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Positive Affectivity is Dampened in Youths with Histories of Major Depression and Their Never-Depressed Adolescent Siblings

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Abstract

While hedonic capacity is diminished during clinical depression, it is unclear whether that deficit constitutes a risk factor and/or persists after depression episodes remit. To examine these issues, adolescents with current/past major depression (probands; n=218), never depressed biological siblings of probands (n=207), and emotionally-well controls (n=183) were exposed to several positively valenced probes. Across baseline and hedonic probe conditions, controls consistently reported higher levels of positive affect than high-risk siblings, and siblings reported higher levels of positive affect than probands (remitted and depressed probands' reports were similar). Extent of positive affect across the protocol predicted adolescents' self-reports of social support network and parental reports of offspring's use of various adaptive mood repair responses in daily life.

Attenuated hedonic responding among youths remitted from depression offers partial support for anhedonia as a trait, while its presence among never depressed high-risk siblings argues for anhedonia as a potential diathesis for clinical depression.

Keywords

positive affect; anhedonia; depression; high-risk siblings; childhood depression

Diminished ability to experience pleasure and joy (anhedonia) has been long recognized by clinicians as a cardinal feature of severe depression (e.g., Kraepelin, 1921); starting in 1980, its importance also has been acknowledged by the official operational diagnostic criteria for depressive disorders (American Psychiatric Association, 1980). Usually joyous events, such as an unexpected present, reaching a coveted goal, being praised, watching a humorous show, or witnessing a beautiful sunset, for example, fail to elicit a sense of pleasure or happiness when a person is suffering from depression. However, it also has been posited that hedonic dysfunction, or a relatively low level of positive affectivity, is not merely a symptom

of depression but is also a trait that might predate the depressive syndrome, signal an elevated risk for it, and/or persist to some extent even after depression has remitted (Hasler, Drevets, Manji, & Charney, 2004; Loas, 1996; Meehl, 1975). Recent research has underscored that depressed people's reports of diminished enjoyment and lack of pleasure in everyday life is one facet of anhedonia, which also includes dysfunction in reward responsiveness and in related motivational, information processing, and decision making neural circuitries (see reviews by Pizzagalli, 2014; Treadway & Zald, 2011), and may identify a depression endophenotype (Hasler, et al., 2004).

While the nature and consequences of anhedonia have been extensively studied, basic questions still remain about its stability across various phases of depression and its role as a risk factor for affective psychopathology. Further, investigations of clinical samples have mostly involved adults, although depression in younger populations is a pressing problem (Kessler & Walters, 1998). All in all, however, numerous laboratory studies involving the presentation of positively-toned stimuli have confirmed the presence of hedonic dysfunction during episodes of clinical depression. Compared to healthy controls, individuals suffering from depression (typically major depressive disorder, MDD) evidence blunted or less intense reactions to pleasant stimuli and potential or actual rewards (for reviews, see Bylsma, Morris & Rottenberg, 2008; Eshel & Roiser, 2010; Zhang, Chang, Guo, Zang, & Wang, 2013). For example, depressed adult patients usually report lower levels of happiness or enjoyment than do controls after viewing standardized positive visual prompts, such as pictures of pleasant scenes or smiling faces (Dunn, Dalgleish, Lawrence, Cusack, & Ogilvie, 2004; Sloan, Strauss, Quirk & Sajatovic, 1997; Sloan, Strauss & Wisner, 2001) or after reading highly positively toned scripts (Horner et al., 2014) and display reduced behavioral responses to monetary reward (Henriques & Davidson, 2000; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2009). Potential monetary reward also elicited lower levels of "joviality" among depressed college students than control peers (McFarland & Klein, 2009). In a similar vein, adolescents with various depression diagnoses were less likely to seek rewards during a gambling task than were healthy peers (Rawal, Collishaw, Thapar & Rice, 2013). Although some studies have reported normative self-rated hedonic experience among adults with MDD (e.g., Osuch, Bluhm, Williamson, Theberge, Densmore & Neufeld, 2009), a meta-analysis found that depressive episodes are associated with blunted positive affective reactions and impaired reward-related behaviors (Bylsma et al., 2008).

In contrast, fewer studies have examined individuals during periods of recovery from depression and the findings have been equivocal regarding whether, and to what extent, hedonic dysfunction persists *after* the depression episode remits. For example, college students remitted from depression and healthy control peers reported comparable levels of positive affect while anticipating monetary rewards (McFarland & Klein, 2009). Similarly, self-rated pleasure in response to chocolate failed to distinguish recovered depressed and never depressed adults (McCabe, Cowen & Harmer, 2009). Recovered depressed and control subjects also had similar behavioral responses to pictures of happy facial expressions (Kerestes et al., 2012). On the other hand, some studies did find adverse residual effects of depression on affective responding. Namely, self-ratings and clinical evaluations of middle-aged outpatients with remitted depression indicated that they had lower hedonic capacity than did community controls (Di Nicola et al., 2013). Likewise, in two separate studies,

adults with remitted depression displayed blunted behavioral responses to monetary incentives and social praise, relative to healthy controls (Pechtel, Dutra, Goetz, & Pizzagalli, 2013). The persistence of hedonic dysfunction after remission of a depression episode supports the proposition that anhedonia has trait-like features, which has ramifications for prevention efforts. Thus, one goal of our study was to address this issue in a clinical sample of youths with prior histories of MDD.

