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**Beyond the Diathesis-Stress Paradigm: Effect of the Environmental Sensitivity x
Pubertal Tempo Interaction on Depressive Symptoms**

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Abstract

Based on the *Differential Susceptibility Theory*, we examined whether the relationship between pubertal maturation and depressive symptoms can be moderated by individual differences in environmental sensitivity. The current article used the three-wave data collected from Japanese adolescents aged from 12 to 15 years (girls = 111, boys = 98). Consequently, a significant Sensitivity x Pubertal Development interaction was observed in 12- to 13-year-old boys, but not girls. Sensitive boys who experienced accelerated physical maturation reported decreased depressive symptoms, while those who experienced less maturation had increased depressive symptoms. The shape of the interaction supported both the *Differential Susceptibility Theory* and the *Diathesis-Stress Model*. Our findings suggest that sensitivity during early puberty among boys could be reconsidered as *susceptibility* rather than *vulnerability*.

Keywords: differential susceptibility; environmental sensitivity, highly sensitive child; temperament; puberty; depression

Beyond the Diathesis-Stress Paradigm: Effect of the Environmental Sensitivity x Pubertal Tempo Interaction on Depressive Symptoms

Adolescence is a period of transition from childhood to adulthood, during which adolescents experience a variety of psychological (e.g., identity construction; McLean, 2005), social (e.g., transition to secondary school; Benner, 2011; Iimura & Kibe, 2020), and biological (e.g., onset of puberty; Ge & Natsuaki, 2009; Peper & Dahl, 2013) changes. Determining how changes experienced during transitional periods affect adolescents' psychological adjustment has traditionally been an important topic of research for developmental researchers. In particular, children experience socio-emotional changes during puberty when they develop their secondary sexual characteristics (Ge & Natsuaki, 2009), and a recent theory regarding differential susceptibility to environmental influences suggested that individual differences in *Environmental Sensitivity* played a vital role in how they experienced these changes (Belsky, 2019). However, while the effects of pubertal development on psychopathology have been well documented (Ge et al., 2001a; Ge et al., 2001b; Ge et al., 2003; Ge & Natsuaki, 2009), the ways in which *Environmental Sensitivity* moderates this relationship has been overlooked. Therefore, the present study aims, for the first time, to clarify the role of *Environmental Sensitivity* in the development of depressive symptoms during puberty.

Environmental Sensitivity in Child Development

Adolescents undergo socio-emotional development through dynamic interactions with different levels of the environment (Bronfenbrenner & Morris, 2006). Importantly, adolescents differ in their susceptibility to environmental influences (Belsky, 1997). The *Environmental Sensitivity Theory* (Greven et al., 2019; Pluess, 2015), an overarching umbrella framework for *Differential Susceptibility Theory* (Belsky, 1997), *Biological Sensitivity to Context Theory* (Boyce & Ellis, 2005), and *Sensory-Processing Sensitivity*

(Aron et al., 2012), is known as an evolutionary-developmental theory and concept, which explains that human and over 100 other species (Aron et al., 2012) differ in their susceptibility to contextual factors—some individuals are more susceptible than others to both positive and negative environmental influences. These individual differences are explained by the concept of *Environmental Sensitivity*, which is postulated to be caused by a heightened sensitivity of certain features in the central nervous system (e.g., amygdala, hippocampus, and insula) (Greven et al., 2019; Pluess, 2015)¹. Therefore, people differ in their ability to process and register external and internal stimuli. Genetic (Keers et al., 2016), neurophysiological (Rudolph et al., 2020), and temperament/personality (Aron & Aron, 1997) factors contribute to individual differences in *Environmental Sensitivity*.

Previous studies that used large samples of children and adolescents often measured *Environmental Sensitivity*, which was observed as temperament or personality traits (Pluess et al., 2018). While difficult temperament and negative emotionality have traditionally been considered as temperamental susceptibility factors, several recent studies have focused on *sensory-processing sensitivity*, which represents the temperament/personality aspect of *Environmental Sensitivity* (Pluess et al., 2018; Pluess et al., 2020; Weyn et al., 2021). Sensory-processing sensitivity is defined as “a fundamental trait found on a spectrum and is defined as the degree to which an individual may register, process, and respond to external factors” and “characterized by greater depth of processing, cognizance of subtleties in the environment, being easily overstimulated, having stronger emotional responses (both positive and negative), and empathy to others’ affective cues (Acevedo et al., 2018, pp. 1–2).” A recent study on adolescent twins reported an estimated heritability of 48% for sensory-

