The Influence of Risperidone on Attentional Functions in Children and Adolescents with Attention-Deficit/Hyperactivity Disorder and Co-Morbid Disruptive Behavior Disorder

Thomas Günther, Ph.D., Beate Herpertz-Dahlmann, M.D., Jellemer Jolles, Ph.D., and Kerstin Konrad, Ph.D.

ABSTRACT

This study aims to examine the influence of risperidone on various attentional functions, including intensity and selectivity aspects of attention plus inhibitory control in children with attention deficit/hyperactivity disorder (ADHD) with co-morbid Disruptive Behavior Disorders (DBD) and normal IQ. Children with ADHD and DBD, aged 8–15 years, were treated with risperidone (mean daily dose: 1.5 mg; n = 23) and examined with three attentional paradigms before and after a 4-week treatment period. Age- and IQ-matched normal controls (n = 23) were also tested without medication on the same two occasions. No influence of the medication could be detected for any neuropsychological variable, neither as a positive enhancement nor as adverse side effects. However, clinical symptoms of ADHD and DBD assessed on the IOWA Conners Scale significantly improved after the 4-week treatment period. Divergent behavioral and cognitive effects of risperidone on ADHD symptoms were observed, with a significant reduction in behavioral symptoms, whereas no positive treatment effects were found on laboratory tasks of impulsivity. Thus, the cognitive effects of risperidone seem to differ from the cognitive effects of stimulant treatments in children with ADHD + DBD. However, no negative impact of risperidone was observed on attentional functions either, i.e., there was no slowing of cognitive speed.

INTRODUCTION

Risperidone is an atypical neuroleptic agent with combined serotonin and dopamine antagonism (Leysen et al. 1992). In child and adolescent psychiatry, risperidone is successfully used to treat neuropsychiatric disorders like schizophrenia (Grcevich et al. 1996; Armenteros et al. 1997), bipolar disorder (Schreier 1998; Frazier et al. 1999), pervasive developmental disorder (Barnard et al. 2002; McCracken et al. 2002; King et al. 2003), tic disorders and obsessive-compulsive disorder (Sc-hill et al. 2003; Gilbert et al. 2004), and disruptive behavior disorders (Barch et al. 2003; Aman et al. 2004; Aman et al. 2005a). Aggression is a common target symptom for which antipsychotics are prescribed to youths.
seen in psychiatric clinics (Kaplan et al. 1994; Turgay 2004), with the best evidence for the efficacy of risperidone treatment for aggression existing for autism (Arnold et al. 2003; McClellan and Werry 2003; Shea et al. 2004; Aman et al. 2005b) and Disruptive Behavior Disorders (DBD; Turgay 2004). For example, the use of risperidone has been suggested in the treatment of aggressive behavior in children with attention-deficit/hyperactivity disorder (ADHD) plus co-morbid severe DBD (Turgay 2005). The DBD of the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) (American Psychiatric Association, 1994) is made up of Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), and DBD-not otherwise specified (American Psychiatric Association 1994). This paper will use the term ‘DBD’ to refer collectively to CD and ODD.

Stimulants are the treatment of first choice for ADHD plus co-morbid DBD (Greenhill et al. 2002), but risperidone has a place in the management of children with ADHD plus severe DBD if other treatments have proven unsuccessful (Kewley 1999; Morant et al. 2001; Simeon et al. 2002; Turgay 2005). Several studies have shown that risperidone is associated with significant behavioral improvement in a high percentage of this difficult-to-treat population (Buitelaar 2000; Findling et al. 2000; Buitelaar et al. 2001; Cesena et al. 2002; Aman et al. 2002; Aman et al. 2004). Recently, Correia Filho et al. (2005) demonstrated that risperidone is associated with even greater reductions in the ADHD total score in children with moderate mental retardation and ADHD than methylphenidate. In clinical terms, children with ADHD and DBD also seem to benefit from a combined treatment of stimulants and risperidone. According to Turgay et al. (2002), the efficacy of risperidone is not affected by type of disorder, level of retardation, presence/absence of ADHD, or use of stimulants.