A further critical issue to be resolved is whether hedonic dysfunction is present prior to the first episode of depressive illness and thus qualifies as a bona fide risk factor for it. The optimal approach to this topic is to establish the presence of hedonic impairment among not-yet-depressed individuals who are known to be at elevated risk for depression (usually by virtue of familial history) and then use follow-up to determine its predictive value for onset of clinical depression. However, studies of young, high-risk offspring (typically of depressed mothers) have yielded inconsistent results about the presence of hedonic impairment and the children have not been followed for a sufficiently long time to model risk factors for first onset depressive disorder. For example, in one study, evocative laboratory tasks elicited lower rates of positive affect displays among high-risk, 3-year-old offspring of depressed mothers than low-risk offspring (Durbin, Klein, Hayden, Buckley, & Moerk, 2005). A longitudinal developmental study also reported consistently lower levels of positive affect displays in the laboratory among high-risk, pre-school, and young school-age children compared to low-risk offspring (Olinio et al., 2011). However, in two other studies of pre-school-age offspring, maternal depression histories were unrelated to children's positive affect (Feng, Shaw, Skuban, & Lane, 2007; Olinio, Klein, Dyson, Rose & Durbin, 2010) and in a small sample of adolescents, those at high- versus low-risk reported comparable levels of positive affect in natural settings (Olinio et al., 2014). Likewise, high- and low-risk 16- to 21-year-olds did not differ in ratings of pleasantness after tasting or viewing pictures of chocolate (McCabe, Woffindale, Harmer, & Cowen, 2012). The inconsistent findings could partly reflect that affect displays (along with behavioral regulation) across childhood are subject to notable developmental mediation and moderation (summarized in Olinio et al., 2011), and such developmental effects probably varied across the studies. In the present investigation, we therefore focused on older high-risk subjects, namely, never depressed adolescents whose brothers or sisters had histories of MDD. Depression in siblings is a robust risk factor for eventual depression in their yet unaffected brothers and sisters (e.g., Farmer, et al., 2000), who have a cumulative probability of a first MDD episode of .42 by the time they are young adults (Ryan et al., 1992). However, with the exception of studies of twin pairs (e.g., Bogdan & Pizzagalli, 2009), to our knowledge, there are no laboratory investigations of positive affectivity involving young, high-risk siblings.

Hedonic capacity or positive affectivity has been shown to facilitate an array of important functions, including subjective well-being, attention allocation and executive processes, as well as intra- and extra-familial social interactions (e.g., Davis, Suveg, & Shaffer, 2014; Fredrickson, 2001; Grol, Koster, Bruyneel & De Raedt, 2014; Smillie, Wilt, Kabbani, Garratt & Revelle, 2015; Wadlinger & Isaacowitz, 2006; Yang & Yang, 2014). The positive affect system also facilitates approach behaviors focused on resources and therefore is associated with a range of adaptive consequences (e.g., Watson & Naragon, 2009). Conversely, attenuated hedonic capacity may have various adverse sequelae, including a

disruption of social relationship and difficulties in maintaining social networks. Deficits in positive affectivity also may compromise emotion regulation repertoires (Kovacs & Lopez-Duran, 2010). For instance, in the presence of chronically low hedonic capacity, a person may be short on regulatory responses that involve positive affect (like recalling happy memories) as a way to lessen current distress. Thus, the final goal of the present study was to examine the functional significance of laboratory-based indices of positive affectivity.

In summary, there is compelling evidence that depression compromises hedonic capacity. However, questions have remained about whether such impairment predates depression among vulnerable people and thus operates as a true risk factor, and if hedonic dysfunction persists after depression. Further, if empirical studies of affectivity are to have meaningful clinical implications, strengths or deficits identified in the laboratory should have functional correlates in daily life. We addressed these issues among adolescents with histories of childhood onset MDD who were either in remission or currently depressed (henceforth referred to as *probands*), never depressed full biological (high-risk) siblings of probands, and healthy controls with no history of psychopathology. After exposure to a sampling of hedonic probes, namely, solvable puzzles, surprising receipt of a desired prize, and a film clip displaying slapstick comedy (presented in random order within a more extensive protocol), subjects repeatedly rated their own positive affect. Separately, youths also provided information about their social support networks, and parents independently reported whether offspring typically used positive affect-related regulatory responses to sadness in daily life.

We hypothesized that, regardless of whether depressed or in remission, probands will evidence lower hedonic capacity across all probes than controls. Based on Meehl's (1975) model, we further hypothesized that high-risk, never-depressed siblings also will report consistently lower levels of positive affect than controls. We expected the differences across groups to be evident in terms of the absolute magnitudes of positive affect (raw scores of affect intensity) and the extent of reactivity to each probe (change scores). Finally, we hypothesized that the magnitude of positive affect elicited in the laboratory will have functional significance and predict the extent of youths' social support networks (by self-report) and their use of mood repair responses that involve positive affect (by parental report).

Method

Subjects

Subjects included *probands*, whose histories of childhood onset major depressive disorder (MDD) were established in a prior study (e.g., Baji et al., 2009; Tamás et al., 2007), never-depressed biological *siblings* of probands, and emotionally well *controls* with no history of major psychiatric disorders. Probands and siblings were a subset of a larger national sample in Hungary, enrolled in a molecular genetic study (the *archival* study) from approximately 1997-2006 (e.g., Burcescu et al., 2006; Dempster et al., 2009). Probands for the archival study were recruited through various child mental health facilities if they: (a) had a current or recent DSM-IV (American Psychiatric Association, 2000) depressive disorder, (b) were 7- to 14-years old at initial screen, (c) were not mentally retarded and had no major systemic

medical disorder, (d) had at least one biological parent who could participate, and (e) had at least one full biological 7-18 year-old sibling (within +/- 3 years of age).

The current investigation of emotion reactivity and regulation enrolled 224 probands and 214 never-depressed siblings from the archival study, aged 11- to 18-years, who lived in the proximity of three research hubs. In this article, we report on $n=218$ probands with MDD histories (6 with bipolar disorder were excluded) and 207 unaffected siblings of probands (7 siblings of bipolar probands were excluded) from altogether 297 families. A total of 102 families contributed a proband and one or more unaffected sibs; 116 families contributed only a proband (when a sibling was not available); and 79 families contributed one or more unaffected sibs (when a proband was not available). Probands were 17.0 years old, on average ($SD=1.4$) and 64.2% were boys; siblings were 15.9 years old ($SD=2.12$), on average, and 44.9% were boys. Parental education served as an indicator of higher socioeconomic standing: 9.2% of proband mothers and 8.2% of fathers had more than secondary school education; for the partly overlapping sibling sample, rates of highly educated mothers and fathers were 17.8% and 11.9%, respectively.