¹ Adolescence is a time when neural sensitivity to reward is heightened by pubertal hormones (e.g., Galván, 2013). The relationship between reward sensitivity and *Environmental Sensitivity* (whether the two are completely independent or partially overlapping concepts) is unclear. However, as *Environmental Sensitivity* is a concept that refers to not only sensitivity to reward but also a wider range of environments, the authors assume that *Environmental Sensitivity* is a distinctly different concept from reward sensitivity.

processing sensitivity as measured by the Highly Sensitive Child Scale (Assary et al., 2020). In other words, this suggested that about 50% of individual differences in sensory-processing sensitivity were shaped by the early environment. In addition, a recent meta-analysis demonstrated that conceptual discrimination between sensory-processing sensitivity and other personality traits (e.g., Big Five) and emotionality (e.g., positive and negative affect) (Lionetti et al., 2019). Furthermore, a review article examined the association between sensitivity and neural activity concluded that autism spectrum disorders, post-traumatic stress disorder, and schizophrenia, which are indicative of sensory atypicality, are qualitatively differentiated from sensory-processing sensitivity (Acevedo et al., 2018). It is important to note that sensory-processing sensitivity moderates the environmental quality youths' experiences and their socio-emotional adjustment. For example, Slagt et al. (2018) found that mother-rated 4- to 5-year-old child's sensitivity moderated, for better and for worse, the relationship between positive/negative parenting and externalizing behavior. This finding supported the *Differential Susceptibility Theory*, suggesting that a child's heightened sensitivity is not simply a vulnerability but a susceptibility. In addition, Noccentini et al. (2018) demonstrated that a school-based anti-bullying intervention program was particularly effective on students with high sensitivity. Taken together, these findings provide evidence that sensory-processing sensitivity functions as a moderator for the association between environmental influences and developmental outcomes. These findings support a central notion of *Environmental Sensitivity* which is - sensitive individuals are more susceptible to both positive and negative environmental influences than others, and thus their socio-emotional development can be moderated, for better and for worse, by the quality of internal or external experience.

Sensitivity x Environmental Stimuli Interaction

Fortunately, the accumulating literature of developmental science appears to have greatly advanced our understanding of who is *vulnerable* to being negatively affected by the

changes in the external and internal environment during adolescence. Research based on the traditional Person x Environment interaction framework, known as the *Diathesis-Stress Model* or *Dual-Risk Model* (Monroe & Simons, 1991; Model A in Figure 1), has contributed to our understanding. These classical models assume that individuals with certain dispositional risks are more likely to be negatively affected by an adversarial environment (Belsky & Pluess, 2009). However, the elaboration of theories since the 1990s has pointed out the limitations of the *Diathesis-Stress* view; currently, the *Differential Susceptibility Theory* derived by the evolutionary reasoning has become the dominant view (Belsky et al., 2007; Model B in Figure 1). The *Differential Susceptibility Theory* views susceptibility to environmental stimuli as more *susceptibility (endophenotype)* rather than *vulnerability*. Therefore, high sensitivity functions negatively for adolescents' psychological adjustment under an adverse environment, while it functions to benefit them under a positive environment. In other words, susceptible individuals are more likely to be affected—for better and for worse—depending on the quality of their experiences, than others. Recently, the *Vantage Sensitivity Model* (Pluess & Belsky, 2013; Model C in Figure 1), which is closely related to “bright side” of the *Differential Susceptibility Theory*, has been proposed, and this framework assumes that, as opposed to *Diathesis-Stress Model*, susceptible individuals are more likely to benefit from a positive environment compared to less susceptible individuals. In contrast, individuals with low susceptibility are less likely to benefit from positive experiences.

