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Callous unemotional traits in children with disruptive behavior disorder: Predictors of developmental trajectories and adolescent outcomes

Pietro Muratori [a,*], John E. Lochman [b], Azzurra Manfredi [a], Annarita Milone [a], Annalaura Nocentini [c], Simone Pisano [d], Gabriele Masi [a]

[a] IRCCS Fondazione Stella Maris, Scientific Institute of Child Neurology and Psychiatry, Calambrone, Pisa, Italy
[b] The University of Alabama, Tuscaloosa, AL, USA
[c] Department of Sciences of Education and Psychology, University of Florence, Firenze, Italy
[d] Department of Mental and Physical Health and Preventive Medicine, Child and Adolescent Psychiatry Division, Second University of Naples, Italy

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ABSTRACT

The present study investigated trajectories of Callous Unemotional (CU) traits in youth with Disruptive Behavior Disorder diagnosis followed-up from childhood to adolescence, to explore possible predictors of these trajectories, and to individuate adolescent clinical outcomes. A sample of 59 Italian referred children with Disruptive Behavior Disorder (53 boys and 6 girls, 21 with Conduct Disorder) was followed up from childhood to adolescence. CU traits were assessed with CU-scale of the Antisocial Process Screening Device-parent report. Latent growth curve models showed that CU traits are likely to decrease linearly from 9 to 15 years old, with a deceleration in adolescence (from 12 to 15). There was substantial individual variability in the rate of change of CU traits over time: patients with a minor decrease of CU symptoms during childhood were at increased risk for severe behavioral problems and substance use into adolescence. Although lower level of socio-economic status and lower level of parenting involvement were associated to elevated levels of CU traits at baseline evaluation, none of the considered clinical and environmental factors predicted the levels of CU traits. The current longitudinal research suggests that adolescent outcomes of Disruptive Behavior Disorder be influenced by CU traits trajectories during childhood.

1. Introduction

Disruptive Behavior Disorders (DBDs), including Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD), are serious mental disorders associated with a host of social, emotional, and behavioral problems, both current and later emerging, with high costs for the community (Kolko et al., 2009). In order to reduce the apparent heterogeneity of DBDs, psychopathic traits have been proposed as a relevant factor in subtyping conduct problems (White and Frick, 2010). The conceptualization of psychopathic traits in children typically focuses on the presence of Callous-Unemotional (CU) traits: lack of empathy and guilt, constricted affects, deceitfulness, shallow and deficient emotions (American Psychiatric Association, 2013).

Previous longitudinal studies showed that CU traits in childhood were concurrently and prospectively associated with severe conduct problems (Lynam et al., 2009; Lopez-Romero et al., 2012), and lower levels of pro-social behavior, social competence skills and emotional regulation (Viding et al., 2009; Masi et al., 2015). In addition, poorer adolescence outcomes for children with high CU traits have been reported not only in children with DBDs, but also in community samples (for a review see Frick et al. (2014)).

Although elevated levels of CU symptoms are associated with future antisocial behavior, not all youths with these symptoms in childhood continue to show them into adolescence. For this reason, several studies have examined the stability of CU traits across childhood or from childhood to early adolescence (Frick et al., 2003; Dadds et al., 2005; Obradivic et al., 2007; Fontaine et al., 2010, 2011). For instance, Fontaine et al. (2010) found that a small proportion of children have unstable levels of CU traits over time, although elevated levels of CU traits (even if unstable) represent a relevant marker for risk of adjustment problems in early adolescence.

All these previous studies focused primarily on normative or at-risk samples; specifically, no studies examined in a clinical sample the association between CU traits in childhood and later

* Correspondence to: IRCCS Stella Maris, Viale del Tirreno 331, 56018 Calambrone (Pisa) Italy.
E-mail address: pietro.muratori@fsm.unipi.it (P. Muratori).

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outcomes using a growth curve analysis. In the current study, we used the growth curve modeling to explore the trajectories of CU traits from childhood to adolescence in a clinical sample. Moreover, individual differences in growth trajectories can predict dysfunctional adolescent outcomes, and early environmental and clinical factors can predict individual differences in CU traits growth trajectories over time. In our opinion, understanding whether CU traits trajectories could be influenced by environmental and/or clinical variables may be relevant to identify possible treatment targets.