The most common side effects in risperidone-treated patients are weight gain, extrapyramidal symptoms, somnolence, sedation, headache, dyspepsia, enuresis, rhinitis, vomiting, and increased prolactin elevation (Buitelaar et al. 2001; Aman et al. 2002; Cesena et al. 2002; Snyder et al. 2002; Aman et al. 2004; Aman et al. 2005b; Hellings et al. 2005). The sedative and amnestic effects of neuroleptic drugs generally occur after the first administration, with children seeming to be much more vulnerable than adults to adverse effects, including sedation (Cheng-Shannon et al. 2004; McConville and Sorter 2004; Eapen and Gururaj 2005).

Most evidence on how risperidone influences cognitive function comes from studies with healthy adults (Allain et al. 2002; Barrett et al. 2004) and schizophrenic patients. Results are mixed with either cognitive improvement (Keefe et al. 1999) or no effect on cognitive functions for patients with schizophrenia (Liu et al. 2000), whereby the neurochemical basis of these effects is poorly understood. Two studies on children with disruptive behavior aged between 5 and 12 years and either borderline intellectual function or mild or moderate mental retardation (IQ between 36 and 84) found neither evidence for any improvement nor for any deterioration of cognitive variables, such as sustained attention and verbal learning (Continuous Performance Test & California Verbal Learning Test) while participants were being treated with risperidone (Snyder et al. 2002; Turgay et al. 2002). However, other studies have reported sedative effects in adolescents with schizophrenia (Grcevich et al. 1996) and children with bipolar disorders and co-morbid ADHD (Schreier 1998). Most of the available studies with risperidone are case reports, small-open label studies, and the samples are often inhomogeneous (for review, see Findling and McNamara 2004). Studies with greater clinical samples are available for tic disorders. Comparative studies with risperidone and pimozide (Brugge-man et al. 2001) or risperidone and clonidine (Gaffney et al. 2002) again reported sedation, somnolence, and fatigue as side effects for risperidone.

Thus, risperidone is an important pharmacological agent in the treatment of children with ADHD plus associated severe DBD when other treatments have proven unsuccessful. Although no negative influences on cognitive functions have been demonstrated for DBD children with subaverage IQ (Snyder et al. 2002; Turgay et al. 2002), little is known about cognitive side effects in ADHD + DBD chil-
Children with average IQ. This is why our study aimed to examine the influence of risperidone on various attentional systems in a sample of ADHD + DBD children with average IQ who had not benefited sufficiently from previous stimulant treatment alone. Because sedation is an adverse event that is frequently associated with risperidone treatment, we expected attentional functions to decline due to the sedative effects of this atypical neuroleptic agent.

Current concepts of attention generally distinguish between the selectivity and intensity of attention (Van Zomeren and Brouwer 1994). Selectivity refers to the process that modulates responsiveness to specific stimuli constellations by giving priority to certain stimuli, whereas intensity describes the ability to activate and maintain attention over time. In addition to the selectivity and intensity components of attention, a supervisory attentional system (SAS) is assumed to act as a control mechanism to modulate the two dimensions of selectivity and intensity. On the basis of previous studies, we assumed that children with ADHD + DBD had deficits within the SAS as reflected by higher cognitive impulsivity (for review, see Sergeant 2005), but also showed deficits in the intensity aspects of attention (for review, see Huang Pollock and Nigg 2003) when compared to normal controls before risperidone treatment. Due to the sedative effect of risperidone, we also expected it to have a negative impact on the intensity aspect of attention. However, we assumed that both aggressive behavior and a cognitive impulsivity task would improve during risperidone treatment.

**METHOD**

*Participants and selection procedure*

A total of 23 children with a lifetime diagnosis of ADHD plus DBD and 23 healthy controls, all aged 8–15 years, participated in this study. In the clinical sample, 18 children met the DSM-IV criteria for ADHD and 5 adolescents had a lifetime diagnosis of ADHD, but currently only fulfilled the ADHD criteria in partial remission. In addition, 13 children met the diagnostic criteria for ODD whereas 10 children had co-morbid CD (American Psychiatric Association 1994). Table 1 summarizes the baseline characteristics of the samples.