[Insert Figure 1]

As mentioned above, developmental researchers have reconsidered sensitivity as *susceptibility* (i.e., plasticity factor) rather than *vulnerability*. Nonetheless, perhaps this depends on the type of environment and developmental outcome being measured. For example, one study may be consistent with the *Differential Susceptibility Theory*, while

another may support the *Diathesis-Stress Model* or the *Vantage Sensitivity Model*. Roisman et al. (2012) proposed a statistical method to quantify the shape of interaction and demonstrated that the shape of interaction between early maternal sensitivity and susceptibility trait was consistent with the *Diathesis-Stress Model* in child development including subjective academic skills and social competence. In contrast, the shape of interaction supported the *Differential Susceptibility Theory* if teacher-rated problem behaviors and social competence were developmental outcomes. Given that supported frameworks vary depending on the variables researchers focus on, we need to explore which framework is most appropriate to explain our research question, that is, the interaction of sensitivity and pubertal maturation on depressive symptoms.

Pubertal Tempo and Psychopathology

Conventionally, puberty has been viewed as a time of increased *vulnerability* due to physiological and physical changes (Ge & Natsuaki, 2009; Peper & Dahl, 2013). Pubertal development, an extremely dramatic biological and physical change in adolescence, is primarily characterized by two major changes (Petersen et al., 1988)—endocrine changes, which includes the release of gonadal hormones, and somatic changes, which includes growth in body size and shape related to secondary sexual characteristics (Corley et al., 2015; Shirtcliff et al., 2009). The processes of pubertal development differ by sex. For example, testosterone, an androgen released from the gonadal axis, causes genital development and changes in voice in males. Estrogen and estradiol promote breast development and fat distribution in females. The growth of body hair is regulated by adrenal hormones in both sexes, while height is regulated by sex steroids and growth hormones.

Questionnaires are often used to assess pubertal development at the somatic level. For example, the Petersen's Pubertal Development Scale (PDS), either self- or other-rated, has been widely used internationally to measure somatic changes. This scale assesses the

physical condition of both boys and girls with respect to sexual hair and height, beard growth in males, and breast development in girls. The validity of the scale has been well studied; for instance, a high correlation between interview ratings and self-ratings based on scale scores has been reported (Petersen et al., 1988). In addition, there is consistency between mother, child, and teacher ratings using the PDS (Carskadon & Acebo, 1993).

Importantly, developmental researchers have reported that individual differences in pubertal status, pubertal timing, and pubertal tempo² can be important factors associated with development of psychopathology, including depressive symptoms and self-control (Ge & Natsuaki, 2009; Ng-Knight et al., 2016). For example, although findings are inconsistent, it has been suggested that early pubertal timing, or early maturation, is associated with a higher risk of depressive symptoms in girls (Keenan et al., 2014). According to some research findings, early maturing girls who had recently experienced stressful life events reported higher depressive symptoms (Ge et al., 2001a). Furthermore, it has been suggested that higher cognitive vulnerability may interact with early pubertal timing in girls to predict higher depressive symptoms (Hamilton et al., 2014). Although the findings for boys are fewer and appear to be less consistent than those for girls, late maturation is the most distressing for boys, whereas early maturation has the most negative impact on girls (Siegel et al., 1999).

Our knowledge of pubertal tempo is minimal compared to pubertal timing. However, studies have suggested that pubertal tempo may also alter the vulnerability of depression. For example, Ellis et al. (2011) pointed out that a faster pubertal tempo could not only cause the events involved in maturation to occur earlier but also be psychologically challenging in and

² Pubertal status refers to “the degree of physical maturation or the level of physical development individuals have reached (e.g., pre-pubertal, mid-pubertal, or post-pubertal) with no necessary reference to the age (Ge et al., 2001b, p.51).” Pubertal timing indicates whether a child’s physical development occurs earlier than, at the same time as, or later than that of his or her same-gender, same-age peers (Ge et al., 2003). Pubertal tempo indicates the rate of within-person progression with respect to physical maturation (Keenan et al., 2014). Pubertal status and timing are state (cross-sectional) indicators of physical maturation, while pubertal tempo is a (longitudinal) indicator of changes in physical status.

