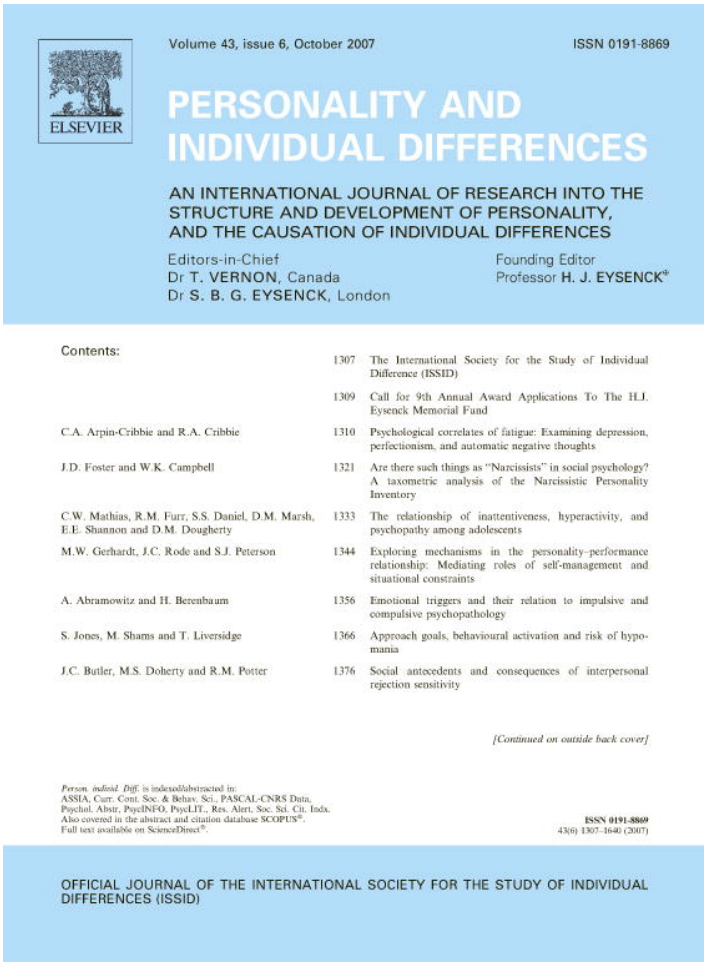

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Differential affective responses in those with aggressive versus non-aggressive antisocial behaviors

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Abstract

Affective dysfunction has been robustly tied to antisocial behavior, but little research has evaluated whether affective responses vary differentially with its aggressive (AGG) and rule-breaking (RB) subtypes. We therefore examined whether changes in negatively valenced affect (elicited via written recollection of one's best and worst life experiences) were linked to level (i.e., low, average, high) and type (i.e., AGG, RB) of antisocial behavior. The sample consisted of 288 undergraduate men. Results suggested a general trait-like association between negative affect and AGG. However, this association varied across experimental conditions. In particular, the potentiation of negative affect following an aversive task was consistently associated with AGG. RB, by contrast, demonstrated little to no association with negative affect in any condition. Such findings imply that the link between antisocial behavior and affective dysregulation is largely specific to its aggressive subtype, and does not persist to non-aggressive antisocial behavior.

Keywords: Negative affect; Antisocial behavior; Aggression; Rule-breaking; Affective dysregulation

1. Introduction

Antisocial behavior (ASB) describes actions and attitudes that violate societal norms and the personal or property rights of others. Though generally operationalized via aggressive

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and rule-breaking behaviors, emotional shallowness and callousness are also considered core components. Consistent with this conceptualization, there is strong evidence of a link between ASB and dysfunctional affective processing/regulation of negatively valenced emotions (Davidson, Putnam, & Larson, 2000; Lorber, 2004), an association that persists across the lifespan (Herpertz, Mueller, Lichterfeld, Konrad, & Herpertz-Dahlmann, 2005; Sterzer, Stadler, Krebs, Kleinschmidt, & Poutska, 2005) and extends to related constructs such as psychopathy (Deeley, Daly, Surguladze, Tunstall, & Mezey, 2006). Specifically, there appear to be functional and morphologic abnormalities in the frontal and limbic regions in those with aggressive/antisocial behaviors. As these areas of the brain play a primary role in the recognition and regulation of emotions (Davidson et al., 2000; Sterzer et al., 2005), and therefore may act to constrain affective outbursts/emotional behavior, such deficits likely increase vulnerability to aggressive and behavioral outbursts.

