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# A Test of Conditioned Blocking and Its Development in Childhood and Adolescence: Relationship to Personality and Monoamine Metabolism

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Conditioned blocking (CB) is the undermining of conditioning to a stimulus by conjoint exposure with one already associated with the unconditioned stimulus. CB is one of several tests of "learned inattention" in which performance has been found to depend on personality features of human participants and monoamine activity in animals. In Part 1, the performance of 25 healthy young adults on a new test form for demonstrating CB is described. From personality inventories and 24-hr urine samples it was proposed that CB may be correlated with extroversion and increased catecholamine utilization. In Part 2, CB was shown to be present in 4 groups of 11 participants with mean ages of 10, 14, 17 and 22 years independent of IQ, but it was least marked in the prepubertal group. No relation of performance with personality features was found. As with the adult group, CB was positively correlated with dopamine activity, but unlike the adults it was negatively correlated with noradrenalin activity. The maturation of attention-related information processing is discussed in terms of the development of limbic structures and dopaminergic versus noradrenergic function.

Learned inattention is a paradigm for studying selective attention. Two of the best-known tasks in this paradigm are latent inhibition (LI) and conditioned blocking (CB). In LI, a stimulus is presented a number of times without consequence before it functions as a conditioned stimulus (CS) requiring a response to be learned. Learning about the consequence is delayed with respect to the situation when the stimulus is a CS from the start. This delay has been attributed to the need

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to "unlearn" that the stimulus means nothing important and learn anew that it is a relevant CS—a process requiring selective attention (Mackintosh, 1975; Lubow & Gewirtz, 1995).

CB is different in that a CS is presented from the start, but similar in that an extra stimulus (CS–2), with the same consequences, is added during conditioning to the original CS (CS–1). Normally CS–2 is initially ignored as irrelevant, but as in LI, its consequences will also eventually be learned. Conditioning to CS–2 is said to be blocked by conditioning to CS–1 (Kamin, 1969; Sutherland & Mackintosh, 1971). CB has also been interpreted in terms of selective attentional mechanisms and the allocation of resources (Mackintosh, 1975; Rescorla & Wagner, 1972).

Both tasks are designed for the study of the way that mechanisms of selective information processing deal with the distinction between relevant and irrelevant stimuli; these mechanisms have been well studied with animals in experimental psychology by the aforementioned authors as well as in psychobiology (e.g., Crider, Blockel, & Solomon, 1986; Oades et al., 1987). But as humans can consciously adjust processing strategies, there have been difficulties in transposing this paradigm to normal psychology (Lubow & Gewirtz, 1995), although it holds promise for the study of psychopathological conditions in which attention disturbance is prominent (e.g., Baruch, Hemsley, & Gray, 1988a; Jones, Gray, & Hemsley, 1992; Oades, Bunk, & Eggers, 1992).

In Part 1 of this article, a learning task is described to show CB in normal young adults. There have been very few attempts to show CB in normal adult humans (e.g., Jones, Gray, & Hemsley, 1990), and no particular test form has established itself.

Part 2 describes the extent to which the same phenomenon occurs in three further groups of children and adolescents. One reason to study developmental aspects concerns the potential instability of the test form for learned inattention in humans. Lubow and Josman (1993) reported LI in a younger but not in an older group of prepubertal children. They attributed this to the participants' application of different cognitive strategies to the task. A second reason for interest reflects the heterogonic development of brain regions that may mediate the processing required for CB. For example, frontal lobe functions undergo marked development in the 9-year-old to 12-year-old age range, resulting in freedom from perseveration displayed on card-sorting tasks, and the development of planning and strategy in the 13-year-old to 15-year-old age range, as shown on the Tower of London task (Levin et al., 1991). However, hippocampal function is required for CB in animals (Rickert, Lorden, Dawson, & Smyly, 1981), and tasks requiring hippocampal function (e.g., trace conditioning or conditioned discrimination reversal) are not generally acquired in children younger than 8 years of age (Woodruff-Pak, Logan, & Thompson, 1990).

The CB test was performed as part of a larger study. As there are reports that personality type can affect performance on LI (Baruch, Hemsley, & Gray, 1988b) and CB (Jones et al., 1990) and that dopaminergic treatments in animals (Crider et

al., 1986) and man (Oades et al., 1992) can influence CB measures, the relation to performance of personality scores and indices of monoamine activity reflected in urinary excretion was examined.

Personality features represented on the extro–introversion and psychoticism–neuroticism dimensions are well known to influence both the general cognitive style and the choice of strategy in task solving (Lynn & Hampson, 1977), and learned inattention performance is no exception. Thus, LI was reported to be attenuated in normal individuals with high scores for psychoticism on the Eysenck scale (Baruch et al., 1988b) and on the Minnesota Multiphasic Personality Inventory (MMPI; De la Casa, Ruiz, & Lubow, 1993). However, this finding depended on the type of scale used (Baruch et al., 1988b), the task parameters, and the test form used (Lipp & Vaitl, 1992; Lubow, Inberg-Sachs, Zalstein-Orda, & Gewirtz, 1992). Indeed, for CB it has been tentatively suggested that attenuation is associated with neurotic features (Jones et al., 1990).

If CB is a function of the breadth of attention, and broad attention has been associated with psychoticism (Hemsley, 1988), then this feature might be expected to influence performance as claimed. However, as incidental learning is often unaffected by psychoticism (Jones et al., 1990), we are more inclined to predict an influence of the extro–introversion dimension on CB performance as seen in IQ measuring tasks (previously mentioned) and dual task conditions requiring parallel processing (as in our form of CB), where introverts are less successful (see Eysenck, 1982, pp. 124–138, for a review).

The neural systems involved in CB have generally been studied in animals. In rodents, an intact hippocampus is thought to be necessary for the expression of CB (Rickert, Bennett, Lane, & Fench, 1978; Solomon, 1977). Intact catecholamine systems involved in tuning and switching functions (Oades, 1985) are also important. Mesolimbic and mesocortical dopamine (DA) activity should be in balance. For example, the attenuation of CB by amphetamine can be prevented by acute neuroleptic treatment (Crider, Solomon, & McMahon, 1982), but chronic haloperidol-induced DA supersensitivity in subcortical regions reduced CB (Crider et al., 1986) and prefrontal 6–OHDA lesions that markedly increased limbic DA utilization upset CB (Oades et al., 1987). Changing the balance between alpha- and beta-noradrenalin (NA) interactions attenuated CB (Caza, 1984). Dorsal NA bundle lesions also attenuated CB, but damage to ascending serotonergic projections did not (Lorden, Rickert, Dawson, & Pelleymounter, 1980).

These comparative studies show that too much or too little catecholamine activity can be detrimental to CB. With respect to development, less CB would be expected in children known to exhibit relatively high DA activity. The situation for NA is harder to predict—first, because a correct balance of receptor type is important and second, because an emphasis on tuning with high NA activity could counteract an emphasis on more DA-mediated switching.

## METHOD

### Participants

Twenty-five high school and college students were recruited through clinic staff or advertisement and were paid for their participation. This group was made up of 9 men and 16 women, whose combined mean age was 21.6 years (range 18–26 years), with a standard deviation of 1.9. Their mean performance IQ on Raven's standard progressive matrices (SPM) was determined to be 121 (range 97–140), with a standard deviation of 12. They had normal or normalized vision, were not color-blind, and preferred to use their right hand (writing and tool-use, first eight questions of Edinburgh handedness inventory, Oldfield, 1971). They claimed they were free of any major illness and had not sought psychological or psychiatric advice.