of itself. According to Keenan et al. (2014), slower tempo was associated with higher depressive symptoms at age 10, and a faster tempo was associated with increased depressive symptoms in girls aged 10–13 years. In addition, Mendle et al. (2010) found that a faster pubertal tempo did not explain higher depression in girls, while it predicted higher depression in boys. Furthermore, Deardorff et al. (2021) demonstrated that a faster pubic hair tempo was associated with higher anxiety among Hispanic girls, but was associated with lower depressive symptoms among Asian girls. Regardless of ethnicity, the tempo of breast development was not associated with mental health problems. As these findings indicated, although the association between pubertal timing and depressive symptoms was relatively robust, the association between faster pubertal tempo and depressive symptoms was not consistent across studies. Specifically, the effects of pubertal tempo seemed to vary not only by age, gender, ethnicity, and body part, but also that a faster tempo did not necessarily correlate with higher depressive symptoms. Although various factors are expected to be involved in the inconsistent effects of pubertal tempo, we believe that an important clue to understanding it is individual differences in sensitivity to physical changes; some adolescents are more susceptible to rapid physical changes than others. However, this perspective, that is, the interaction between individual differences in susceptibility and pubertal tempo, has not been sufficiently examined to date (as an exception, the accentuation hypothesis (Caspi & Moffitt, 1991) examined the interaction between prepubertal behavior problems or vulnerable temperament and puberty).

The Current Study: Beyond Diathesis-Stress Paradigm

Conventionally, developmental researchers have assumed that *vulnerable* adolescents are more likely to experience aggravating depressive symptoms, depending on their pubertal status and tempo. In other words, the association between pubertal development and depressive symptoms has been traditionally examined within the framework of the *Diathesis-*

Stress Model. However, since the introduction of the *Differential Susceptibility Theory*, traditional *Diathesis-Stress* thinking may need to be reconsidered. In other words, individual susceptibility may moderate the effects of puberty on their psychological adjustment, for better or worse.

Given that research on differential susceptibility has traditionally focused on the interaction between a child's sensitivity and the *external* environment (primarily parenting), this study's emphasis on the interaction between sensitivity and *internal* stimuli (i.e., physical maturation) is novel. Importantly, as the concept of *Environmental Sensitivity* is defined as sensitivity to both external and internal environmental stimuli (e.g., Greven et al., 2019), children with higher sensitivity may be more responsive, for better or worse, to changes within their own bodies compared to those with lower sensitivity. In a broader sense, physical status can be understood as part of the (internal) environment. However, this perspective has been overlooked in existing research on differential sensitivity.

Based on the above discussion, the present study exploratively examines whether sensory-processing sensitivity moderates the relationship between pubertal maturation and depressive symptoms, and if so, which of the *Diathesis-Stress* or *Differential Susceptibility* or *Vantage Sensitivity* frameworks best explains depressive symptoms in puberty. We used longitudinal data from 209 Japanese adolescents who were 12–15 years of age, collected from an ongoing longitudinal survey project with three time points and one-year intervals, for our analysis. As mentioned above, early adolescence is a period in which individual differences in pubertal development can be observed, and data from adolescents aged 12–15 are suitable for examination of our research objectives (for example, see Baker et al. (2012), Copeland et al. (2019), Joinson (2012), and Swarr and Richards (1996) for studies that focused on adolescents in the same age range as our study participants). Based on Roisman's approach (see the Data Analysis section), we examined the appropriate shape of interaction between

sensitivity and pubertal growth that explains changes in depressive symptoms. Given the novelty of this study, we did not formulate any hypotheses regarding the interaction between sensitivity and puberty.

Method

Procedure and Participants

We recruited children and their mothers who participated in the ongoing longitudinal [blinded] project. For this project, a total of 209 Japanese children and their mothers were initially recruited from all over Japan through a research company. A paper-and-pencil questionnaire survey had been administered once a year and completed at three time points.

At Time 1, 209 children (111 girls; $M_{\text{age}} = 12.8$, $SD_{\text{age}} = 0.5$) and their mothers ($M_{\text{age}} = 44.8$, $SD_{\text{age}} = 6.3$) completed the questionnaire. At Time 2, 95 children (47 girls; $M_{\text{age}} = 13.7$, $SD_{\text{age}} = 0.6$) and their mothers participated; and at Time 3, 93 children (45 girls; $M_{\text{age}} = 14.7$, $SD_{\text{age}} = 0.6$) and their mothers were included. Retention rates for the participants were 46% at Time 2, and 45% at Time 3. The most frequent annual household income ranged from 4,000,000 to 6,000,000 JPY (approximately 37,280 to 55,920 USD). The institutional review board of the University of Tokyo approved all procedures in the current study (No. 17-122, 17-179, 18-192, and 19-160).

Measures

Children rated their own depressive symptoms at all three-time point and reported sensory-processing sensitivity at Time 3. Mothers evaluated their child's pubertal status on each occasion. Detailed information regarding the distribution of variables we assessed can be found at the Open Science Framework (<https://osf.io/aqd2f/>). The following three questionnaire were used in the current study.

