Online-Only Text

In post-hoc analyses, age was added as a covariate in the L amygdala ROI analysis and there remained a main effect of *Group* $F(2,53)=3.78$, $p<.03$, but no main effect of *Age* $F(1, 53)=1.84$, $p=.18$. Similarly, we included *Age* as a covariate in the analysis of the one whole brain cluster for N→A and the four clusters for N→H that showed significant between-group differences. Again, all the main effects of *Group* remained significant (all Fs>9, all ps<.0001), with no effects of *Age* (all Fs<1.2, all ps>.3).

To investigate the effect of *Sex*, it was included as a between-groups variable in the amygdala ROI and whole-brain clusters. In the L amygdala ROI for N→A there was a significant main effect of *Sex*, $F(1,56)=6.83$, $p<.02$, with males having a lower average beta weights (-.01) than females (.05). There was also a trend for a main effect of *Sex* in the L middle/superior frontal gyrus for N→H, $F(1,56)=3.78$, $p=.06$, with females having lower average beta weights (-.03) than males (.02). However, in neither the amygdala ROI nor any of the whole-brain clusters was there a significant *Group* x *Sex* effect.

Possible group differences in pubertal status were evaluated by analyzing Tanner stage data. Unfortunately, Tanner stage data was available for some, but not all, of the participants (Ns BD=18, SMD=13, HV=7). Unfortunately, the majority of HV did not have Tanner Breast/Testes (TannerBT) or Tanner Pubic Hair (TannerPH) data. Using the available data, we assessed possible group differences for both TannerBT and TannerPH. There were no group differences on either measure (TannerBT: $F(2,37)=2.28$, $p=.12$; TannerPH: $F(2, 37)=2.11$, $p=.14$.

Since BD and SMD differed significantly on CDRS score, CDRS score was used as a covariate in post-hoc analyses in the four whole brain N→H clusters where these two patient groups differed from each other. (HV, by definition, were not depressed and CDRS ratings were not obtained, so they were not included in these analyses.) With CDRS scores covaried, BD and SMD still differed significantly from each other in all four clusters (all Fs>10, all ps<.0001), and there was no effect of CDRS (all Fs<.05, all ps>.8).

In regions where BD and SMD differed in their BOLD response patterns, an analysis of the effect of ADHD in BD was conducted in order to aid in the interpretation of the N→H results. For the L Amygdala ROI, an ANOVA was conducted including only BD subjects, with the between-groups factor being with or without ADHD. There were 10 BD with ADHD and 9 BD without ADHD. There was no difference in the N→A slope for BD with vs. without ADHD, $F(1,17)=.26$, $p=.62$. A similar ANOVA was also conducted on the one whole brain cluster identified in the whole-brain analysis for N→A (i.e., in the posterior cingulate). Again, we found no difference in BD with vs. without ADHD, $F(1,17)=.83$, $p=.34$. The four whole brain clusters that were significant for N→H also showed no ADHD effects, all Fs<.3, ps>.38.

An additional analysis examined correlations between behavioral and neural measures in the amygdala ROI and in the clusters from the whole-brain analysis that showed significant between-group differences. Specifically, we examined correlations between the slope of the behavioral ratings (i.e., hostile and nose ratings) and the slope of the beta weights in each of these rating conditions. The only significant finding was in the amygdala for the BD youth,

where there was a positive correlation between the slope of neural activation during nose width ratings of angry faces and the slope of the nose width ratings themselves (r=.53, p=.02). Of note, this finding would not remain significant after correction for multiple comparisons. All other correlations between neural activation and behavioral ratings (hostile ratings of angry faces, hostile ratings of happy faces, nose width ratings of angry faces, nose width ratings of happy faces) were not significant.

Since the samples of medicated vs. unmedicated patients are very small and thus prone to Type II error, we covaried medication status (on/off medication) within BD vs. SMD (since HV were not medicated) in the four whole brain N→H clusters where the two patient groups differed from each other. With medication status scores covaried, BD and SMD still differed significantly in all four clusters (all Fs>13, all ps<.001), and there was no effect of medication status (all Fs<1.5, all ps>.2).

A *Group* x *Attention* ANOVA for N→A in the whole-brain cluster of the posterior cingulate, conducted in euthymic patients only, showed a main effect of *Group*, driven by BD vs. HV (p<.005), with a trend for SMD vs. HV (p=.06). For N→H in the R inferior parietal lobule (BA40/7), SMD continued to differ from both BD and HV. In the L Middle Occipital/Fusiform Gyrus (BA37) the main effect of group remained, while the post-hoc analyses were not significant. In the R Middle Occipital Gyrus/Cuneus (BA18/19) and the L Middle/Superior Frontal Gyrus (BA6/8) the results of the main effect of *Group* and post-hoc analyses were unchanged from the primary analysis.