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Abstract

Objective: Oppositional Defiant Disorder (ODD) is highly prevalent in Attention-Deficit/Hyperactivity Disorder (ADHD) and may account for inconsistencies in findings on neurocognitive functioning in ADHD. Our aim was to assess cool and hot executive functioning (EF) and temporal processing in ADHD with and without comorbid ODD to elucidate the effects of comorbid ODD. **Method:** ADHD-only (n = 82), ADHD + ODD (n = 82), and controls (n = 82), with mean age 16 years (SD = 3.1), matched for age, gender, IQ, and ADHD type (clinical groups) were assessed on cool EF (inhibition, working memory), hot EF (reinforcement processing, emotion recognition), and temporal processing (time production and reproduction). **Results**: Individuals with ADHD + ODD showed abnormalities in inhibition, working memory, facial emotion recognition, and temporal processing, whereas individuals with ADHD-only were solely impaired in working memory and time production. **Conclusion:** Findings suggest that ODD carries a substantial part of the EF deficits observed in ADHD and contrast with current theories of neurocognitive impairments in ADHD. *(J. of Att. Dis. 2020; 24(9) 1317-1329)*

Keywords

ADHD, ODD, comorbidity, executive functioning, temporal processing, emotion processing

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common childhood-onset psychiatric disorders and is associated with a range of deficits in neurocognitive functioning. Specifically, neurocognitive abnormalities in executive functioning (EF) and temporal processing have been intensively studied and have become central to leading theories on ADHD (Castellanos & Tannock, 2002; De Zeeuw, Weusten, van Dijk, van Belle, & Durston, 2012; Sonuga-Barke, Bitsakou, & Thompson, 2010). EF is the sum of neurocognitive processes that maintain an appropriate problem-solving set to attain a goal (Pennington & Ozonoff, 1996; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). A well-known distinction in EF is that between cool and hot EF. Cool EF refers to goal-directed and problem-solving behaviors, as well as self-regulation, not involving affective or motivational aspects. Two functions central to cool EF are inhibition and working memory (Diamond, 2013). In contrast, hot EF is characterized by affective and motivational aspects of cognitive processing, such as reinforcement learning and emotional processing (V. A. Anderson, Jacobs, & Anderson, 2008; Blair & Lee, 2013; Kerr & Zelazo, 2004; Zelazo & Carlson, 2012). The neurocognitive domain of temporal processing is the ability to order sequential events in time and to create rhythms by using information from time perception and (re)production (Castellanos & Tannock, 2002; Ivry, 1996). However, even though abnormalities in aforementioned domains have been repeatedly reported in ADHD, findings remain inconsistent.

Individuals with ADHD show high levels of comorbid Oppositional Defiant Disorder (ODD), with up to 60% of clinically referred children with ADHD qualifying for a

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diagnosis of comorbid ODD (Connor & Doerfler, 2008; Quay, 1965, 1993). Compared with individuals with only ADHD or ODD, individuals with ADHD + ODD show an earlier age of symptom onset, exhibit more physical aggression and delinquency, show more functional impairments, and have a considerably worse future prognosis (N. E. Anderson & Kiehl, 2012; Biederman et al., 2008; Loeber, Burke, Lahey, Winters, & Zera, 2000). These findings have raised the question whether ADHD with comorbid ODD can be seen as a combination of the symptoms of ADHD and ODD or should be considered a separate disorder, with familiality studies seemingly supporting the latter (Christiansen et al., 2008; Petty et al., 2009). However, studies with a specific focus on ADHD with comorbid ODD are scarce, making it difficult to verify this claim. In addition, the high comorbidity between ADHD and ODD may have confounded previous studies into ADHD, given that ODD is also associated with abnormalities in neurocognitive functioning (Hobson, Scott, & Rubia, 2011; Sergeant, Geurts, & Oosterlaan, 2002). Surprisingly, many of the previous neurocognitive studies in ADHD did not address ODD comorbidity. Therefore, it is unclear whether previous findings truly reflect neurocognitive dysfunction in ADHD or whether the reported abnormalities actually relate to comorbid ODD.

In terms of cool EF, a meta-analysis on inhibition deficits showed medium- to large-sized impairments in ADHD and small- to medium-sized impairments for ADHD + ODD and ODD, implying abnormalities in inhibition being strongest in groups with only ADHD (Lipszyc & Schachar, 2010). However, this meta-analysis only investigated results from the Stop Signal task and reported a publication bias for both ADHD with and without comorbid ODD. For working memory, recent meta-analyses showed large working memory deficits for children with ADHD that persist into adulthood (Alderson, Kasper, Hudec, & Patros, 2013; Kasper, Alderson, & Hudec, 2012). Studies in ADHD + ODD groups are scarce and report both absence and presence of working memory abnormalities (Burt, McGue, & Iacono, 2009; Hicks, South, Dirago, Iacono, & McGue, 2009; Saarinen, Fontell, Vuontela, Carlson, & Aronen, 2015; Walden, McGue, Lacono, Burt, & Elkins, 2004). Only two studies investigated working memory in ODD and found the disorder to be associated with a working memory deficit (Rhodes, Park, Seth, & Coghill, 2012; Sergeant et al., 2002). Taken together, this leaves open the possibility that abnormalities in both domains of cool EF in ADHD + ODD are most strongly related to ADHD, and that (comorbid) ODD may be not or only weakly associated with these abnormalities.

