Construct Validity of the Animal Latent Inhibition Model of Selective Attention Deficits in Schizophrenia

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Latent inhibition (LI) is demonstrated when a previously unattended/inconsequential stimulus is less effective in a new learning situation than a novel stimulus. In rats and humans, LI is reduced by dopamine agonists and increased by dopamine antagonists. In addition, LI is attenuated in actively psychotic schizophrenia patients, thus conferring strong predictive validity to the animal LI preparation for schizophrenia. However, the validity of the attentional construct in the LI model of schizophrenia dysfunction depends on confirming two assumptions: that animal and human LI share a common process, and that the process is related to selective attention. Evidence to support both assumptions is presented, followed by a description of a conditioned attention theory that emphasizes the role of initial levels of attention elicited by repeated relevant and irrelevant stimuli, and the differences between these levels in schizophrenia and normal groups.

Keywords: Schizophrenia/schizotypal/dopamine/latent inhibition/attention/construct validity/predictive validity/animal model

Introduction

Latent inhibition (LI) is defined as poorer evidence of learning for a stimulus that previously was presented without consequence, as compared to a novel or previously attended stimulus. The present article puts forth the evidence and arguments for the validity of the attentional construct in animal LI models of schizophrenia. The introduction briefly reviews animal models of psychopathology, in general, and more specifically, of schizophrenia. Following a description of the LI paradigm, evidence for the predictive validity of LI for schizophrenia is offered. To support the validity of the attentional construct, evidence for the similarities of animal and human LI and for the modulation of LI by attentional processes is described. The final sections delineate the operation of attentional processes in normal LI and describe the nature of its dysfunction in schizophrenia.

Construct Validity of Animal Models of Human Psychopathology. Construct validity refers to the degree to which a set of related procedures generates data that reflect the essential process that those procedures purport to assess. As such, there are two critical aspects of construct validity, the relationships of the manipulations (independent variables) and of the measurements (dependent variables) to the theoretical hypotheses that are being tested. Consequently, one cannot talk about the construct validity of a particular animal model of psychopathology without first specifying the core process that is postulated to be common to the model and to the disorder.

Animal models of human psychopathology have had a prominent role in research, particularly in regard to schizophrenia (e.g., Gray 1998; Lipska and Weinberger 2000; Moser et al. 2000; Weiner et al. 2000; Kilts 2001). In general, there is agreement that a valid animal model of a disease should be reflected in terms of etiology, treatment, and underlying neurophysiological processes (e.g., McKinney and Bunney 1969), to which, at least for psychopathological states, one should add “underlying psychological constructs.”

Most animal models of schizophrenia emphasize the conditions that produce behavioral effects that are purportedly related to schizophrenia. The induction conditions have included neonatal hippocampal lesions (e.g., Lipska et al. 2002), prenatal stress (e.g., Shaley and Weiner 2001), postnatal stress (e.g., Shaley et al. 1998), early handling (e.g., Peters et al. 1991), maternal separation (e.g., Ellenbroek and Cools 1995), and early social isolation (e.g., Wilkinson et al. 1994). These treatments are chosen because they are stressful and they engage dopamine agonistic processes (e.g., Salamone et al. 1997). According to the still popular dopamine hypothesis of schizophrenia (e.g., Kapur and Mamo 2003), the positive symptoms of that disorder (e.g., delusions, hallucinations, disorganized thought) are based on hyperactivation of the mesolimbocortical dopamine system (e.g., Carlsson 1988; Carlsson and Carlsson 1990). Thus, for example, early social isolation results in an increase in dopamine transmission in the ventral striatum (Wilkinson et al. 1994). Notably, more recent models of disrupted LI and schizophrenia relate to biphasic dopamine dysregulation and both positive and negative symptoms (Weiner 2003).
Although there may be some general agreement as to the rationale for the procedures used to induce schizophrenia-like behaviors in animals, there is less of a consensus as to which behaviors best characterize schizophrenic symptoms. In most cases, the behavioral tests of manipulation effectiveness have been LI, prepulse inhibition of the acoustic startle response, or social interaction. Of these, for reasons described below, LI tests offer the strongest case for providing data relevant for a theoretically valid model for schizophrenia-like deficits in animals, particularly because the neural substrates of LI, disruptions of which have been related to schizophrenia, have been the subject of intense investigation (for recent reviews, see Gray 1998; Weiner 2000; Schmajuk 2001).

**LI.** When an organism is repeatedly exposed to a stimulus that is not followed by a significant consequence, the stimulus subsequently becomes less effective, as compared to a novel stimulus, in the acquisition/performance of a new association. This negative transfer phenomenon, LI, has been demonstrated in a wide variety of learning paradigms and in many different species (for a review, see Lubow 1989). The ubiquitous nature of LI suggests that it has an adaptive significance. Indeed, it would appear that LI represents a biasing of the organism to more fully process new inputs than older, unimportant ones. As such, it helps preserve limited attentional resources, providing a defense against processing overload. Indeed, it is this characterization of LI, with its focus on promoting selective attention, that provides a link to schizophrenia (for reviews, see, e.g., Lubow and Gewirtz 1995; Gray 1998; Weiner 2000).

That LI has become a topic of interest in the human literature is in part because of an old behaviorist tradition, the goal of which was to demonstrate that basic learning processes uncovered at the level of the lower animal also would be found in humans. Such continuity has been demonstrated for LI, albeit with qualifications, because in most human LI studies the preexposed stimulus is presented while the subject is engaged in another task (for a review of such masking tasks, see Lubow and Gewirtz 1995). Although continuity by itself might be of little interest, the fact that animal LI was believed to be modulated by attentional processes (Lubow 1973, 1989; Mackintosh 1975; Wagner 1978; Pearce and Hall 1980) led to the use of LI procedures to study human pathologies that are characterized by attentional dysfunctions, particularly schizophrenia (e.g., Lubow 1989; Weiner 1990; Gray et al. 1991). Indeed, such research is based on the assumption that LI effects in animals and humans are governed by the same attentional processes. In an explicit statement of this position, Lubow (1989) proposed that the attentional mechanism that is responsible for LI in normal subjects is dysfunctional in schizophrenia patients.

