Though schizophrenia and normal aging differ in many ways, Faubert & Bertone's argument reminds us that there may be important similarities. If confirmed by future research, this could have important practical implications because it could lead to improvements in cognitive and pharmacotherapies aimed at reversing the impairments in both populations.

R2.1. Age-related changes in coordinating perceptual processes

As reviewed by Faubert (2002) there is evidence for a specific pattern of age-related changes in the perception of symmetry, motion, size, spatial frequency, and stereoscopic depth, and so forth. Faubert (2002) suggested that the common pattern running through all these changes is that age-related decline is greater when processing is in some way or other more "complex." To account for the various findings, the notion of complexity was outlined in various ways: (i) less hardwired is more complex; (ii) some tasks and spatial configurations require more complex processing (e.g., symmetry detection and tasks requiring comparison of figures at different locations); (iii) inter-attribute processing is more complex than intra-attribute; (iv) complexity increases with the number of stages of processing required; and, (v) long-range interactions are more complex than short-range ones. Faubert's emphasis on processes that are less hardwired is in accord with our conception of dynamic grouping, which also emphasizes processes that cannot be prespecified (Watt & Phillips 2000). The central issue here, however, is whether the findings Faubert reviews provide evidence for a specific age-related decline in processes of perceptual coordination.

Symmetry detection as studied by Herbert et al. (2002) is a clear example of a task that requires dynamic grouping (Watt & Phillips 2002, Fig. 2). On each trial a novel pattern of many dots was presented, with the discrimination to be made depending on the discovery of a symmetry axis that maximized the coherence of pairing across the axis for that particular stimulus. As this pairing could not be prespecified, it had to be discovered by processes of dynamic organization. The large age-related deficits that Herbert et al. observed in this task could therefore be due to deficits in dynamic grouping. To establish more firmly whether that is the case, it would be useful to develop versions of the symmetry detection task in which dynamic grouping is not necessary, and then to use those to determine whether age-related deficits depend specifically on the need for dynamic grouping. Knight et al. (2000) studied symmetry perception in a paradigm not requiring dynamic grouping. No dynamic grouping was required in their paradigm because simple familiar letter forms were shown on a blank background, with no need to discover new groupings defined only by symmetry. Using this paradigm, Knight et al. (2000) found no evidence of any abnormality in the use of symmetry as a figural descriptor by schizophrenic patients. Thus, Herbert et al. (2002) found evidence that symmetry detection requiring dynamic grouping tends to decline with aging, and Knight et al. (2000) found evidence that symmetry perception not requiring dynamic grouping is normal in schizophrenic patients. If both have deficits that are specific to paradigms requiring dynamic grouping, then aging will not be associated with deficits in the paradigm used by Knight et al. (2000) and schizophrenia will be associated with deficits in the paradigm used by Herbert et al. (2002).

In the case of motion perception we have well-developed paradigms with two versions, one that requires dynamic grouping and one that does not – that is, paradigms that compare the perception of local motion with the perception of global or coherent motion. Habak and Faubert (2000) found greater age-related decline for the perception of motion defined by second-order texture cues than for that defined by first-order cues. This is evidence for a specific deficit in the perception of global or coherent motion, but it requires confirmation using other paradigms for comparing the perception of local and global motion.

Prima facie, comparisons of figures at different locations may seem to have little to do with the dynamic processing implemented by coordinating interactions. Receptive fields in general integrate information from different locations and are primarily achieved by the prewired feed-forward connections with which coordinating interactions are contrasted. On further consideration, however, it seems that

the size-comparison task used by Sara and Faubert (2000) may well have involved processes of dynamic coordination as it required an arbitrary comparison of figures across locations that were arbitrary and task dependent.

Interattribute processing is by far the most frequently used example of the need for "binding." Prima facie then, the evidence for an increased age-related decline in interattribute comparisons (Faubert & Bellefeuille 2002) is evidence for a specific decline in coordinating interactions. Though we are skeptical of this frequently used example, we can interpret the task used as requiring dynamic coordination because that too required comparisons across attributes that were arbitrarily selected and task-dependent.