### Materials

The CB task was presented with a personal computer using a color monitor. Most of the physical and timing aspects had been preselected after pilot studies using specially written menu-driven software. (Copies may be purchased by writing to R. D. Oades.) Responses were made with an SVI joystick, Spectravideo, Hong Kong. The form and content were designed to motivate young children to solve a problem—hence it was called the "mouse-in-house" game. The game includes movement, color, and the possibility of winning points. It was designed to be difficult enough to solve only after a number of learning trials but to be soluble in 5 to 20 min for individuals between 8 and 80 years of age.

The basic format of the task (Figure 1) consisted of a rectangle 13 cm high × 24 cm long that could be subdivided into 17 × 8 squares (each 1.5 × 1.5 cm, invisible during task performance). This was described as an apartment or house, divided into two rooms by a wall down the middle, with a door in the center. On each side of the door were walls three squares long and one square thick. Each room was divided into four chambers by three walls, each three squares long.

Above and outside the "house" (13 mm), 1 to 3 color panels (18 × 7 mm) could appear together in the middle at the start of each trial. Above the left and right ends of the "house" were small panels that showed the minus and plus points obtained during the task. The red cursor was shaped somewhat like the head of a mouse.

### Procedure

Testing consisted of two sessions starting between 0900 and 1100, with one day between the first (CB–A) and the second (CB–B). CB–A was the participant's own

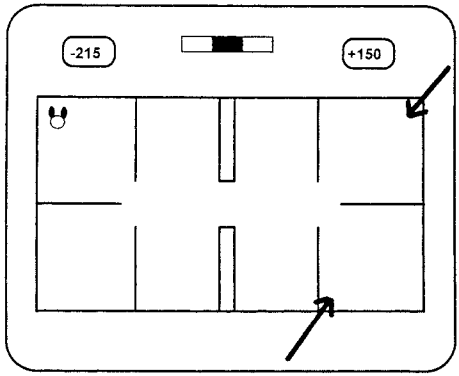


FIGURE 1 The game-plan is divided into two rooms and four chambers by walls that the "mouse" (upper-left) cannot cross. The participant looks for a safe spot (e.g., lower arrow) cued briefly by three colors (upper middle). On finding this, the area flickers yellow and +15 points are awarded (upper right). Delays are punished with minus points accumulating every sec (upper left). The upper arrow (right) shows an alternate mirror-image starting point for one of the two learning tasks per session. Each task has different colors and separate safe spots.

learning control session, and CB–B was the blocking test session. Individuals were seated in a soft upright stool with their eyes 65 cm from the screen.

Participants were told that this was a learning game and were shown the floor plan. The point was to bring the mouse with the joystick from the start square to one of *two possible "safe" spots.* (This was the size of a square, demonstrated during pretest calibration of the joystick.) After the safe spot was found, the "square" would shimmer yellow, and they would get +15 points (intertrial interval = 2 sec). The mouse might start in either room and the safe spot was always in the other room, so they would have to steer around the walls, through which the mouse could not go, and explore the other room.

At the start of each trial, color panels lit up briefly (1 sec). These were cues as to whether the one or the other safe spot was the goal on this trial. In CB–A (Phase i), there were three colors throughout training (pale green, pale blue, and pale red—Safe Spot 1; and mauve, white, and yellow—Safe Spot 2). Participants were warned to attend to these, because if they did not learn to steer directly to the safe spot, after a short period of grace (10 sec), they would start to accumulate 5 minus points per sec on the other counter. They were told that the game would finish

automatically when they had achieved a certain number of trials without scoring further minus points.

Having achieved the learning criterion (88% over 8 trials), the participants proceeded automatically to 21 test trials (CB–A Phase ii) in which the first and third colors were presented alone for response (alternately first–third and third–first for each three-color cue series). The main dependent variable was the latency to find the safe spot after the stimulus originally presented on the right, minus the latency following the stimulus originally presented on the left. Data for the first test trial were discarded due to the inevitable surprise of seeing only one color cue.

CB–B was presented 48 hr later, starting with two pairs of colors (royal blue–deep green; turqoise–brick red). After achieving a 50% criterion over eight trials (CB–B Phase i), a third color was added (gray or brown) until the full learning criterion had been reached (CB–B Phase ii). CB would be expected to the third (added) color when tested alone (CB–B Phase iii) with respect to the first color (alone) that had been presented from the start of the session. CB was measured by the latency to the third minus first stimulus in CB–B less the same measure obtained in the learning control session, CB–A (i.e., CB–B Phase iii [third minus first stimulus] – CB–A Phase ii [third minus first stimulus]). This was calculated for the 1st pair of test stimuli, BA(1); the mean of the first 3 pairs, BA(1–3); 5 pairs, BA(1–5); and all 10 pairs of stimuli, BA(1–10). Positive scores show blocking.

Participants were not told that the safe spots were the same (mirror-image) in each room or that the starting points alternated from side to side while the color panels were presented in a pseudo-random sequence. They were not informed whether two or three colors would appear during training or that there would be a test phase with only one per trial. All participants accumulated minus points at first, but on this measure came out (more or less) winners at the end. Points weightings were adjusted to maintain motivation if a participant seemed to be getting too many minus points.

## Personality and Monoamine Measures

Personality features were assessed on the short version of the MMPI (Gehring & Blaser, 1982) and the Hamburger Neuroticism/Extroversion Scale (HANES; Buggle & Baumgürtel, 1975). The MMPI was given to a subgroup of 16 participants aged over 16 years, but the simpler HANES, with norms up to 16 years, was administered to all participants. The results of both (and their intercorrelations in which responses to both were obtained) are described in an attempt to provide continuity of personality dimensions across adolescence. The MMPI was used (a) to provide results comparable to previous learned-inattention studies, as cited earlier and (b) because of its widespread acceptance in clinical settings and our proposed extension of this study (Oades, Zimmermann, & Eggers, 1996) to patients.

DA, NA, serotonin (5HT), and their metabolites (homovanillic acid, HVA; 3–methoxy–4–hydroxy-phenylglycol, MHPG; 5-hydroxyindole acetic acid, 5HIAA) were measured in 24-hr urine collections (low monoamine diet) on the day between CB–A and CB–B performance. Acidified samples were deep frozen until they were analyzed with ion exchange liquid chromatography and fluorescence detection. Measures were corrected for volume and expressed in ng per g creatinine per $m^2$ body area to correct for general somatic metabolic rates and variations incurred in the study of children less than half the size of some adults (see Oades, Roepcke, & Eggers, 1994, for full description).

### Data Treatment

In the description of the new test (Part 1), exploratory Pearson rho correlations were calculated separately for learning parameters, CB latencies, and personality and monoamine measures. Results are cited as significant ($a < 5\%$) or as trends ($a = 5–10\%$) with respect to Bonferroni corrections. A potential gender effect was assessed by a $2 \times 4$ (Gender × CB Measures) analysis of variance (ANOVA).

## RESULTS (PART 1)

### CB

After describing the general features of learning in this version of the CB paradigm, the features showing CB are illustrated and correlates between learning parameters are described to show their specificity (dependency and independency of measures).

Participants improved from the first to the second session, learning CB–A with a mean of 23 and CB–B in 16 trials (CB–B Phase i, 6 trials; and Phase ii, 10 trials, Table 1). This was confirmed by the time spent exploring before finding the safe spot (punished with minus points). This averaged 98 sec during initial learning (CB–A Phase i) and 22 sec during the test phase (CB–A Phase ii) versus 32 sec while learning CB–B and 11 sec in the test phase (CB–B Phase iii).

