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 a 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-

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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-

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