**Predictive Validity of LI Measures for Schizophrenia.** Independent of the construct validity of the animal LI attentional model of schizophrenia, there is strong evidence for the predictive validity of the LI procedure. Thus, amphetamine, an indirect dopamine agonist, which by itself produces positive symptoms of schizophrenia in normal subjects (e.g., Ellinwood 1967; Zahn et al. 1981) and exacerbates such symptoms in schizophrenia patients (e.g., Angrist et al. 1980; Sato et al. 1992), attenuates LI in rats (e.g., Weiner et al. 1984, 1988). On the other hand, nonselective dopamine-receptor antagonists such as chlorpromazine and haloperidol, effective neuroleptics, reverse this attenuation (e.g., Solomon et al. 1981; Weiner and Feldon 1987). Relatedly, in normal human subjects, low doses of amphetamine attenuate LI (e.g., Gray et al. 1992b; Swerdlow et al. 2003), while chlorpromazine produces a super-LI effect (McCartan et al. 2001), as do low doses of haloperidol (Williams et al. 1996, 1997) but not high doses (Kumari et al. 1999; also see Williams et al. 1998; McCarten et al. 2001). This relationship between haloperidol dosage and LI is similar to that in rats and parallels the drug’s therapeutic effectiveness for treating schizophrenia (Baldessarini et al. 1988; Van Putten et al. 1990).

The correspondence between dopamine agonists that produce psychotic-like effects in humans and reduce LI, and dopamine antagonist neuroleptics that counteract effects of dopamine agonists and produce super LI, also can be found with atypical antipsychotic drugs such as clozapine (e.g., Moran et al. 1996), olanzapine (e.g., Gosselin et al. 1996), remoxipride (Trimble et al. 1997; also see Nadal 2001), and risperidone (e.g., Alves et al. 2002), all of which, in rats, produce the expected increase in LI, or reversal of the LI-reducing effects of indirect dopamine agents (for reviews, see Moser et al. 2000; Weiner 2000; Tschentke 2001). Indeed, effective dosages of most clinically effective neuroleptics are similar to those that enhance LI (Dunn et al. 1993).

As one might predict from the above, nonmedicated acute schizophrenia patients show reduced LI compared to normals (Baruch et al. 1988a; Gray et al. 1992a, 1995; Rasle et al. 2001; Sitskoorn et al. 2001; but see Swerdlow et al. 1996; Williams et al. 1998). As opposed to this, chronic medicated schizophrenia patients and, of course, normals exhibit LI. Although most studies report no difference in LI between these latter two groups (Lubow et al. 1987; Baruch et al. 1988a; Leumann et al. 2002), super-LI effects have been reported for chronic medicated schizophrenia patients with high levels of negative symptoms (Rasle et al. 2001), particularly when combined with low levels of positive symptoms (Cohen et al. 2004).

All of the above studies used variations of the LI procedure developed by Ginton et al. (1975), with correct responses as the dependent variable. Importantly, unlike with most other tests, with the Ginton et al. procedure, the attenuated LI that characterizes schizophrenia is a result
of better learning in the schizophrenia stimulus-preexposed groups as compared to the normal control groups. Thus, the LI procedure avoids a major failing of other preparations, which yield performance deficits of limited diagnostic value because of their lack of specificity.

Nevertheless, several LI-schizophrenia experiments have used other dependent measures, including electrodermal responding (GSR; Vaitl et al. 2002), event-related potentials (ERP; Guterman et al. 1996; Kathmann et al. 2000), and response times (RTs; Lubow et al. 2000a; RTs were also supplementary measures in the ERP and GSR studies). The results of the LI experiments with schizophrenia patients are summarized in table 1.

The study of the relationship between LI and schizophrenia has been extended to healthy populations that are differentiated on the basis of scores on tests of schizotypality and psychosis proneness. The rationale for conducting experiments with psychometrically identified schizotypal subjects is based on evidence from family studies that indicate that the genetic vulnerability to schizophrenia may be manifested in nonpsychotic individuals as a schizophrenia-like personality (Gottesman and Shields 1982; Kendler et al. 1993; Nuechterlein et al. 2002). This vulnerability has been called schizotypy (Meehl 1962; Claridge and Broks 1984) or psychotic proneness (Chapman et al. 1980). Investigating cognitive dysfunctions and schizotypal traits in healthy groups has the advantage of isolating the predisposition to schizophrenia from possible confounding factors, such as hospitalization, medication, and social stigma (Mednick and McNeil 1968).

As would be expected from the preceding discussion, otherwise normal high psychotic-prone/schizotypal subjects exhibit reduced LI compared to low psychotic-prone subjects (e.g., Baruch et al. 1988b; Lipp and Vaitl 1992; Lubow et al. 1992, 2001; De la Casa et al. 1993; Lipp et al. 1994; Braunstein-Bercovitz and Lubow 1998a; Braunstein-Bercovitz 2000; Gray et al. 2002; Lubow and De la Casa 2002; for a review, see Braunstein-Bercovitz et al. 2002). Relatedly, a recent study has reported the absence of a significant LI effect in both high and low schizotypals who were first degree relatives of chronic schizophrenia patients, who themselves did not show LI (Serra et al. 2001).

In summary, attenuated LI in acute, nonmedicated schizophrenia patients as well as high schizotypal normals, and the pharmacological correspondence between the effects of drugs on LI and the efficacy of these same drugs in ameliorating the symptoms of schizophrenia, attest to the predictive validity of the LI-schizophrenia relationship.