In CB–A, initial responses were biased to the right (Trials 1–3, latency to left-stimulus minus right-stimulus alone, $M = -1.28$ sec, Table 2), but in CB–B there was a small left bias (Trials 1–3, $M = 0.76$ sec). This provides evidence against either a general response bias to one side or a consistent bias due to conditioning (overshadowing), in which the use of two sets of stimulus panels both requiring left and right responses protects against overshadowing effects, thus the comparison of the two sessions demonstrated CB. This was most marked on the initial trial(s) and decreased over the 10 pairs of left-stimulus and right-stimulus test trials (Table 2; positive latencies represent CB). This was confirmed by comparing the mean latencies over the first and last three trials in the CB–B test phase: ([B,1–3] –

TABLE 1
Number of Learning Trials, Duration of Learning, and "Punished" Exploration Time

| *Learning Trials* | *No.* | *95%* | *Cumulative Mean Time (Min)* | *95%* | *Punished-Latency (Sec)* | *95%* |
|---|---|---|---|---|---|---|
| CB–A | | | | | | |
| i Learn | 23.4 | | 5.4 | | 98.0 | |
| | | *31/16* | | *7/4* | | *136/60* |
| ii Test | | | 8.8 | | 22.2 | |
| | | | | *11/7* | | *32/13* |
| CB–B | | | | | | |
| i Learn | 6.0 | | 1.2 | | 16.6 | |
| | | *7/5* | | *1/1* | | *23/10* |
| ii Learn | 10.4 | | 2.9 | | 15.5 | |
| | | *14/7* | | *4/2* | | *26/5* |
| iii Test | | | 5.5 | | 11.2 | |
| | | | | *6/5* | | *20/3* |

[a]Total time across trials punished with minus points to find safe spot (i.e., exploration not including unpunished latency); 95% (upper/lower limits) shown in italics (for comparison with Part 2 of this article).

TABLE 2
Conditioned Blocking (Relative) Latency Measures for 25 Young Healthy Adults (1/100ths Sec)

| *CB–A (1-3)* | *CB–A (1-5)* | *CB–B (1-3)* | *CB–B (1-5)* | |
|---|---|---|---|---|
| −128.1 | −62.1 | +75.7 | +58.4 | |
| *48/−304* | *71/−195* | *180/−29* | *124/−7* | |
| **CB B–A (1)** | **CB B–A (1-3)** | **CB B–A (1-5)** | **CB B–A (1-10)** | **CB B (1-3/8-10)** |
| +578.8 | +203.8 | +118.2 | +98.9 | +40.4 |
| *1030/128* | *431/−24* | *284/−47* | *184/14* | *159/−7* |

*Note.* 95% (upper/lower limits) shown in italics.

[B,8–10], +0.4 sec). This measure may be viewed as the development of unblocking.

With respect to the correlates of learning, two separate measures of learning intercorrelated only for the same phase of learning and not with later phases. First, the number of trials to learn the discrimination correlated with the length of time spent searching for the safe spot (punished latency; Table 3). Second, there was no relation between either the time taken to learn or the number of trials required with any CB measure (e.g., BA[1–3]; Table 3). A nonsignificant trend (after correction) between the number of trials in CB–B Phase ii (third color added) and the number of nonblocking trials in the test phase ($r = .4$, $p = .05$, data not shown) indicates a

TABLE 3
Pearson Rho Correlations Between Trials to Learn CB–A and CB–B With Exploration Latency and SPM IQ

| | *CB–A* | | *CB–B* | | | | |
|---|---|---|---|---|---|---|---|
| *Trials* | *i* | *ii* | *i* | *ii* | *iii* | *CB (BA 1–3)* | *IQ* |
| CB–A | 0.77** | 0.03 | 0.26 | 0.29 | 0.41 | −0.10 | −0.05 |
| CB–B | | | | | | | |
| i | 0.03 | 0.10 | 0.60* | 0.11 | 0.17 | −0.12 | 0.12 |
| ii | −0.04 | −0.02 | 0.21 | 0.78** | 0.04 | −0.09 | −0.14 |
| IQ | −0.10 | 0.04 | −0.09 | −0.30 | −0.14 | 0.07 | |

*Note.* CB–A Phase ii and CB–B Phase iii refer to the test phase of the two learning sessions and CB (BA 1–3) is a blocking measure on the first 3 test presentations.
*$p < .03$.**$p < .000$ (after Bonferroni correction).

sensitive relation between the number of added-stimulus exposures and CB (i.e., the learning criterion for CB–B Phase i may perhaps be raised above 50%).

Was there any relation between initial learning performance and CB? There was a trend for the exploration time (punished latency) in CB–A Phase i to correlate with exploration during the CB test phase (CB–B Phase iii, $r = .5$, $p = .01$) but not with any of the CB measures ($r < .13$). However, the time spent exploring in the CB–A test phase correlated with all measures of CB ($r = .51$–$.60$, $p = .009$–$.001$) and negatively with latency measures on the CB–A session (CB–A[1–5] $r = .66$, $p < .001$). This could be interpreted as showing that those participants initially favoring left-sided stimuli were naturally biased toward showing CB. But this hypothesis is negated by finding no relation between the difficulty in finding the safe spot (punished latency) in the CB–B test phase and the relative latency between left and right stimuli either on the CB–A or the CB–B phase (for Trials 1–5, $r = -.09$ to $+.03$).

Finally, performance IQ was not found to be related to any measure, and an ANOVA for Gender (2) × Blocking Measures (BA[1], [1–3], [1–5] and unblocking B[1–3/8–10]) was not significant, $F(4, 20) = .045$, $p = .99$.

## Personality Features

Was there a relation between HANES and MMPI measures? HANES extroversion measures correlated negatively with MMPI social introversion ($r = -.49$ to $-.64$, $p = .05$–$.008$), whereas the HANES neuroticism scores correlated positively with social introversion, psychasthenia, the three neurotic scales, and the schizotypal 2–7–8 scales of the MMPI ($r = .52$ to $.60$, $p = .04$–$.01$).

There were no CB correlations ($p < .1$, $n = 16$) with MMPI features of social introversion, depression or the three neurotic dimensions, psychopathic deviance, paranoia, psychasthenia, psychoticism, hypomania, or three combinations repre-

senting psychopathy (4–9) or schizotypal features (2–7–8, 8–9; Merritt & Balogh, 1990). The mean values on these dimensions varied close around the norm (47–53, $SD = 5.3$–$9.8$). The HANES sum scores were also close to the norms, with some variation attributable to the participants being too mature for the scale ($Es = 6.6$, $SD = 2.3$; $Ns = 4.7$, $SD = 2.3$). From the HANES features of neuroticism (*N1, Ns*) and extroversion (*E1, E2, Es*) and the combination (*E1–Ns*), the only correlation achieving better than 10% significance was *E2* (active outgoing behavior, $n = 25$). The CB measures BA(1), (1–3) and (1–5) correlated with *E2* ($r = .35$, $r = .39$, $r = .38$, respectively, $p < .05$–$.08$). These trends would not be significant after a Bonferroni correction ($3 \times 6$ comparisons).

### Monoamine Metabolism

CB measures did not correlate with the level of any monoamine or metabolite excreted. But increased CB was related to increased DA and NA (not 5HT) utilization (Table 4). In a multiple linear regression model, partial correlations for age, DA utilization, and NA utilization were, respectively, –.27, .48, and .59; with $ts(21) = -1.25$, 2.45, and 3.25; and $ps = .23$, .02, and .004.