In terms of the reinforcement processing domain of hot EF, a preference for smaller immediate rewards over larger delayed rewards is generally reported in individuals with ADHD, although a substantial amount of these studies did not account for the possible effects of comorbid ODD (for a review, see Luman, Tripp, & Scheres, 2010). For ADHD + ODD, only one study investigated reinforcement processing and reported an association with larger performance improvements in the face of rewards compared with ADHD-only and controls, implying that a heightened sensitivity to reward might be carried by ODD rather than by ADHD (Luman et al., 2009). This preference for smaller immediate rewards over larger delayed rewards was also found for ODD, in addition to a decreased sensitivity to penalty compared with controls (Humphreys & Lee, 2011; Loeber, Slot, Van der Laan, & Hoeve, 2008; Matthys, Vanderschuren, & Schutter, 2013). Concluding, it may be that comorbid ODD negatively influences reinforcement processing in ADHD, but the scarcity of studies with a focus on ADHD + ODD calls for more research in this group.

In terms of the emotion recognition domain of hot EF, ADHD has been associated with abnormalities (Da Fonseca, Seguier, Santos, Poinso, & Deruelle, 2009; Pelc, Kornreich, Foisy, & Dan, 2006; Sinzig, Morsch, & Lehmkuhl, 2008; Sjowall, Roth, Lindqvist, & Thorell, 2013; Yuill & Lyon, 2007). However, only two studies assessed emotion recognition in ADHD-only groups. One of these two studies showed abnormalities in emotion recognition due to the inability to correctly focus attention (Cadesky, Mota, & Schachar, 2000), whereas the other did not show any abnormalities (Schwenck et al., 2013). Only one study investigated an ADHD + ODD sample and showed abnormalities in emotion recognition compared with controls (Downs & Smith, 2004). In contrast, for individuals with ODD, abnormalities in emotion recognition have been repeatedly studied and reported (Loeber et al., 2008; Matthys, Vanderschuren, Schutter, & Lochman, 2012). In summary, it seems plausible that previously reported abnormalities in emotion recognition in ADHD may be accounted for by (comorbid) ODD rather than by ADHD, but more studies in ADHD-only and ADHD + ODD are needed.

In the domain of temporal processing, including time estimation and time (re)production, several studies have reported abnormalities for ADHD (for a review, see Noreika, Falter, & Rubia, 2013). However, so far only one study investigated temporal processing abnormalities for ADHD + ODD and showed that these were more pronounced in ADHD + ODD compared with ADHD-only (Luman et al., 2009). This is in line with other studies that report an association between aggression and a bias to perceive time to elapse more quickly (Dougherty et al., 2007). To conclude, it is unclear whether the findings of temporal processing deficits in ADHD are confounded by the presence of comorbid ODD and the one study on comorbid ODD suggests that ADHD + ODD is associated with at least similar, and likely more severe, abnormalities in temporal processing compared with ADHD-only, conceivably due to both disorders carrying temporal processing deficits.

The aim of this study was to elucidate the effects of comorbid ODD on neurocognitive functioning in ADHD and investigate whether the heterogeneity in previous ADHD studies may be due to comorbid ODD. To this end, individuals with ADHD without ODD (ADHD-only), individuals with ADHD + ODD, and typically developing controls were compared on cool EF, hot EF, and temporal processing. Improving on previous work, groups were matched on age, gender, IQ, and ADHD type (clinical groups only) to control for the uncalled effects of these variables, as (a) neurocognitive performance develops with age (Best & Miller, 2010; Uekermann et al., 2010); (b) lower IQ scores, as seen in ADHD and ODD, are related to worse neurocognitive performance (Loeber et al., 2008); (c) gender is associated with differences in ODD comorbidity rates in ADHD (heightened levels in males; Skogli, Teicher, Andersen, Hovik, & Oie, 2013), as well as in EF performance (females show better working memory and emotion recognition; V. A. Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; Skogli et al., 2013); and (d) different ADHD types (predominantly inattentive, predominantly hyperactive/impulsive, and combined) express specific abnormalities in neurocognitive functioning (Adams, Derefinko, Milich, & Fillmore, 2008; Shuai, Chan, & Wang, 2011). We hypothesized that (a) abnormalities in cool EF would be more strongly associated with ADHD than with comorbid ODD, and therefore equally pronounced in both ADHD-only and ADHD + ODD (Lipszyc & Schachar, 2010; Luman et al., 2009); (b) abnormalities in hot EF would be more strongly associated with comorbid ODD than with ADHD, and therefore more pronounced in ADHD + ODD than in ADHD-only (Matthys et al., 2012); and (c) abnormalities in temporal processing would be associated with both ADHD and comorbid ODD and therefore more pronounced in ADHD + ODD than in ADHD-only (Luman et al., 2009; Noreika et al., 2013).

Method

Participants

A total of 246 participants took part in this study, including (a) participants with ADHD + ODD (n = 82), (b) participants with ADHD-only (n = 82), and (c) typically developing controls (n = 82). Groups were one-to-one matched on age, gender, IQ, and ADHD type (clinical groups only). The mean age was 16 years (SD = 3.1 years). Further group characteristics are shown in Table 1.