Importantly, however, studies of affective dysfunction have yet to fully consider differences *within* ASB. Indeed, given the pronounced heterogeneity within ASB, such studies would offer a significant refinement of current theories (Raine, 2002). Physical aggression is found largely in males and decreases precipitously from early childhood to adulthood, with only a slight increase during mid-adolescence (Stranger, Achenbach, & Verhulst, 1997). In contrast, non-aggressive rule-breaking increases sharply over the course of adolescence (Burt, Carter, McGue, & Iacono, 2007), and is found in males and females (Moffitt, 2003; Stranger et al., 1997). Factor analytic studies (Frick et al., 1993) further support this distinction, finding evidence for independent but correlated aggressive and rule-breaking dimensions within ASB. Aggression also appears to be more heritable than rule-breaking (Eley, Lichtenstein, & Moffitt, 2003; Hudziak et al., 2003), while rule-breaking may be more highly influenced by shared environmental factors (Deater-Deckard & Plomin, 1999; Eley et al., 2003) (though the magnitude of non-shared environmental influences, generally 40–50% of the variance, does not differ across sub-types).

To date, however, studies have only indirectly considered whether affective dysfunction varies by sub-type. Raine and colleagues (1997) found that low resting heart rate (a variable tied to disinhibition) at age 3 predicts aggressive but not non-aggressive ASB at age 11 (Raine, Venables, & Mednick, 1997), offering tentative (but provocative) support for differences in affective regulation across sub-type. Similarly, studies have found that aggressive ASB is more closely linked to indices of autonomic and neuroendocrine functioning than is non-aggressive ASB (Lahey, Hart, Pliszka, Applegate, & McBurnett, 1993). As low autonomic arousal and lack of autonomic responsiveness represent a form of affective deficit (Raine, 2002), such findings offer additional support for aggression-specific affective dysfunction. Finally, recent work suggests that affect dysregulation may be particularly important for precipitating aggressive behavior (Verona, Patrick, & Lang, 2002), again supporting an aggression-specific role for affective dysfunction.

The above findings provide strong circumstantial evidence that affective dysfunction may be specific to aggressive ASB. The current study sought to more explicitly evaluate this hypothesis, examining whether affective responses were differentially associated with aggressive and non-aggressive ASB. We expected that those with high levels of aggression would exhibit both higher levels of negative affect in general and a more pronounced negatively valenced affective response to an aversive task (i.e., recollection of their worst memory) than those with lower aggression, and that these associations would not persist to rule-breaking.

2. Methods

2.1. Participants

The sample consisted of 288 undergraduate men (average age 19) at a large research university in Michigan who participated in exchange for course credit or extra credit. Data were collected over the department internet using an anonymous, web-based interface. Participants were representative of the ethnic composition of the area; approximately 81% were European-American, with the remaining participants consisting of African-American (5%), Asian or Pacific Rim (9%), Hispanic/Latino (3%), and other (2%) ethnicities. Results from this sample have not previously been reported. Because participants were allowed to omit up to 10% of items on each scale, we are not missing any data.

2.2. Measures

2.2.1. Physical aggression

Participants reported on their aggressive behaviors using the Aggression Questionnaire (AQ) (Buss & Perry, 1992), a 29-item inventory of physically aggressive behaviors, verbally aggressive behaviors, hostility, and anger. The current study made use of the nine-item physical aggression scale, thereby avoiding possible predictor-criterion overlap between the AQ hostility scale and negative affect (''hostile'' is on the negative affect scale). Participants were asked to rate on a five-point scale the extent to which a series of statements generally described them. Items were averaged to index physical aggression (AGG). Higher scores reflect endorsement of more aggressive behaviors. AGG demonstrated good internal consistency reliability ($\alpha = .80$).

2.2.2. Non-aggressive, rule-breaking behaviors

Participants reported on their delinquent behaviors using the Delinquent Behavior Index (DBI) (Farrington & West, 1971), a 36-item inventory of minor (e.g., skipping school) and more serious (e.g., stealing a car) delinquent behaviors. Participants were asked how frequently they had engaged in that behavior in their lifetime (0 = never; 1 = once; 2 = more than once). Those nine items assessing aggressive acts (e.g., using a weapon in a fight) were not included. The remaining 27 items were averaged to index rule-breaking (RB). Higher scores reflect endorsement of more rule-breaking behaviors. RB demonstrated good internal consistency reliability ($\alpha = .78$).