Prior to the study, all children underwent an extensive psychiatric examination conducted by an experienced child psychiatrist. A further psychiatric classification was then determined on the basis of a German semi-structured interview with the parents and the child (K-DIPS; Unnewehr et al. 1995) performed by a second independent rater who was blind to the first rater’s diagnosis.

Exclusion criteria were general IQ below 80 (WISC-III) and any kind of neuroleptic medication prior to the study. In addition to the ADHD + DBD diagnosis, the co-morbid disorders were dysthymic disorder ($n = 1$; DSM-IV: 300.4), major depression with a recent episode of minor severity ($n = 2$; DSM-IV: 296.21), and anxiety disorders (social phobia [$n = 3$; DSM-IV: 300.23], separation anxiety disorder [$n = 2$; 309.21]) in the clinical group. Of the 23 patients, 21 subjects were males and 2 females. All of the patient children had the following characteristics in common: Attentional problems, hyperactive-impulsive symptoms, and a repetitive and persistent pattern of socially dysfunctional, aggressive, or defiant behavior that was sufficiently severe to disturb their schooling and everyday life.

The control group (NC) was recruited from elementary schools and junior–senior high schools. Only Caucasian participants were included in the study, and school education of the participants and socioeconomic status (Moore and Kleining, 1968), evaluated according to the paternal profession, did not differ between both groups.

The 23 children were matched to the ADHD + DBD group on age and IQ. The 23 partici-
pants in the NC group (18 male, 5 females) had no history of psychiatric diagnosis. The two groups were not different with respect to sex distribution ($X^2_{(1)} = 1.52; p = NS$), age ($t_{(44)} = 0.06; p = NS$) or IQ ($t_{(54)} = 1.19; p = NS$). Informed parental and patient consent was obtained for all participants.

Medication log

The ADHD + DBD group was recruited from our in-patient ward at the Department of Child and Adolescent Psychiatry. All children showed severe aggressive behavior and had a long history of disruptive and impulsive behavior in various social settings. They received risperidone in addition to a standardized cognitive behavioral treatment program, including parental education and single and peer group therapy. The treatment program incorporated training in problem solving, anger management, and interpersonal skills and was continued during the study. In addition, behavioral techniques, such as token economy systems, and time-out procedures, were used to increase desirable behavior and decrease undesirable behavior.

Risperidone was started at 0.25 mg/day and increased until a positive clinical response was obtained or side effects emerged. The daily dose varied between 1 mg and 2.5 mg [mean dose = 1.5 mg; standard deviation (SD) = 0.5] and was between 0.03 mg and 0.04 mg/kg per day. 18 children with ADHD + DBD were treated with a combination of risperidone and stimulants [methylphenidate ($n = 7$), methylphenidate HCl ($n = 9$), and D-amphetamine ($n = 2$)]. The stimulant medication did not change during the study, and the duration of the medication before the study varied between 8 weeks and 7 years. In addition, 5 adolescents diagnosed with ADHD in partial remission and CD were given a monotherapy with risperidone. The risperidone medication was not discontinued in any of these patients during the period of the study and no other drugs [e.g., (SSRIs), anticonvulsants, etc.] were used before, during, or after the study. The score on the IOWA Conners rating scale (Pelham et al. 1989; Collett et al. 2003) was averaged across 5 days before risperidone treatment and the last 5 days of in-patient treatment to control for the efficacy of the treatment. The rating scale was filled out by specialized staff members. If the children received also stimulants, these were discontinued 48 hours before the neuropsychological examinations and before the 5-day-interval of behavioral ratings at both times of measurement, T1 and T2. This procedure was chosen to ensure that the dependent variables were not influenced by the stimulant treatment of the children.