Participants were selected from the NeuroIMAGE cohort (Von Rhein et al., 2015). Inclusion criteria for the current study that applied to all participants were European Caucasian descent, IQ ≥ 80 (as estimated with the Vocabulary and Block Design subtests of the Wechsler Intelligence Scale for Children–III [WISC-III] or Wechsler Adult Intelligence Scale–III [WAIS-III], depending on the participant's age), no diagnosis of autism, Asperger's, anxiety disorder, depression, epilepsy, general learning difficulties, brain disorders, or known genetic disorders (such as Fragile X syndrome or Down syndrome). Furthermore, typically developing controls were not allowed to have a past or current diagnosis of ADHD, ODD, or any other psychiatric disorder. Individuals in the ADHD + ODD group were only allowed to have an ADHD diagnosis and comorbid ODD, whereas individuals in the ADHD-only group were only allowed to have an ADHD diagnosis. A total of 1,069 participants contributed data to NeuroIMAGE: 751 participants from ADHD families and 318 participants from control families (Von Rhein et al., 2015). ADHD families consisted of participants in the ADHD-only or ADHD + ODD group and their biological brothers or sisters, control families consisted of participants in the control group and their biological brothers or sisters. Of all these participants, 82 participants were diagnosed with both ADHD and ODD and met inclusion criteria. These participants were one to one matched to typically developing controls and to participants with ADHD-only on gender, age (≤1 year), full-scale estimated IQ (≤10 points), and ADHD type (for clinical groups), resulting in a total of 246 participants in the study.

Diagnostic Assessment

Diagnostic assessment of all participants included the comprehensive assessment of ADHD and ODD symptoms (Von Rhein et al., 2015). To determine ADHD and ODD diagnoses, participants were assessed using the Dutch translation of the Kiddie–Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997). In addition, each child was assessed with a teacher rating (Conners' Teacher Rating Scale–Revised: Long version [CTRS-R:L]; Conners, Sitarenios, Parker, & Epstein, 1998, applied for children <18 years) or a self-report questionnaire (Conners' Adult ADHD Rating Scales–Self-Report:Long Version [CAARS-S:L]; Conners, Erhardt, & Sparrow, 1999, applied for children ≥18 years). The CTRS-R:L assesses both ADHD and ODD symptoms, whereas the CAARS-S:L assesses only ADHD symptoms. For participants using medication, ratings were done of children's functioning off medication.

For ADHD, a diagnostic algorithm was applied to combine symptom counts on the K-SADS and CTRS-R:L (for participants <18 years) or CAARS-S:L (for participants ≥18), both providing operational definitions of ADHD defined by the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV)* (American Psychiatric Association, 2000). Participants with ADHD were required to obtain a combined symptom count of ≥6 symptoms of hyperactive/impulsive behavior and/or inattentive behavior, provided they (a) met the *DSM-IV* criteria for pervasiveness and impact of the

Table 1. Group Characteristics.

Measure	ADHD + ODD (*n* = 82)		ADHD-only (*n* = 82)		TD (*n* = 82)		Group comparisons
	M	*SD*	*M*	*SD*	*M*	*SD*	
Age (years)	16.3	3.1	16.3	3.0	16.1	3.3	*ns*
IQ	97.5	11.2	96.9	11.0	98.3	7.3	*ns*
Gender (% male)	67	—	67	—	67	—	*ns*
SES (average of both parents)[a]	11.3	2.1	11.3	2.2	12.8	2.5	TD > ADHD, ADHD + ODD**
ADHD type (I/HI/C)	29/4/49	—	29/4/49	—	—	—	*ns*
ADHD total symptoms[b]	18.9	5.9	18.2	6.3	0.7	1.0	ADHD, ADHD + ODD > TD**
Hyperactive symptoms[b]	8.5	4.0	8.0	4.0	0.3	0.6	ADHD, ADHD + ODD > TD**
Inattentive symptoms[b]	10.4	3.1	10.2	3.5	0.4	0.8	ADHD, ADHD + ODD > TD**
ODD symptoms[b]	5.2	1.1	0.4	0.9	0.0	0.0	ADHD + ODD > ADHD > TD**
Global functioning score[c]	5.9	1.0	6.2	1.2	8.9	0.3	TD > ADHD > ADHD + ODD***

Note. ADHD = attention deficit hyperactivity disorder; ODD = oppositional defiant disorder; TD = typically developing; SES = socioeconomic status; I = predominantly inattentive type; HI = predominantly hyperactive-impulsive type; C = combined type; K-SADS-PL = Kiddie–Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version.
[a]As measured using the average level of maternal and paternal education (see Buis, 2010)
[b]As measured using the combination of K-SADS-PL and Conners' scales Total, Inattentive, Hyperactive/Impulsive.
[c]As measured using the Global Assessment Scale score of K-SADS-PL.
**$p < .01$. ***$p < .001$.

disorder (K-SADS), (b) showed an age of onset before 12 (K-SADS), and (c) received a $T \geq 63$ on at least one of the DSM ADHD scales (Total, Inattentive behavior, Hyperactive/Impulsive behavior) on either one of the Conners' questionnaires. Likewise, for ODD, a diagnostic algorithm was applied to combine symptom counts on the K-SADS and CTRS-R:L (for participants <18 years), both providing operational definitions of ODD defined by the *DSM-IV* (American Psychiatric Association, 2000). Participants with ODD were required to obtain a combined symptom count of ≥4 symptoms of oppositional behavior, provided they (a) met the *DSM-IV* criteria for pervasiveness and impact of the disorder (K-SADS), and (b) received a $T \geq 63$ on the DSM Oppositional behavior scale of the CTRS-R:L.