Prima facie, the suggestion that complexity can be related to the number of stages in a feature extraction hierarchy does not fit well with our distinction between the primary driving receptive field (RF) interactions that specify semantic content, and the coordinating interactions that modulate the primary processing to make it as coherent and task-relevant as possible (Phillips & Singer 1997). Within perceptual systems the RF synaptic connections are predominantly mediated by feed-forward connections, though some may be lateral, and the modulatory contextual field (CF) connections are predominantly mediated via lateral and descending connections. As the CFs have a distinct role, they have a distinct synaptic physiology – the NMDA-receptor channels, via which they modulate the primary RF processing. Therefore, in contrast to Faubert's notion of complexity, coordinating interactions are defined by their distinct function and physiology, not by where they occur in a hierarchy of processing. The findings interpreted by Faubert (2002) as evidence for greater age-related decline in higher-order features could nevertheless be interpreted as being due to deficits in coordinating interactions. This is because under certain conditions processing at higher levels depends in part on the coordinating interactions that occur at lower levels, and the higher the level the greater the possibility that failures in lower level coordination will affect processing. The conditions under which this is most likely are those in which dynamic grouping or contextual disambiguation at lower levels, or both, are most needed.

Similar arguments apply to the suggestion that long-range interactions are more complex than short-range interactions. Some RF connections may be long-range, for example, those necessitating callosal connections or those linking different cortical regions. Nevertheless, it is likely that cortical anatomy is such that RF connections are short so that a first sweep of primary feed-forward processing can be achieved rapidly, with coordinating interactions involving longer-range lateral and descending connections applying later. Overall, therefore, there will be a correlation between connection length and type of interaction, with coordinating CF connections tending to be longer than the primary feed-forward RF connections.

In summary, the evidence cited by Faubert (2002) supports the suggestion that age-related perceptual decline involves coordinating interactions. This raises many specific issues that require further research. For example, are the deficits in symmetry and coherent motion perception specific to versions of the paradigms that require dynamic grouping? Are there age-related disturbances of top-down influences on perceptual processes that are reversible by contextual and psychological manipulations as seen in schizophrenia (Silverstein et al. 1996a; 1996b; 1998)? Are there age-related deficits in context-sensitivity?

R2.2. Age-related NMDA-receptor malfunctions

Faubert & Bertone suggest that age-related NMDA-receptor malfunctions may cause the deficits in perceptual coordination. Before discussing this suggestion we must clarify two aspects of our perspective that may have been misunderstood. First, malfunction of the NMDA-receptor system does not necessarily involve any reduction in the number of NMDA-receptors. There are many other possibilities. Different subtypes have different subunit compositions, conferring subtly different physiological properties. It could therefore be that in some disorders it is the subunit composition that is abnormal, rather than the total number of receptors. Another important possibility is that it is not the NMDA-receptors themselves that are abnormal but the way in which they are modulated. NMDA-receptor function is dynamically modulated by several complex mechanisms, some pre- and some postsynaptic, and several genes that encode components of these mechanisms or regulate their expression have been shown to be susceptibility genes for schizophrenia (Moghaddam 2003). Although our target article emphasized the coordinating role of NMDA-receptors, it is now becoming clear that they are themselves modulated in various ways by several distinct mechanisms. This may explain why disorders of NMDA-receptor function can be subtle and specific to some aspects of cognition despite the fact that NMDA-receptors in general are widespread and fundamental to many aspects of brain function ranging from early embryological development to learning, memory, and coordination (Moghaddam 2003).