Depressive symptoms were assessed using the Japanese short version of the

Depression Self-Rating Scale for Children (Namikawa et al., 2011). This scale consisted of nine items (e.g., “*I look forward to things as much as I used to (reverse)*,” “*I think life is not worth living*,” “*I feel like crying*”) and was rated using a three-point Likert scale (1 = *most of the time*, 2 = *sometimes*, 3 = *no/never*). The internal consistency was good for both girls and boys, with Cronbach’s alpha = .78 to .84 over three time points. Given that the interest of this study was the socio-emotional development of adolescents, the change scores for depressive symptoms from Time 1 to Time 2, Time 2 to Time 3, and Time 1 to Time 3 were calculated and used in the analysis.

The 12-item Japanese version of the Highly Sensitive Child Scale for Childhood (Kibe & Hirano, 2020) was used to measure student’s sensitivity at Time 3³. As with the English scale (Pluess et al., 2018), this scale included items that reflected the temperament/personality aspects of *Environmental Sensitivity* such as ease of excitation (e.g., “*loud noises make me feel uncomfortable*”), low sensory threshold (e.g., “*I get nervous when I have to do a lot in little time*”), and aesthetic sensitivity (e.g., “*some music can make me really happy*”). Each item was rated using a seven-point Likert scale, ranging from 1 (*not at all*) to 7 (*extremely*). The internal consistency of the 12 items was somewhat low with Cronbach’s alpha = .54 at Time 3. As in previous studies (Iimura & Kibe, 2020; Pluess et al., 2018; Weyn et al., 2021), the bifactor model, which consisted of a general sensitivity factor and the three subscales, showed an acceptable fit to the data (root mean square error of approximation [RMSEA] = .09, standardized root mean square residual [SRMR] = .06, comparative fit index [CFI] = .79).

The Japanese version of the Pubertal Development Scale (Petersen et al., 1988; Mukai & Ito, 1995) was used to assess pubertal maturation. Mothers were asked the extent to

³ Sensory-processing sensitivity, a dispositional marker of *Environmental Sensitivity*, is a stable concept within individuals, as are other temperament or personality traits. In fact, studies that used this scale reported high test-retest reliability (e.g., Pluess et al., 2018). Therefore, measuring this at Time 3 was not expected to pose any measurement problems.

which their children had experienced pubertal growth in several domains (e.g., body hair development, growth spurt, skin changes, and breast development) using five items for boys and four items for girls⁴. While some studies have questioned the validity of parental ratings (Rasmussen et al., 2015), a recent study reported a high association between parental ratings and the Tanner scale for children and adolescents aged 7-18 years (Koopman-Verhoeff et al., 2020). The latter finding supports the validity of the measurement method employed. Each item was rated using a four-point Likert scale, ranging from 1 (*has not begun*) to 4 (*development completed*). We also added the option 5 (*unknown*), and if this option was chosen, the item was assigned as a missing value. Cronbach's alphas were .61 to .75 for girls and .71 to .84 for boys across three time point. Similar to the depression scale, difference scores between each time point were calculated to evaluate changes in pubertal status (i.e., pubertal tempo)⁵.

Data Analysis

Based on the recently recommended procedure known as Roisman's exploratory approach (Roisman et al., 2012), we conducted a hierarchical multiple regression analysis for predicting changes in depressive symptoms by gender to evaluate the optimal shape of Person x Environment interaction (i.e., *Differential Susceptibility* framework vs. *Diathesis-Stress* framework vs. *Vantage Sensitivity* framework). In the first model, we examined only the main effects of sensitivity and pubertal tempo (Model 1). The second model tested the interaction between sensitivity and pubertal tempo (Model 2). Bayesian information criterion (BIC) and Akaike information criterion (AIC) were used to compare which model fitted the data. With

⁴ In this study, the item on the onset of menarche was not used. Given that this item was a categorical variable (and furthermore, its method of scoring differed from the other items) and the other items were ordinal or ratio scales, we judged that there would be serious statistical problems in calculating their total scores and using them in the analysis.