We assessed the concurrent validity of our AGG and RB measures in an independent sample of 199 undergraduates (50% female) at a large Midwestern research university. Participants completed the AGG and RB scales described above and the Achenbach Adult Self-Report (ASR) (Achenbach & Rescorla, 2003) Rule-Breaking and Aggression Scales. We examined relations between AGG and RB and these alternate indices, performing ordinary least squares multiple regressions in which RB and AGG were simultaneously entered as predictors of each ASR scale. As seen in Table 1, AGG was the only significant predictor of the ASR Aggression scale. Both AGG and RB predicted the ASR Rule-Breaking scale. However, the contribution of RB was significantly stronger, as evidenced by the predictors' non-overlapping confidence intervals. These results thus offer confirmation that AGG and RB, as operationalized in the current study, tap distinct components of the broader construct of ASB.

Table 1
Associations between AGG, RB, and other measures of aggressive and delinquent behaviors

	Standardized beta weights	
	RB	AGG
ASR rule-breaking scale	**.53**** (.41, .65)	.25** (.13, .36)
ASR aggression scale	.12 (−.02, .26)	**.39**** (.25, .53)

Note: These validation data were collected in an independent sample of 199 undergraduates. AGG and RB represent the present study's constructs of aggressive and rule-breaking behaviors, respectively. ASR represents the Achenbach Adult Self-Report. Standardized beta weights from ordinary least square regressions are presented. The outcome-relevant predictors are highlighted in bold. $^{*}p < .05$; $^{**}p < .01$.

2.2.3. Affect

Affect was assessed at four time points using the 20-item Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). In the PANAS, positive affect (PA) and negative affect (NA) are each composed of 10 affect states. PA indicates the extent to which an individual feels enthusiastic, active, and alert, while NA captures aversive mood states, including hostility, guilt, disgust, and fear. Participants were asked to rate on a five-point scale the extent to which they were experiencing each mood state "right now, that is, at the present time". The 10 items comprising NA were averaged. The scale demonstrated good internal consistency reliability (all $\alpha > .80$).

2.3. Procedure

Participants first established baseline levels of affect via the PANAS. We then attempted to alter participants' affect by asking them to describe their most traumatic life experience in detail ("Please describe in detail your worst or most upsetting life experience (i.e., the worst thing that has ever happened to you)"). To ensure adequate description and a more thorough recollection, we followed the free response with specific follow-up questions (e.g., "What were the circumstances surrounding this experience?", "Did you think you might die or be seriously injured during this experience?"). We then asked participants to complete the PANAS for a second time. We next repeated this process for their best or most rewarding life experience, again following the free response with specific follow-up questions. Participants then completed the PANAS for a third time. Lastly, participants completed the AQ and the DBI, after which they completed the PANAS for a fourth and final time. Roughly half the sample ($n = 142$) completed the experiment in the above order (i.e., baseline, worst experience, best experience, questionnaires). For the remaining participants ($n = 146$), affect induction was reversed (i.e., baseline, best experience, worst experience, questionnaires).

2.4. Data analyses

Two sets of analyses were performed. First, correlations were computed to determine whether and how AGG and RB, as measured continuously, were associated with NA at each of the four assessments. To evaluate whether associations with NA varied between AGG and RB, correlations were then statistically compared across sub-type. Analyses were conducted for both full

AGG and RB scores and their residuals (e.g., AGG and RB were entered as a predictor of the other in two regressions. The residuals comprise that variance remaining in a given sub-type once we statistically removed overlap with the other). The residuals are particularly important as they index variance unique to each subtype (because sub-types are correlated at .37, $p < .01$, the full scores are not as "pure").

We next examined mean differences in NA within and across sub-type. Full and residual AGG and RB were first independently trichotomized, such that individuals with scores in the bottom, middle, and top tertiles were, respectively, designated as those with low, average, and high levels of these behaviors. We then conducted a repeated measures analysis of variance (ANOVA) with NA as the within-subject factor and AGG and RB as the between-subject factors. These analyses allowed us to evaluate not only whether we were able to successfully manipulate affect, but also whether affective responses varied differentially by sub-type (i.e., did those high in aggression have a stronger NA response to the recollection of their worst memory than those lower in aggression?). Analyses were conducted first on the full scores and then repeated on the residuals. Huynh–Feldt corrected p values are reported for all within-subject effects to correct for possible violations of sphericity.