1.1. Predictors of CU traits

A number of variables emerged from previous studies as risk or protective factors for high and stable levels of CU traits during childhood, both child and environmental related. Among the former, genetic and temperamental variables, early-onset conduct problems and hyperactivity comorbidity have been reported as mostly influential; regarding environmental variables, low family's socio-economic status is the most important predictor of high CU traits (Viding et al., 2005; Fontaine et al., 2011). Further, growing evidence indicates that parenting practices may also influence the maintenance of CU traits in children over time. Although harsh and coercive discipline has been associated with conduct problems in youths with normal levels of CU traits (Pasalich et al., 2011), some studies suggest that these dysfunctional parenting practices may affect CU traits themselves (Barker et al., 2011). However, (Viding et al., 2009) showed that during the transition to early adolescence, negative parental discipline operates as a non-shared environmental risk factor for development of conduct problems, but not for the development of CU traits. Previous studies suggested also that high parental involvement is associated to a decrease in CU traits over time (Pardini et al., 2007), whereas parental monitoring may be the most relevant dimension of parenting in the adolescence period (Munoz et al., 2011).

The present study aims to explore growth trajectories of CU traits in a sample of children with DBD diagnosis referred to a mental health service. The trajectories of CU features were investigated in children followed-up from childhood to adolescence (ages 08–09 to 14–15 years), using a growth curve analysis. We firstly investigated the growth curve of CU traits and inter-individual variability. Secondly, we explored the role of several predictors of these trajectories, including socio-economic and parenting variables, baseline diagnosis (ODD or CD), comorbidity (ADHD and Mood Disorder-MD), general functioning, and additional pharmacological treatment. Finally, we included in the model clinical outcomes in adolescence. Overall, we hypothesized that a slower decrease of CU traits during childhood is associated to a higher risk for poorer clinical outcomes into adolescence (severe aggression and antisocial behaviors in early adolescence, such as externalizing symptoms, substance use and CD diagnosis).

2. Method

2.1. Participants and procedure

A sample of children firstly referred for behavioral problems to a pediatric psychiatric hospital and received a systematic evaluation. Trained child psychiatrists administered separately to parents and youths a diagnostic clinical interview, the Schedule for Affective Disorders and Schizophrenia for School- Age Children-Present and Lifetime Version (K-SADS-PL) (Kaufman et al., 1997). Cognitive abilities in all the participants were assessed with the Wechsler Intelligence Scales for Children – 3rd Ed (WISC-III) (Wechsler, 1991).

A sample of 63 children fitted the following inclusion criteria: (01) DSM-IV-TR main diagnosis of Oppositional Defiant Disorder (ODD) or Conduct Disorder (CD) according to K-SADS-PL and DSM-IV criteria; (02) a Full Scale IQ greater than 85; (03) a Child Behavior Check List externalizing score above 63; (04) Children Global Assessment Scale (C-GAS) score below 60. Exclusion criteria were the presence of acute neurological or medical disease. Four patients were lost in the follow-up, and the remaining 59 were included in the study. The same 59 children were assessed at each follow-up; they were 53 boys and 06 girls, 48 (82%) Caucasian and 11 (18%) African, 38 (65%) with ODD and 21 (35%) with CD; 18 (28%) children presented also an ADHD comorbidity. Regarding family socioeconomic status (SES), assessed with the Hollingshead and Redlich scale (1958), 19 (29%) of families resulted with low SES, and 30 (50%) with medium SES. Location of the sample was the west coast of Tuscany (Italy), urban context.

All the participants were treated with a multi-component treatment using cognitive behavioral practices (see Masi et al., 2013; 2014). The treatment lasted 15 months, organized in weekly sessions including individual psychotherapy for children and individual parent training. 21 Patients received an additional pharmacotherapy: 10 an antipsychotic, 03 a mood stabilizer, and 08 methylphenidate.

The participants were 09 years of age at the beginning of the study, and were followed-up until the age of 15 years. Data were collected at Time 01 (before treatment; 09 years old), Time 02 (at the end of the treatment: 18 months after the pretest; 10.5 years old), Time 03 (first follow up: 34–36 months after the pretest; 12 years old) and Time 04 (second follow up; 70–72 months after the pretest; 15 years old). Written consent was obtained from parents at initial enrollment and in each of the following assessments through the course of the study. The Ethical Committee of our Hospital approved the study.