⁵ In general, the latent growth curve model is often used to statistically represent pubertal tempo (e.g., Koopman-Verhoeff et al., 2020). However, in this study, due to the small sample size, this was alternatively represented by calculating the difference scores from Time n to Time n-1.

this criteria, a lower values indicated a better model. If Model 2 yielded better fit to the data than Model 1 and the interaction term was significant, we proceeded to a simple slope test to examine the slope of sensitivity at $M \pm 1SD$ using the free application

(<https://www.yourpersonality.net/interaction/ros.pl>).

To further quantify the shape of the interaction, we then estimated the following four indicators. 1) The regions of significance on predictor (RoS on X): RoS on X test examines how Y (i.e., changes in depressive symptoms) and Z (i.e., sensitivity) are correlated both at the high and low ends of the distribution of X (i.e., pubertal tempo) bounded by a conventional range of interest that is $\pm 2SD$ from the mean of X. In RoS on X test, if the relationship between sensitivity (moderator) and changes in depressive symptoms (outcome) was significant at the high but not low end of pubertal growth, our data supported the *Diathesis-Stress* framework. In addition, if the association was significant at both the low and high ends of pubertal tempo, our data supported the *Differential Susceptibility* framework. Further, if the association was significant at the low but not high ends of pubertal tempo, our data underpinned the *Vantage Sensitivity* framework (Roisman et al., 2012). 2) The RoS on moderator (RoS on Z): the upper/lower bound shows the value of sensitivity above/below, where the regression of outcome (i.e., changes in depressive symptoms) on pubertal tempo is statistically significant. 3) The crossover point where the lines of the high and low sensitivity groups crossed on pubertal growth; and 4) the proportion of interaction (PoI) index, which was the ratio of the *better* area to the total area (i.e., $better / [better + worse]$: see Appendix Figure A1 for visual representation). According to Roisman et al. (2012) and Del Giudice (2017), if the value of PoI index⁶ was between about 0.40 and 0.60, it would represent an

⁶ According to Roisman et al. (2012), a crossover point is not a criterion for determining *Differential Susceptibility* framework. On the other hand, according to Widaman et al. (2012), the location of the crossover point is a criterion for identifying *Differential Susceptibility* framework. In this study, the crossover points were calculated, but the interpretation of the shape of the interaction was based on the PoI. For more information on how to calculate the PoI index, see Roisman et al. (2012).

interaction effect highly consistent with *Differential Susceptibility* framework. If it approached 1.00, this suggested strong evidence for *Vantage Sensitivity* framework. If it approached 0.00, it then indicated *Diathesis-Stress* framework.

Missing Data Analysis. To examine the missing patterns in the current data, we performed Little's missing completely at random (MCAR) test, which suggested that our longitudinal data, which included depressive symptoms, pubertal status, and sensitivity, were not MCAR ($\chi^2 = 318.19$, $df = 242$, $p < .001$). Perhaps the reason for this is that the dropout rate increased after Time 1 as this study did not have sufficient research funding. For participants who responded to all the surveys, the missing values for depressive symptoms were 2.3%, 6.7%, and 0.0% at Time 1, 2, and 3, respectively. The missing values for pubertal status were 7.9%, 16.9%, and 21.4% at Times 1, 2, and 3, respectively. Sensory-processing sensitivity did not contain any missing values. Participants who completed all the surveys had higher depressive symptoms at Time 1 compared to those who dropped out ($t(204) = 4.74$, $d = .67$, $p < .001$). However, there was no difference in pubertal status between the genders (boys: $t(82) = 1.52$, $d = .33$, $p = .132$; girls: $t(99) = 1.66$, $d = 0.34$, $p = .099$). In the present study, the missing data were supplemented using a multiple imputation method rather than a listwise deletion to deal with the reduced statistical power. The *mice* package in R was used to perform the multiple imputation method: "the method is based on Fully Conditional Specification, where each incomplete variable is imputed by a separate model (van Buuren & Groothuis-Oudshoorn, 2011 p.72). We imputed the missing values by generating ten datasets based on a normal distribution. A supplementary table showing the differences in the results of the correlation coefficient tests based on multiple imputation methods and listwise methods has been uploaded to the Open Science Framework (<https://osf.io/aqd2f/>). The results of the post-hoc power analysis using G*Power (Faul et al., 2009) showed that when a moderate correlation coefficient was detected ($r = .30$, $\alpha = .05$), the statistical power was .87 for boys (n

= .98) and .91 for girls ($n = 111$), which indicated adequate power. All analyses were conducted on R, version 3.6.3 (R Core Team, 2018) and its interface RStudio, version 1.2.5033.