Probands had the onset of their first MDD episode when they were 9.07 years old, on average ($SD=1.90$ years). At enrollment in the current study, 56.0% had one MDD episode, 31.2% had 2 episodes, and 12.9% had 3 or more episodes. Further, 32 probands (14.7%) were in a depressive episode, while 186 (85.3%) had recovered from their last MDD episode. Recovery was operationally defined as being essentially free of depressive symptoms for at least two consecutive months (Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984a). Rates of comorbid psychiatric disorders were consistent with the literature (Birmaher et al., 1996): 39.0% of the probands had some type of anxiety disorder, 36.24% had some behavioral disorder (e.g., ADHD, oppositional defiant disorder); altogether 70.2% had one or more comorbid major psychiatric disorder.

The 183 control subjects (mean age: 16.1 years, $SD=2.13$ years; 64.5% boys) were recruited for the present study to approximate the sex and age distribution of probands; they were identified in public elementary and secondary schools in the 3 cities in which most of the probands resided. Enrollment required lifetime history free of any major psychiatric or systemic medical disorder, having a parent willing to participate, and having at least one sibling (to approximate probands' family units). For these youths, 48% of the mothers and 39% of the fathers had more than secondary school education.

As can be seen in Table 1, probands were approximately 1 year older than never-depressed siblings and controls ($F(3,565) = 15.31, p<.001, R_p^2$). The preponderance of boys among probands was matched by design in the controls (as already noted), while the sex distribution of the sibling group was more even (44.9% boys, $\chi^2(3)=21.1, p<.001, W = .19$). Ethnic composition of the proband/sibling group was 95% Caucasian, 2% multiracial/other, and 3% Roma (representative of the population of Hungary), and did not significantly differ from that of the control sample, which was 99% Caucasian, 1% multiracial/other and 0% Roma.

The current research study was approved by the institutional review boards of the University of Pittsburgh and the Hungarian clinical research sites. Parents provided written informed

consent, and depending on their ages, young subjects provided either assent or consent. All procedures, schedules, rating scales, and instruments used in this study were first developed in English, translated into Hungarian, and then back translated by bi-lingual child psychiatrists and clinical psychologists. An iterative procedure was used to resolve any discrepancies between original and back-translated versions of documents.

Clinical and Diagnostic Assessment

Caseness of probands was originally established in the archival study via standardized psychiatric diagnostic evaluations using a semi-structured interview by trained interviewers. The information was gathered via the Interview Schedule for Children and Adolescents: Diagnostic version (ISCA-D). The ISCA-D, described previously (Baji et al., 2009; Kiss et al., 2007; Tamás et al., 2007), uses DSM-IV criteria (American Psychiatric Association, 2000). The clinician first interviews the parent about the child, and then separately the child about him or herself, and then assigns an overall rating to each symptom. Those ratings, along with the child's clinical history, determine whether DSM-IV criteria for any of the covered disorders are met. The diagnostic process also includes operational definitions of onset and recovery and psychiatric comorbidity (Kovacs et al., 1984a, b); diagnoses were always finalized by consensus among pairs of senior raters. Evaluations were conducted by child psychiatrists and psychologists trained in the ISCA-D, who had met predefined symptom rating goals on individually supervised interviews and reliability ratings against videotaped “gold-standard” diagnostic interviews. We have previously reported acceptable inter-rater reliability coefficients on ISCA-D symptom ratings (Tamás et al., 2007). A follow-up version of the ISCA-D, which queries about symptoms and adjustment since the prior evaluation, was used to establish the youth's diagnostic status at the time of the current study. Sociodemographic information about the youths was obtained from the parents in accordance with a structured set of questions.

Experimental Procedures

After the youth was familiarized with the experimental procedures and equipment, he or she was asked to rank order, from the least to the most preferred, 7 age-appropriate prizes (e.g., high-tech earphones, gift certificate to a multi-media store, combinations of snacks, each of comparable monetary value). The subject then was told that, at some point, the “computer will randomly decide” which prize he or she will receive. However, unbeknownst to the youth, he or she was to receive his/her most preferred prize. The protocol, which was introduced to subjects as an experiment to better understand how children and adolescents react to different tasks and stimuli, included probes of physiological and psychological reactions to affectively pleasant, unpleasant, and stressful stimuli/tasks, and took about 1 hour, with a 5- to 10-minute mid-point break. To minimize potential order-of-task effects, subjects were randomized to predefined task sequences.

At the start of the protocol, the subject was seated in a comfortable chair in front of a table, and was asked to provide affect ratings (see below) for the first time (baseline). After that, he or she was connected to sensors for physiological recording (data not reported here) and then engaged in the specified series of tasks. In this article, we focus on affect self-ratings from baseline (pre-stimuli) and after three experimental probes that were designed to induce

positive affect (PA): presentation of solvable puzzles, provision of a preferred prize, and watching a funny film clip. The solvable puzzle and getting the prize were presented in the first 30 minutes of the protocol to 50% of the subjects and in the second half of the protocol to the other 50% of cases (interspersed with other tasks, not reported here). The happy film clip was the last stimulus for all cases.

Positive Affect Probes—The Happy Film Clip, one of three conditions used to induce positive affect, was a 186 second segment from *Mr. Bean*, a slapstick style comedy, which has been used with adults (e.g., Joormann & Siemer, 2004; Rottenberg, Kasch, Gross & Gotlib, 2002). Film clips also have been shown to elicit the desired valenced emotion in children (von Leupoldt et al., 2007). We nonetheless piloted the *Mr. Bean* clip with 240 Hungarian youth (53% girls; 62% of the sample aged 14- to 18-years, recruited from 6 schools in two cities). Youngsters rated each of 5 emotion words (from 0 to 8) after watching *Mr. Bean*, as well as neutral and sad film clips, presented in a randomized order. The *Mr. Bean* clip elicited robust reports of happiness ($M=6.45$; $SD=1.77$) but relatively low reports of non-target emotions (e.g., anger, $M=1.35$, $SD=1.15$), as well as significantly higher ratings of happiness than the neutral ($t(203)=13.25$, $p<.001$, $d_z=0.93$) and sad film clips ($t(203)=19.15$, $p<.001$, $d_z=1.34$).