Construct Validity of the Animal Attentional Model of LI for Schizophrenia

Dysfunctional attention, particularly distractibility, has long been recognized as a prime candidate for explaining certain features of schizophrenic behavior, particularly positive symptoms (Kraepelin 1919, p. 5). Although Bleuler’s (1911) description emphasized disorders of thought, “the loosening of associative threads,” modern accounts portray such behaviors primarily as being a consequence of distraction (e.g., Chapman and Chapman 1987). Indeed, today many writers regard a deficit in selective attention as one of the core psychological aspects of schizophrenia (e.g., Nuechterlein and Dawson 1984; Braff 1993; Gray 1998).

Although it is apparent that a selective attention disorder, as represented by high distractibility, is central to schizophrenia, the role of attention in LI is more controversial and requires some explication. This is particularly true in light of recent claims that attentional theories of LI are not adequate to explain certain LI effects, particularly the recovery of conditioning with long retention intervals and the disruption of LI when the test context is different from the preexposure context (e.g., Bouton et al. 1999; Escobar et al. 2002b). In addition, because the procedures for inducing LI in animals and humans are very different (see below), it has been claimed that the LI in the two groups reflects two different phenomena and processes. The construct validity of the animal attentional LI model for schizophrenia, then, depends on verifying two assumptions: that animal and human LI represent the operations of the same underlying process, and that that process is governed by attentional variables.

Evidence for the Similarities of Animal and Human LI. Although there are hundreds of animal LI studies, the number of human LI experiments is quite small. Table 2 lists the various preparations that have been used in animal and human LI experiments. As can be seen, the two sets of experiments differ on two counts: (1) the type of learning assessed in the test phase, associative learning in animals and rule learning in humans (with a few exceptions); and (2) in general, three-stage preparations for animal experiments versus two-stage preparations for human experiments. All LI experiments with schizophrenia patients and schizotypal normals are two-stage, and all but one have used a rule learning test.

Masking. In addition to the differences described above, animal and human LI procedures differ in regard to the use of a masking task. Briefly, human LI appears to require that the preexposed stimulus be presented while the subject is engaged in another task (masking). Such a procedure is not overtly employed in animal LI studies. Because the issue of masking has raised the strongest argument against the process identity of animal and human LI, I will describe a typical masked LI experiment, one that was used with schizophrenia patients (Lubow et al. 1987).

There are two groups, stimulus preexposed (PE) and not preexposed (NPE). During the preexposure stage, both groups listen to and attend to a continuous stream
Table I. LI studies with schizophrenia patients as a function of dependent variable, diagnosis, gender, medication, and magnitude of LI in the schizophrenia groups relative to the healthy control groups

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Dependent variable</th>
<th>Group</th>
<th>Males/females</th>
<th>Medicated</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baruch et al. (1988)</td>
<td>Correct R</td>
<td>Control</td>
<td>24/29</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute schizophrenia</td>
<td>18/8</td>
<td>Yes</td>
<td>Reduced LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic schizophrenia</td>
<td>16/11</td>
<td>Yes</td>
<td>LI</td>
</tr>
<tr>
<td>Cohen et al. (2004)</td>
<td>RT visual search</td>
<td>Control</td>
<td>16/14</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizophrenia</td>
<td>24/6</td>
<td>Yes</td>
<td>LI</td>
</tr>
<tr>
<td>Gray et al. (1992a)</td>
<td>Correct R</td>
<td>Control</td>
<td>10/10</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute schizophrenia</td>
<td>12/4</td>
<td>No</td>
<td>No LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic schizophrenia</td>
<td>12/4</td>
<td>Yes</td>
<td>LI</td>
</tr>
<tr>
<td>Gray et al. (1995)</td>
<td>Correct R³</td>
<td>Control</td>
<td>7/6</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute schizophrenia</td>
<td>2/4</td>
<td>No</td>
<td>Reduced LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic schizophrenia</td>
<td>3/4</td>
<td>No</td>
<td>LI</td>
</tr>
<tr>
<td>Guterman et al. (1996)</td>
<td>RT and ERP</td>
<td>Control</td>
<td>8/6</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizophrenia</td>
<td>11/3</td>
<td>Yes</td>
<td>See text</td>
</tr>
<tr>
<td>Kathmann et al. (2000)</td>
<td>RT and ERP</td>
<td>Control</td>
<td>9/11</td>
<td>—</td>
<td>See text</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute schizophrenia</td>
<td>12/4</td>
<td>Yes</td>
<td>See text</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Remitted schizophrenia</td>
<td>8/8</td>
<td>Yes</td>
<td>See text</td>
</tr>
<tr>
<td>Leumann et al. (2002)</td>
<td>Correct R</td>
<td>Control</td>
<td>13/5</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizophrenia</td>
<td>8/2</td>
<td>Yes</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizophrenia</td>
<td>11/1</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td>Lubow et al. (1987)</td>
<td>Correct R</td>
<td>Control</td>
<td>20/28</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paranoid schizophrenia</td>
<td>18/2</td>
<td>Yes</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nonparanoid schizophrenia</td>
<td>12/7</td>
<td>Yes</td>
<td>LI</td>
</tr>
<tr>
<td>Lubow et al. (2000a)</td>
<td>RT visual search</td>
<td>Control</td>
<td>15/17</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizophrenia</td>
<td>17/15</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td>Rasce et al. (2001)</td>
<td>Correct R</td>
<td>Control</td>
<td>14/26</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute schizophrenia</td>
<td>23/12</td>
<td>Yes</td>
<td>No LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic schizophrenia</td>
<td>21/9</td>
<td>Yes</td>
<td>Super LI</td>
</tr>
<tr>
<td>Serra et al. (2001)</td>
<td>Correct R</td>
<td>Control</td>
<td>13/14</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic schizophrenia</td>
<td>15/6</td>
<td>Yes</td>
<td>No LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nonschizotypal</td>
<td>6/13</td>
<td>—</td>
<td>No LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizotypal³</td>
<td>9/13</td>
<td>—</td>
<td>No LI</td>
</tr>
<tr>
<td>Swerdlow et al. (1996, experiments 1 and 2)</td>
<td>Correct R</td>
<td>1 control</td>
<td>63⁶</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 acute schizophrenia</td>
<td>24⁶</td>
<td>No⁶</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 chronic schizophrenia</td>
<td>40⁶</td>
<td>Yes</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 control</td>
<td>44⁶</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 acute schizophrenia</td>
<td>18⁷</td>
<td>No⁷</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 chronic schizophrenia</td>
<td>33⁷</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td>Vaitl et al. (2002)</td>
<td>GSR, RT³</td>
<td>Control</td>
<td>8/8</td>
<td>—</td>
<td>LI⁸</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizophrenia</td>
<td>8/8</td>
<td>No</td>
<td>No LI⁸</td>
</tr>
<tr>
<td>Williams et al. (1998)</td>
<td>Correct R</td>
<td>Control</td>
<td>39/34</td>
<td>—</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizophrenia</td>
<td>14/9</td>
<td>No</td>
<td>LI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizophrenia</td>
<td>28/6</td>
<td>Yes</td>
<td>Reduced LI</td>
</tr>
</tbody>
</table>