Results

Preliminary Analysis

Table 1 shows descriptive statistics for changes in depressive symptoms and pubertal tempo, and sensitivity by gender. While no significant mean differences in depressive symptoms between each occasion were found in boys ($F(2, 194) = 0.08$, $\eta_p^2 = 0.00$, $p = .927$); the means at Times 2 and 3 were higher than that at Time 1 in girls ($F(2, 220) = 4.69$, $\eta_p^2 = 0.41$, $p = .011$). An expected significant increase in the means of pubertal status was observed for both boys ($F(2, 194) = 57.36$, $\eta_p^2 = 0.37$, $p < .001$) and girls ($F(2, 220) = 128.35$, $\eta_p^2 = 0.54$, $p < .001$) over time.

[Table 1]

Table 2 presents the bivariate correlations between depressive symptoms and pubertal tempo, and sensitivity by gender. There was no significant correlation between changes in depressive symptoms and pubertal tempo for either gender. A weak positive correlation between changes in depressive symptoms from Time 2 to Time 3 and sensitivity was found only in girls ($r = .28$, 95%CI [.10, .45], $p = .002$). No significant correlation between pubertal tempo and sensitivity was observed in boys. For girls, pubertal tempo from Time 1 to Time 2 were positively correlated with sensitivity ($r = .21$, 95%CI [.03, .38], $p = .027$), while pubertal tempo from Time 2 to Time 3 were negatively associated with sensitivity ($r = -.26$, 95%CI [-.42, -.07], $p = .007$).

[Table 2]

Testing Sensitivity x Pubertal Tempo Interaction

Boys

A significant interaction was observed between sensitivity and pubertal tempo only in boys aged 12 to 13 (Table 3)⁷. The right side of Table 3 shows that sensitivity ($\beta = -0.03$, $p = .637$) and pubertal tempo ($\beta = 0.07$, $p = .234$) did not predict changes in depressive symptoms in the main effects model (Model 1), but their significant interaction ($\beta = -0.19$, $p = .031$) was detected in the interaction model (Model 2)⁸. In addition, both BIC and AIC supported that Model 2 (BIC = 103.45, AIC = 90.57) had a better fit than Model 1 (BIC = 103.82, AIC = 93.47).

[Table 3]

Figure 1 depicts the shape of the interaction as identified by the simple slope test. Highly sensitive (i.e., +1SD) boys reported decreased depressive symptoms with accelerated pubertal maturation around 12 to 13 years of age ($\beta = -0.21$, $p = .036$). In contrast, no such association was identified for the low-sensitivity (i.e., -1SD) boys ($\beta = 0.17$, $p = .064$). The RoS on Z test showed that the association between pubertal tempo and depressive symptoms was significant with standardized scores on the sensitivity ranging from -1.15 to 0.80. In addition, in the RoS on X test, the association between sensitivity and changes in depressive symptoms was significant in areas with pubertal tempo of -11.28 or less and 0.15 or more; the lower threshold was not included in the possible range of X in this study. Hence, the RoS on X test supported *Diathesis-Stress* framework. Finally, the point at where both slopes crossed on pubertal tempo was 0.26, and the estimated PoI index was 0.54, which suggested that the

⁷ The results of the Benjamini and Hochberg test (1995), which controlled for the false discovery rate (FDR) for the three tests of interaction (T1-T2, T2-T3, and T1-T3) in boys, suggested that the T1-T2 test was not a false positive (q-value = 0.093; with the level of the FRD set at 0.10).

⁸ As an additional analysis, we examined whether the interaction between sensitivity and pubertal development (intercept and slope) predicted the development of depressive symptoms based on the Latent Growth Curve model, but the solutions did not converge.

shape of the interaction was consistent with *Differential Susceptibility* framework rather than *Diathesis-Stress* framework or *Vantage Sensitivity* framework. This meant that in areas where the pubertal tempo was greater than 0.26 (Figure 2), boys with higher sensitivity tended to have lower depressive symptoms on average compared to those with lower sensitivity. Taken together, a series of analyses suggested that, based on the two criteria (RoS on *X* and PoI index), the optimal shape of the Sensitivity x Pubertal Tempo interaction among 12- to 13-year-old boys can be interpreted as *Differential Susceptibility* framework as well as *Diathesis-Stress* framework.