2.2. Measures

To evaluate CU traits in children across time, the CU-scale of the Antisocial Process Screening Device-parent report (APSD) (Frick and Hare, 2001) was completed by parents at each assessment points. The APSD is a 20-item behavior rating scale with each item scored 0 (not at all true), 01 (sometimes true), or 02 (definitely true). A factor analysis revealed three APSD dimensions, a 07-item Narcissism dimension, a 05-item Impulsivity dimension, and a 06-item CU dimension, which could fit in both community and clinic-referred samples of children (Frick et al., 2000). The current study used the CU-subscale of parent report version of the APSD; in our sample, Cronbach α for assessment points from Time 01 to Time 04 for this scale was .77, .75, .73, and .79 respectively.

2.3. Pre-treatment predictors

All following measures were administered at the baseline assessment point:

2.3.1. Categorical diagnosis

Child psychiatrists administered separately to the patients and their parents the clinical interview K-SADS PL (Kaufman et al., 1997), which explores the presence or absence of each symptom according to DSM-IV. The rate of patient-parent K-SADS diagnosis agreement was 89%. The predictor was dichotomous variable, ODD vs CD. Comorbidity with ADHD or MD was also considered.

2.3.2. Level of functioning

Children's Global Assessment Scale (C-GAS) (Shaffer et al., 1983) was used to describe the severity of functional impairment. The clinician coding the CGAS on the basis of your patient's worst

level of functioning in the past three months by selecting the lowest level, which describes his/her functioning on a hypothetical continuum of health-illness; scores above 70 indicate normal functioning.

2.3.3. Family socioeconomic status

Was assessed with the Hollingshead and Redlich scale (Hollingshead and Redlich, 1958).

2.3.4. Parenting practices

The parent-reported Alabama Parenting Questionnaire (APQ – Frick (1991)) is designed to assess several parenting practices associated with the development of conduct problems in children. For the purpose of this investigation, the 03-item corporal punishment scale, the 10-item parental involvement scale and the 10-item monitoring scale were used. For each statement, parents are asked to indicate how often each behavior typically occurs in their home on a scale from 01 (never) to 05 (always). The Cronbach α for these subscales was .82, .81, and .82 respectively.

2.3.5. Use of pharmacotherapy

One month before the beginning of the multimodal treatment, patients were evaluated by clinicians unaware about the aims of the study, and, when necessary, they received a medication, continued during the whole period of multimodal treatment. Drug dosage was adjusted naturalistically during the treatment period, based on efficacy and tolerability assessed by the clinician. This predictor was dichotomous variable.

2.4. Adolescent outcomes

All following measures were administered at the final assessment point:

2.4.1. Children's behavioral problems

All patients were assessed with the Child Behavior Check List (Achenbach and Rescorla, 2001), a 118-item scale, completed by parents, with two broad-band scores designated as Internalizing Problems and Externalizing Problems. In the current study the Externalizing Problems score was used, and the Cronbach α for this scale was .81.

2.4.2. Categorical diagnosis

At last follow-up child psychiatrists administered separately to the parents and to the patients the clinical interview K-SADS PL (Kaufman et al., 1997). These clinicians were blind for the objectives of the current research. The rate of child-parent K-SADS diagnosis agreement was 86%. Only the diagnosis of CD was used in this study as an adolescent outcome.

2.4.3. Substance use

The CSAP (Center for Substance Abuse Prevention) Student Survey is a 14-item child-report questionnaire adapted from the California Student Survey (Pentz et al., 1989). The CSAP Student Survey measures students' attitudes toward, and use of, alcohol, tobacco and other drugs. Self-report survey assessments of youths' substance use have been found to be reliable and valid (MacKinnon and Dwyer, 1993). The items assessing children's use of alcohol, tobacco, or marijuana in the past month were aggregated in this study to produce the Substance Use score.

2.5. Statistical analyses

All the analyses were conducted using Mplus 7 (Muthén and Muthén, 2010). Since the variables did not show consistent values of skewness and kurtosis, Maximum Likelihood estimator was used. To avoid bias due to the limited attrition in the sample, we estimated all models using the direct maximum likelihood procedure available in Mplus. Bootstrap technique has been used given the small sample (10,000 samples). Model fit was evaluated using the maximum likelihood ratio test statistic (chi square), the root mean square error of approximation (RMSEA) and the comparative fit index (CFI). Recommended cut-off points for these measures are: for RMSEA the cut-off is .08 (Browne and Cudeck, 1993) or .06 (Hu and Bentler, 1999); for CFI the cut-off is .95 (Hu and Bentler, 1999).