Note.—ERP = event-related potential; GSR = electrodermal responding; LI = latent inhibition; RT = response time.

¹Overall LI effect was due to super LI in patients with a combination of low-positive and high-negative symptoms. The other three symptom combination groups did not have significant LI.

²Patients were tested within 14 days of start of neuroleptics medication and were drug-free at least 6 months prior to that.

³Within-subjects design.

⁴Defined as illness duration of less than 12 months.

⁵First degree relatives divided into low and high schizotypals.

⁶As reported by Swerdlow et al. (1996) in their figure 1 caption.

⁷As reported by Swerdlow et al. (1996) in their figure 3 caption.

⁸GSR, conclusions are compromised by absence of interaction with groups.
of meaningless syllables. The masking task consists of counting the number of times that the list repeats itself. For the PE group, a number of short bursts of white noise are presented concurrently with the syllables. The NPE group hears only the syllables. In the test stage, the syllables continue to be presented to both groups. However, now there is a numerical counter in front of the subject, and the subject is told to press a button when he or she thinks the counter is about to change. In fact, the counter change is immediately preceded by the white noise. If the subject learns the connection, indicated by a key press immediately before the counter changes, he or she is reinforced.

The role of the masking task is to divert attention from the white noise. In fact, at least for rule learning, LI is generated only with the use of a masking task (Ginton et al. 1975; Braunstein-Bercovitz and Lubow 1998a). The masking task requirement for human LI is directly related to our interpretation of the attentional dysfunction in schizophrenia. If schizophrenia patients are highly distractible, they, unlike healthy controls, will continue to allocate attention to the irrelevant white noise during the preexposure phase. As a consequence, the schizophrenia PE group, as compared to the healthy control PE group, will begin the test phase with a higher level of attention allocated to the previously irrelevant stimulus, with the result that the former will learn the new association faster than the latter.

The fact that LI requires a masking task in humans but not animals raises a question in regard to the similarities of processes. However, animal LI procedures may contain an inherent masking task. When the animal is placed in the apparatus for the first time during the preexposure stage, it actively explores the new environment, as can be seen by locomotion, rearing, and sniffing behaviors. It is while the animal is engaged in these behaviors that the to-be-conditioned stimulus is presented. In other words, exploratory behavior may serve as a masking task for the rat (for an alternative explanation of the animal-human LI differences, see below).

**Variables having the same effects on animal and human LI.** The construct validity of the animal attentional LI model of schizophrenia would be compromised by significant differences in outcomes between animal and human LI experiments that are attributable to either two versus three stages, masking versus nonmasking, or rule learning versus associative learning. However, in practice, the three issues are confounded: most human studies are two stage, are masked, and use rule learning tests, and most animal studies are three stage, are unmasked, and use associative learning tests. Consequently, if one can show that, in spite of the differences described above, the same variables have the same effects in both the human and the animal LI preparations, then the argument for different underlying processes is considerably weakened. Indeed, not only does irrelevant stimulus preexposure result in poorer performance on a subsequent learning task in both animal and human LI preparations, but, as described below, the same variables produce the same effects in animals and humans.

**Context manipulations.** Among the variables that consistently have been shown to modulate LI, and perhaps the most important theoretically, is that of context. In LI studies, the context, unless specifically an experimental variable, remains the same in the stimulus preexposure and test phases. However, if the context is changed from the preexposure to the acquisition/test phase, then LI is severely attenuated, in both animals (e.g., Lubow et al. 1976a; Lovibond et al. 1984) and humans (e.g., Zalstein-Orda and Lubow 1995; Gray et al. 2001). In addition, for context and stimulus preexposure to be effective in producing LI, the two must be preexposed conjointly, again in both animals (e.g., Hall and Channell 1986) and humans (Zalstein-Orda and Lubow 1995).

**Other variables.** LI increases as a function of the number of stimulus preexposures (animals: for a review, see Lubow 1989, pp. 59–63; humans: e.g., Lipp et al. 1992; De la Casa and Lubow 2001; but see Lipp 1999), and it is attenuated when a second stimulus is paired with the preexposed stimulus (animals: e.g., Lubow et al. 1976b; Matzel et al. 1988; humans: Lubow et al. 1975), or when an additional stimulus is presented toward the end of the preexposure session (animals: Lantz 1973; humans: Braunstein-Bercovitz and Lubow 1998b). LI is reduced by stress (animals: Hellman et al. 1983; Shalev et al. 1998; humans: Braunstein-Bercovitz et al. 2001). Finally, as already described, LI is reduced by dopamine

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**Table II.** Learning paradigms used to study LI in animals and humans