## DISCUSSION (PART 1)

The test as presented was difficult enough to require a number of trials to solve in which most participants showed neither ceiling nor floor effects (performance IQ range = 97–140).

CB was shown by the relatively longer latency to find the safe spot indicated by the stimulus added in the second phase of learning CB–B (i.e., CB–B Phase ii), when presented alone in the test phase (CB–B Phase iii vs. the learning control, CB–A Phase ii). The gradual decrease of the CB measure across test trials is indicative of the breakdown of initial blocking as the participant learned about the added stimulus (i.e., unblocking). CB was found to be sensitive to exposure to the to-be-blocked stimulus. Having considered if participants were biased from the start in processing left-sided versus right-sided stimuli, it was concluded that there was no unequivocal bias that could explain the CB result. Simple interpretations in terms of overshadowing were not supported by the data and were controlled for by (a) the use of two sets of stimuli in each learning phase, (b) both stimulus sets requiring both left and right responses, and (c) the use of a learning control session for each participant.

Normal CB was seen more clearly in individuals showing outgoing behavior (extroversion) within the normal range. But those with more introverted features did not show impaired CB.

TABLE 4
Pearson Rho Correlations of Four Measures of Blocking[a]

| | *Ad* | *DA* | *HVA* | *util HVA/DA* | *NA* | *MHPG* | *util MHPG/NA* | *5HT* | *5HIAA* | *util 5HIAA/5HT* |
|---|---|---|---|---|---|---|---|---|---|---|
| Blocking | | | | | | | | | | |
| CB–BA (1) | .06 | −.17 | .14 | .63*** | −.19 | .23 | .47* | .14 | −.01 | −.18 |
| (1–3) | .10 | −.08 | .16 | .40* | −.24 | .26 | .53** | .15 | .01 | −.17 |
| (1–5) | .40 | −.09 | .20 | .40* | −.13 | .23 | .45* | .12 | −.03 | −.18 |
| CB–B (1–3/8–10) | .23 | −.13 | .31 | .23 | .01 | .30 | .34 | .28 | .06 | −.27 |
| Mean values[b] | 2.7 | 244 | 2023 | 12.1 | 16 | 988 | 58.1 | 78 | 1113 | 22.1 |
| 95% up/down | *4.2* | *306* | *2661* | *17.8* | *19* | *1437* | *77.8* | *120* | *1688* | *29.1* |
| | *1.3* | *182* | *1384* | *6.4* | *14* | *539* | *38.4* | *35* | *538* | *15.0* |

*Note.* Util = utilization measure of metabolism: CB–BA (x–y) = conditioned blocking, mean latency on trial pairs on x–y trials (CS vs. added stimulus) in Test Phase B less Test Phase A. AD = adrenalin.

[a]Mean values given in 24 hr urine collections from 24 young adults. [b]units - ng per mg creatinine per square meter body area.

*$p < .05$. **$p < .01$. ***$p < .001$ (Bonferroni correction $p < .002$).

Only utilization measures of DA and NA activity (and not 5HT) were positively related to CB. Increased NA metabolism is consistent with a role of increased efficiency of tuning-in pathways carrying significant information (Oades, 1985) and, by implication, tuning out irrelevant information. Oades argued for a similar consequence for increased DA metabolism but via a switching mechanism: Increased DA activity facilitates another pathway assuming control of the output of information processing and by implication the irrelevant source is shut off. Within limits, the activity of both transmitters could facilitate CB by decreasing the influence of extra distracting stimuli. (Outside these limits perseveration or excessive distractibility may be expected.)

## METHOD (PART 2)

In Part 2 there were 44 participants divided into four age groups with 11 in each (selection as in Part 1). C1, age 20–23 years ($M = 21.9$, $SD = 0.9$; 4 men, 7 women) was selected from participants in Part 1 who showed BA(1–3) measures within 95% confidence levels of the mean. They all had a Tanner maturity rating on a scale of 1–6 of greater than 5. The other groups were (a) C2, age 16–19 years, ($M = 17.1$, $SD = 1.0$, 7 male adolescents, 4 female adolescents, mean Tanner score = 4.9); (b) C3, age 12–16 years ($M = 14.5$, $SD = 1.2$, 9 male adolescents, 2 female adolescents, mean Tanner score = 2.8); and (c) C4, age 8–12 years ($M = 10.1$, $SD = 1.0$, 5 male children, 6 female children, mean Tanner score = 1.4). Mean SPM IQ scores were 124 (C1), 109 (C2), 106 (C3), and 111 (C4). Somatic age was assessed from hand X-rays for epiphysis closure for groups C2 (16.6 years, $SD = 1.3$) and C3 (14.2 years, $SD = 1.4$) and usually gave marginally lower values than chronological age (approx. 0.5 years). Most parents of C4 participants did not consent to this measure. Signed informed consent was obtained from the parent or guardian for each procedure for participants under 18 years of age.

The procedure was the same as in Part 1 except that the MMPI was not carried out with participants less than 16 years old and, as members of groups C2 and C3 were accomplished joystick users, the prepunishment latency to find the safe spot was reduced to 8 sec.

Data analysis, additional to Part 1, included ANOVA for four age groups versus the three types of learning parameters and the five main CB measures (cf. Tables 5 and 6). As the CB measures were partially related, initially a repeated-measures analysis was conducted. This established the principal measures to be used in further analyses (e.g., HANES and monoamines). Prior testing for CB parameters showed a normal distribution (e.g., BA(1–3), Kolmogorov-Smirnov $d = .105$) and a homogeneity of variance that did not correlate with the means across groups ($r = .062$). As IQ was related to age ($r = .36$, $p < .02$), it was used as a covariate. Significant group comparisons were ascertained post hoc with the conservative

TABLE 6
Conditioned Blocking (Relative) Latency Measures (Means) (1/100ths Sec)

| | *CB B–A (1)* | *CB B–A (1–3)* | *CB B–A (1–5)* | *CB B–A (1–10)* | *CB B (1–3) - (8–10)* |
|---|---|---|---|---|---|
| C1[a] | 708 | 298 | 205 | 148 | 58 |
| sem* | 370 | 122 | 73 | 42 | 89 |
| C2[b] | 322 | 117 | 104 | 68 | 54 |
| sem* | 118 | 54 | 36 | 29 | 104 |
| C3[b] | 458 | 346 | 211 | 115 | 59 |
| sem* | 255 | 126 | 82 | 48 | 68 |
| C4[c] | −13 | 22 | 101 | 91 | 34 |
| sem* | 225 | 142 | 105 | 64 | 121 |

*Note.* Intergroup comparisons were not significant (see text).
[a]Young adults. [b]Adolescents. [c]Children.
*Standard error.