The estimation and the prediction of longitudinal development of CU trajectory were analyzed through the growth latent curve model (Bollen and Curran, 2006). Within this framework, the repeated observed variables can be used to estimate the unobserved underlying trajectory defined by latent growth factors (i.e. the intercept, the slope and the quadratic factors). This model can be extended in order to evaluate whether individual variability of intercept and slope can be predicted by a set of explanatory variables. A series of latent trajectory models of increasing complexity were constructed. First, an unconditional growth model was estimated. Two models were tested and compared with each other: a linear model and a quadratic model. A linear model assumes that the development of CU is linear across time and represents a constant change over time, (i.e. CU would decrease or increase constantly across time). A quadratic model assumes that the development is not linear (i.e., CU would decrease or increase for certain groups of individuals after a period of decrease or increase). In this way, we could determine the parameterization that best fit the data. In all the models tested, we defined the intercept as CU at age 09 by fixing the factor loading relating this variable to the slope at 0. Second, a conditional model was estimated where the unconditional model was extended including the main effects of time-invariant variables. The latent growth factors with significant variability around the mean were regressed on the background variables. Third, the second model was extended testing also the direct effects of the latent growth factors on different outcomes in adolescence, controlling for the predictors. In this model the outcomes constructs were simultaneously regressed into the CU intercept and the CU linear slope factors. Covariance was freely estimated between the outcomes. Correlation table, means and standard deviations of all the variables included in the models are presented in Table 1.

3. Results

3.1. Unconditional latent growth curve

The first model tested was a linear model representing a constant change over time: to define the linear metric of time, the factor loadings for the slope were set to 0, 1, 2 and 4. The model did not fit the data well (χ^2(05)=13.977; p=.016; RMSEA=.177; CFI=.800). The second model tested was a quadratic model. The model fit the data well (χ^2 (01)=.335; p=.050; RMSEA=.000; CFI=1.00). However, given that the model showed a non–significant variance of the quadratic slope, and considering the small sample, this variance factor was fixed to 0. The final model (χ^2 (4)=5.795; p=.215; RMSEA=.08; CFI=.960) showed a significant mean of the intercept (intercept mean=5.649; t=22.535, $p<.001$), indicating that the overall group reported a positive average starting point at age 09, and a significant variance of the intercept (intercept variance=1.373; t=2.356; $p<.05$) reflecting inter-individual variability around this mean group. The mean of the slope was significant and negative (linear slope mean=-.893; $t=-3.182$; $p<.001$), showing on average a tendency to linear decrease across time. A significant variance of the slope was found

Table 1
Correlations, means and standard deviations of the measures of CU and all covariates

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. CU T01	1.00															
2. CU T02	.39	1.00														
3. CU T03	.21	.30	1.00													
4. CU T04	.10	.53	.39	1.00												
5. SES	−.28	−.10	.06	−.13	1.00											
6. Diagnosis T01 (ODD/CD)	.09	−.03	−.06	.05	−.13	1.00										
7. Pharmacotherapy	−.24	.05	.05	.16	.00	.16	1.00									
8. C_GAS	−.10	−.45	−.17	−.21	.02	−.27	−.39	1.00								
9. ADHD comorbidity	−.01	.00	.10	−.26	.13	.00	.25	−.26	1.00							
10. MD comorbidity	−.08	.27	.29	.40	−.10	.05	.37	−.28	−.21	1.00						
11. Parental corporal punishment	−.06	−.08	−.04	−.04	.04	.34	.12	−.12	−.14	.07	1.00					
12. Parental involvement	−.27	.00	−.22	.02	.00	.03	−.23	.21	−.14	.05	.07	1.00				
13. Parental monitoring	.14	−.10	.04	.14	.05	.16	.04	.07	−.10	−.01	.33	−.07	1.00			
14. Externalizing behaviors T04	.01	.24	.39	.52	−.05	.10	.31	−.26	−.11	.20	−.03	.02	.22	1.00		
15. Diagnosis CD T04	.02	.46	.44	.69	−.00	−.03	.24	−.29	−.10	.31	−.15	.02	.11	.67	1.00	
16. Substance use T04	.17	.44	.40	.66	−.20	.12	.33	−.44	.00	.35	−.07	−.16	.08	.57	.70	1.00
Mean	5.89	4.75	4.52	4.59	2.90	1.10	1.20	43.06	1.20	1.14	6.02	35.38	13.25	59.61	.46	6.51
SD	1.83	2.09	2.18	2.18	.79	.30	.40	5.86	.40	.35	1.57	2.79	3.34	7.19	.71	2.43