<table>
<thead>
<tr>
<th>Humans</th>
<th>Rats/rabbits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conditioned suppression</td>
<td>Conditioned suppression</td>
</tr>
<tr>
<td>Conditioned taste aversion</td>
<td>Conditioned taste aversion</td>
</tr>
<tr>
<td>—</td>
<td>Conditioned avoidance</td>
</tr>
<tr>
<td>Electrodermal (GSR)</td>
<td>—</td>
</tr>
<tr>
<td>Classical defensive conditioning (eye blink)</td>
<td>Classical defensive conditioning (eye blink, pinna response, leg flexion, freezing)</td>
</tr>
<tr>
<td>—</td>
<td>Appetitive conditioning</td>
</tr>
<tr>
<td>Discrimination learning</td>
<td>Discrimination learning</td>
</tr>
<tr>
<td>Affective/evaluative conditioning</td>
<td>—</td>
</tr>
<tr>
<td>Rule learning</td>
<td>—</td>
</tr>
</tbody>
</table>

*Note.—* GSR = electrodermal responding; LI = latent inhibition.
agonists such as amphetamine (animals: e.g., Weiner et al. 1984, 1988; humans: e.g., Gray et al. 1992b; Salgado et al. 2000), and it is increased by dopamine antagonists such as haloperidol (animals: Weiner and Feldon 1987; Christison et al. 1988; humans: Williams et al. 1996, 1997).

In summary, in spite of the differences in procedures, the fact that so many different variables have the same effects on animal and human LI supports the contention that the two sets of data represent similar phenomena governed by the same underlying processes.

Evidence That LI Is Modulated by Attentional Processes. The construct validity of the animal attentional LI model of schizophrenia depends on showing that normal LI, in both animals and humans, is governed by attentional processes and that disrupted LI in schizophrenia can be attributed to some dysfunction in those same attentional processes. The earliest theories of LI were, indeed, attentional. They asserted that LI is due to a stimulus-specific decline of attention as a function of repeated irrelevant stimulus preexposures, one consequence of which is that stimulus preexposure affects the subsequent associability of that stimulus (e.g., Mackintosh 1975; Lubow et al. 1976b, 1981; Pearce and Hall 1980; Wagner 1981). Because more recent models of LI, based on retrieval/competition concepts, have questioned the attentional-associability explanations of LI (e.g., Miller and Matzel 1988; Bouton et al. 1999; Denniston et al. 2001), it seems appropriate to review the evidence for such involvement.

LI is reduced by manipulations that maintain or restore attention. If LI is the result of repeated nonreinforced stimulus preexposures that produce a decline in attention to that stimulus, then any manipulation that maintains or restores attention to that stimulus should reduce the magnitude of LI. Such LI attenuations are obtained in rats when a second, neutral, stimulus is paired with the target stimulus on each of its preexposure presentations (Lubow et al. 1976b; Szakmary 1977), or irregularly (Matzel et al. 1988), and when a second stimulus is added to the target stimulus at the end of the preexposure session (Lantz 1973). Similar results have been obtained with humans (Lubow et al. 1975; Braunstein-Bercovitz and Lubow 1998b, experiment 2).

Another way to maintain attention to the preexposed stimulus, and thereby to preclude LI, is to omit the masking task. As already noted, the masking task ensures that the subject does not fully allocate attention to the preexposed stimulus that will subsequently become the test-phase target (see above). Indeed, with the exception of electrodermal conditioning studies, LI has not been obtained when stimulus preexposures occurred in the absence of a masking task (for reviews, see Lubow 1989; Lubow and Gewirtz 1995; but see Escobar et al. 2002a). The importance of attention during the stimulus preexposure period is also attested to by the fact that LI varies with the processing demands of the masking task (Braunstein-Bercovitz and Lubow 1998a).

There is yet one more situation that demonstrates that LI is modulated by attentional processes. In LI studies using visual stimuli, the masking task stimuli (M) are presented at the center of the computer screen, flanked by the currently irrelevant but to-be-target stimuli (T), creating a T M M T configuration (e.g., Lubow et al. 1992, experiment 2; Zalstein-Orda and Lubow 1995). Braunstein-Bercovitz and Lubow (1998b, experiment 1) showed that the peripheral position of the irrelevant preexposed stimulus is necessary to produce LI. When the preexposed stimuli were located centrally, between the masking stimuli (M T T M), LI was not obtained. These results follow from the fact that attention is allocated automatically to the preexposed stimulus because it falls on the fovea of the retina during the course of scanning the two masking stimuli, or because with such a configuration “attention forms a unitary zone that may expand to encompass multiple relevant locations, but must also include the area between them” [italics added] (Usai et al. 1995, p. 411). In either case, when the preexposed stimuli are central to the masking task stimuli, more attention will be allotted to them than if they are peripheral, thereby interfering with the acquisition of LI.

Visual search analog of LI. The attentional model of LI has been questioned on the basis of the fact that LI is attenuated when there is a change of context from preexposure to acquisition/test (e.g., Lovibond et al. 1984). Such context specificity of LI has been used to support a retrieval explanation of LI (e.g., Escobar et al. 2002b). However, the context specificity of LI also can be explained by attentional constructs, on the basis that a change of context from the preexposure to test phase creates a test setting in which a familiar stimulus is presented in a novel context. Such a situation serves to attract attention to the target stimulus, a consequence of which, according to attentional theories of LI, would be to attenuate LI. Indeed, the perceptual learning effect, namely, better learning after stimulus preexposure (for reviews, see Gibson 1969; Hall 1991), as opposed to the poorer learning that characterizes LI, has been accounted for by such an analysis (Lubow et al. 1976a) and has been tested directly in a visual search study (Lubow and Kaplan 1997). The latter study is particularly important for strengthening the claim that LI is governed, at least in part, by attentional processes.

Basic visual search LI procedure. As with the standard LI task, the visual search analog of LI has two phases, preexposure and test. In both phases, the participant has to report the presence (or location) of a unique meaningless shape among a field of identical distractors.
However, unlike the standard LI studies, which use a between-subject design with number of correct responses as the dependent variable, the visual search studies use a within-subject design and response time.