TABLE 5

Number of Learning Trials, Duration of Learning, and "Punished" Exploration Time and Age Correlation

| | *Learning Trials No.* | | | *Cumulative Mean Time (Min)* | | | | | *Punished Latency (Sec)* | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *CB–A* | *CB–B* | | *CB–A* | | *CB–B* | | | *CB–A* | | *CB–B* | | |
| | *i* | *i* | *ii* | *i* | *ii* | *i* | *ii* | *iii* | *i* | *ii* | *i* | *ii* | *iii* |
| C1[a] | 18**** | 6 | 10 | 4**** | 7** | 2 | 8* | 10* | 72*** | 17 | 15 | 14 | 3 |
| up | *25* | *6* | *14* | *6* | *9* | *1* | *4* | *6* | *105* | *29* | *27* | *34* | *6* |
| down | *11* | *5* | *5* | *2* | *5* | *1* | *1* | *4* | *37* | *4* | *4* | *−7* | *0* |
| C2[b] | 25* | 9 | 14 | 5* | 8* | 1 | 4 | 6 | 77 | 13 | 26 | 16 | 11 |
| up | *37* | *11* | *21* | *8* | *11* | *2* | *5* | *7* | *134* | *19* | *37* | *32* | *18* |
| down | *14* | *6* | *8* | *2* | *4* | *1* | *2* | *4* | *20* | *5* | *15* | *1* | *3* |
| C3[b] | 25* | 7 | 15 | 5* | 8* | 1 | 3 | 6* | 74 | 26 | 17 | 20 | 15 |
| up | *35* | *10* | *22* | *7* | *10* | *2* | *5* | *7* | *113* | *40* | *27* | *39* | *27* |
| down | *15* | *5* | *7* | *3* | *6* | *1* | *2* | *4* | *35* | *13* | *7* | *2* | *3* |
| C4[c] | 46 | 9 | 23 | 11 | 14 | 2 | 6 | 9 | 205 | 23 | 38 | 53 | 16 |
| up | *63* | *13* | *36* | *15* | *19* | *19* | *3* | *9* | *301* | *35* | *64* | *101* | *24* |
| down | *28* | *6* | *11* | *7* | *10* | *1* | *3* | *6* | *110* | *10* | *12* | *5* | *8* |
| $\rho$ | −.53 | | −.43 | −.51 | | −.37 | | −.48 | | −.19 | | −.37 | |
| | | −.37 | | | −.48 | | −.46 | | −.48 | | −.30 | | −.44 |

*Note.* 95% (upper/lower limits) shown in italics. Pearson rho correlations with age, $\geq .48$, $p < .01$; $\leq .48$, $p < .05$; $\leq .42$ not significant after Bonferroni correction for 13 measures.

[a]Young adults. [b]Adolescents. [c]Children.

*$p < .05$ (vs. C4). **$p < .01$. ***$p < .01$ (vs. C4), $p < .05$ (vs. C2 and C3). ****$p < .005$.

Scheffé test ($\alpha = 5\%$). Potential gender interactions were assessed by an exploratory ANOVA, $4 \times 2 \times 4$ (Age Group × Gender × CB Measure) and linear regression analysis. In view of the small numbers involved, this has the purpose of generating hypotheses for future study.

## RESULTS (PART 2)

### CB

Measures of learning for the four groups are shown in Table 5. The number of learning trials correlated negatively with age, particularly in CB–A. Younger participants (especially C4) required more trials, $F(9, 90) = 2.5$, $p = .01$. Accordingly, C4 also spent more time learning, $F(15, 97) = 1.84$, $p = .04$. This was partly accounted for by the increased time to explore (punished latency), which correlated negatively with age on initial learning and in the test phase. Group exploration differences, $F(15, 97) = 1.94$, $p = .03$, were only evident during original learning (CB–A Phase i), whereby the C1 adults were more efficient than any of the younger groups (Table 5).

The five latency measures of CB (and the development of unblocking, B(1–3/8–10) were not correlated with age. Repeated-measures analysis ($df = 4, 37$) found neither a main effect of group nor an interaction; $F = 1.13/1.30$, $p = .35/.23$, respectively. However, a main effect of measure, $F(4, 160) = 4.89$, $p = .001$, showed that the relative latency across the 10 test trials (BA[1–10]) and the development of unblocking were less strong markers of blocking than BA(1) (Scheffé $p < .02$). Thus, BA(1), (1–3) or (1–5), were taken as measures that usefully reflect CB. The means for all measures in all groups were all well within the 95% confidence limits of the larger adult group (Table 2) or in the case of C3, slightly more positive. Descriptively, the data in Table 6 confirm that BA(1–3) and (1–5) were the more reliable data, that CB in the adolescent groups (C2 and C3) can be variable, and that the children (C4) showed least CB, albeit nonsignificantly different from the other groups. One way to view this trend is to say that if $F$-values in the ANOVA remained constant, group size would have to exceed 25 to achieve conventional significance.

There was no significant interaction of gender on a $2 \times 4 \times 4$ (Age Group × Gender × CB) ANOVA, $F(12, 84) = 1.19$, $p = .31$, and gender did not significantly influence CB measures across the four age groups (partial correlation = –.085, $p = .6$). However, it should be cautioned that the power to reject a role of gender is weak as numbers representing each gender are small. But it may be noted that covariates for the previous ANOVA of CB latencies ranged from 1.2 to 1.6 between groups, altering nonsignificant probabilities for one-way effects by only 0 to 2 percentage

points. This suggests a trivial influence of gender, but this needs to be confirmed in future work.

Having shown there were no group differences, $F(15, 97) = .58$, $p = .89$ (Table 6), we explored the removal of the covariate for IQ and the use as a covariate of the Göttinger Form-Reproduction Test (GFT; Schlange, Stein, von Boetticher, & Taneli, 1977; copy simple drawings, used as a soft sign for neurological and developmental problems), where errors, as expected, correlated negatively with age ($r = .42$, $p = .005$). Neither of these analyses proved significant, $F(15, 99) = .69$, $p = .79$; $F(15, 97) = .74$, $p = .74$, respectively. Of note, however, was that the $F$-values for the one-way analyses across the three ANOVAs were more stable for the BA(1–3) and (1–5) measures than for the other three blocking measures (4% and 16% vs. 27%–60%). Finally, IQ (range = 77–145) did not correlate significantly with CB (e.g., BA(1–3), $r = .22$, $p = .15$).

## Personality Features

Personality features reflected in the HANES dimensions did not vary across groups ($E2 = 5.1$–$6.2$), and were not correlated with age (e.g., for $E2$, $r = -.04$, $p = .8$), or to the main measures of CB (e.g., $E2$ with BA(1–3) $r = .18$, $p = .24$).

## Monoamine Metabolism

As there were no group differences in CB to be explained, the urinary analysis is restricted to group monoamine differences and their relation to learning speed and the degree of CB.

Given that adrenalin, DA, NA, their metabolites (HVA and MHPG), and DA utilization correlated with age changes, a multivariate analysis of variance analysis was performed separately on levels and utilization, $F(21, 98) = 1.87$, $p = .021$ (Table 7). One-way analyses confirmed increases for all monoamine levels in the young except those for 5HT and 5HIAA, $F(3, 40) = 4.1$–$8.3$, $p = .01$–$.0002$. Metabolism, however, reflected by utilization, did not differ between groups, $F(9, 92) = 1.88$, $p = .064$. An apparent increase in NA utilization was responsible for the trend, $F(3, 40) = 2.53$, $p = .071$.

As may be seen in Table 7, CB measures correlated positively with DA activity, less so and negatively with NA utilization, to a lesser extent with levels of DA and NA but not with other measures. In contrast, measures of learning (number of trials on CB–A and CB–B Phase i) correlated with levels of DA and HVA ($r = .55$ to $.52$, $p < .05$ after Bonferroni correction), adrenalin ($r = .46$ to $.39$, $p > .05$), and NA ($r = .46$ to $.35$, $p > .05$), and not with any measure of utilization.