Notes. CU= Callous Unemotional; SES=Socio Economic Status; ODD/CD, 01= Oppositional Defiant Disorder, 02=Conduct Disorder; C_GAS= Children's Global Assessment Scale; MD= Mood Disorder comorbidity, 01=no comorbidity, 02=MD comorbidity; ADHD=Attention Deficit Hyperactivity Disorder comorbidity, 01=no comorbidity, 02=ADHD comorbidity; Pharmacotherapy, 01=no additional pharmacotherapy, 02=additional pharmacotherapy; Diagnosis CD T04, 00=no CD diagnosis, 01=CD diagnosis.

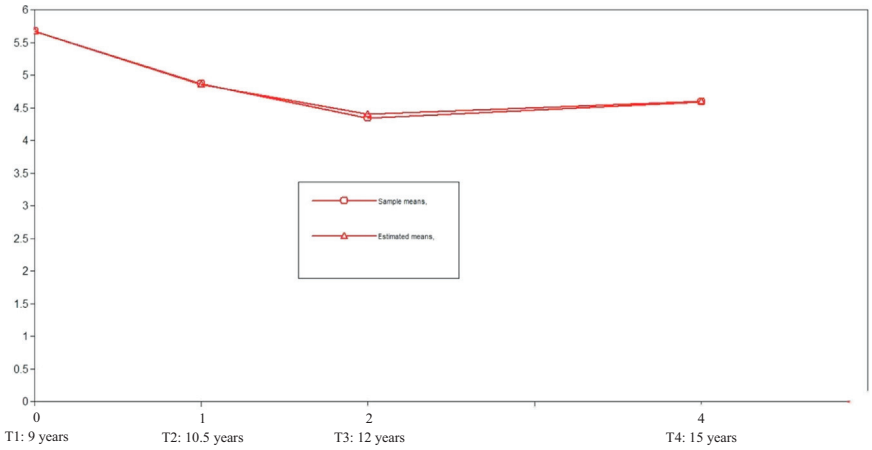

Fig. 1. Sample and estimated means for the CU trajectory. *Note*. The *y*-axis represents scores on the measure of Callous Unemotional traits.

(linear slope variance=.249; $t=1.968$; $p<.05$), showing inter-individual variability in growth over time. Finally, the mean of the quadratic factor was significant and positive (quadratic slope mean=.158; $t=2.571$; $p<.01$), showing on average a tendency to upturn from T03 to T04 beyond what is predicted by the linear decrease (see Fig. 1).

3.2. Conditional growth model with time-invariant predictors at T01

In the conditional model the growth factors with significant inter-individual variability around the mean (intercept and linear slope) were regressed on all the predictors described above (see Table 2). The model fit the data well (χ^2 (22)=13.591; p=.040; RMSEA=.080; CFI=.90). Results showed that family socio-economic status was significantly associated with intercept: higher level of socio-economic status was related to lower level of CU traits at T01. The general functioning was marginally associated with CU traits at T01: those with higher levels of functioning reported lower levels of CU at T01. Finally, regarding parenting practices, an effect of parenting involvement on CU was found at

Table 2
Conditional model with time-invariant covariates: multivariate predictors at baseline of CU growth curve trajectories.

	Intercept beta (SE)	Linear slope beta (SE)
SES	−.51 (.26)*	.21 (.14)
Diagnosis T01 (ODD/CD)	.12 (.79)	−.24 (.40)
Pharmacotherapy	–	.40 (.51)
C_GAS	−.08 (.04)****	.00(.02)
MD comorbidity	.28 (.71)	.38 (.32)
ADHD comorbidity	.27 (.59)	−.53 (.28)
Parental involvement	−.16 (.08)*	.06 (.04)
Parental corporal punishment	−.03 (.14)	−.05 (.07)
Parental monitoring	.03 (.10)	−.11 (.08)

Notes. CU=Callous Unemotional; SES=Socio Economic Status; ODD/CD, 01=Oppositional Defiant Disorder, 02=Conduct Disorder; C_GAS=Children's Global Assessment Scale; MD=Mood Disorder comorbidity, 01=no comorbidity, 02=MD comorbidity; ADHD=Attention Deficit Hyperactivity Disorder comorbidity, 01=no comorbidity, 02=ADHD comorbidity; Pharmacotherapy, 01=no additional pharmacotherapy, 02=additional pharmacotherapy.
Values are unstandardized estimates.