Faubert & Bertone's commentary also shows that a second aspect of our perspective needs further emphasis. This concerns long-term potentiation (LTP). Neither LTP nor any other form of plasticity is involved in the direct effect of NMDA-receptor-mediated neurotransmission on current activity. This was discussed briefly in section 3 of the target article, but needs further emphasis because our view differs from the standard textbook view that is still too frequently held by many researchers in the area. This standard view is that the primary role of NMDA-receptors is to facilitate learning. We disagree. Their primary effect, and their primary role, is to directly alter ongoing processing, and in particular to increase its coherence. In so doing they greatly increase postsynaptic depolarization, and thereby initiate the complex cascade of processes that lead to LTP and learning. Thus, what is learned are coherent, rather than incoherent, patterns of activity. NMDA-receptors have an important role in learning because they directly influence processing. If all forms of learning were suddenly suppressed, then this would not of itself directly alter the effect of NMDA-mediated neurotransmission on current activity. In the long-run, and in accord with Hemsley's (2003) perspective, it could, however, have an effect by suppressing adaptation to new statistical regularities. Our perspective sees this adaptation as including learning what predicts what, with the RF connections embodying the "whats" and the CFs the predictive relations between them. Therefore, in addition to their direct effect on current processing, NMDA-receptors can have an effect on later processing via their effect on learning. For formal and computational demonstrations of the possibility of neural networks that simultaneously discover

both the predictive relations and the entities between which they hold, see Phillips et al. (1995) and Kay et al. (1998).

We can now ask whether NMDA-receptor malfunction is likely to produce perceptual and other cognitive disorders associated with aging, and, if so, whether these malfunctions are likely to be the same as those producing cognitive disorganization in schizophrenia. In brief, the answer to the first question is "probably" and to the second is "prob-ably not." We are not well acquainted with the evidence on changes in NMDA-receptor function with aging, but agree that some changes seem likely. As NMDA-mediated neurotransmission has a central role in both coordination and learning, impairments in several aspects of cognition are a likely consequence. NMDA-receptor malfunctions in aging are not likely to be the same as those in schizophrenia, however. As there are various subtypes of NMDA-receptors, and several ways in which they are modulated, it is highly likely that they can malfunction in many different ways. This would lead to a large family of disorders which are similar in that all are disorders of some aspects of learning and coordination, but which differ in the particular aspects involved. This predicts that though there may be similarities between NMDA-receptor malfunction in schizophrenia and aging, there are also likely to be important differences.

An analogy with color vision may be useful here. There are several different forms of color blindness, some inherited, some acquired, but, despite these differences, they are all impairments of color vision. Similarly, there may be several different disorders of coordination, with both similarities and differences. In contrast to color vision, however, coordination is not of relevance only to certain specialized domains of cognition, but to all.

R3. Analogies between schizophrenia and autism

Despite differences in clinical symptomatology between schizophrenia and autism there may be similarities in underlying cognitive deficits. We agree with many others in considering schizophrenia to be a developmental disorder, and therefore assume that our understanding of schizophrenia and other developmental disorders, such as autism, will be advanced by focusing on similarities and differences in underlying mechanisms rather than focusing only on outward signs and symptoms, which is the current approach adopted in DSM-IV. Therefore, when **Bertone et al.** state, for example, that the clinical symptoms of autism and schizophrenia are evident at different ages, this is technically correct only to the extent that DSM-IV criteria are used to define the onset of these disorders. There is much evidence, however, that abnormalities are evidenced at a very early age in people who go on to meet DSM-IV criteria for schizophrenia in adolescence or adulthood. For example, children born to mothers with schizophrenia demonstrate abnormalities in infancy, which has been called a pandysmaturation syndrome (Fish 1977; Fish et al. 1992). High-risk children also demonstrate cognitive and social functioning deficits in childhood and early adolescence (Erlenmeyer-Kimling 2000; Neumann 1995). Interestingly, it is the subgroup of schizophrenia patients that demonstrate perceptual organization deficits after illness onset (as defined by DSM-IV) who have the histories of poorest "pre-illness" functioning of all schizophrenia patients (Knight & Silverstein 1998). Therefore, although there are clearly differences between schizophrenia and autism, their different ages of onset, as defined by DSM-IV, should not be taken as evidence that their underlying illness mechanisms are operative at distinctly different ages.