Neurocognitive Tests

Table 2 provides a description of the neurocognitive tests and accompanying dependent variables.

Cool EF: Inhibition and working memory. The Stop task was used to assess inhibition. The dependent measure was stop signal reaction time (SSRT), measuring the latency of the inhibitory process (Logan, 1994). To assess working memory, Digit Span Backwards of the WISC-III or WAIS-III (participants ≥17 years) was used. The maximum sequence length was used as dependent measure, providing a measure of verbal working memory.

Hot EF: Reinforcement processing. The Temporal Discounting task was used to assess temporal discounting of rewards (Scheres et al., 2006). The subjective value of the delayed rewards was calculated for each individual and used as dependent measure. The Motor Timing task (see the complete description below) was assessed to measure the effects of reward and penalty on performance. Median reaction time (RT) and consecutive variability of reaction times (for calculation, see Luman, Oosterlaan, & Sergeant, 2008) were compared between a feedback-only condition and two reinforcement types: reward and penalty.

Emotion recognition: Facial and vocal emotion recognition. The Identification of Facial Emotions (IFE) task was used to assess recognition of facial affect, and the Prosody (PR) task was used to assess recognition of vocal intonation. For both tasks, dependent measures were percentage of correct responses and mean RT for each of the emotions (IFE: happy, angry, afraid; PR: happy, sad, angry, afraid).

Temporal processing: Time production and time reproduction. The Motor Timing task was used to assess the accuracy and consecutive variability of time production (Van Meel, Oosterlaan, Heslenfeld, & Sergeant, 2005). Median RT and consecutive variability of response times were used as dependent measures (Luman et al., 2008). Median RT provides a direct measure of internal clock functioning that is less vulnerable to extreme data points than the mean RT, and consecutive variability of responses reflects a measure of the variability of motor output. The Timetest was used to assess the time reproduction aspect of temporal processing (Barkley, 1998). The precision of the reproduction, calculated as the aggregated absolute discrepancy between the

Table 2. Assessed Tasks.

Task and key reference	Domain	Task aim and description	Dependent measure(s)
Stop task Logan (1994)	Cool EF	*Motor inhibition.* The task consisted of go-trials and stop-trials. Go-trials required execution of a two-choice reaction time task, requiring either a left or right button press. Stop-trials were identical to go trials, but in addition a visual stop-signal was presented, instructing children to withhold their response. The delay between go- and stop-signal was dynamically adjusted to accomplish 50% successful inhibition on stop-trials	SSRT measuring the latency of the inhibition process
Digit Span Backwards Wechsler (2000 [WAIS-III], 2002 [WISC-III])	Cool EF	*Working memory.* Participants listened to a sequence of numbers and had to repeat these numbers in reverse order. The length of the sequences increased from two to eight numbers	Length of the longest successfully reproduced sequence measuring (verbal) working memory
Timetest Barkley (1998)	Temporal processing	*Time reproduction.* Participants were shown a light bulb that was illuminated during either 4-, 8-, 12-, 16-, or 20-s intervals and had to reproduce these intervals as accurately as possible by pressing the space-bar.	Mean absolute discrepancy between the presentation interval and response interval
Motor Timing Van Meel et al. (2005)	Temporal processing	*Time production.* Participants were presented a sound after which they had to press a button, producing a 1-s interval. For each trial, visual feedback was given: correct (for responses between lower and upper boundary), too short (for responses below lower boundary) or too long (for responses above upper boundary). Boundaries were set at 500 and 1,500 ms at the beginning of the task, and were dynamically adjusted during the task to accomplish 50% positive and 50% negative feedback trials	Median of response times measuring timing precision. Consecutive variability of response times measuring response consistency
	Hot EF	*Reward and punishment sensitivity.* In addition to performance feedback (neutral trials), the task contained a rewarded reinforcement type where reward (15 cents gain) was added to positive feedback on correct trials, and a penalized reinforcement type where penalty (15 cents loss) was added to negative feedback on incorrect trials	Median response time and response time variability were compared between neutral, reward, and penalty trials, measuring reinforcement sensitivity
Temporal discounting Scheres et al. (2006)	Hot EF	*Delay of gratification.* Participants had to choose between small variable rewards (1, 2, 3 or 4 cents) that were delivered immediately, and a larger reward (5 cents) that was delivered after a variable delay (0, 5, 10, 20, or 30 s). Each small immediate reward was paired twice with every delay for the large reward	Subjective value of the delayed reward measuring delay gratification
Identification of facial emotions De Sonneville (2005)	Emotion recognition	*Recognition of facial emotion.* Participants were shown a picture of an adult face displaying an emotion and had to compare the expressed emotion with the target emotion (happy, sad, and angry), by pressing a yes/no button. Pictures remained on screen until a response was given. For every emotion, a 50/50 distribution of pictures that contained the target emotion and pictures that contained a non-target emotion was shown. The sequence of the tested target emotions was randomly assigned	Percentage of correct responses and mean reaction time measuring accuracy and speed of facial emotion recognition
Prosody De Sonneville (2005)	Emotion recognition	*Recognition of vocal emotion*. Participants were presented spoken sentences by an adult with a neutral content, with a happy, sad, angry, or scared voice intonation. Participants had to identify the emotion by naming the emotion	Percentage of correct responses and mean reaction time measuring accuracy and speed of vocal emotion recognition

Note. EF = executive functioning; SSRT = stop signal reaction time.

response length and the stimulus length across all interval lengths, was used as dependent measure.