* $p<.05$
**** $p=.07$.

Table 3
Conditional model with time-invariant covariates and outcomes.

	Intercept beta (SE)	Slope beta (SE)	B beta (SE)
SES	−.48 (.24)*	.17 (.12)	
Diagnosis T01 (ODD/CD)	.05 (.73)	−.17 (.35)	
Pharmacotherapy	–	.30 (.24)	
C_GAS	−.09 (.04)*	−.00 (.02)	
MD comorbidity	.49 (.67)	.19 (.31)	
ADHD comorbidity	.47 (.56)	−.39 (.36)	
Parental involvement	−.16 (.07)*	.07 (.04)	
Parental corporal punishment	−.05 (.14)	−.08 (.07)	
Parental monitoring	.05 (.24)	.03 (.06)	
Intercept→Externalizing behaviors T04			1.91 (1.06)
Slope→Externalizing behaviors T04			7.72 (1.94)***
Intercept→CD Diagnosis T04			.32(.09)***
Slope→CD Diagnosis T04			.98(.17)***
Intercept→Susbtance use risk T04			.48(.14)***
Slope→Susbtance use risk T04			1.04(.24)***

Notes. CU=Callous Unemotional; SES=Socio Economic Status; ODD/CD, 01=Oppositional Defiant Disorder, 02=Conduct Disorder; C_GAS=Children's Global Assessment Scale; MD=Mood Disorder comorbidity, 01=no comorbidity, 02=MD comorbidity; ADHD=Attention Deficit Hyperactivity Disorder comorbidity, 01=no comorbidity, 02=ADHD comorbidity; Pharmacotherapy, 01=no additional pharmacotherapy, 02=additional pharmacotherapy; Diagnosis CD T04, 00=no CD diagnosis, 01=CD diagnosis.
Values are unstandardized estimates.

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

T01, meaning that higher level of parenting involvement at T01 is associated with lower levels of CU at T01. No significant effects were found for harsh parenting and monitoring.

3.3. CU growth curve model predicting outcomes in adolescence

The model predicting the outcomes (externalizing behavioral problems, CD diagnosis and risk of using substances) fitted the data well, (χ^2 (56)=62.814; p=.025; RMSEA=.050; CFI=.95) (see Table 3). Results showed a significant role of intercept and linear slope on predicting externalizing behaviors at T04, diagnosis at T04, and risk of using substances at T04. Only the effect from intercept to externalizing behavioral problems at T04 was not significant.

Higher levels of CU at T01 and a minor decrease from T01 to T03 predicted higher levels of externalizing behaviors at T04, the probability of receiving a diagnosis of CD at T04, and of using substances at T04. The model explained 37% of externalizing variance, 64% of diagnosis, and 46% of risk of substance use.

4. Discussion

The results from the current investigation provide significant insights into the developmental trajectories of CU traits during childhood in patients with DBD diagnosis referred to a mental health service for receiving a treatment. Children's levels of CU traits decreased during the treatment period, and this decrease continued during years thereafter; on the contrary, when patients became adolescents (from 12 to 15 years), the CU traits showed a decelerated decrease with a substantial stability. Previous studies indicated that CU traits more frequently tend to decrease during development, whereas an increase is significantly more rare (Pardini and Loeber, 2008; Fontaine et al., 2010). Regarding the substantial stability of CU features at the last point of the growth curve, it may raise the issue of the influence of the adolescent developmental features on CU traits. For example, increased reward sensitivity in adolescence may increase in vulnerable individuals, the proneness to antisocial behavior, using illicit means to achieve their goals, with poor sensitivity for the possible negative consequences on others (Blair, 2013).