TABLE 7

Mean Levels of Monoamines and Metabolites in 24-hr Urine Samples[a] From Four Age Groups and Pearson Correlation Coefficients With Age and With Two Latency Measures of Blocking

| | *Monoamines and Metabolites* | | | | | | |
|---|---|---|---|---|---|---|---|
| | *Ad* | *DA* | *HVA* | *NA* | *MHPG* | *5HT* | *5HIAA* |
| *r* (age) | −.48[d] | −.48[d] | −.43[d] | −.41[d] | −.41[d] | −.24 | −.11 |
| *r* | | | | | | | |
| BA (1) | −.27 | −.37[c] | −.11 | −.30 | −.25 | −.02 | −.02 |
| BA (1-3) | −.08 | −.27 | −.30 | −.13 | −.35 | .00 | .05 |
| Group | | | | | | | |
| C1 | 2.1 | 201 | 2135 | 16 | 772 | 62 | 730 |
| *SD* | 1.5 | 100 | 1560 | 8 | 646 | 56 | 306 |
| C2 | 2.5 | 249 | 1595 | 15 | 583 | 67 | 1010 |
| *SD* | 1.1 | 211 | 744 | 5 | 252 | 102 | 1955 |
| C3 | 3.2 | 212 | 2120 | 16 | 555 | 46 | 637 |
| *SD* | 1.9 | 71 | 599 | 5 | 209 | 29 | 452 |
| C4* | 7.2 | 543 | 3663 | 36 | 1688 | 145 | 1238 |
| *SD* | 6.5 | 332 | 1746 | 31 | 997 | 207 | 1091 |

| | *Monoamine Utilization* | | |
|---|---|---|---|
| | *HVA/DA* | *MHPG/NA* | *5HIAA/5HT* |
| *r* (age) | .32 | −.19 | −.07 |
| *r* | | | |
| BA (1) | .66[c] | −.06 | −.12 |
| BA (1-3) | .38[b] | −.33[b] | −.05 |
| Group | | | |
| C1 | 17 | 47 | 20 |
| *SD* | 18 | 27 | 15 |
| C2 | 10 | 41 | 13 |
| *SD* | 5 | 15 | 14 |
| C3 | 11 | 35 | 14 |
| *SD* | 4 | 15 | 8 |
| C4 | 8 | 70 | 26 |
| *SD* | 3 | 52 | 21 |

*Note.* *r* (age), $p = .006$-$.001$ (Bonferroni correction requires .5%). *r* (blocking), $p < .01$ (correction requires .05% = *). Partial correlations for age/DA-util/NA-util with blocking BA (1-3) were .03, .37, −.34; $t = .17, 2.49, -2.29$; $p = .86, .017, .027$. Partial correlations for age/DA-util/NA-util with learning (CB-A trials) were −.49, −.12, −.07; $t = -3.57, -.73, -.44$; $p = .001, .47, .67$.

[a]ng per mg creatinine per square meter body area. [b]$p < .05$. [c]$p < .01$. [d]$p < .006$.

*$p < .01$, C4 levels Ad, DA, HVA, NA, MHPG higher than in C1.

An alternative way to express the relation of DA and NA utilization to CB is the linear regression of these measures with age. The partial correlations were significant for CB (BA[1–3] and DA utilization, .37, $t = 2.49$, $p < .02$); and with NA utilization (–.34, $t = -2.29$, $p < .03$); but not for learning (CBA trials, Table 7).

## DISCUSSION

CB was found in young healthy adults in the "mouse-in-house" test according to the relative latencies to find the goal on the CB test versus the participant's own learning control test. The presence of CB was confirmed in groups of older and younger adolescents and in largely prepubertal 10-year-old children, albeit nonsignificantly reduced in this latter group.

With the maturation of which cognitive ability or which brain region does this match? In the absence of studies of brain-damaged humans and, with the exception of neurochemical lesions (Oades et al., 1987), the absence of studies of the frontal cortex on learned inattention in animals, arguments for the involvement of the frontal lobe are largely precluded. The present lack of such studies is unfortunate, as the frontal cortex is important for mediating nonepisodic rule learning (Winocur, 1991) and the application of rules in stimulus selection strategies (Diamond, 1990), the flexibility of which is developing in the late prepubertal stage (Levin et al., 1991), concurrently with large changes in the number and connectivity of synapses (Huttenlocher, 1990).

In animals, however, an intact hippocampus is necessary for CB (Rickert et al., 1981), although the basolateral amygdala may not be necessary for LI (Weiner, Tarrasch, & Feldon, 1995). We suggest that, in humans, our prepubertal age group (which showed less CB than the adolescent groups) is representative of the period of development when limbic areas mature (e.g., Altman, Brunner, & Bayer, 1973). In humans, the developmental migration of hippocampal neurons is complete prenatally, and the relative size of the hippocampus to the rest of the brain is stable by the second half of the first decade of life, yet brain weights (including myelination processes) continue to increase by 5% in the second decade (Benes, Turtle, Khan, & Farol, 1994; Jakob & Beckmann, 1994). Most important, the prepubertal period in Group C4 also matches with age for the maturation of the ability to perform discrimination and trace conditioning in the eye-blink conditioning paradigm that also depends on hippocampal function (see review by Woodruff-Pak et al., 1990).

A more important substrate for the consideration of cognitive development in humans is the parahippocampal gyrus. Although entorhinal development is relatively rapid (Rakic, 1988), Benes et al. (1994) reported that the myelination-area to brain-weight ratio doubles in the second decade for presubicular and parasubicular areas, possibly reflecting development of the cingulum. Indeed, there is good reason to suggest that cingulate function is crucial. First, it has been argued that the

cingulate plays an important role in the assumption of limbic attentional functions by the frontal lobe during primate evolution (Oades, 1982, pp. 123–128). Second, an important attentional function has been described for the cingulate in general (Morecroft, Geula, & Mesulam, 1993), and in mediating the inhibitory processes of the Stroop interference condition in particular (Pardo, Pardo, Janer, & Raichle, 1990). Last, doubt on the contribution of mesial temporal lobe structures to learned inattention has been raised recently by the finding of normal LI in a group of 11 patients with temporal lobe epilepsy (Gray et al., 1995).

It seems likely that it is the functional interaction among temporal, cingulate, and frontal areas that is maturing around 8 to 12 years of age—a proposal that is also supported by the importance of the monoaminergic interdependence seen between these areas in animals performing CB (Oades et al., 1987) and the marked frontal cortical DA (Oades & Halliday, 1987) and temporal cortical DA innervation found in humans (Smiley, Williams, Szigeti, & Goldman-Rakic, 1992).

However, it is also important to emphasize the stability of the CB measures (particularly BA(1–3)) across the age groups studied in view of clear developmental improvements in learning speed (number of trials, duration and amount of exploration of the "house") and in improved visuospatial abilities involved in drawing (GFT). Further, CB was independent of IQ in the range of 77–140.

The association of CB with extroversion, tentatively suggested on the basis of HANES scores from young adults, was not borne out in Part 2 across the age groups for which the HANES was designed. This could reflect the late development of parallel processing abilities needed in a dual-task situation (and part of our form of CB test) and said to be better expressed in the extroverted (Eysenck, 1982). It is of interest that introversion on the HANES scale was correlated to schizotypal features that have been found to relate to the attenuation of learned inattention in an LI task (Baruch et al., 1988b; De la Casa et al., 1993). However, this relation disappeared in Part 2, and in the only other pertinent study of personality and CB, no relation among psychoticism, extroversion, and CB was found (Jones et al., 1990).