3. Results

As seen in Table 2, AGG and RB were both generally normally distributed in the current sample, with some participants exhibiting quite high levels of these behaviors. The broad range of AGG and RB behaviors endorsed suggests that we are able to meaningfully study these phenomena in our normative college sample. Moreover, these rates are quite consistent with the high frequency of these behaviors in late-adolescence, particularly in males (i.e., roughly 25% of late adolescents display clinically significant antisocial behavior; Moffitt, 2003). NA also varies across the sample, particularly following recollection of one's worst memory.

3.1. Correlations

As seen in Table 3, AGG was positively associated with NA across all assessments. The association with NA following recollection of the worst memory, and to a lesser extent after the questionnaires, appeared to be particularly salient. RB, by contrast, displayed variable patterns of

Table 2
Descriptive statistics

	Mean	SD	Observed range	Possible range	Skew
Aggression	2.18	0.72	1.0–4.9	1.0–5.0	1.06
Rule-breaking	0.46	0.28	0.0–1.4	0.0–2.0	0.73
Negative affect					
Baseline	1.51	0.52	1.0–3.5	1.0–5.0	1.41
After recollection of worst memory	1.72	0.72	1.0–4.3	1.0–5.0	1.14
After recollection of best memory	1.38	0.49	1.0–3.5	1.0–5.0	1.99
After questionnaires	1.53	0.61	1.0–3.8	1.0–5.0	1.41

Table 3
Correlations

Negative affect (NA)	AGG	RB	AGG (w/o RB)	RB (w/o AGG)
Baseline	.11	.01	**.12***	−.04*
After recollection of worst memory	**.29****	.05**	**.29****	−.06**
After recollection of best memory	.11$^{\sim}$	−.04$^{\sim}$	**.13****	−.08**
After questionnaires	**.22**	.11	**.19***	.03*

Note: Correlations that are significantly different from zero at $p < .05$ are indicated via bold font. Correlations that vary significantly across AGG and RB are indicated as follows: $^{*}p < .05$; $^{**}p < .01$; $^{\sim}p < .10$.

association, with some positive and some negative correlations. None, however, were significantly different from zero. Moreover, the associations between AGG and NA were significantly larger than those between RB and NA, particularly once we controlled for sub-type overlap. The differences between sub-type were particularly pronounced following the worst memory condition, regardless of whether we controlled for sub-type overlap (both $p < .005$). Such findings collectively highlight differential associations with NA across sub-type, such that only AGG appears to be generally associated with NA. Moreover, this association appears to be especially robust following recollection of the worst memory, offering preliminary support for our hypothesis that one's negatively valenced affective response to an aversive task is moderated by his AGG.

3.2. Repeated measures

Repeated measures ANOVA results for the full AGG and RB scales are presented graphically in Fig. 1. The within-subject effect for NA was highly significant, $F(3,739) = 40.76$, $p < .001$, indicating that reported levels of NA varied significantly across conditions (e.g., baseline, recollection of worst memory, etc.). Consistent with the above correlations, the between-subjects main effect was significant for AGG, $F(2,285) = 5.35$, $p = .005$, but not for RB, $F(2,285) = 0.52$, $p = .60$, indicating that NA varies significantly across level of AGG but does not vary across level of RB. Moreover, the interaction between NA and AGG was highly significant, $F(5,739) = 3.55$, $p = .003$, indicating that the variation in NA across conditions was moderated by level of AGG. The corresponding interaction between NA and RB, by contrast, was not statistically significant, $F(5,739) = 1.87$, $p = .09$. Finally, the three-way interaction of NA*AGG*RB was not significant, $F(10,2216) = 0.85$, $p = .58$, suggesting that AGG and RB do not interactively moderate changes in NA across conditions.[1]

Analyses were then repeated for the residual AGG and RB scores. Results are presented graphically in Fig. 2. As before, the between-subject main effect was significant only for AGG, $F(2,285) = 7.45$, $p = .001$ (for RB, $F(2,285) = 0.70$, $p = .50$), further supporting the notion that NA varies across level of AGG, but not RB. The three-way interaction of NA*AGG*RB was

[1] Associations with positive affect were also examined. None of the correlations with AGG or RB were significantly different from zero. Further, although the repeated measures within-subject effect for PA was highly significant ($p < .001$), none of the between-subjects or PA* between-subject interactions were significant (all $p > .35$). Such findings thus suggest that although our manipulation of PA was successful, the variability in PA across conditions had no association with either AGG or RB.