Procedure

The current study was part of a comprehensive assessment protocol encompassing phenotypic, neurocognitive, and magnetic resonance imaging (MRI) assessments (Von Rhein et al., 2015). To ensure that medication effects did not influence neurocognitive task performance, individuals on medication (70 in the ADHD-only group, 63 in the ADHD + ODD group) were assessed after a washout period. For individuals using psychostimulants, the use was discontinued for at least 48 hr before measurement to allow washout. In line with standard procedures, other medication to suppress ADHD symptoms (such as atomoxetine) was tapered off gradually to achieve washout. All neurocognitive tests were planned on one day. Standardized task instructions were used. Informed consent was signed by all participants and their parents in case of participants below 18 years (for participants below the age of 12, only parents signed informed consent), and the study was approved by the local ethics committees.

Statistical Analyses

Dependent variables were screened for outliers, which were transformed in accordance with Tabachnick and Fidell to a value one unit smaller than the most extreme non-outlier (Tabachnick & Fidell, 2001). Groups were compared on group characteristics using analysis of variance or chi-square tests. All analyses that tested differences in neurocognitive functioning between participants with ADHD + ODD or ADHD-only and typically developing controls were performed using SPSS Mixed Models (IBM SPSS Statistics version 21.0). Mixed model analyses were performed with a random intercept, with an exchangeable structure for family, to account for the hierarchical structure due to family relations (siblings) in the data. Group differences were examined as a fixed effect. To correct for multiple testing, the alpha level of the main group comparisons was adjusted according to the Bonferroni method per outcome domain (cool EF, hot EF, temporal processing). When a significant main effect of group was found, post hoc pairwise group comparisons were used to locate the nature of the group effect. We report Bonferroni adjusted results. For the Motor Timing task, an additional fixed within-subject effect of reinforcement type (neutral, reward, penalty) was tested as well as the interaction between group and reinforcement type. For this reinforcement type effect, two separate contrasts were tested comparing (a) reward with feedback-only trials and (b) penalty with feedback-only trials. Effect sizes are reported in terms of Cohen's f^2, which indexes the independent effect sizes of variables of interest within a multivariate model that includes other variables (Selya, Rose, Dierker, Hedeker, & Mermelstein, 2012).

For the Stop task, data were available for 54% of the 246 participants due to the task being assessed in a subsample participating in an MRI scanning session. For other tasks, there were some missing data (7% Motor Timing, Temporal Discounting; 8% Timetest; 12% IFE; 15% PR) due to technical issues (e.g., software licensing, voice recognition problems). Missing data were randomly distributed over the three groups. Furthermore, excluding the participants with missing data did not affect the group comparisons (see Table 1).

Results

Table 3 shows an overview of the results of the group comparisons on all neurocognitive tests. As shown in Table 1, the two clinical groups did not differ in terms of number of ADHD total, hyperactive, or inattentive symptoms, or in socioeconomic status (SES). However, both clinical groups showed lower SES compared with typically developing controls. Furthermore, all differences as reported below were replicated when covarying for SES (data not shown).

Cool EF

Inhibition: Stop task. Groups differed on SSRT, $F(2, 128) = 4.39$, $p = .014$, $f^2 = .08$, with post hoc group comparisons showing larger SSRTs, indicating poorer inhibitory control, in the ADHD + ODD group compared with controls ($p = .013$). There were no differences between the ADHD + ODD group and the ADHD-only group ($p = .140$), nor between the ADHD-only group and controls ($p = 1.000$).

Verbal working memory: Digit Span Backwards. Groups differed on maximum sequence length, $F(2, 202) = 5.50$, $p = .005$, $f^2 = .05$. Post hoc group comparisons showed that both the ADHD-only ($p = .006$) and ADHD + ODD ($p = .034$) group showed shorter maximum sequence length than controls, indicating poorer verbal working memory abilities, with no differences between the two clinical groups ($p = 1.000$).

Hot EF

Motor timing - Reward/penalty. Participants responded less accurate (and thus more impulsive) in terms of median RT in reward trials than in feedback only trials, $F(1, 251) = 11.05$, $p = .001$, but the effects of reward did not differ between groups, as shown by the absence of an interaction between group and the reward contrast, $F(2, 251) = 0.24$, $p = .787$. For the penalty trial contrast there was no difference in median RT, $F(1, 252) = 0.12$, $p = .726$, nor was there an interaction between group and the penalty contrast, $F(2, 252) = 0.09$, $p = .917$. For the consecutive variability of

Table 3. Results of Group Comparisons on Neurocognitive Tests per Domain.