Solvable Puzzles—Puzzles have been used as mood induction tools with youths (e.g., Berger, Miller, Seifer, Cares & Lebourgeois, 2012; Cole et al., 2007; Hokoda & Fincham, 1995; Nolen-Hoeksema, Wolfson, Mumme, & Guskin, 1995). Our solvable puzzles were a series of computerized tasks in which the youth had to replicate a particular configuration of numbered tiles by sliding them around. The 5 puzzles (lasting for 60 seconds) were programmed to present a bit of a challenge but be relatively easy to complete. A training phase was provided.

As another probe of positive affectivity, youths were unexpectedly provided with their Preferred Prize. We pilot tested the desirability of 25 potential prizes (roughly equivalent in monetary value) with younger (9- to 13-years old) and older (14- to 18-years old) volunteer samples of convenience. The final list of 7 prizes (e.g., earphones, snacks) was based on the rankings these subjects provided.

Psychometric Questionnaires—Adolescents completed the widely used 20-item *Positive and Negative Affect Schedule* (PANAS; Watson, Clark, & Tellegen, 1988), which we edited slightly for clarity with younger age groups. The factorial and concurrent validity of the PANAS was reported in the original article by Watson et al., (1988); further psychometric information, including on construct validity, has been reported by Crawford and Henry (2004), among others. PANAS items were rated (using a 1 to 5 Likert scale) for the past 2 weeks and were averaged to create the positive affect (PA) and negative affect (NA) scores. Level of depressive symptoms was quantified by the *Children's Depression Inventory-2* (CDI-2), a widely used, self-rated questionnaire for youngsters, with documented concurrent, construct, and predictive validity (Kovacs & MHS Staff, 2011). The CDI also was completed to reflect the prior 2 weeks. We assessed adolescents' social support networks via the 12-item *Multidimensional Scale of Perceived Social Support* (MSPSS; Zimet, Dahlem, Zimet, & Farley, 1988), which queries about friends, family, and others.

MSPSS items are rated on a 7 point Likert scale and reflect both network size and support availability; the instrument has been used cross-culturally (e.g., Steese et al., 2006; Terzi-Unsal & Kapci, 2005; Zimet, Powell, Farley, Werkman, & Berkoff, 1990) and has good psychometric properties, including construct and concurrent validity (e.g., Kazarian & McCabe, 1991; Zimet et al., 1988, 1990). Pubertal stage was determined via self-report on a questionnaire that uses drawings to depict stages of puberty along the lines established by Tanner (Morris & Udry, 1980). The validity of such self-rated pubertal staging against physicians' assessments has been established (Duke, Litt & Gross, 1980; Morris & Udry, 1980) as well as its correlation with chronological age (e.g., Angold, Costello & Worthman, 1998).

Finally, parents completed a trait questionnaire of their offspring's mood repair response repertoires (*Feelings and My Child*), which surveys a range of helpful (adaptive) and not helpful (maladaptive) regulatory responses to sadness and distress, and has good psychometric properties (Bylsma et al., in press; Gentzler, Santucci, Kovacs, & Fox, 2009). Using the 6 items that reflect adaptive responses involving the self-mobilization of positive affect (e.g., when my child feels sad, he/she tries to think of happier times in the past), we created a subscale. Total scores for this 6-item scale ranged from 0 to 12, with higher scores indicating greater reliance on adaptive regulatory responses that mobilize positive affect to counter sadness. Based on $n=607$ parental reports, the Cronbach alpha of .72 revealed acceptable internal consistency. The construct validity of this 6-item questionnaire is supported by its significant correlation with youths' self-ratings on the Positive Affect (PA) scale of the PANAS (Watson et al., 1988) ($r(397)=.22, p<.001$) and their self-ratings on the Distraction subscale of the Children's Response Style Questionnaire (Ziegert & Kistner, 2002) ($r(366)=.17, p<.01$), which taps another dimension of adaptive emotion regulation. The scale's discriminant validity is evidenced by the finding that depressed probands in this study ($n=32$) had significantly lower scores than did a subset of controls ($n=32$), matched for age and sex ($(M_{depressed}=2.97, SD=2.12; M_{control}=5.53, SD=1.88; t(62)=5.12, p<.001, d=1.28$).

Positive Affect Self-Rating (PASR) During the Protocol—At baseline, and after each hedonic probe, subjects indicated on a 0 (not at all) to 7 (very much) Likert scale the extent to which several discrete feeling states characterized them. The items included three manifestations of PA: *happy*, *interested*, and *enthusiastic* (along with items pertaining to negative affect). Because the three items were significantly intercorrelated at each measurement point (Cronbach alphas from .75 to .86), the scores were averaged at each rating, which yielded the Positive Affect Self-Rating (PASR) composite, used in statistical analyses.

Statistical Analyses

Variables were compared across groups using ANOVAs, t -tests, or χ^2 tests, as appropriate. Mixed effects, repeated measures analysis of covariance (rANCOVA) was used to test for group (depressed proband, remitted proband, sibling, control) by task (baseline, prize, solvable puzzle, happy film) effects on PASR scores, while accounting for dependence between siblings. In these models, group was a between-subjects factor, experimental task

was a within-subjects factor, age (in years) and sex were covariates, and family was a random effect. Nonsignificant covariate-by-task interactions were removed, and the best-fitting repeated measures' covariance matrix was chosen (Gueorguieva & Krystal, 2004). The method of Kenward and Roger (Gomez, Schaalje & Fellingham, 2005) was used to compute fixed effect denominator degrees of freedom which, along with the corresponding F statistic, was used to compute the effect size, namely, the partial R_{β}^2 (Edwards, Muller, Wofinger, Qaquish, & Shabenberger, 2008). The group-by-task interaction was further examined by tests of simple effects sliced by task. That is, within each task, covariate-adjusted group means were tested for differences using Tukey's correction for Type I error in one-sided hypotheses.