Outcome measures

All subjects received a standardized computerized neuropsychological assessment before treatment with risperidone (T1) and again 4 weeks after treatment onset with risperidone (T2). The dose of the risperidone medication was kept stable over 2 weeks before T2. To control for retest effects, the normal controls were also tested twice without medication at the start and at the end of the 4-week test period. The order of the neuropsychological tests was randomized. Attentional functions were assessed by the following computerized tests in accordance with Van Zomeren and Brouwer’s framework of attention (1994).

Intensity of Attention: The Sustained Attention Task involved the continuous and consecutive presentation of 50 series of 12 different dot patterns (600 signals; de Sonneville 2000). In each series, an equal number of 3-, 4-, or 5-dot patterns was presented in pseudo-random order. The child was instructed to push the ‘yes’ button with the dominant hand whenever a 4-dot pattern (target) was shown and to press the ‘no’ button with the nondominant hand when the pattern contained 3 or 5 dots (nontargets).

Selectivity of Attention: Divided Attention was a dual task that combined a visual and acoustic discrimination task (Fimm and Zimmermann 2001). Children were asked to respond as quickly as they could whenever a square appeared and if an alternating high and deep tone was repeated. One hundred stimuli were presented containing 17 visual and 16 acoustic goal targets.

SAS: In the Go/No-Go Paradigm (Fimm and Zimmermann 2001) that assesses response
selection/inhibition, a motor response with the dominant hand was either initiated (go) or inhibited (no-go) depending on whether an “x” (go) or a “+” (no-go) stimulus appeared. Visual stimuli appeared in random order for 200 msec with a variable intertrial interval of a maximum of 1,600 msec. Half (50%) of the 40 stimuli were go trials. The design of this task triggers impulsive reactions (false alarms) and the number of false alarms is a good measurement for impulsive behavior (Van Zomeren and Brouwer 1994).

The dependent measures in all three tasks were reaction time (RT) and its SD, the number of misses (MIS), and false alarms (FA).

In addition to the neuropsychological examination, behavioral changes were also assessed by the IOWA Conners rating scale (Pelham et al. 1989; Collett et al. 2003) filled out by trained staff on the inpatient wards. The IOWA Conners rating scale contains two subscales, Inattention-Overactivity (IO) and Aggression (AG), each of which consists of five items.

**Data analysis**

Data were analyzed using SPSS 12 (SPSS Inc., 2003). The demographic characteristics were assessed by an independent t-test (IQ and age) and \( \chi^2 \)-Pearson (sex distribution). Changes to the IOWA score within the risperidone group were analyzed with a paired sample t-test. Repeated measure group differences were evaluated using repeated measure analysis of variance (ANOVAR), with the diagnostic group as the independent variable, time of measurement as the within-subject factor, and neuropsychological test scores as the dependent variables. It was decided not to adjust for multiple testing to avoid Type II errors and to enhance sensitivity for detecting even milder cognitive side effects (Tabachnick and Fidell 2000). All tests were two-tailed.

**RESULTS**

As expected, the total score on the IOWA Conners scale for the ADHD + DBD group decreased significantly from T1 to T2 (T1: M = 11.2 (±4.5), T2 = 8.4 (±2.9), \( t_{(22)} = 3.34; p = 0.003 \)). Significant improvement was found for both subscales of the IOWA Conners scale (IO and AG; \( t_{(22)} > 2.2; p < 0.039 \)). For more details see Table 2.

**Intensity of Attention: Sustained Attention Task**

No group difference was found between NC and ADHD + DBD (\( F_{(1/44)} < 0.63; NS \)) in any of the variables. The results of RT and its SD did not change significantly at T2 (\( F_{(1/44)} < 3.16; NS \)), whereas both error rates, FA (\( F_{(1/44)} = 9.42; p = 0.004 \)) and the number of MIS (\( F_{(1/44)} = 5.51; p = 0.024 \)) were higher in the NC group. No significant group \( \times \) time interaction was observed, indicating no significant impact of the risperidone medication on any of the dependent measures for the sustained attention task (\( F_{(1/44)} < 1.81; NS \)). For details see Table 3.