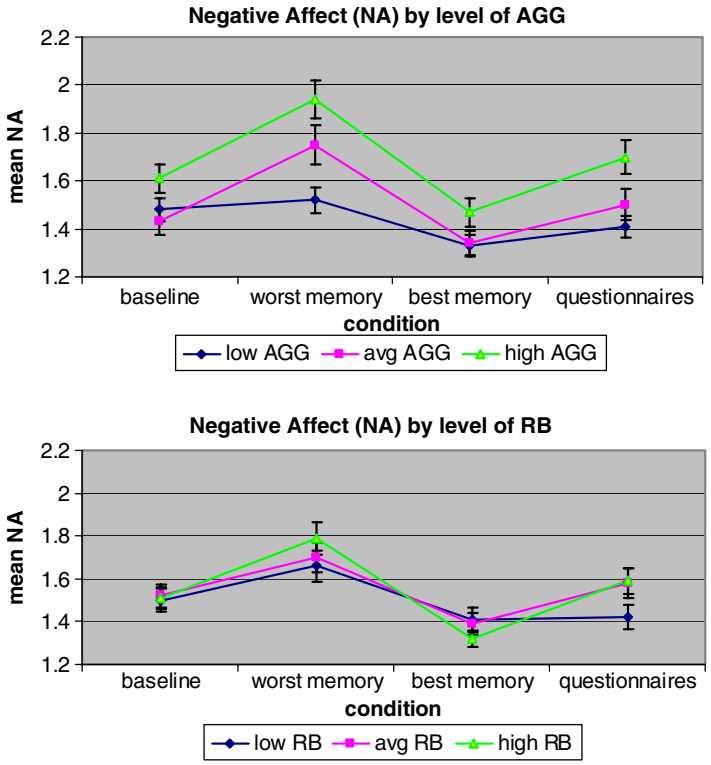

Fig. 1. NA for those with low, average or high levels of aggressive (AGG) or non-aggressive (RB) antisocial behaviors. *Note:* Numeric means are presented in Table 2.

similarly non-significant, $F(10,2232) = 1.0$, $p = .44$. Finally, the interaction between NA and AGG remained highly significant, $F(5,744) = 3.73$, $p = .002$, while that for RB remained non-significant, $F(5,744) = 2.00$, $p = .08$. Such findings thus collectively indicate that negatively valenced affective responses to our emotional manipulation varied primarily with level of AGG, and had little to no association with RB.[2]

3.3. Comparisons of mean NA

Post hoc independent *t* tests were conducted to determine the specific conditions under which NA varied with AGG and AGG (controlling for RB), and the specific groups that varied (see Table 4).[3] For the full AGG scale, NA varied for the worst memory condition. Those with low AGG

[2] To ensure that results for AGG were not measure-specific, analyses were repeated for the aggression items on the DBI (those omitted from the RB scale), as well as the verbal aggression and anger scales on the AQ. As with AGG, the correlations with NA following the worst memory and the questionnaires were uniformly positive and significant ($p < .01$). Moreover, all of the NA* between-subject interactions were significant (all $p < .05$). Such findings suggest that the variability in NA across level of AGG generalizes both to other measures of physical aggression and to related forms of aggression.

[3] When independent *t* tests were conducted on RB, results were uniformly non-significant. The only exception was the comparison of low and high RB following the questionnaires (ES = .29, $p = .049$). However, once we controlled for overlap with AGG, this association dissipated ($p = .22$), undercutting its significance.