Results

Characteristics of Subject Groups

Table 1 includes the mean (SD) affect ratings across subject groups at baseline. PANAS positive affect scores revealed a group effect ($F(3, 548)=12.09, p<.001, R_{\beta}^2=.06$): controls reported the highest level of PA, probands reported the lowest level, and siblings were positioned in between probands and controls (each group differed from the others, correcting for type I error using Tukey's test). Baseline PASR scores also showed a group effect ($F(3, 557)=21.97, p<.001, R_{\beta}^2=.11$): again, controls reported the highest PASR scores, while probands reported the lowest ones. However, remitted and currently depressed probands did not differ on extent of positive affect at baseline, whether measured by the PANAS or PASR composite rating (Table 1).

Not surprisingly, probands reported higher levels of negative affect, in general (PANAS NA, Table 1) than did siblings or controls ($F(3, 568)=27.99, p<.001, R_{\beta}^2=.13$), and higher levels of depressive symptoms on the CDI ($F(3, 543)=56.68, p<.001, R_{\beta}^2=.24$). Further, currently depressed probands ($n=32$) reported more negative affect on the PANAS than their remitted counterparts ($n=186$). PASR scores and sex were modestly correlated ($r(602)=-.12, p<.01$). All in all, sex and age were controlled in all subsequent analyses.

Manipulation Check

Changes in mean PASR scores between baseline and each task indicated that the hedonic probes elicited the intended effect: *getting a prize* was associated with the greatest increase in PA ($M=0.42, SD=1.02, t(607)=10.22, p<.001, d=0.41$), followed by watching the *happy film clip* ($M=0.23, SD=1.15, t(607)=4.90, p<.001, d=0.20$). The *solvable puzzles* elicited trend-level increases in positive affect 1 ($M=0.08, SD=1.06, t(607)=1.78, p=.08, d=0.07$). Very few subjects endorsed absolutely no positive affect (composite score=0) in response to the probes. Specifically, 3 depressed probands, 12 remitted probands, 3 siblings, and 2 controls reported no positive affect to one or more hedonic probes, with probands being overrepresented as non-responders ($\chi^2(3)=11.15, p=.01$, vs. siblings: $OR_{Depressed}=7.04, OR_{Remitted}=4.69, OR_{Controls}=0.75$).

As a further manipulation check, we examined PASR ratings subsequent to other tasks interspersed throughout the protocol and found that they were consistently associated with

decreases in positive affect. For example, relative to levels of positive affect at baseline, PASR scores declined after a physical challenge (face exposed to ice: $M=0.31$, $SD=0.83$, $t(307)=-6.55$, $p<.001$, $d=-.37$), a task involving unsolvable puzzles ($M=0.77$, $SD=1.09$, $t(607)=-17.48$, $p<.001$, $d=-.71$), and watching a sad film clip ($M=0.87$, $SD=1.10$, $t(607)=-19.55$, $p<.001$, $d=-.79$). Thus, PASR scores reflect changing levels of positive affect as a function of experimental task.

Patterns of Positive Affect Experience

The mean PASR scores are graphically portrayed in the Figure 1. Consistent with our first hypothesis, a repeated measures ANCOVA (with age and sex as covariates) yielded effects for task ($F(3, 606)=12.87$, $p<.001$, $R_{\beta}^2=.06$) and group ($F(3,574)=23.56$, $p<.001$, $R_{\beta}^2=.11$), but the group-by-task interaction was not significant ($F(9,949)=1.07$, $p=.38$, $R_{\beta}^2<.01$). Thus, the different probes varied in the levels of positive affect they elicited, and subject groups *consistently* differed in their reactions to tasks. Across tasks, controls reported the greatest intensity of positive affect, followed by lower levels reported by siblings, remitted probands, and currently depressed probands, in that order (linear contrast $F(1,594) = 34.75$, $p<.001$, $R_{\beta}^2=.06$). This linear pattern was supported by within-task, pair-wise contrasts that controlled for age and sex. For all tasks, mean PASR scores of controls were higher than the scores of never depressed siblings (all $t(367)s > 3.39$, corrected 2-sided $ps<.01$, $ds: 0.34-0.47$), and the scores of siblings were higher than the scores of remitted probands (all $t(391)s > 2.92$, $ps<.01$, $ds: .29-.39$), while the scores of depressed and remitted probands did not differ ($t(216)s: 0.47-1.08$, $ps>.05$, $ds: 0.09-0.21$).

To examine positive affect *reactivity* (change from baseline to each task), PASR scores were modeled via rANCOVA, with group (four levels) as the independent variable, and baseline PASR rating, age, and sex as covariates. As expected, there was a main effect of task ($F(2, 607) = 11.43$, $p<.001$, $R_{\beta}^2=.04$). The group effect was also significant ($F(3, 560) = 2.95$, $p=.03$, $R_{\beta}^2=.02$), but the group by task interaction was not ($F(6, 808) = .47$, $p=.83$, $R_{\beta}^2<.01$). A test of a linear trend in reactivity across group means (controls > siblings > remitted > depressed) was not significant ($F(1, 581) = 2.70$, $p=.10$, $R_{\beta}^2<.01$). To better understand the group effect, least squares estimates of overall reactivity were compared via one-sided t -tests. Overall, remitted probands exhibited the lowest levels of reactivity ($M=0.32$, $SE=0.13$) and were similar to depressed probands in that regard ($M=.40$, $SE=.20$, $t(216)=0.45$, $p>.40$, $d=0.09$). Compared to remitted probands, reactivity was higher in siblings ($M=0.55$, $SE=0.13$, $t(391)=2.47$, $p=.03$, $d=0.25$) and controls ($M=.59$, $SE=.15$, $t(367)=2.65$, $p=.02$, $d=.28$). Thus, in so far as change scores reflect affective flexibility, controls and high-risk siblings evidenced the most flexibility, and probands evidenced the least flexibility.