R3.1. Impairments of cognitive coordination in autistic spectrum disorders

Evidence that schizophrenia and autism may, at least in the case of an impairment in cognitive coordination, be on a continuum comes from several sources. One is that there is a group of children, typically called multidimensionally impaired or multiplex developmentally disordered (Kumra et al. 1998), that is characterized by several features of schizophrenia (e.g., psychotic symptoms, cognitive deficits, elevated rated of schizophrenia in first-degree relatives) *and* by features characteristic of pervasive developmental disorders such as autism (e.g., impaired social skills, linguistic impairment, cognitive decline). There is also evidence for elevated rates of psychosis and disorganization in people with autism spectrum disorders (Dykens et al. 1991; Konstantareas & Hewitt 2001) and in those with milder developmental disorders such as dyslexia (Duggan & Brylewski 2002; Sanderson et al. 1999), along with evidence of failures in contour integration and mismatch negativity in the latter group (Demonet & Habib 2001; Simmers & Bex 2001). Autistic-like problems may also antedate the "onset" of schizophrenia in many cases (Asarnow et al. 1988), and cases of DSM-IV-defined schizophrenia can occur in childhood. Therefore, as the commentaries suggest, it may be fruitful to examine commonalties between these various developmental disorders, and to define the most relevant dimensions on which to conceptualize the similarities and differences other than simply the age at "onset of illness."

In pursuing this goal, however, we should not assume that similar test performance is necessarily due to similar abnormal mechanisms. For example, it has been suggested that the superior part processing in autism may be due to an enhanced ability to process elementary physical properties of stimuli (Mottron et al. 2000), whereas in schizophrenia the tendency to process parts over wholes has been attributed to a deficit in processing holistic stimulus properties (Knight & Silverstein 1998). Similarly, whereas visual perceptual learning deficits in autism are related to a reduced ability to discriminate familiar forms compared to controls, an increased ability to learn novel stimuli has also been observed (Plaisted et al. 1998). This contrasts with the finding that in schizophrenia learning for shape-like forms was equivalent to that of controls, whereas learning of novel patterns was impaired (Silverstein et al.1998).

We agree with **Bertone et al.**'s view that the similarities between the deficits of motion perception in autism and schizophrenia are particularly striking. Rigorous psychophysical tests have now established that in both disorders local motion perception is preserved but global coherent motion perception is impaired. This is strong evidence for a specific deficit in dynamic grouping in each of the disorders. Furthermore, this echoes the findings in studies of cognitive aging discussed above, indicating that deficits of dynamic grouping can be part of several different disorders.

There is also evidence for deficiencies of context-sensi-

tivity in autism. Happé (1997) found evidence that autistic subjects are less susceptible to several standard illusions. Some of the illusions used depend on context-sensitivity, and this applies in particular to the Ebbinghaus size perception illusion in which the size of a target figure is made to appear larger or smaller by surrounding it with smaller or larger figures respectively. Happé's evidence for a reduction in this illusion in autism is therefore evidence for reduced context-sensitivity in autism. There have been failures to replicate this finding, however, so its status is not yet clear (e.g., Ropar & Mitchell 1999).

As deficiencies in theory-of-mind tests are also prominent in autism and can also be related to impaired coordination (Brock et al. 2002), Uhlhaas (2003) studied the performance of schizophrenic patients on theory-of-mind tasks and on a psychophysical test of context-sensitivity based on the Ebbinghaus size perception illusion. He found a clear relation between the two in that poor performance on a particular theory-of-mind task (the hinting test) was associated with reduced context-sensitivity (and thus better performance) on the size perception task. Others (e.g., Safarti et al. 1999) have also found that disorganized schizophrenics have deficits in theory-of-mind tasks. This strengthens the grounds for assuming that a useful analogy can be drawn between cognitive impairments in schizophrenia and autism.

Brock et al. (2002) have proposed an "impaired temporal binding" hypothesis of autism that is very similar to our hypothesis of impaired cognitive coordination in schizophrenia. From a psychological perspective the hypothesis of a cognitive coordination deficit in autism has much in common with the far better known theory of reduced "central coherence" (Frith 2003). However, the cognitive coordination hypothesis has the great advantage that the fundamental concepts are given a precise formal specification and are related in detail to physiological mechanisms. The psychological concepts can therefore be developed and tested in a far more principled way.