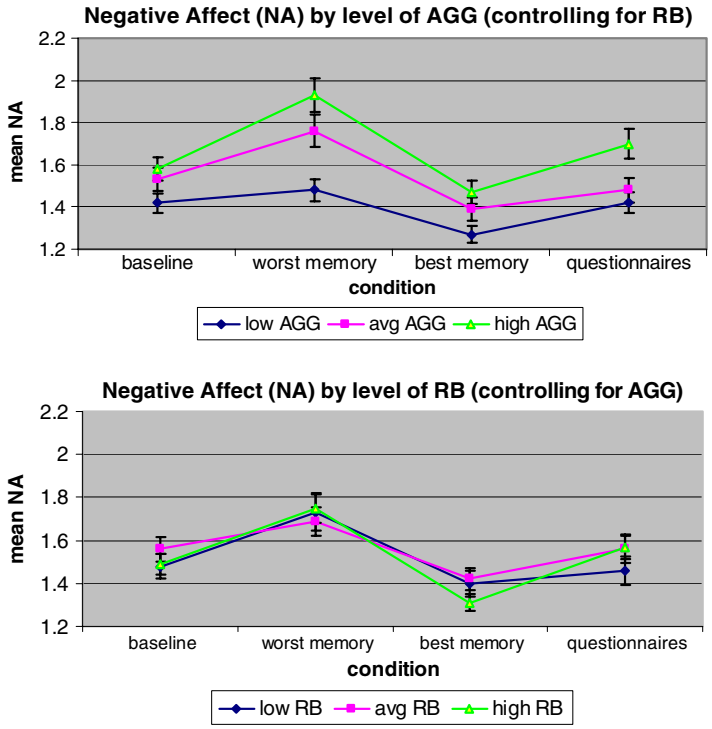

Fig. 2. NA for those with low, average or high levels of aggressive (AGG) or non-aggressive (RB) antisocial behaviors, controlling for the other. Numeric means are presented in Table 2.

Table 4
Mean self-reports of negative affect (NA) assessed under four conditions as a function of low, average, and high levels of aggressive (AGG) antisocial behavior

NA	Mean (SD)		
	Low	Average	High
AGG			
Baseline	1.48 (.50)	**1.43 (.48)**$_{b}$	**1.61 (.57)**$_{b}$
After recollection of worst memory	**1.52 (.56)**$_{a,c}$	**1.75 (.75)**$_{a}$	**1.94 (.79)**$_{c}$
After recollection of best memory	1.33 (.45)	1.34 (.45)	1.47 (.57)
After questionnaires	**1.41 (.49)**$_{e}$	**1.50 (.59)**$_{d}$	**1.70 (.71)**$_{d,e}$
AGG (controlling for RB)			
Baseline	**1.42 (.46)**$_{f}$	1.53 (.54)	**1.58 (.55)**$_{f}$
After recollection of worst memory	**1.48 (.52)**$_{j,g}$	**1.76 (.73)**$_{j}$	**1.93 (.81)**$_{g}$
After recollection of best memory	**1.27 (.39)**$_{h}$	1.39 (.51)	**1.47 (.55)**$_{h}$
After questionnaires	**1.42 (.49)**$_{i}$	**1.48 (.57)**$_{k}$	**1.70 (.71)**$_{i,k}$

Note: Bolded items with the same subscript are significantly different at $p < .05$. These means are presented graphically in Figs. 1 and 2.

reported less NA following recollection of their worst memory than did those with average or high levels (standardized effect size (ES) = .35, $p = .02$ and ES = .61, $p < .001$, respectively), although those with average and high levels of AGG did not differ significantly (ES = .25, $p = .11$).

Furthermore, those with low or average AGG reported less NA than those with high AGG after the questionnaires (ES = .48, $p = .001$ and .31, $p = .04$, respectively), though those with average and low levels did not differ. Finally, at baseline, those with average and high levels of AGG differed (ES = .34, $p = .02$), though neither differed from those with low AGG.

Analyses were then repeated for the AGG residual. Consistent with earlier results, examining this "cleaner" measure of aggression served primarily to augment our results. Those with low AGG endorsed significantly less NA than those with high levels, across all conditions (ES = .32, $p = .03$ at baseline, ES = .66, $p < .001$ following worst memory, ES = .42, $p = .004$ following best memory, and ES = .46, $p = .002$ following questionnaires). Furthermore, those with low AGG endorsed less NA than those with average levels following recollection of their worst memory (ES = .44, $p = .003$), although those with average and high levels did not differ (ES = .22, $p = .13$). Similarly, although those with average and high levels of AGG differed following the questionnaires (ES = .34, $p = .02$), those with low and average levels did not (ES = .11, $p = .46$). When viewed in combination with the full AGG scale results, these findings are thought to offer strong support for the notion that AGG is related to increased negative affect in general, and that this association is particularly pronounced following aversive experiences.

4. Discussion

The current study sought to determine whether negatively valenced affective responses differentially varied with aggressive and non-aggressive antisocial behavior. We therefore elicited changes in negative affect via recollection of one's best and worst life experiences, and evaluated whether these changes were linked to level (i.e., low, average, high) and type (i.e., AGG, RB) of ASB. Results revealed that AGG was associated with NA across conditions (particularly once we controlled for overlap with RB), but this association was particularly robust following recollection of one's worst life experience and completion of a relatively boring set of questionnaires. Such findings suggest that aggression is not only associated with NA more generally, but is also specifically linked to the potentiation of NA following completion of an aversive task. Importantly, these results generalized to both another measure of physical aggression and to other forms of aggression. By contrast, none of these associations persisted to RB, collectively supporting our contention that the affective dysfunction associated with ASB may instead be specific to its aggressive sub-type.