After a common preexposure phase, in which the same target and distractors appear on every trial, but in different locations, there is a test phase. On any given test trial, the target is either congruent with its status during the preexposure phase (a target in both phases, T → T), incongruent (a distractor during preexposure becomes a target in test, D → T), or novel (N). Similarly, distractors are congruent (D → D), incongruent (T → D), or novel (N) in relationship to their status in the preexposure phase. A description of a test-phase trial, then, requires two expressions, one for the target and one for the distractor. The status of the test-phase target is designated by the first term, and the status of the distractor by the second term. Thus, for example, (D → T):(T → D) indicates that the test-phase target was previously a distractor (D → T) and the test-phase distractor was previously a target (T → D).

**Visual search LI data.** The PE and NPE conditions in a standard LI procedure correspond to the (D → T): (T → D) and (N):(T → D) visual search conditions (for a detailed explanation, see Lubow and Kaplan 1997). These conditions have consistently produced LI-like effects, with response times in the PE condition being slower than in the NPE condition (Lubow and Kaplan 1997; Lubow et al. 1999, 2000a, 2000b; Gibbons et al. 2001; Kaplan and Lubow 2001; Cohen et al. 2004). In addition, as with standard LI, a change of context from preexposure to test disrupts the LI-like effect (Kaplan and Lubow 2001). Furthermore, again as with standard LI, schizophrenia patients (Lubow et al. 2000b; Cohen et al. 2004) and high-schizotypal normals (Lubow et al. 2001) show similarly modulated LI-like effects as compared to controls.

Finally, Gibbons et al. (2001) directly compared performance of the same subjects on visual search and standard LI tasks. If performance on the two tasks is governed by a common process, then one would expect a significant positive correlation between RTs from a visual search LI index (PE minus NPE) and performance of the PE group in a standard LI learning task. Such a correlation was obtained for the PE group (0.47; RT from block 1 of visual search and number of correct responses in the rule learning LI task). For subjects in the NPE condition/group, the correlation was not significant and was negative, −0.20. The correlational data, together with the data showing that similar variables have similar effects on standard LI and the visual analog of LI, substantiate the claim that the visual search procedure taps the same underlying attentional process as that involved in LI.

**Electrophysiological evidence.** It is well documented that repeated presentation of a neutral stimulus results in the diminution of one or more of the ERPs that are associated with stimulus-specific attention and its associability (e.g., contingent negative variations [CNV], N100, P300; for reviews, see e.g., Donchin et al. 1986; Pritchard 1986). Although these data are consistent with the claim that normal LI depends on the reduction of attention to the preexposed stimulus during the preexposure phase, a stronger case for that position could be made if one could find differences in the attention-related ERP indicators that are associated with similar differences in LI. More specifically, because a failure to disattend to irrelevant stimuli is one of the fundamental characteristics of schizophrenia (e.g., Garmezy 1977; Mirsky and Duncan 1986; Anscombe 1987), the construct validity of the attentional model of LI for schizophrenia would be further enhanced if the attention-related ERP associated with repeated irrelevant stimuli were less attenuated in schizophrenia patients than controls. However, only two ERP studies have used an LI procedure with schizophrenia subjects (Guterman et al. 1996; Kathmann et al. 2000).

Although LI with RT measures was obtained by Kathmann et al. (2000) but not Guterman et al. (1996), both studies provided several significant ERP effects from stimulus preexposure. Guterman et al. (1996) reported that the increase in CNV amplitude, a widely accepted index of the extent of association between the probe and the imperative stimulus (Rohrbaugh et al. 1986), was delayed across test-trial blocks in PE as compared to NPE healthy controls. The CNV-stimulus preexposure effect was absent in the schizophrenia group, perhaps related to the group members’ inability to develop a prominent CNV at any stage, irrespective of preexposure. Although Kathmann et al. (2000) did not find any effect of stimulus preexposure on CNV amplitudes, they did not analyze their data across blocks of trials.

Both Guterman et al. (1996) and Kathmann et al. (2000) reported lower test-phase N100 amplitudes in the PE than the NPE control group. Because N100 amplitude covaries with the level of early attentional involvement with a stimulus (e.g., Naatanen 1990), the relatively low N100 amplitude in the PE group suggests that behavioral LI effects are preceded by preexposure-induced changes in the early attention recruiting stages. Guterman et al. (1996) did not find similar differences in N100 for the PE and NPE schizophrenia groups, both of which consisted of medicated outpatients. However, Kathmann et al. (2000) reported that the acute (inpatients, admitted 2–3 weeks prior to test) but not the partially remitted outpatient schizophrenia patients displayed higher amplitude N100 than the comparable NPE groups.

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1 These correlations were not reported in the original article.
P300 amplitude, which increases as a function of task relevance and stimulus novelty, is related to conscious updating of stimulus interpretation (e.g., Donchin and Coles 1988). As such, it may be the ERP component most closely related to the recognition of associations between events. In Guterman et al. (1996), the PE control group compared to the NPE control group tended to have lower test-phase P300 amplitudes across the entire session. However, the effect was significant in only the first block of trials. This pattern may be the result of habituation of P300 amplitude during the preexposure phase, or a delay in P300 enhancement in the test phase, perhaps related to retardation in the recognition of the probe.

Nature of Attentional Dysfunction in Schizophrenia

A complete theory of LI and related stimulus preexposure effects would encompass serially ordered, but overlapping, subprocesses that involve initial primitive stimulus feature encoding (Treisman 1988), a selective attention process followed by higher order stimulus property encoding, stimulus-specific conditioning of inattention, acquisition of context associations, and context-based retrieval of previously unattended stimuli (Lubow 1989; Lubow and Gewirtz 1995). Because the present article is primarily concerned with the validity of the attentional construct in the explanation of LI deficits in schizophrenia, only selective attention, which provides the basis for higher cognitive processing, will be addressed. The data to support the proposition that LI is modulated by selective attentional processes have already been presented. The next section describes how selective attention operates to create LI in normally healthy subjects and how those processes malfunction in schizophrenia patients.