To examine whether the above noted group differences in positive affectivity were due to residual depression symptoms, we repeated the analyses with CDI scores added to the models. Controlling for depression symptoms, the group effect remained significant in the rANCOVA of PASR scores ($F(3,570)=15.34$, $p<.001$, $R_{\beta}^2=.07$), as did the test for linear trend across four groups ($F(1,610)=16.23$, $p<.001$, $R_{\beta}^2=.03$). Similarly, when reactivity scores were considered by adding CDI to the prior model, the group effect was still significant ($F(3,553)=2.96$, $p=.03$, $R_{\beta}^2=.02$) while the linear effect was at a trend level

($F(1,582)=2.78, p<.10, R_{\beta}^2<.01$). A revised linear contrast comparing the average of remitted and depressed probands versus the average of siblings and controls was significant ($F(1,549)=4.28, p=.04, R_{\beta}^2=.01$). Overall, therefore, differences in depression symptoms did not account for probe-related differences in positive affectivity across the subject groups.

We also considered whether the results were affected by pubertal stage, comorbid anxiety and externalizing disorders, and the interaction of sex-by-group. However, neither pubertal stage, comorbid anxiety disorder, nor the sex-by-group interaction term emerged as statistically significant in the repeated measures ANCOVA of the raw PASR scores or the reactivity scores. Comorbid externalizing disorder was significant in the rANCOVA of PASR scores ($F(1,607)=10.34, p<.01, R_{\beta}^2=.02$), but not in the model of reactivity scores. Nonetheless, in the model of PASR scores with comorbid externalizing disorders, the group effect still remained significant ($F(3,639)=9.90, p<.001, R_{\beta}^2=.04$). Thus, impaired positive affectivity in subjects with a personal or family history of depression was not due to pubertal stage or comorbid disorders and was consistent across boys and girls.

Hedonic Responding in the Laboratory and Daily Functioning

As hypothesized, lower levels of positive affect in response to experimental probes predicted the extent of adolescents' perceived social support networks and parental reports of the adolescents' utilization of mood repair responses that mobilize positive affect. Adjusting for age, sex, and family effect in a random effects model, perceived social support was predicted by average level of positive mood across the hedonic probes ($b \pm SE=0.08 \pm 0.02$; $F(1,596)=10.73, p<.01, R_{\beta}^2=.02$) and subjects' group membership ($F(3,551)=10.09, p<.001, R_{\beta}^2=.05$). Likewise, adjusting for age, sex, and family effect in a random effects model, offspring's positive affect across the various hedonic probes predicted parental reports of that offspring's emotion regulation responses that mobilized positive affect ($b=0.22, SE=0.06, F(1,557)=14.37, p<.001, R_{\beta}^2=.03$) along with offspring's group membership ($F(3,483)=8.78, p<.001, R_{\beta}^2=.05$). In other words, adolescents with lower levels of laboratory-based positive affect had more constrained social support networks and also were characterized by diminished use of positive affect in daily life to counteract dysphoria.

Discussion

We found that currently and previously depressed adolescent probands, as well as their never-depressed high-risk adolescent siblings, reported diminished hedonic responses to a variety of laboratory probes of positive affect, as compared to emotionally healthy control peers. In fact, the three groups of youths differed in a linear fashion in levels of positive affect at both baseline and across the hedonic probes: controls reported the highest levels of positive affect, followed (in decreasing order) by high-risk siblings, and then by probands. Remitted and depressed probands reported comparable levels of positive affect. The results were similar when we examined changes in affect ratings, which are believed to reflect affective flexibility (Bylsma et al., 2008). Thus, personal history of clinical depression and familial history of depression in adolescence both appear to diminish the intensity of subjective positive affect and attenuate the magnitude of that affective response. Importantly, the experimental probes modeled some daily life situations that presumably trigger positive

emotion and mood, such as receiving a desired object, solving a mild challenge, and watching a funny movie. The fact that probands and high-risk siblings exhibited some degree of hedonic dysfunction in response to all probes suggests that they may not be able to capitalize on the varied opportunities in daily life to experience positive affect. Crucially, across-group differences in positive affect persisted when we controlled for levels of current depression symptoms. Moreover, the extent of reported positive affect in the laboratory had real-world functional significance; it predicted adolescents' perceived social support networks and their habitual use of adaptive mood repair responses that involve positive affect.

The finding of hedonic dysfunction among our remitted probands is in line with reports of attenuated positive affectivity among adult patients recovered from depression (e.g., Di Nicola et al., 2013), and extends the literature to clinical samples of adolescents. While no longer depressed, the fact that these adolescents still experienced lower levels of positive affect than controls did does support the thesis that hedonic dysfunction has trait-like features. Given reports of impaired mood repair in these very same subjects (Bylsma et al., in press; Kovacs et al., 2015), remitted major depression in youths appears to be associated with various dysfunctions in affective processing.

Never depressed siblings reported higher levels of positive affect than probands did, but yet failed to match the hedonic responses of control peers. Thus, the attenuated positive affectivity that tends to characterize young offspring at elevated risk for depression (e.g., Dietz et al., 2008; Durbin et al., 2005; McMakin et al., 2011) also is detectable in older youths who were identified via a different definition of familial risk. Given that high-risk siblings were also found to display impairment in the tone and accessibility of positive autobiographical memories (Begovic et al., submitted), and that high-risk adolescent offspring evidenced attenuated positive affectivity during mother-child interactions (Dietz et al., 2008; McMakin et al., 2011), familial risk for depression may involve multiple forms of hedonic dysfunction. Further, the finding of lowered levels of positive affectivity among never depressed youths at elevated risk for eventual depression supports the proposition that impaired hedonic capacity is a plausible risk factor or diathesis for clinical depression (Meehl, 1975; Watson & Naragon, 2009). While the actual risk of an episode of clinical depression posed by reduced hedonic capacity in our sibling sample is yet to be documented, low positive affectivity in other samples of youths has predicted subsequent depression symptoms (Lonigan, Phillips, & Hooe, 2003; Wetter & Hankin, 2009). And low reward seeking (defined via behavioral indices) reportedly prognosticated onset of depressive disorder among high-risk adolescents (Rawal et al., 2013). Thus, there is accumulating evidence that hedonic impairment may serve as a diathesis for clinical depression.