	ADHD + ODD			ADHD-only			TD			Main effects of group	Post hoc group comparisons (Bonferroni)[a]
Measure	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*		
Cool EF—Inhibition/Working memory											
Stop task											
SSRT (ms)	40	293	60	42	263	70	52	250	63	$F(2, 128) = 4.39$*	TD > ADHD+ODD; TD = ADHD; ADHD = ADHD+ODD
Digit Span Backwards											
Maximum sequence length	82	5.4	1.9	82	5.3	1.6	82	6.1	1.7	$F(2, 202) = 5.50$**	TD > ADHD, ADHD+ODD
Hot EF—reinforcement processing[b,c]											
Motor timing reward											
Median RT (ms)	77	14.3	68.0	76	17.8	82.6	76	22.5	56.5	$F(2, 251) = 0.24$	*ns*
Variability (ms)	77	27.1	118.1	76	17.4	98.0	76	28.9	67.4	$F(2, 250) = 0.35$	*ns*
Motor timing penalty											
Median RT (ms)	77	−0.6	77.4	76	−5.5	74.0	76	1.1	53.2	$F(2, 252) = 0.09$	*ns*
Variability (ms)	77	16.4	102.3	76	32.1	72.7	76	33.5	66.6	$F(2, 251) = 0.33$	*ns*
Temporal discounting											
Subjective value delayed reward	75	3.19	1.21	76	2.70	1.10	79	2.94	1.07	$F(2, 220) = 2.82$	*ns*
Emotion recognition—facial											
IFE correct responses (%)											
Happy	77	94.6	5.0	78	95.4	4.5	62	95.7	4.8	$F(2, 217) = 1.02$	*ns*
Angry	77	89.0	8.8	78	87.4	8.8	62	89.9	8.1	$F(2, 217) = 1.66$	*ns*
Afraid	77	90.1	9.9	78	89.0	10.5	62	91.6	8.3	$F(2, 210) = 1.51$	*ns*
IFE mean RT (ms)											
Happy	77	642	149	78	623	133	62	604	123	$F(2, 165) = 1.30$	*ns*
Angry	77	882	200	78	832	203	62	770	150	$F(2,199) = 6.29$**	TD > ADHD+ODD; TD = ADHD; ADHD = ADHD+ODD
Afraid	77	874	234	78	830	200	60	769	185	$F(2, 191) = 4.08$	*ns*
Emotion recognition—vocal											
PR correct responses (%)											
Happy	73	82.5	12.0	72	81.1	14.9	64	83.3	13.5	$F(2, 204) = 0.44$	*ns*
Sad	73	75.6	20.6	72	72.0	22.6	64	77.0	19.0	$F(2, 206) = 1.60$	*ns*
Angry	73	87.2	14.5	72	85.0	16.2	64	87.8	12.4	$F(2, 209) = 0.74$	*ns*
Afraid	70	45.8	16.9	70	44.4	19.7	63	40.1	17.8	$F(2, 196) = 1.49$	*ns*
PR mean RT (ms)											
Happy	73	3,063	643	72	3,070	624	64	2,823	617	$F(2, 192) = 3.29$	*ns*
Sad	73	3,355	940	72	3,598	965	64	3,144	946	$F(2, 184) = 3.60$	*ns*
Angry	73	2,757	554	72	2,802	487	64	2,664	503	$F(2, 185) = 1.19$	*ns*
Afraid	70	3,174	641	70	3,214	646	63	3,004	722	$F(2, 185) = 1.76$	*ns*
Temporal processing											
Motor timing neutral											
Median RT (ms)	77	979	96	76	982	89	76	994	80	$F(2, 229) = 0.61$	*ns*
Variability (ms)	77	286	124	76	273	97	76	233	90	$F(2, 191) = 5.33$**	TD > ADHD+ODD; TD = ADHD; ADHD = ADHD+ODD
Timetest											
Mean absolute discrepancy (s)	79	1.9	1.1	79	1.9	1.0	68	1.3	0.6	$F(2, 212) = 8.23$***	TD > ADHD, ADHD+ODD

Note. ADHD = attention deficit hyperactivity disorder; ODD = oppositional defiant disorder; TD = typically developing; EF = executive functioning; SSRT = stop signal reaction time; RT = reaction time; ns = not significant; IFE = Identification of Facial Emotions; PR = Prosody.

[a]Higher scores reflect better performance.

[b]For the reward and penalty contrasts, difference scores are provided (neutral–reinforcement).

[c]Results are provided for the group by condition interaction.

*$p < .05$. **$p < .01$. ***$p < .001$.

response times, individuals showed lower consecutive variability, thus responded less variable, during reward, $F(1, 250) = 14.89$, $p < .001$, and penalty trials, $F(1, 251) = 21.70$, $p < .001$, compared with feedback only trials. The effects of reinforcement did not differ between groups, as shown by the absence of an interaction between group and both the reward contrast, $F(2, 250) = 0.35$, $p = .707$, and the penalty contrast, $F(2, 251) = 0.33$, $p = .718$.

Temporal discounting. Groups did not differ in the subjective value of delayed reward, $F(2, 220) = 2.82$, $p = .062$.

Emotion Recognition

Identification of facial emotions. Groups did not differ in the percentage of correct responses during happy, $F(2, 217) = 1.02$, $p = .361$; angry, $F(2, 217) = 1.66$, $p = .193$; or afraid, $F(2, 210) = 1.51$, $p = .222$, trials. However, mean RT for angry trials did differ between groups, $F(2, 199) = 6.29$, $p = .002$, $f^2 = .07$. Post hoc group comparisons revealed that only the ADHD + ODD group showed slower mean RTs for correct responses compared with controls ($p = .002$), indicating difficulties in correctly identifying angry facial emotions. The ADHD-only group did not differ from controls ($p = .196$) or from the ADHD + ODD group ($p = .199$). For fearful trials, there appeared to be a group difference in mean RT, $F(2, 191) = 4.08$, p .018, but this effect did not survive Bonferroni correction for multiple testing. For happy trials no group differences in mean RT were present, $F(2, 165) = 1.30$, $p = .275$.