Though we know of no studies that have separately examined the role of affective dysfunction in AGG and RB, our results are consistent with several studies examining affective dysfunction in aggression and ASB (Davidson et al., 2000; Lorber, 2004; Sterzer et al., 2005; Verona et al., 2002). In spite of this replicability, there are several limitations to the present study. Affective responses were assessed via self-reports, rather than direct observation, and were induced via recollection of one's worst memory, rather than standardized stimuli. To ensure that results were not a function of the type or severity of life event recalled, the content of each participant's worst story was evaluated by coders blind to the participants' AGG, RB, and NA status. Each story was assigned to one or more of 24 negative life event categories (Ferguson, Lawrence, & Matthews, 2000), with categories ranging from relatively innocuous (e.g., settling into the university) to more severe (e.g., sexual assault, physical assault, death). Interrater reliability, established on thirty stories,

was found to be quite good (percent agreement was 99%, Pearson correlation was .86). Importantly, correlations revealed that NA following one's worst memory had no significant association with any category of life events (results available upon request), suggesting that affective responses to one's worst memory are not a function of the type or severity of life event experienced. Moreover, although both AGG and RB were associated with a higher incidence of legal problem life events ($r = .26$, $p < .001$; $r = .16$, $p < .01$), neither displayed any association (all $p > .20$) with the most severe negative life events (i.e., death of a close friend or relative, physical attack, sexual attack, health concerns, infidelity). Such findings are thus thought to highlight the external validity of our AGG and RB measures, while simultaneously suggesting that our findings are not a function of differentially aversive recollections. Even so, future research should seek to replicate these findings using standardized emotional stimuli.

The present results are also uninformative regarding the specific neural mechanisms involved. Thus, although the current findings certainly imply that the neural abnormalities previously linked to ASB are instead related to aggression in particular, this remains a hypothesis for future testing. Future research should seek to identify the specific neural abnormalities involved in AGG. Further, the current sample is a normative one, composed solely of undergraduate men. It thus remains unclear whether and how the current results may generalize to more deviant populations. Future research should seek to extend current findings in an adjudicated or incarcerated sample.

Next, it is unclear how age-of-onset impacted the results. As noted by Moffitt (2003) however, the aggressive/non-aggressive distinction investigated here maps neatly onto her developmental taxonomy for ASB (Moffitt, 1993). For example, research has indicated the median age-of-onset of aggressive behaviors is earlier than that of rule-breaking behaviors (Lahey, Loeber, Quay, Frick, & Grimm, 1992). Further, as compared to those whose ASB began in adolescence, those whose onset was prior to age 10 exhibited higher rates of aggressive behaviors but roughly the same prevalence of rule-breaking behaviors (Lahey et al., 1998). Accordingly, current results are thought to tentatively suggest that affective dysregulation may be specific to life-course persistent ASB (by virtue of their common overlap with AGG), though future research should evaluate this issue more specifically.

Finally, to ensure that our findings were not biased by the ordering of the emotional manipulation, the ordering was counterbalanced across subjects. Comparisons revealed that, although they generally did not vary, those whose order was "best, worst" reported more NA following recollection of their worst memory (ES = .27, $p = .024$) than those whose order was "worst, best". Importantly however, those whose order was "best, worst" also reported somewhat more AGG (ES = .23, $p = .055$). Moreover, when examined individually, repeated measures results were fully replicated across both orderings (e.g., the interaction between NA and AGG was significant, while that between NA and RB was not), suggesting that this difference did not impact our final conclusions. Rather than undercutting our conclusions then, such findings are instead thought to highlight the robust association between AGG and negatively valenced affective responses to aversive tasks.

5. Conclusion

The current results serve to further illuminate etiological distinctions between aggressive and non-aggressive antisocial behavior. Namely, the faulty affective regulation previously linked to

antisocial behavior in general appears instead to be specific to its aggressive sub-type. Previous work indicates that unpleasant stimuli or events, such as pain or social stress, increase impulsive aggressive behaviors via the activation of high state-NA (Berkowitz, 2003). Within this formulation, the present study's finding of affect dysregulation only in those higher in AGG is suggestive of an AGG-specific vulnerability for NA-mediated aggression. Moreover, as gene–environment interactions appear to contribute to NA-mediated aggression (Verona, Joiner, Johnson, & Bender, 2006), the current results further imply that gene-environment interplay may also vary by ASB sub-type (at least in men). Future research should further explore the possibility of differential gene–environment interplay within ASB.