Attention and LI. Although contemporary psychology has increasingly used attentional concepts in explanatory networks, there is still only limited agreement as to what constitutes attention, except for a phenomenological description and the general notion that the amount of attention allocated to a stimulus is equivalent to the amount or depth of processing that it receives. Indeed, a common assumption of animal learning theory is that the associative strength that accrues to a conditioned stimulus depends on the extent of conjoint processing it receives with the unconditioned stimulus in a central processor (e.g., Wagner 1978, 1981; Pearce and Hall 1980). If one accepts the idea that preexposure of an irrelevant stimulus reduces its ability to enter into associative relationships, then one will not be surprised to find that these theories use, either explicitly or implicitly, attentional constructs to account for LI. According to Mackintosh (1975), if a stimulus is not reinforced, it is deprived of access to the central processor. For Lubow (1989) and Pearce and Hall (1980), if a stimulus reliably predicts no consequence, the amount of processing that it receives is decreased. In Wagner’s (1978) theory, the context of stimulus preexposure “primes” the stimulus into short-term memory (STM), or into a secondary state of activation (Wagner 1981), which, in turn, reduces the amount of processing it receives when it is re-presented.

Role of the masking task. According to these theories, the attention allocated to a preexposed stimulus at the beginning of the acquisition phase is less than that given to a novel stimulus, thereby accounting for the relatively slow acquisition of associative strength by the preexposed stimulus. The difficulty in obtaining LI in adult humans in the absence of a masking task is accounted for in the same manner. When there is no masking task during preexposure, attention to the to-be-conditioned stimulus is not reduced (Lubow and Gewirtz 1995).

The role of masking in producing LI in adult humans, and its apparent irrelevance for infrahumans, gains additional clarity when one considers the distinction between automatic and controlled processing (e.g., Shiffrin and Schneider 1977). Automatic processes are pre-attentive (not available to conscious awareness), are relatively effortless and rapid, and operate in a parallel mode. Controlled processes are attention demanding, resource limited, and effortful, operating relatively slowly and serially. Species differences in LI may be explained by the assumption that animals and humans share the automatic processes of attention but that controlled processing is to be found only in humans. In addition, humans have a controlled processing bias that, when fully engaged, overrides automatic processing. This formulation allows for LI in animals without the use of a masking task, as opposed to the earlier speculation (see above).

When the preexposed stimulus is first presented in stage 1, it is novel. As such, it elicits an orienting response that acts as a “call” for controlled attentional processing (Ohman 1979). When that stimulus is repeatedly presented but not followed by an event of consequence, the call goes unanswered and the preexposed irrelevant stimulus enters the automatic processing mode. The masking task serves to engage controlled attention/processing so that the only information processing mechanism operating with respect to the currently irrelevant but to-be-relevant stimulus is automatic. In the absence of a masking task, adult humans would continuously process the irrelevant stimulus in the controlled attentional mode, a situation that would preclude the development of LI.

It is proposed, then, that distraction is characterized by a failure to shift from controlled to automatic processing under conditions of repeated irrelevant stimulation. For the schizophrenia patient, irrelevant stimuli attract attention; when repeated, the stimuli continue to be the subject of controlled processing. As a consequence, STM/conscious awareness is inundated with experimentally
familiar but phenomenally novel stimuli. Frith (1979) has described the end product of this collapse in similar terms, referring to the inability of schizophrenia patients to limit the contents of consciousness. Indeed, the positive symptoms of schizophrenia are congruent with the notion that, for schizophrenia patients, STM is “preoccupied” with irrelevant stimuli.

In short, normal LI depends on a shift in processing mode from controlled to automatic. The disrupted LI in schizophrenia patients and high-schizotypal normals, as well as in adults without a masking task, is due to a failure to shift from controlled to automatic processing. Some evidence for this contention is provided by De la Casa et al. (1993). In those conditions in which high schizotypals exhibited less LI than low schizotypals, they also had superior recall and explicit recognition memory scores for the preexposed irrelevant stimuli. This suggests that the attenuation of LI in the high- as compared to the low-schizotypal subjects was a result of increased controlled processing of the irrelevant preexposed stimuli.

**Masking task load.** The above principles were instantiated by Braunstein-Bercovitz and Lubow (1998a). They found that with a zero-load masking task, LI was not obtained, as would be expected because the absence of a masking task allows maintenance of attention to the preexposed stimulus. On the other hand, low-load conditions (judgments of letter-pair similarities/differences) produced LI, as has been repeatedly demonstrated in other adult human LI studies with relatively low-load masking conditions (e.g., Zalstein-Orda and Lubow 1995; Gray et al. 2001, 2002). Importantly, the low-load condition does not exhaust available attentional resources and therefore allows for some initial encoding of irrelevant stimulus properties, in turn to be followed by the acquisition of a stimulus–no consequence association. In the high-load condition (letter orientations were varied), LI was attenuated, as would be expected if attentional resources were exhausted, precluding any processing of the preexposed irrelevant stimulus, and thereby making it functionally novel at the test stage. Notably, the pattern of results was influenced by whether the participants were low or high schizotypals.

As shown in figure 1, with zero load (no masking), neither the low- nor the high-schizotypal group exhibited LI. With the low-load masking task, low-, but not high-, schizotypals exhibited LI, as in many other studies (for reviews, see Lubow and Gewirtz 1995; Braunstein-Bercovitz et al. 2002). The attenuation of LI in high schizotypals is congruent with the data from LI studies with amphetamine-treated healthy subjects (Gray et al. 1992b; Thornton et al. 1996; Salgado et al. 2000) and schizophrenia patients (e.g., Baruch et al. 1988a; Gray et al. 1992, 1995; Rascle et al. 2001; Serra et al. 2001; Sitskoorn et al. 2001), all of which used low-load masking conditions.

In the high-load condition, the LI effect was reversed; LI was obtained in high but not in low schizotypals. For low-schizotypal subjects, the relatively high-load masking task fully engages controlled processing, and the automatic attentional response, normally elicited during the first few presentations of the nominally irrelevant stimulus, is not elicited, thereby precluding the development of LI. For the high schizotypals, the increase in masking task load only partially uses processing resources (as was the case with normals in the low-load condition), allowing some attention to be directed to the irrelevant stimuli, thereby providing the basis for the decline of attention with repeated stimulus presentations and the subsequent acquisition of LI.