Any interpretation of the role of monoamine metabolism should take two points into consideration: First, there was no pharmacological manipulation or any significant CB difference to be explained and second, any relation with age, be it negative or positive, may be spurious in terms of function, as there are a large number of developmental changes in cerebral structure in the young that influence the cognitive and metabolic measures. We did not find that gender contributed to the results reported for adults or those found in development. However, neither the design of the experiment nor the power of the analysis can exclude an hypothetical influence. This, along with features that change during development, should be examined in future studies. Finally, we briefly consider one of these—namely, our explorative analysis of indicators of the status of monoaminergic activity.

The youngest children showed higher levels of all monoamines and their metabolites, as is usual in standard clinical practice. However, utilization measures

were not uniformly elevated (or depressed). The increased number of learning trials (CB–A or CB–B Phase i) required by young children thus correlated with increased monoamine levels, in particular those of the catecholamines. However, CB only correlated with increased DA utilization and decreased NA utilization, which were not significantly related to age. We therefore suggest that (a) increasing levels of catecholamines could be related to the ease of problem solving, but are just as likely to be a spurious cohabitant of early development; and (b) the separate relations of DA and NA utilization with CB, relatively independent of age and learning abilities, are related to the operation of the selective attention mechanism resulting in normal CB.

Why did CB correlate positively with NA activity in adults but negatively in younger individuals? Curiously, there is an interesting parallel in the animal literature. Isoprotorenol, a beta agonist, facilitated CB in adolescent rats but reduced it in adults (Caza, 1984). Clearly, such potential functional changes must be carefully studied in the future. Tentatively, we would propose a loose relation. NA activity may relate to the high levels of adrenalin recorded in the youngest participants. In turn, this may interfere with the effort required to use the adult CB strategy, as at this stage of development NA can only be used to tune in stimuli (cf. Oades, 1985). In adults, however, NA activity can also be used to tune out stimuli from an influence on learning (cf. discussion of the role of NA and adrenalin in learning with or without emotional arousal; Cahill, Prins, Weber, & McGaugh, 1994).

## CONCLUSIONS

CB is about not attending to stimuli that are redundant, even if they are relevant in the sense of predicting consequences as accurately as the CS that blocks. CB is usually transient for what is temporarily taken by the participant to be an irrelevant stimulus. A central nervous mechanism for CB appears to be present, according to data from the current test, from the age of 8 years in a form similar to that seen in adults. However, there are indications of it not being marked before 10 to 12 years of age.

This period of development coincides with maturation of limbic function but precedes complete maturation of frontal lobe function. The present data also suggest that consolidation of the adult CB "strategy" coincides with settling down to mature levels of catecholamine activity. This fits well with results from animal studies showing that CB is disturbed by changes of NA and DA activity, particularly in mesolimbic and mesocortical projection regions (Crider et al., 1986; Lorden et al., 1980; Oades et al., 1987).

The present data suggest that DA and NA have opposing influences in development on processing relevant to CB. This supports, in principle, the functions attributed by Oades (1985) to DA of promoting the switching between inputs that can influence the outputs from areas processing information relevant to the ongoing situation and NA in tuning in or out inputs relevant or irrelevant to the demands of the situation. The data do not support suggestions that both DA and NA are involved in tuning (Cohen & Servan-Schreiber, 1993). Whether tuning and switching are appropriate descriptions of the processes involved could be studied with the present task by comparing the CB situation with one where the added stimulus has additional important consequences.

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## REFERENCES

Altman, J., Brunner, R. L., & Bayer, S. A. (1973). The hippocampus and behavioral maturation. *Behavioral Biology, 8,* 557–596.

Baruch, I., Hemsley, D. R., & Gray, J. A. (1988a). Differential performance of acute and chronic schizophrenics in a latent inhibition task. *Journal of Nervous and Mental Disease, 176,* 598–606.

Baruch, I., Hemsley, D. R., & Gray, J. A. (1988b). Latent inhibition and "psychotic proneness" in normal subjects. *Personality and Individual Differences, 9,* 777–783.

Benes, F. M., Turtle, M., Khan, Y., & Farol, O. (1994). Myelination of a key relay zone in the hippocampal formation occurs in the human brain during childhood, adolescence and adulthood. *Archives of General Psychiatry, 51,* 477–484.

Buggle, F., & Baumgürtel, F. (1975). *Hamburger Neuroticismus- und Extroversionsskala für Kinder und Jugendliche 3(HANES, KJ)* [Hamburg Neuroticism/Extroversion Scale for Children and Adolescents]. Göttingen: Hogrefe.

Cahill, L., Prins, B., Weber, M., & McGaugh, J. L. (1994). B-adrenergic activation and memory for emotional events. *Nature, 371,* 702–704.

Caza, P. A. (1984). Noradrenergic influences on blocking: Interactions with development. *Pharmacology Biochemistry and Behavior, 21,* 9–17.

Cohen, J. D., & Servan-Schreiber, D. (1993). A theory of dopamine function and its role in cognitive deficits in schizophrenia. *Schizophrenia Bulletin, 19,* 85–104.

Crider, A., Blockel, L., & Solomon, P. R. (1986). A selective attention deficit in the rat following induced dopamine receptor supersensitivity. *Behavioral Neuroscience, 100,* 315–319.

Crider, A., Solomon, P. R., & McMahon, M. A. (1982). Disruption of selective attention in the rat following d-amphetamine administration: Relationship to schizophrenic attention disorder. *Biological Psychiatry, 17,* 351–361.

De la Casa, L. G., Ruiz, G., & Lubow, R. E. (1993). Latent inhibition and recall/recognition of irrelevant stimuli as a function of preexposure duration in high and low psychotic-prone normal subjects. *British Journal of Psychology, 84,* 119–132.

Diamond, A. (1990). The development and neural bases of memory functions as indexed by the AB and delayed response tasks in human infants and infant monkeys. *Annals of the New York Academy of Sciences, 608,* 267–318.

Eysenck, M. W. (1982). *Attention and arousal.* Heidelberg: Springer-Verlag.

Gehring, A., & Blaser, A. (1982). *Minnesota multiphasic personality inventory (MMPI); Dt. Kurzform für Handauswertung; Handbuch* [MMPI; German Short Form for Hand-Scoring; Manual]. Bern: Huber.

Gray, N. S., Mellers, J. D. C., Morton, N., Hemsley, D. R., Goldstein, L. H., & Toone, B. K. (1995). Latent inhibition in temporal lobe epilepsy and the schizophrenia-like psychoses of epilepsy. *Schizophrenia Research, 15,* 177.

Hemsley, D. R. (1988). Psychological models of schizophrenia. In E. Miller & P. Cooper (Eds.), *Adult abnormal psychology* (pp. 101–127). London: Churchill Livingstone.

Huttenlocher, P. R. (1990). Morphometric study of human cerebral cortex development. *Neuropsychology, 28,* 517–527.

Jakob, H., & Beckmann, H. (1994). Circumscribed malformation and nerve cell alterations in the entorhinal cortex of schizophrenics. *Journal of Neural Transmission, 98,* 83–106.

Jones, S. H., Gray, J. A., & Hemsley, D. R. (1990). The Kamin blocking effect, incidental learning and psychoticism. *British Journal of Psychology, 81,* 95–110.

Jones, S. H., Gray, J. A., & Hemsley, D. R. (1992). Loss of the Kamin blocking effect in acute but not chronic schizophrenics. *Biological Psychiatry, 32,* 739–755.