R3.2. The pathophysiology of impaired cognitive coordination in autism

Bertone et al. suggest that as the clinical symptomatology in autism differs from that in schizophrenia their underlying pathophysiologies must also differ. We agree. We must note at the outset, however, that although section 6 of the target article did suggest that genetically specified variations in the strength of coordinating neuronal interactions may play an important role in autism, this does not necessarily imply a primary deficit in NMDA-receptors, and certainly not one that is the same as any associated with schizophrenia. This in no way implies that they cannot both involve NMDA-receptor malfunctions, however. As outlined in R2.2, coordination within the glutamatergic system via the various subtypes of NMDA-receptor and their modulators provides a rich source of possibilities for many different malfunctions. It remains to be discovered which malfunctions produce which disorders, but even at this stage it seems likely that there is not only one kind of NMDA-receptor malfunction, but many.

Another point requiring clarification here is that we do not assume that NMDA-receptor malfunction necessarily produces a hypoglutamatergic state. Our working hypothesis is that it produces dysregulation of glutamatergic activity. In some conditions this will lead to an overall excess of activity, in others to an overall decrease, and perhaps in others to little or no net change in overall activity. However, in all cases this dysregulation will produce activity that is less internally coherent and less relevant to the current stimulus and task conditions.

Is there any evidence for NMDA-receptor malfunction in autism? Very little is known about its pathophysiology, but two main neurotransmitter systems have been implicated: the serotonergic and the glutamatergic. Genetic linkage studies indicate that one of the susceptibility genes for autism encodes a serotonin receptor protein, and increased serotonin (5HT) levels have been found in both blood and urine (reviewed in Gerlai & Gerlai 2003). Risperidone is a potent 5HT-2A receptor antagonist and has been reported to ameliorate some autistic symptoms (Hunsinger et al. 2000). In mice, serotonin (5HT)-2A receptor antagonists are effective in counteracting the psychotomimetic effects of NMDA (Carlsson et al. 1999). Based in part on the similarity of these psychotomimetic effects to autism, Carlsson (1998) hypothesized that autism is a hypoglutamatergic disorder that involves the underactivity of NMDA-receptors and that may therefore be treatable with agents acting on the glycine site that modulates their activity, just as Javitt (2003) proposed for schizophrenia. Though still speculative, Carlsson's hypothesis clearly merits further investigation and development.

Any similarities between the neurochemical disorders in autism and schizophrenia must not be allowed to obscure their clear differences, however. The remitting and relapsing time course of schizophrenia and its good response to neuroleptics contrasts with the enduring and treatment-resistant character of autism, indicating that the underlying malfunction in autism has more profound and lasting effects on brain development. Furthermore, the above suggests that serotonin-NMDA interactions may be particularly important for autism. This contrasts with schizophrenia, where dopamine-NMDA interactions seem more important. Any such contrast is not absolute, however, as both classes of interaction are probably relevant to both disorders to some extent.

R4. The importance of individual differences

In the above we have referred to aging, autism, and schizophrenia as though they were essentially homogeneous, with no important internal variation. This may be misleading. It is better to think of each subject at any time as impaired to a greater or lesser extent on each of a number of basic aspects of mental function. From this perspective the goal is to discover the physiological bases for each of those aspects and their malfunctions. This way of looking at the problem of heterogeneity can be related to the notion of subtypes but is not equivalent to it.

There is clear evidence for distinct dimensions of variation within each of the disorders. The distinct dimensions of reality distortion, negativity, and cognitive disorganization in schizophrenia were outlined in section 1 of the target article, where we made clear that we were primarily concerned with the latter. In relation to aging, the evidence clearly shows that variance both within and between sub-

jects increases with age, and to account for this Rabbitt (1993) concluded that there are several different patterns of cognitive aging. In relation to the balance between local and global processing, Massman et al. (1993) found pronounced dissociations in performance in a global-local task among Alzheimer's disease subgroups. Those with greater impairment in verbal than spatial skills had particular difficulties in processing the local forms, whereas patients who had greater spatial than verbal impairment exhibited deficits in processing the configural (global) forms.