These data provide additional confirmation that attenational processes are involved in LI and that low and high schizotypals—and by extension, schizophrenia patients—differ in the operation of those processes. It appears, then, that the attainment of LI requires a certain amount of processing capacity to be devoted to the relevant *and* to the irrelevant stimuli during the preexposure phase. Furthermore, the acquisition of LI requires a diminution of attention to the irrelevant stimulus in the preexposure phase. In short, a major dysfunction in high-schizotypal normals and at least some schizophrenia subgroups (together, SZs) concerns the manner in which nominally irrelevant stimuli are processed under different levels of competition from a relevant task. When SZs are engaged in a low-load task, they allocate more free attentional
Conditioned attention theory. The above data are all compatible with the position that normal LI is modulated by an attentional process that is dysfunctional in schizophrenia. Of the many theories that adopt attentional constructs, conditioned attention theory (CAT) is the one that explicitly addresses LI (Lubow et al. 1981). In brief, CAT posits that repeated nonreinforced preexposures to a stimulus (S₁) retard the acquisition of subsequent associations to that stimulus because, during those preexposures, the subject learns not to attend to the stimulus. The theory treats inattention as a classically conditioned response, and it specifies the conditions of reinforcement that modulate that response. Furthermore, during the preexposure phase, when S₁ is followed by a second stimulus (S₂) that is in a conditioning relationship to the first one (S₁-S₂; contingent and temporally contiguous), the magnitude of the attentional response to S₁ is temporarily increased; this increase of attention is also conditionable. Finally, a minimum level of attention to a stimulus is a prerequisite for it to be able to enter into new associations. As attention to a stimulus increases, its associability also increases (for a detailed description of the theory and a review of the supporting evidence, see Lubow 1989; Lubow and Gewirtz 1995).

There have been several modifications of CAT since its original formulation (Lubow et al. 1981). First, there was acknowledgment of a stimulus encoding stage that precedes or overlaps with the acquisition of subsequent associations during preexposure. The encoding of stimulus properties is necessary to account for the stimulus specificity of the LI effect. Second, although the old CAT emphasized the level of the attentional response at the end of the stimulus preexposure phase as the factor determining LI, it is now clear that the initial level of the attentional response is also important (Braunstein-Bercovitz and Lubow 1998a, 1998b). It is not only where you are but how you got there. Thus, LI will not be attained if there is no attentional response to the early presentations of the irrelevant stimulus. The conditioned decrementing of that response is the basis for the LI effect. Third, the conditioning of inattention that accompanies the stimulus—no consequence association, in turn, becomes associated with the preexposure context, such that the subsequent elicitation of the stimulus—no consequence association depends on the presence of the preexposure context.

The above description of CAT naturally leads to the question of which process or processes are disrupted in schizophrenia. Given the sequential nature of these processes, data from the end products of typical LI experiments are not informative. An answer must await an examination of each processing stage independently. The visual search LI experiments described above were motivated by this question, and their results strongly support attentional involvement in normal LI and dysfunctional attentional processes in the disrupted LI in some schizophrenia subgroups and in high-schizotypal normals. However, they too do not reveal the juncture in the processing sequence at which normal attention breaks down. Thus, the vaunted distraction of schizophrenia patients may, indeed, account for their disrupted LI and thereby support the construct validity of the animal LI model of selective attention deficits in schizophrenia. However, the locus of the effect remains to be determined.²

Summary and Conclusions

The present article relates LI to the operation of selective attentional processes that are dysfunctional in schizophrenia. It was shown that the animal LI preparation has excellent predictive validity for schizophrenia, primarily because LI is attenuated by dopamine agonists and increased by dopamine antagonists, as well as atypical neuroleptics, both in rats and humans, and because schizophrenia patients and psychometrically identified high-schizotypal normals exhibit reduced LI. Evidence for the construct validity of the attentional model of LI for schizophrenia was provided by showing that animal and human LI share a common process that is related to selective attention. Such evidence includes the fact that the same variables—pharmacological as well as extrinsic manipulations such as context change from preexposure to test, number of stimulus preexposures, and second-stimulus presentations—have the same effects on animal and human LI.

The evidence for the involvement of selective attention processes in the acquisition of LI consists of data from a number of manipulations that relate to distraction, a major descriptive variable for schizophrenia. Thus, experimental operations that maintain or restore attention to the otherwise irrelevant preexposed stimulus (and

²The confirmation of an attentional component for LI does not preclude a contribution of retrieval/competition processes to LI, particularly in three-stage procedures. However, for reasons enumerated above, the two-stage human LI effects, and its disruption in SZs, are still best understood within the attentional framework.
therefore are distraction producing) attenuate LI; these operations include omitting the masking task, pairing the preexposed stimulus with another stimulus either throughout the preexposure session or at its termination, and using foveal placement of the irrelevant stimulus. Furthermore, manipulation of the load of the masking task during the stimulus preexposure phase, which influences the amount of attention allocated to the irrelevant stimulus, has differential effects on low and high schizophrenia. Finally, a visual search procedure that was designed to directly assess attentional processes with LI-related conditions produced LI-like effects that were attenuated in some groups of schizophrenia patients and in high-schizotypal normals.

A modified CAT was introduced to integrate the data from the LI experiments, with their extensive manipulations of attention/distraction-related variables, and the differences in LI between SZs and controls. The theory emphasizes the role of the initial level of attention elicited by an irrelevant stimulus, and its subsequent conditioned decline with stimulus repetition. It was concluded that SZs allocate more attention to irrelevant stimuli and maintain that attention over more irrelevant presentations than do normal control groups. Given all of the above, there appears to be good reason to endorse the construct validity of the animal attentional LI model of schizophrenia. As such, lest we be distracted, animal LI experimentation should continue to play an important role in uncovering the neuropsychopharmacological basis of schizophrenia.

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