Attenuated hedonic capacity may increase the risk of a depression episode through various psychosocial and physiological routes (e.g., Dockray & Steptoe, 2010; Forbes & Dahl, 2012). For instance, based on our findings, low hedonic capacity may contribute to depression risk by adversely affecting social support networks. Social support networks, in turn, play an important role in buffering the effects of stress events that may trigger depression. Our findings also suggest the possibility that lower levels of positive affect may compromise the availability of certain mood repair responses, such as the recall of positive

memories to attenuate sadness (Isen, 1985; Josephson, Singer, & Salovey, 1996). In turn, a constricted mood repair response repertoire may make it more difficult to manage the affective consequences of depressogenic triggers (Kovacs & Lopez-Duran, 2010).

Because both the ecological validity and practical implications of laboratory probes of affect have been questioned, it is particularly notable that we documented various extra-laboratory functional correlates of experimentally induced positive affect. Youths who reported higher levels of positive affect in the laboratory had better social support networks and were more likely to deploy adaptive mood repair responses in daily life that mobilize positive affect. These findings are in line with the facilitating role of positive affect in various areas of functioning (Fredrickson, 2001) and were based on reports provided independently by youths and parents, thereby increasing confidence in the results.

The present study has various strengths, including several large samples, its extension of the traditional high-risk study design to juvenile siblings, thorough diagnostic evaluation of the subjects across the years, and the use of multiple hedonic probes and their randomized presentation. Further, the probes included a sampling of stimuli/tasks that might be encountered in daily life, increasing the validity of the results. As well, some of the findings may have practical implications. For example, receipt of a preferred prize was the most robust hedonic probe, suggesting that personal relevance is an important dimension to be considered in protocols using positive affect probes.

Along with similar work by others in this area (reviewed above), our findings complement a corpus of investigations that point to blunted neural responses to positive stimuli or reward related tasks in high risk compared to low risk young offspring (Kujawa, Hajcak, Torpey, Kim, & Klein, 2012; Monk et al., 2008), adolescents and young adults (Foti, Kotov, Klein & Hajcak, 2011; Gotlib et al., 2010; McCabe et al., 2012), and adult offspring and older adult siblings (Macoveanu et al., 2013). A recent study also found that blunted neural response to reward predicted onset of the first major depression episode among adolescent girls at variable familial risk for depression (Bress, Foti, Kotov, Klein, & Hajcak, 2013). However, neural, subjective, and behavioral dimensions of hedonic abnormalities have not yet been compellingly linked within and across studies.

Recent approaches (particularly those focused on neural mechanism) have emphasized demarcating the anticipatory/motivational and consummatory (i.e., wanting versus liking) phases of hedonic processing (Treadway & Zald, 2011; Whitton, Treadway, & Pizzagalli, 2015). The present study did not address hedonic function during task anticipation but instead focused on the experiential or “liking” aspect of pleasurable experience. Contrary to some work indicating no impairment during the consummatory phase of hedonic processing among depression-prone adults (reviewed by Treadway & Zald, 2011; Whitton et al., 2015) and similar findings in a small sample of high-risk adolescents (Olinio et al., 2014), but in line with various reports we cited above, probands and high-risk youths in our study did evidence “consummatory” hedonic impairment. Such discrepant results probably reflect important methodological differences across studies, including varying definitions of the outcome and the type of experimental hedonic probe that was used.

The findings of this study should be evaluated in the context of several limitations. First, it is possible that we failed to find significant differences between depressed (n=32) and remitted probands (n=186) in positive affectivity because of low statistical power. Further, while there is compelling evidence that positive affectivity is strongly heritable (e.g., Watson & Naragon, 2009), our study was not designed to disentangle the influence of familial depression versus low familial positive affectivity on the results. Finally, our study focused on subjective experience as indexed by self-report, and did not consider other response systems.

Given the accumulating findings and the ongoing groundswell of interest regarding hedonic dysfunction in depression, what questions should be prioritized and what studies should be conducted as the logical next steps? One basic question still to be conclusively answered is whether hedonic dysfunction in the context of depression is a trait or a state. While the best interpretation of extant findings (based on cross-sectional comparisons of depressed, remitted, and control subjects) is that hedonic dysfunction has both trait and state components, a definitive answer can only be based on longitudinal studies that repeatedly test the same high-and low-risk individuals across varying mood states. Is hedonic dysfunction a bona fide risk factor that precedes and predicts first onset of clinical depression? Based on accumulating evidence, the answer probably is “yes,” although further supportive findings are needed. However, the relevant literature reflects group differences across samples at high versus low risk for depression, while hedonic capacity is clearly an individual differences variable, impairment in which is only one route to clinical depression (e.g. Meehl, 1975). Thus, the challenge is to identify those who exhibit this characteristic and then intervene to remediate the presumed dysfunction.

How can individuals with low hedonic capacity best be identified? On the one hand, there is consensus that (regardless of how it is assessed) the construct of hedonia has various facets beyond experiential or consummatory pleasure and positive affect. The complexity is well reflected by the numerous studies of reward processing, which have underscored the additional importance of anticipation, stimulus salience, as well as related motivational, decision making, and learning processes (e.g., Treadway & Zald, 2011; Whitton et al., 2015). On the other hand, the practical ramifications of this broad literature for the identification of vulnerable cases are yet to be established. For example, it is unclear how the various facets of hedonic functioning relate to positive affect (Treadway & Zald, 2011), which has long been considered a fundamental dimension of pleasure and joy. Consequently, it is unclear whether early identification of high-risk cases should be based on attenuated responses to positive stimuli, impaired incentive motivation, problems in reward-related learning, some other index of performance, or combinations of these variables. Importantly, little is known about how these facets “play out” in everyday life and which facet is most predictive of functional impairment or clinical course.

Several of these issues could be addressed in samples of interest by monitoring hedonic related responses and processes in daily life (along the lines of ecological momentary assessment designs). Such studies could explore the relative importance of affective, cognitive, motivational, and information processing mechanisms and their interactions in hedonic responding, as well as the roles of hedonic stimulus access and salience *in situ*, and

how contextual features may attenuate or facilitate hedonic dysfunction. The resultant information could yield integrated guidelines about which parameters best identify hedonically-challenged individuals and also inform existing approaches to remediate anhedonia in depressed people (e.g. McMakin, Siegle & Shirk, 2011).