Kamin, L. J. (1969). Predictability, surprise, attention and conditioning. In R. Church & B. Campbell (Eds.), *Punishment and aversive behavior* (pp. 279–296). New York: Appleton-Century-Crofts.

Levin, H. S., Culhane, K. A., Hartmann, J., Evankovich, K., Mattson, A, J., Harward, H., Ringholz, G., Ewing-Cobbs, L., & Fletcher, J. M. (1991). Developmental changes of performance on tests of purported frontal lobe functioning. *Developmental Neuropsychology, 7,* 377–395.

Lipp, O. V., & Vaitl, D. (1992). Latent inhibition in human Pavlovian differential conditioning: Effect of additional stimulation after preexposure and relation to schizotypal traits. *Personality and Individual Differences, 13,* 1003–1012.

Lorden, J. F., Rickert, E. J., Dawson, R., & Pelleymounter, M. A. (1980). Forebrain norepinephrine and the selective processing of information. *Brain Research, 190,* 569–573.

Lubow, R. E., & Gewirtz, J. C. (1995). Latent inhibition in humans: Data, theory and implications for schizophrenia. *Psychological Bulletin, 117,* 87–103

Lubow, R. E., Inberg-Sachs, Y., Zalstein-Orda, N., & Gewirtz, J. C. (1992). Latent inhibition in low and high "psychotic prone" normal subjects. *Personality and Individual Differences, 13,* 563–572.

Lubow, R. E., & Josman, Z. E. (1993). Latent inhibition deficits in hyperactive children. *Journal of Child Psychology and Psychiatry, 84,* 959–975.

Lynn, R., & Hampson, S. L. (1977). Fluctuations in national levels of neuroticism and extraversion, 1935–1970. *British Journal of Social and Clinical Psychology, 16,* 131–138.

Mackintosh, N. J. (1975). A theory of attention: Variations in the associability of stimuli with reinforcement. *Psychological Review, 82,* 276–298.

Merritt, R. D., & Balogh, D. W. (1990). Backward masking as a function of spatial frequency: A comparison of MMPI-identified schizotypics and control subjects. *Journal of Nervous and Mental Disease, 178,* 186–193.

Morecroft, R. J., Geula, C., & Mesulam, M-M. (1993) Architecture of connectivity within a cingulo-frontal-parietal neurocognitive network for directed attention. *Archives of Neurology, 50,* 279–284.

Oades, R. D. (1982). *Attention and schizophrenia: Neurobiological bases.* London: Pitman Press.

Oades, R. D. (1985). The role of noradrenaline in tuning and dopamine in switching between signals in the CNS. *Neuroscience and Biobehavioral Reviews, 9,* 261–283.

Oades, R. D., Bunk, D., & Eggers, C. (1992). Paranoid schizophrenics may not use irrelevant signals: The use of measures of blocking and of urinary dopamine. *Acta Paedopsychiatrica, 55,* 183–184.

Oades, R. D., & Halliday, G. M. (1987). Ventral tegmental (A10) system: Neurobiology. 1. Anatomy and connectivity. *Brain Research Reviews, 12,* 117–165.

Oades, R. D., Rivet, J-M., Taghzouti, K., Kharouby, M., Simon, H., & Le Moal, M. (1987). Attentional blocking is delayed by depletion of septal dopamine but remains attenuated after frontal depletion. *Brain Research, 406,* 136–146.

Oades, R. D., Roepcke, B., & Eggers, C. (1994). Monoamine activity reflected in urine of young patients with obsessive compulsive disorder, psychosis with and without reality distortion and healthy subjects: An explorative analysis. *Journal of Neural Transmission, 96,* 143–159.

Oades, R. D., Zimmermann, B., & Eggers, C. (in press). Conditioned blocking in patients with paranoid, nonparanoid psychosis, or obsessive compulsive disorder: Association with symptoms, personality and monamine metabolism. *Journal of Psychiatric Research.*

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh Inventory. *Neuropsychologia, 9,* 97–113.

Pardo, J. V., Pardo, P. J., Janer, K. W., & Raichle, M. E. (1990). The anterior cingulate cortex mediates processing selection in the Stroop attentional conflict paradigm. *Proceedings of the National Academy of Sciences (USA), 87,* 256–259.

Rakic, P. (1988). Specification of cerebral cortical areas. *Science, 241,* 170–176.

Raven, J. C. (1960). *Guide to the Standard Progressive Matrices.* London: Lewis.

Rescorla, R. A., & Wagner, A. R. (1972) A theory of Pavlovian conditioning: Variations in the effectiveness of conditioned but not of unconditioned stimuli. In A. H. Black & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory* (pp. 64–69). New York: Appleton-Century-Crofts.

Rickert, E. J., Bennett, T. L., Lane, P., & Fench, J. (1978). Hippocampectomy and the attenuation of blocking. *Behavioral Biology, 22,* 147–160.

Rickert, E. J., Lorden, J. F., Dawson, R., & Smyly, E. (1981). Limbic lesions and the blocking effect. *Physiology and Behavior, 26,* 601–606.

Schlange, H., Stein, B., von Boetticher, I., & Taneli, S. (1977). *Göttinger Formreproduktionstest: zur Diagnose der Hirnschädigung im Kindesalter. 3* [Göttinger Form-Reproduction Test: Manual for the diagnosis of brain-damage in childhood, 3rd edition]. Göttingen: Hogrefe.

Smiley, J. F., Williams, S. M., Szigeti, K., & Goldman-Rakic, P. S. (1992). Light and electron microscopic characterization of dopamine-immunoreactive axons in human cerebral cortex. *Journal of Comparative Neurology, 321,* 325–335.

Solomon, P. R. (1977). Role of the hippocampus in blocking and conditioned inhibition of the rat's nictitating membrane response. *Journal of Comparative and Physiological Psychology, 91,* 407–417.

Sutherland, N. S., & Mackintosh, N. J. (1971). *Mechanisms of animal discrimination.* New York: Academic.

Weiner, I., Tarrasch, R., & Feldon, J. (1995). Basolateral amygdala lesions do not disrupt latent inhibition (LI). *Behavioural Brain Research, 72,* 73–82.

Winocur, G. (1991). Functional dissociation of the hippocampus and prefrontal cortex in learning and memory. *Psychobiology, 19,* 11–20.

Woodruff-Pak, D. S., Logan, C. G., & Thompson, R. F. (1990). Neurobiological substrates of classical conditioning across the life span. *Annals of the New York Academy of Sciences, 608,* 150–174.

## APPENDIX

### List of Abbreviations

*Task/Tests:*
LI = Latent inhibition
CB = Conditioned Blocking
BA(1) / BA(1–3) = CB measure (relative latency for session B - A) for first / first 3 test trials
B 1–3/8–10 = development of unblocking on session B from the first to the last 3 trials, positive scores represent CB
GFT = Göttinger Form Test (copy figures)
SPM = Raven's Standard Progressive Matrices (Performance IQ)
*Participants:*
C1 = young adults ($M$ = 21.9 years)
C2 = older adolescents ($M$ = 17.1 years)
C3 = younger adolescents ($M$ = 14.5 years)
C4 = children ($M$ = 10.1 years)
*Personality/Symptoms:*
MMPI = Minnesota Multiphasic Personality Inventory
HANES = Hamburg Neuroticism–Extroversion Scale
*Monoamines:*
DA = dopamine
HVA = homovanillic acid
NA = noradrenaline
MHPG = 3–methoxy–4–hydroxyphenylglycol
5HT = serotonin
5HIAA = 5–hydroxyindoleacetic acid