**Selectivity of Attention: Divided Attention Task**

No difference could be detected between NC and ADHD + DBD (\( F_{(1/44)} < 0.63; NS \)) in any of the variables. The results of RT and its SD did not change significantly at T2 (\( F_{(1/44)} < 3.16; NS \)), whereas both error rates, FA (\( F_{(1/44)} = 7.4; p = 0.009 \)) as well as the number of MIS

| Table 2. Improvement on the IOWA Conners Rating Scale for the Risperidone Group (n = 23) |
|---------------------------------|------------------|------------------|------------------|
|                                | \( T1^a \)      | \( T2^b \)      | Group difference |
| IOWAa                          | 11.2 (4.5)      | 8.4 (2.9)       | \( p = 0.008 \)  |
| IOa                            | 5.9 (2.7)       | 4.3 (1.9)       | \( p = 0.023 \)  |
| AGa                            | 5.4 (2.7)       | 4.1 (1.5)       | \( p = 0.039 \)  |

SD = standard deviation; IO = Inattention-Overactivity; AG = Aggression; M = mean.
^aAssessment before treatment.
^bAssessment 4 weeks after treatment onset.
^cIOWA Conners rating scale sum score.
^dInattention-Overactivity (IO).
^eAggression (AG).
### Table 3. Neuropsychological Performance of the Control Group and Risperidone Group, Shown Separately for Time of Assessment

<table>
<thead>
<tr>
<th></th>
<th>Controls (n = 23)</th>
<th>Risperidone (n = 23)</th>
<th>Between-subject effect</th>
<th>Within-subject effect</th>
<th>Interaction (group × time)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>F (p)</td>
<td>F (p)</td>
<td>F (p)</td>
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<tr>
<td><strong>Sustained Attention</strong></td>
<td></td>
<td></td>
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<tr>
<td>Speed (seconds)</td>
<td>13.7 (3.9)</td>
<td>11.7 (3.4)</td>
<td>14.1 (4.2)</td>
<td>13.3 (4.4)</td>
<td>0.90 (NS)</td>
</tr>
<tr>
<td>Speed fluctuation (seconds)</td>
<td>2.5 (1.3)</td>
<td>2.1 (1.3)</td>
<td>3.7 (1.8)</td>
<td>3.3 (1.5)</td>
<td>9.24 (p = 0.004)</td>
</tr>
<tr>
<td>False alarms</td>
<td>21.7 (14.6)</td>
<td>13.7 (8.7)</td>
<td>24.3 (18.3)</td>
<td>18.8 (16.7)</td>
<td>0.97 (NS)</td>
</tr>
<tr>
<td>Misses</td>
<td>27.7 (18.1)</td>
<td>31.5 (21.5)</td>
<td>47.1 (33.1)</td>
<td>44.8 (29.3)</td>
<td>5.51 (p = 0.024)</td>
</tr>
<tr>
<td><strong>Divided Attention</strong></td>
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</tr>
<tr>
<td>Reaction time (msec)</td>
<td>757 (119)</td>
<td>747 (115)</td>
<td>809 (260)</td>
<td>726 (91)</td>
<td>0.16 (NS)</td>
</tr>
<tr>
<td>Standard deviation (msec)</td>
<td>294 (95)</td>
<td>283 (75)</td>
<td>318 (115)</td>
<td>267 (83)</td>
<td>0.04 (NS)</td>
</tr>
<tr>
<td>False alarms</td>
<td>4.4 (4.5)</td>
<td>2.8 (3.9)</td>
<td>3.8 (4.6)</td>
<td>1.9 (1.7)</td>
<td>0.63 (NS)</td>
</tr>
<tr>
<td>Misses</td>
<td>6 (4.8)</td>
<td>4.8 (5.3)</td>
<td>5.9 (4.2)</td>
<td>4 (3.3)</td>
<td>0.12 (NS)</td>
</tr>
<tr>
<td><strong>Go/No-Go</strong></td>
<td></td>
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<tr>
<td>Reaction time (msec)</td>
<td>431 (81)</td>
<td>445 (67)</td>
<td>454 (120)</td>
<td>437 (105)</td>
<td>0.08 (NS)</td>
</tr>
<tr>
<td>Standard deviation (msec)</td>
<td>112 (46)</td>
<td>113 (39)</td>
<td>120 (37)</td>
<td>127 (49)</td>
<td>0.84 (NS)</td>
</tr>
<tr>
<td>False alarms</td>
<td>3 (2.8)</td>
<td>3.1 (2.7)</td>
<td>6.2 (4.6)</td>
<td>5.6 (4)</td>
<td>10.01 (p = 0.003)</td>
</tr>
<tr>
<td>Misses</td>
<td>1 (1.1)</td>
<td>0.7 (1)</td>
<td>2 (2.5)</td>
<td>0.9 (1.6)</td>
<td>2.74 (NS)</td>
</tr>
</tbody>
</table>