Prosody. Similar to the facial emotion recognition task, groups did not differ in percentage of correct responses for happy, $F(2, 204) = 0.44$, $p = .644$; sad, $F(2, 206) = 1.60$, $p = .204$; angry, $F(2, 209) = 0.74$, $p = .477$; or fearful, $F(2, 196) = 1.49$, $p = .227$, trials. Groups did appear to differ on mean RT during happy, $F(2, 192) = 3.29$, $p = .039$, and sad, $F(2, 184) = 3.60$, $p = .029$),vocal emotion recognition, but these effects did not survive Bonferroni correction for multiple testing. No group differences were observed for the mean RT during angry, $F(2, 185) = 1.19$, $p = .306$, or fearful, $F(2, 185) = 1.76$, $p = .174$, trials.

Temporal Processing

Time production: Motor timing task. Groups did not differ on the median RT, $F(2, 229) = 0.61$, $p = .546$, suggesting no abnormalities in the quality of time productions in the ADHD and ADHD + ODD groups. Groups did differ on consecutive variability of response times, $F(2, 191) = 5.33$, $p = .006$, $f^2 = .05$, indicating abnormalities in consistency of time productions. Post hoc group comparisons revealed again that only individuals with ADHD + ODD differed from controls ($p = .006$), showing larger consecutive variability in producing the 1-s interval. Individuals with ADHD-only did not differ from controls ($p = .058$) or from individuals with ADHD + ODD ($p = 1.000$).

Time reproduction:Timetest. Groups differed in absolute discrepancy between presentation and response interval, $F(2, 212) = 8.23$, $p < .001$, $f^2 = .08$. Both the ADHD-only ($p = .001$) and ADHD + ODD ($p = .001$) groups showed larger absolute discrepancy than controls, indicating poorer time reproduction. The two clinical groups did not differ from each other ($p = 1.000$).

Discussion

The current study investigated the effects of comorbid ODD on individuals with ADHD on key domains of neurocognitive functioning: cool EF, hot EF, and temporal processing (De Zeeuw et al., 2012; Sonuga-Barke et al., 2010; Willcutt et al., 2005). Groups were closely matched on age, gender, IQ and for the clinical groups, ADHD type. Our results showed that, compared with typically developing controls, the ADHD + ODD group exhibited more impairments in all domains than the ADHD-only group. Our findings are not in line with a number of theories of neurocognitive impairments in ADHD, as we found no evidence for the well-documented abnormalities in inhibitory control and reinforcement processing in our ADHD-only group (Castellanos & Tannock, 2002; De Zeeuw et al., 2012; Sonuga-Barke et al., 2010). This suggests that previously reported abnormalities and heterogeneity of findings in ADHD may partially be explained by the presence of comorbid ODD, rather than by heterogeneity of ADHD itself. Furthermore, our findings emphasize the importance of accounting for comorbid ODD, as individuals with ADHD-only showed fewer abnormalities in neurocognitive functioning than those with ADHD + ODD.

Our first hypothesis that ADHD would be associated with cool EF abnormalities and that ADHD would carry the abnormalities in the cool EF domain in ADHD + ODD was not confirmed by our results. Instead, we found that the ADHD + ODD group showed abnormalities in both inhibition and working memory, whereas the ADHD-only group only showed abnormalities in working memory. Thus, individuals with ADHD + ODD showed more impairments on cool EF compared with controls than subjects with ADHD-only. This suggests that the inhibitory abnormalities in the ADHD + ODD group may be caused by the presence of comorbid ODD rather than ADHD. This idea is supported by a recent study showing larger inhibitory abnormalities in an ADHD + ODD group than in an ADHD-only group (Pauli-Pott, Dalir, Mingebach, Roller, & Becker, 2014). As comorbid ODD is reported to be prevalent in up to 60% of the individuals with ADHD (Connor & Doerfler, 2008), this may partially explain the heterogeneity in previous findings of inhibitory abnormalities in ADHD (Lipszyc & Schachar, 2010).

Our second hypothesis that hot EF impairments would be related to comorbid ODD and would therefore be more pronounced in ADHD + ODD was partially confirmed. Although none of the clinical groups showed abnormalities in the reinforcement processing domain of hot EF, individuals with ADHD + ODD did show abnormalities in the emotion recognition domain. The absence of group differences between both clinical groups and controls in reinforcement

processing was not in line with our hypothesis, as previous studies did show impairments in this domain (Humphreys & Lee, 2011; Loeber et al., 2008; Luman et al., 2010; Matthys et al., 2013). The absence of group differences on both the motor timing and temporal discounting task might be due to the relatively low amount of money that we used to manipulate reinforcement type. A recent review showed that improved task performance in ADHD was especially evident with high intensities of reinforcement (Modesto-Lowe, Chaplin, Soovajian, & Meyer, 2013). Compared with the amounts of money used in the studies reported in the review of Modesto-Lowe et al. (2013), the amounts of money used in our motor timing task were fairly low (1-5 eurocent). In our temporal discounting task, both the difference between immediate (1 eurocent) and delayed (2-5 eurocent) rewards as well as the maximum possible total gain were smaller compared with previous studies (Scheres et al., 2006). An explanation in terms of the intensity of reinforcement for the absence of group differences on our measures of reinforcement processing is further supported by a recent study into the effects of maximum total gain and reward magnitude in individuals with ADHD. That study showed no abnormalities in temporal discounting with relatively small reward magnitudes compared with relatively large reward magnitudes (Scheres, Tontsch, Thoeny, & Kaczkurkin, 2010).