Moreover, the current study shows that the slower rates of decrease in CU traits across time in DBD population are associated with more severe outcomes in early adolescence. Our findings indicate that patients with higher levels of CU traits at the baseline evaluation and a slower decrease of CU symptoms during childhood are at increased risk for serious externalizing behavioral problems and substance use into adolescence. Consistently with previous studies, children who maintained elevated levels of CU traits were at higher risk for severe aggression or other measures of serious antisocial adolescence outcomes, compared with children with lower or decreasing levels of CU traits (Muñoz and Frick, 2007; Obradivic et al., 2007; Rowe et al., 2010; Barker and Salekin, 2012). In the current study, children who showed a slower decrease of the CU traits were at higher risk for poorer behavioral prognosis in early adolescence, in terms of externalizing symptoms and substance use. Regarding the risk of substance abuse, our results are consistent with those of Wymbs et al. (2012), who showed that CU traits (along with CD symptoms) at grade 6th predicted substance abuse in 9th grade. Both Wymbs' and our findings may provide useful information for substance abuse prevention and interventions in at-risk youths. Overall, although this study suggests that the parent-report measure of CU traits (APSD) is a valid indicator of future risk for more severe outcomes, future studies should examine whether the findings hold with other measures of CU traits in children, including those that use different informants (e.g teachers, self-report).

The present study aimed also at examining the role of different predictors on shaping the CU-traits curve trajectory. Although lower level of socio-economic status and lower level of parenting involvement were related to elevated levels of CU traits at baseline evaluation, none of the considered clinical and environmental factors predicted the linear rates of decrease of CU traits over time. Specifically, in contrast to previous studies, higher level of parental involvement, lower level of harsh parenting and the use of additional pharmacotherapy did not predict a steeper decrease of CU during development (Pardini et al., 2007; Waschbusch et al., 2007; Barker et al., 2011; Pasalich et al., 2011). In line with Viding et al. (2005), a possible stronger influence of genetic factors than environmental ones on CU traits during the development arose from our data. But the small number of subjects in the sample prevents us from drawing firm conclusions. Future studies should continue to search for the drivers of developmental changes in CU traits.

However, a lower family socio-economic status was significantly associated with higher levels of CU traits at the baseline. A recent meta-analysis have showed that low family socio-economic status is associated with higher levels of children's antisocial behavior, and indicated that this relationship is stronger when CU traits are considered as outcome variables (Piotrowska et al., 2015).

Similarly to several previous studies, the lack of parental involvement appears associated with the presence of elevated levels of children's CU traits (for a review see Waller et al. (2013)). From a developmental psychopathology perspective, also Kochanska (1997) suggested that a supportive parent–child relationship is associated with morality development in childhood. Nonetheless, child-driven effects may contribute to such processes, with high levels of CU traits found to predict reduced parental involvement (Larsson et al., 2008; Hawes et al., 2011).

4.1. Limitations and clinical implications

The current study presents several methodological limitations, firstly the small number of participants. However, bootstrap technique allowed to assign more accuracy to sample estimates. Secondly, our study used a parent-report measure of CU traits; as a result, the entire range of affective characteristics associated with CU traits may not be adequately assessed, particularly features associated with a lack of empathy and shallow emotions in children.

However, our findings described a sample of children with DBD as primary diagnosis, treated as needed, and followed-up in an ordinary clinical setting, which may actually be one of the strengths of our study. For this reason, our findings have some meaningful clinical implications. Firstly, although the definition of CU used in this study was not identical to that of the DSM-5 definition of "lack of pro-social emotions" (American Psychiatric Association, 2013), the importance of the DSM-5 sub-typing criterion is supported by our finding that the trajectory of CU traits may identify at-risk patients, irrespective for diagnosis, comorbidity and environmental variables (Rowe et al., 2010). Children's levels of CU traits may strongly affect their psychosocial needs, and thus they must be carefully considered, along with other emotional and behavioral indicators, throughout the different periods of development. We suggest that treatment approaches DBD children would benefit from an assessment of CU traits, given their reliable association with later severe outcomes.

Overall, our findings show that many DBD children have unstable levels of CU traits, consistent with the emerging consensus that these personality traits are subject to change across development and they do not represent an un-modifiable route to psychopathy. In our sample levels of CU may be ameliorated by treatment in some children, suggesting that CU traits in DBD patients may decrease if children are treated with timely and specialized combinations of intensive behavioral and pharmacological treatments (Dadds. et al., 2012; Lochman et al., 2014; Muratori et al., 2015).

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Conflict of interest

Dr. Masi was in the advisory boards for Eli Lilly, Shire and Angelini, has received research grants from Eli Lilly and Shire, and has been speaker for Eli Lilly, Shire, Lundbeck, and Otsuka. All the other authors do not have conflicts of interest to declare.

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