SD = standard deviation; NS = not significant.

aMean (standard deviation).
bAssessment before treatment.
cAssessment 4 weeks after treatment onset.
(F\(_{1/44}\) = 8.15; \(p = 0.007\)) decreased. No significant influence of the risperidone medication was detected for any of the divided attention variables (F\(_{1/44}\) < 1.75; NS).

**SAS: Go/No-Go-Paradigm**

No within (F\(_{1/44}\) < 0.56; NS) or between subject effect (F\(_{1/44}\) < 0.84; NS) could be detected for the RTs and the within-subject variability of RTs. The number of MIS was comparable for both groups (F\(_{1/44}\) = 2.74; NS), whereas the number of FA, a measure of impulsivity, was significantly higher in the ADHD + DBD group (F\(_{1/44}\) = 10.01; \(p = 0.003\)). Furthermore, all children made fewer MIS at T2 (F\(_{1/44}\) = 5.58; \(p = 0.023\)). Again, no significant influence of the risperidone medication was detected for any of the Go/No-Go variables (F\(_{1/44}\) < 1.81; NS).

**DISCUSSION**

Our goal was to examine the influence of the neuroleptic agent risperidone on various attentional functions, including intensity of attention, selectivity of attention, and inhibitory control in children with ADHD and co-morbid DBD with normal intelligence. We expected a negative impact on the intensity aspect of attention but an improvement in the impulsivity related task. Previous research did not focus on the cognitive adverse effects of risperidone or were based either on adults or included only children with ADHD + DBD with subaverage IQ. In agreement with previous research, we found that unmedicated children with ADHD + DBD showed intensity deficits of attention and were more impulsive in laboratory tasks than normal controls (e.g., Halperin et al. 1990; Lynam 1996; Oosterlaan et al. 1998; Oosterlaan and Sergeant 1998; Hill 2002; Nigg 2003; van Goozen et al. 2004). Note, however, that dependent variables were interpreted according to Van Zomeren and Brouwer’s framework of attention (1994), linking deficits in the sustained attention task primarily to intensity functions of attention and false alarms in the Go/No-Go Task to cognitive impulsivity. Furthermore, fewer errors and greater speed were detected in all parts of the sustained attention task due to the repetition of the tasks. However, contrary to our hypotheses, no differential influence of the risperidone medication could be detected on the neuropsychological task variables, neither as a positive enhancement nor as an adverse side effect.

The decrease in aggression and inattention-overactivity behavior scales under risperidone treatment in our sample is consistent with an emerging body of literature in pediatric psychopharmacology documenting the effectiveness of risperidone in the treatment of ADHD and/or aggression in children and adolescents (e.g., Buitelaar et al. 2001; Findling and McNamara 2004). However, in addition to the pharmacotherapy, ADHD + DBD children received a cognitive-behavioral treatment program. Furthermore, our study was an open clinical trial, which meant that Rosenthal’s “expectation effect” could not be ruled out (Goodwin 1998). Thus, the improvement in the behavior scales could not be solely interpreted in the light of risperidone treatment. In addition, behavioral improvement might interact with neuropsychological measures used in this study. Note, however, that our study found equal behavioral improvement for both subscales of the IOWA Conners rating scale, although it has been demonstrated that cognitive-behavioral therapy is less effective in reducing overactive and inattentive behavior than aggressive symptoms (Pelham et al. 1998; Kutcher et al. 2004). So, it seems to be unlikely at least that the behavioral improvement observed is primarily due to the cognitive behavioral treatment procedure.