As knowledge of the various forms of autism has increased they have become known as autistic spectrum conditions. This suggests that it is only severity on a single dimension that varies, however, and there are at least three distinct areas of mental function on which severity of impairment can vary: social interaction, communication, and cognitive coherence. Emphasis on this multidimensionality may help explain puzzling and unpublished findings from five studies of the context-sensitivity of size perception in autism that have been conducted at Stirling. The studies have used various tests based on the Ebbinghaus illusion, and include rigorous psychophysical paradigms that unequivocally provide sensitive and specific measures of context-sensitivity (Phillips et al. 2003). The first three of these studies showed that, in accord with Happé (1997), autistic subjects as a group are less context-sensitive, and thus they performed significantly better than controls. When parents of the autistic subjects were also studied, they too showed significantly reduced context-sensitivity. The last two studies found no such differences, however. One of these studied university students who scored either high or low on the Autistic Quotient questionnaire (Baron-Cohen et al. 2001). There was no sign of low context-sensitivity in those with high autistic quotient scores, even though those subjects did have relative lengths of the second and fourth fingers indicative of high prenatal testosterone. Furthermore, a subject diagnosed with Asperger's syndrome, and who clearly has difficulties with social interaction, showed significantly greater context-sensitivity than the mean for students as a whole. Further analysis of the Stirling studies that did find reduced context-sensitivity in autism clearly shows that the group difference was due to just a subset of autistic subjects, with the remainder showing normal effects of context. Our current working hypothesis is therefore that impairments of social interaction, and possibly also of communication, can occur independently of any impairments in cognitive coordination, even though impairments in all three aspects of mental function often co-occur. One possibility is that malfunctions of a "mind-reading" module can occur independently of any general deficit in cognitive coordination, but widespread deficits in cognitive coordination can also occur, and have particularly strong effects on "mind reading" processes because they tend to rely strongly on coordinating interactions.

R5. Conclusion

In brief, we can build on the title of **Bertone et al.**'s commentary to state our conclusion: cognitive decline in aging, autism, and schizophrenia have similar but different perceptual consequences, and similar but different pathophysiological bases.

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The letter "r" before author's initials stands for Authors' response to CC references

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Commentary on **J. D. Smith, W. E. Shields & D. A. Washburn (2003). The comparative psychology of uncertainty monitoring and metacognition. BBS 26(3): 317–339.**

Abstract of the original article: Researchers have begun to explore animals' capacities for uncertainty monitoring and metacognition. This exploration could extend the study of animal self-awareness and establish the relationship of self-awareness to other-awareness. It could sharpen descriptions of metacognition in the human literature and suggest the earliest roots of metacognition in human development. We summarize research on uncertainty monitoring by humans, monkeys, and a dolphin within perceptual and metamemory tasks. We extend phylogenetically the search for metacognitive capacities by considering studies that have tested less cognitively sophisticated species. By using the same uncertainty-monitoring paradigms across species, it should be possible to map the phylogenetic distribution of metacognition and illuminate the emergence of mind. We provide a unifying formal description of animals' performances and examine the optimality of their decisional strategies. Finally, we interpret animals' and humans' nearly identical performances psychologically. Low-level, stimulus-based accounts cannot explain the phenomena. The results suggest granting animals a higher-level decision making process that involves criterion setting using controlled cognitive processes. This conclusion raises the difficult question of animal consciousness. The results show that animals have functional features of or parallels to human conscious cognition. Remaining questions are whether animals also have the phenomenal features that are the feeling/knowing states of human conscious cognition, and whether the present paradigms can be extended to demonstrate that they do. Thus, the comparative study of metacognition potentially grounds the systematic study of animal consciousness.

Let's not forget about sensory consciousness

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Abstract: The metacognitive stance of Smith et al. (2003) risks ignoring sensory consciousness. Although Smith et al. rightly caution against the tendency to preserve the uniqueness of the human mind at all costs, their reasoned stance is undermined by a selective association of consciousness with high-level cognitive operations. Neurobiological evidence may offer a more general, and hence more inclusive, basis for the systematic study of animal consciousness.