Finally, another critical topic is the developmental origins of anhedonia in depression-prone youths. For example, while affective processes appear to be most salient in the consummatory (liking) phases of hedonic experience, cognitive processes appear to predominate in the motivational/anticipatory phases; this is important because components of affective and cognitive processing (and associated neural mechanisms) have different developmental trajectories. Thus, it would be helpful to know how early in development children at high risk for depression evidence atypical cognitive (e.g., motivational) versus the affective (e.g., experiential) facets of hedonic processing, which type of impairment is most likely to derail subsequent development, and how these processes relate to early life stress, which has been implicated in eventual affective abnormalities (e.g., Pechtel & Pizzagalli, 2011). Information about the developmental course of dysfunctional hedonic processes, and the conditions under which they persist, could yield new approaches to intervention with depression-prone youths in the search to lower the risk of clinical depression among them.

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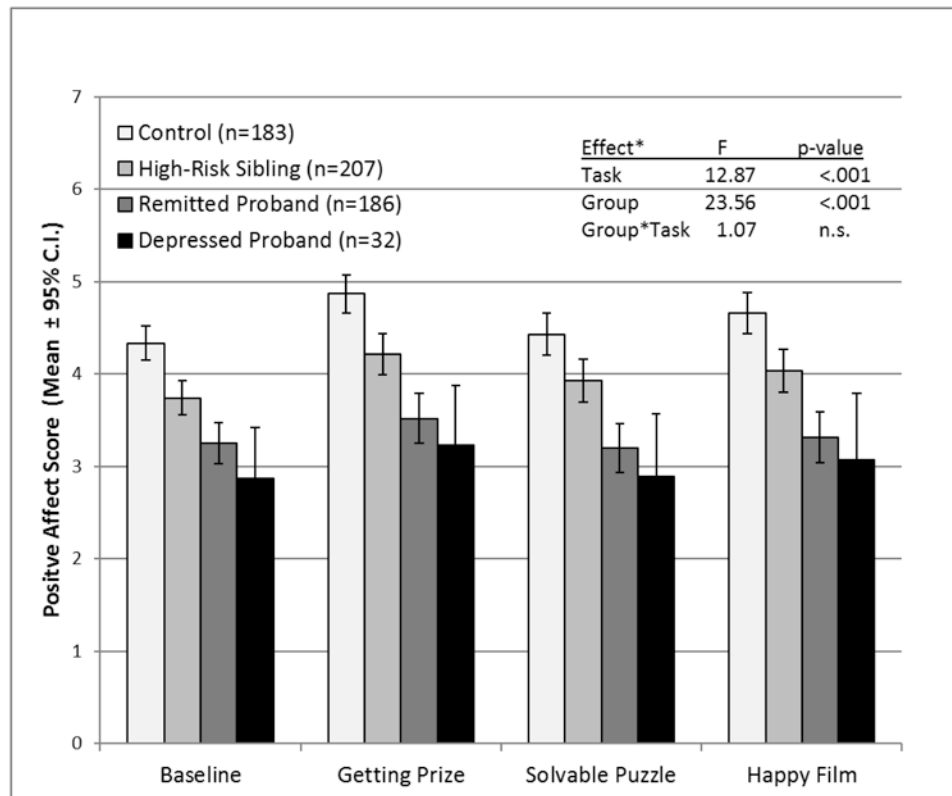


Figure 1. Self-Rated Positive Affect Composite Score Across Baseline and Three Hedonic Probes^a

Note: Effects are from the full factorial model of group and time, adjusting for age and sex fixed effects, family random effect, and within-subject unstructured correlation.

^a The probes were presented in randomized order across cases

Table 1
Selected Demographic Characteristics and Baseline Self-Ratings of Affect

Variable	Controls (n=183)	High-Risk Siblings (n=207)	Remitted Probands (n=186)	Depressed Probands (n=32)	Statistic [†]	Effect Size [‡]
Age (years)	16.10 ± 2.13 _a	15.94 ± 2.12 _{a,b}	17.14 ± 1.35 _b	16.27 ± 1.47 _{a,b}	15.3 ^{***}	.08
Sex (% Male)	64.5 _a	44.9 _b	63.4 _a	68.8 _{a,b}	21.1 ^{***}	.19
PANAS						
Positive Affect Score	30.98 ± 5.91 _a	29.11 ± 6.22 _b	27.51 ± 6.66 _{c,d}	25.68 ± 8.12 _d	12.1 ^{***}	.06
Negative Affect Score	15.28 ± 4.29 _a	17.95 ± 6.20 _b	17.37 ± 5.52 _{b,c}	25.19 ± 9.34 _d	28.0 ^{***}	.13
CDI Total Score	4.74 ± 4.23 _a	8.67 ± 5.96 _b	9.24 ± 6.32 _{b,c}	18.53 ± 8.17 _d	56.7 ^{***}	.24
Positive Affect Self-Ratings (PASR)						
Happy	4.35 ± 1.54 _a	4.00 ± 1.69 _a	3.54 ± 1.83 _{b,c}	2.72 ± 1.82 _c	12.3 ^{***}	.06
Enthusiastic	3.95 ± 1.74 _a	3.27 ± 1.76 _b	2.73 ± 1.82 _{b,c}	2.28 ± 1.90 _c	17.8 ^{***}	.09
Interested	4.70 ± 1.58 _a	3.96 ± 1.92 _b	3.48 ± 1.92 _{b,c,d}	3.59 ± 2.05 _d	14.5 ^{***}	.07
Composite of 3-items	4.15 ± 1.45 _a	3.63 ± 1.43 _b	3.13 ± 1.60 _{b,d}	2.50 ± 1.60 _d	22.0 ^{***}	.11

Note: Mean ± SD unless otherwise indicated. PANAS = Positive and Negative Affect Schedule. CDI = Children's Depression Inventory.

[†] For χ^2 , adjusted for family effect;

[‡] f^2 or W

p < .001.

Group means with common subscripts did not differ significantly from one another in post-hoc tests (Tukey, $p > .05$).