The abnormalities in angry facial emotion recognition for the ADHD + ODD group were not reflected in lower levels of accuracy, but in slower reaction times. This fits with previous studies reporting similar problems in individuals with ODD (Collin, Bindra, Raju, Gillberg, & Minnis, 2013; Loeber et al., 2008). The absence of emotion recognition abnormalities in the ADHD-only group was expected and is in line with the study by Schwenck (2013) that showed no abnormalities in an ADHD-only group. The lack of abnormalities for the ADHD + ODD group on vocal emotion recognition was not expected, but may have been the result of our use of adult voices and not child voices. A study in children with ADHD compared vocal emotion recognition using child and adult voices and showed only abnormalities using child voices (Cadesky et al., 2000). Taken together, our findings support our hypothesis that comorbid ODD, and not ADHD, is associated with abnormalities in emotion recognition.

Our third hypothesis, that individuals with ADHD + ODD would show more abnormalities in the temporal processing domain than individuals with ADHD-only compared with controls (Luman et al., 2009; Noreika et al., 2013), was confirmed by our results. We found that the ADHD + ODD group showed abnormalities in both time production and reproduction compared with controls. In contrast, the ADHD-only group only showed abnormalities in time reproduction compared with controls. Hence, individuals with both disorders appear to show a double burden of temporal processing abnormalities.

In contrast to our hypotheses that the ADHD-only group would show abnormalities on inhibition and reinforcement processing, we found no differences between this group and the control group on these domains. This may have been due to a normalization in these EF domains in individuals with ADHD as they grow older, as stated in the maturational delay theory stating (Rubia, 2007; Shaw et al., 2011; Sripada, Kessler, & Angstadt, 2014). Individuals in our sample were on average 16 years old, whereas most previous studies into ADHD have used samples of children in the age range between 8 and 12 years and to a far lesser extend adolescents. Indeed, a previous study of our group on inhibition in a sample partially overlapping with the current study seems to confirm a maturational delay for ADHD, as that study did report inhibitory abnormalities (Rommelse et al., 2008). Furthermore, reinforcement sensitivity has been found to develop with age, with younger adults showing lower reinforcement sensitivity than children (Nigg & Breslau, 2007). For delay aversion in ADHD, a recent comprehensive meta-analysis showed a transition period around puberty, when deficits that are present in younger individuals with ADHD seem to disappear (Pauli-Pott & Becker, 2015).

Compared with controls, the comorbid group showed a greater diversity of neurocognitive impairments than the ADHD-only group. We found that individuals with ADHD + ODD showed impairments in all cool EF tasks and temporal processing, as well as in the emotion recognition domain of hot EF. However, we found no evidence for impairments in reinforcement. In contrast, individuals with ADHD-only showed abnormalities only on half of the cool EF tasks and no abnormalities in any of the hot EF domains. The specificity of an impairment in emotion recognition for ADHD + ODD implies that neurocognitive testing may be of value in distinguishing between ADHD-only and ADHD + ODD. However, studies documenting the diagnostic accuracy of such testing would be needed. Interestingly, the differences in neurocognitive functioning paralleled differences observed in terms of global functioning. Even though both groups showed similar ADHD symptom levels, individuals with ADHD + ODD showed worse scores in terms of global functioning than individuals with ADHD-only (see Table 1). Possibly, worse neurocognitive functioning in individuals with ADHD + ODD may translate into cognitive and social difficulties in settings such as home and school, which may explain their worse outcomes in terms of global functioning. However, we did not find any strong correlations between neurocognitive and global functioning, so this should be further investigated.

A strength of the current study is the large, well-defined sample, matched on important possibly confounding characteristics. Furthermore, we assessed an extensive battery of neurocognitive tests. A possible limitation is that groups differed on SES that has been found associated with difficulties

in EF (Pettersson et al., 2015). However, there was only a difference in SES between both clinical groups and controls. Therefore, differences in neurocognitive functioning between both clinical groups and controls cannot be attributed to differences in SES. Moreover analyses covarying for SES replicated all group differences. To further clarify differences and specificity of neurocognitive abnormalities in ADHD-only and ADHD + ODD, future studies should include an ODD only group. This would clarify whether ADHD + ODD is indeed, as our findings suggest, the accumulation of abnormalities in neurocognitive functioning associated with both ADHD and ODD, or that ADHD + ODD should be considered as a separate disorder as has been reported by family study data (Petty et al., 2009).

In summary, our results support the idea that ADHD with comorbid ODD is a more severe type of ADHD in terms of neurocognitive functioning (cool EF, hot EF, emotion recognition, and temporal processing). For cool EF and temporal processing, individuals with ADHD + ODD showed abnormalities on all tests, whereas individuals with ADHD-only showed abnormalities only on half of these tests, compared with controls. Abnormalities in facial emotion recognition were specific for comorbid ODD. Our findings clearly indicate that future studies should carefully account for comorbid ODD. Moreover, our findings challenge findings from previous studies that did not account for comorbid ODD and, by extension, weaken the support for current theories on neurocognitive impairments in ADHD (Castellanos & Tannock, 2002; De Zeeuw et al., 2012; Sonuga-Barke et al., 2010).

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