Smith et al. (2003) say "we invite colleagues to take seriously the claims of a hundred years of cognitive scientists who noted that *the highest levels of information processing and particularly consciousness* present themselves when difficulty, complexity, and indeterminacy are encountered" (■■■, emphasis added). It may well be true that environmental complexity evokes complex cognitive responses, but it is entirely another matter to assume that these are the only situations in which consciousness may be involved. Conscious events can have both sensory content, relating to entities in the world, and metacognitive content, relating to the contents of sensory consciousness or other mental contents. Metacognitive conscious content therefore assumes sensory consciousness, but the converse is not true: sensory content need not be overlain by metacognitive content in order to constitute a conscious event.

Smith et al. focus on the (metacognitive) states of uncertainty elicited by the discrimination tasks they cite, overlooking the fact that these discriminations also involve sensory phenomenal features. Presupposing that consciousness is grounded in metacognition, they can then claim that "the comparative study of metacognition potentially grounds the systematic study of animal consciousness" (■■■).

Such a "metacognitive stance" implausibly assumes that metacognition arose in evolution before, or at least in parallel with, sensory consciousness. The phylogeny of consciousness cannot, however, be restricted to a history of metacognitive adaptations. Consciousness may have been good for many things, such as adaptively linking current perceptual categorizations to a previous individual history (Edelman 1989), distilling a stable representation of the external world from an enormously variable succession of sensorimotor transformations (Merker, in press), or mobilizing and integrating otherwise separate and independent brain functions by enabling "global access" of conscious information throughout the nervous system (Baars 1988). Tononi and Edelman (1998) have proposed that the occurrence of one particular conscious state over billions of others is *informational,* in the specific sense of a reduction in uncertainty. These examples represent just a small sample of the current literature.

Relinquishing the strong metacognitive stance of Smith et al. reveals new opportunities for grounding the systematic study of consciousness in animals (Edelman et al., in press; Seth et al., in press). One such opportunity is provided by neurobiological evidence for sensory consciousness.

Physiologically, two fundamental facts stand out about sensory consciousness. First, there is the finding, dating back to Hans Berger in 1929, that waking consciousness seems to be associated with low-amplitude, irregular gamma-range activity in cortex (John et al. 2001). Conversely, unconscious states like deep sleep, coma, general anesthesia, and epileptic states of absence show slow-wave, high-amplitude, and more regular voltages. Conscious perceptual input also appears to involve greater spatial coherence in gamma activity than matched input that does not become conscious (Srinivasan et al. 1999).

The second fundamental fact is that sensory consciousness seems to be distinctively associated with the thalamus and cortex. Damage to the brainstem, including the thalamus, can abolish the state of consciousness, and damage to posterior cortex appears to delete specific conscious features such as color vision, visual motion, the perception of objects and faces, and the like. No other brain areas show these effects when damaged.

These basic facts are unlikely to be limited to humans. For one thing, posterior cortex is structurally and functionally conserved across a wide range of mammalian species (Rosa 1999). Thalamic midline and intralaminar nuclei are also widely conserved and have been associated with conscious events in both humans and nonhuman animals (Ysbrand et al. 2000). Furthermore, gamma-range coherence has been observed in diverse species, including rabbits, rats, cats, and monkeys (see Bragin et al. 1995; Bressler et al. 1993). Finally, disturbing the integrity of either the thalamocortical system or gamma coherence in animals leads to changes in both conscious state (e.g., coma; Bogen 1995) and conscious perception (Cowey & Stoerig 1995).

In summary, it should not be assumed that metacognition is necessary for consciousness. It is likely that metacognitive adaptations played an important role in the evolution of human consciousness. However, the assumption that such adaptations are required for the emergence of consciousness in general occludes a great deal of evidence suggesting both the early emergence of sensory consciousness in evolution and its presence in many extant species. Smith et al. are quite right to warn us away from the tendency to preserve the uniqueness of the human mind, but their

In 1st Column:

Jonathan, please check quote against Smith et al. T.A. in printed version of BBS 26(3), 2003 and insert section and paragraph numbers for quote here. Thanks. – S.M.

Jonathan, please check quote against T.A. original and add section and paragraph numbers here. – S.M.

In 2nd Column

Query Seth: reference list gives year 2002 for this reference – which date is correct, 2000 or 2002? Correct in text or in CC reference list, accordingly. – S.M.

own metacognitive stance leads them back toward this very mistake.

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