Sedation could be a troublesome side effect (Buitelaar 2000; Eapen and Gururaj 2005), and we assumed that sedation may particularly affect performance in a computerized sustained attention task. It was hypothesized that the sedative effect of risperidone might cause a cognitive slowing in this attentional task. However, no negative influence of the risperidone medication could be detected in our study. Nevertheless, the results are in agreement with previous research on children with subaverage IQ, demonstrating that cognitive functions were not affected by risperidone treatment (Snyder et al. 2002; Turgay et al.
The sedative and amnestic effects of psychotropic drugs occurred mainly after the first administrations (Barrett et al. 2004). In addition, the doses of risperidone prescribed for children with DBD are much lower than those typically administered to schizophrenic patients who had been included in the majority of studies. It is also possible that the sedative effects of risperidone might disappear after a few weeks (Aman et al. 2002) or that the duration of the neuropsychological testing was too short to assess sedation effects. However, in summary and in agreement with previous research (e.g., Frazier et al. 1999; Findling et al. 2000; Aman et al. 2002), our data demonstrate that the efficacy of risperidone in treating aggressive behavior in children was not due to the sedative effect of antipsychotics as had been hypothesized by other authors (e.g., Campbell et al. 1992).

Furthermore, no changes were found to the supervisory attentional system and selectivity of attention during risperidone treatment. This is in line with a recent study by Snyder et al. (2002) and Turgay et al. (2002). In their study of more than 100 children with subaverage IQ, no deterioration of Verbal Learning Test and Continuous Performance Test abilities could be detected during risperidone treatment.

Taken together, risperidone seems to be useful in treating impulsivity and aggression in children and adolescents with ADHD + DBD and average IQ, and we did not find any negative impact on attentional functions. However, more importantly, although risperidone reduces ADHD symptoms in behavioral terms (see also Correia Filho et al. 2005), it does not show the same positive cognitive effects as produced by stimulants. Stimulants have been demonstrated to reduce significantly deficits in vigilance, within-subject variability, and impulsivity in laboratory tasks (e.g., Scheres et al. 2003; Konrad et al. 2004). So, our results indicate that the behavioral and cognitive effects of risperidone might be even more divergent than has been previously described for stimulants in ADHD (e.g., Solanto 2002). Additional stimulant treatment might be useful to treat the core attentional deficits in children with ADHD + DBD. In line with this, Aman and colleagues (2004) demonstrated that the addition of risperidone to a psychostimulant treatment resulted in significantly better control of hyperactivity than was achieved with stimulant treatment alone, without causing any increase in adverse events. However, to examine the differential effects of stimulants and risperidone in children with ADHD and DBD, comparative studies with either stimulants and risperidone or treatment with risperidone alone are needed.

In conclusion, the present study found that risperidone did not affect attention in children with ADHD + DBD and normal IQ. This interesting finding suggests that risperidone does not decrease attentional functions in children with ADHD + DBD with average intelligence at dosages below 2.5 mg. However, like all antipsychotics, risperidone should be used with caution, and any risks associated with the use of risperidone need to be carefully weighed up against the poor prognosis of the untreated disruptive behavior disorder.

**DISCLOSURES**

Drs. Günther, Herpertz-Dahlmann, Jolles, and Konrad have no financial ties or conflicts of interest to report.

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Address reprint requests to:

Thomas Günther, Ph.D.
Department of Child and Adolescent Psychiatry
University Hospital Aachen
Neuenhofer Weg 21
D – 52074 Aachen, Germany

E-mail: thomas.guenther2@post.rwth-aachen.de