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References

Achenbach, T. M., Rescorla, & L. A. (2003). *Manual for ASEBA adult forms & profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.

Berkowitz, L. (2003). Affect, aggression, and antisocial behavior. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 804–823). New York: Oxford University Press.

Burt, S. A., Carter, L. A., McGue, M., & Iacono, W. G. (2007). The different origins of stability and change in Antisocial Personality Disorder symptoms. *Psychological Medicine, 37*, 27–38.

Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology, 63*, 452–459.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation – A possible prelude to violence. *Science, 289*, 591–594.

Deater-Deckard, K., & Plomin, R. (1999). An adoption study of the etiology of teacher and parent reports of externalizing behavior problems in middle childhood. *Child Development, 70*, 144–154.

Deeley, Q., Daly, E., Surguladze, S., Tunstall, N., Mezey, G., et al. (2006). Facial emotion processing in criminal psychopathy. *British Journal of Psychiatry, 189*, 533–539.

Eley, T. C., Lichtenstein, T., & Moffitt, T. E. (2003). A longitudinal behavioral genetic analysis of the etiology of aggressive and nonaggressive antisocial behavior. *Development and Psychopathology, 15*, 383–402.

Farrington, D. P., & West, D. J. (1971). A comparison between early delinquents and young aggressives. *British Journal of Criminology, 11*, 341–358.

Ferguson, E., Lawrence, C., & Matthews, G. (2000). Associations between primary appraisals and life-events while controlling for depression. *British Journal of Clinical Psychology, 39*, 143–155.

Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L., Van Horn, Y., Christ, M. A. G., et al. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review, 13*, 319–340.

Herpertz, S. C., Mueller, B., Lichterfeld, C., Konrad, K., & Herpertz-Dahlmann, B. (2005). Response to emotional stimuli in boys with Conduct Disorder. *American Journal of Psychiatry, 162*, 1100–1107.

Hudziak, J. J., van Beijsterveldt, C. E. M., Bartels, M., Rietveld, M. J. H., Rettew, D. C., Derks, E. M., et al. (2003). Individual differences in aggression: Genetic analyses by age, gender, and informant in 3-, 7-, and 10-year-old Dutch twins. *Behavior Genetics, 33*, 575–589.

Lahey, B. B., Hart, E. L., Pliszka, S., Applegate, B., & McBurnett, K. (1993). Neurophysiological correlates of conduct disorder: A rationale and a review of research. *Journal of Clinical Child Psychology, 22*, 141–153.

Lahey, B. B., Loeber, R., Quay, H. C., Applegate, B., Shaffer, D., Waldman, I., et al. (1998). Validity of DSM-IV subtypes of conduct disorder based on age of onset. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 435–442.

Lahey, B. B., Loeber, R., Quay, H. C., Frick, P. J., & Grimm, J. (1992). Oppositional defiant and conduct disorders: Issues to be resolved for DSM-IV. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 539–546.

Lorber, M. F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: A meta analysis. *Psychological Bulletin, 130*, 531–552.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Reveiw, 100*, 674–701.

Moffitt, T. E. (2003). In B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Life-course persistent and adolescence-limited antisocial behavior: A research review and a research agenda*. New York: Guilford.

Raine, A. (2002). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry, 43*, 417–434.

Raine, A., Venables, P. H., & Mednick, S. A. (1997). Low resting heart rate at age 3 years predisposes to aggression at age 11 years: Evidence from the Mauritius Child Health Project. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*, 1457–1464.

Sterzer, P., Stadler, C., Krebs, A., Kleinschmidt, A., & Poutska, F. (2005). Abnormal neural responses to emotional visual stimuli in adolescents with conduct disorder. *Biological Psychiatry, 57*, 7–15.

Stranger, C., Achenbach, T. A., & Verhulst, F. C. (1997). Accelerated longitudinal comparisons of aggressive versus delinquent syndromes. *Development and Psychopathology, 9*, 43–58.

Verona, E., Joiner, T. E., Johnson, F., & Bender, T. W. (2006). Gender specific gene-environment interactions on laboratory-assessed aggression. *Biological Psychiatry, 71*, 33–41.

Verona, E., Patrick, C. J., & Lang, P. J. (2002). A direct assessment of the role of state and trait negative emotion in aggressive behavior. *Journal of Abnormal Psychology, 111*, 249–258.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070.