[Insert Figure 2]

Girls

In girls, while no significant interaction between sensitivity and pubertal tempo was identified at any age, only the main effect of pubertal tempo was confirmed (Tables 3 and 4). Interestingly, depressive symptoms decreased with greater acceleration of pubertal growth in girls around 12–13 years of age ($b = -0.12$, $p = .048$; Table 3), while depressive symptoms increased with accelerated maturation in those around 14–15 years of age ($b = 0.15$, $p = .003$; Table 4). Hence, no linear association between pubertal development and depressive symptoms was observed for the entire period of 12–15 years ($b = 0.05$, $p = .357$).

[Table 4 and 5]

Discussion

Differential Susceptibility Theory has suggested that individuals differ in their adaptative system in their given contexts depending on their *Environmental Sensitivity* (Belsky & Pluess, 2009). Adolescents experience dynamic contextual changes externally and internally, and puberty entails drastic physiological changes, thus challenging psychological adjustment. The current study aimed, for the first time, to quantify the shape of the interaction

between pubertal tempo and sensitivity, and examine whether their interaction could be interpreted as traditional *Diathesis-Stress* framework or *Differential Susceptibility* framework or *Vantage Sensitivity* framework in development of depression during early adolescence. Consequently, our study yielded two major findings. First, a moderating effect of sensitivity was observed only in early puberty among boys; sensitive boys showed a decrease in depressive symptoms when their maturation progressed. In contrast, pubertal tempo was not clearly related to depressive symptoms among boys with low sensitivity. Second, although no significant interaction was observed, sensitivity and pubertal tempo in girls were partially and positively correlated with depressive symptoms, which replicated previous findings (Ge et al., 2001a; Liss et al., 2005). From the perspective of the *Differential Susceptibility Theory*, our findings suggest that pubertal tempo and sensitivity can be related to depressive symptoms differentially for girls and boys.

Gender Differences in Sensitivity x Pubertal Tempo Interaction

Interestingly, our study demonstrated that personality trait of *Environmental Sensitivity* moderated the relationship between pubertal tempo and depressive symptoms, for better and for worse, only in boys during early puberty (i.e., 12–13 years old; see Table 3). Specifically, for sensitive boys, accelerated physical maturation was associated with decreased depressive symptoms, while a slow progression was associated with increased depressive symptoms. In contrast, low-sensitivity boys reported stable depressive symptoms, regardless of individual differences in speed of physical maturation.

Regarding this finding, a quantitative evaluation based on Roisman's approach revealed that the Sensitivity x Pubertal Tempo interaction supported both *Differential Susceptibility* framework and *Diathesis-Stress* framework (see Figure 2). While the PoI, which represents the ratio of better to worse areas, was consistent with *Differential Susceptibility* framework, the RoS on X, which refers to the area of significant association

between sensitivity and depressive symptoms on the level of pubertal tempo, suggests that the shape of the interaction could be interpreted as *Diathesis-Stress* framework. Particularly in light of the *Differential Susceptibility* findings obtained in our study, it may be more appropriate to reconsider boys with high sensitivity in early puberty as individuals with high *susceptibility*, rather than simply as individuals with high *vulnerability* to the development of psychopathology.

Compared to girls, researchers have pointed out that the association between individual differences in pubertal development and psychopathology in boys are inconsistent. For example, accumulating evidence has indicated that boys with faster pubertal tempo reported higher depressive symptoms (e.g., Ge et al., 2003), that pubertal status did not contributed to change in depressive symptoms (e.g., Conley & Rudolph, 2009), and that later maturation was related to more symptoms (e.g., Siegel et al., 1999). In our data of Japanese boys, the main effect of pubertal tempo did not clearly predict the development of depressive symptoms as in some previous studies, but when examining the interaction with susceptibility, 12- to 13-year-old sensitive boys benefited from accelerated physical maturation while being negatively affected by slow progression.

Intuitively, the aforementioned findings regarding highly sensitive boys may support the traditional deviance hypothesis—later maturers in boys tend to place themselves in the category of socially “deviant” and are at risk for the difficulties associated with this (Petersen & Crockett, 1985; Siegel et al., 1999), which is similar to *Diathesis-Stress* viewing. However, these traditional views could not adequately explain our data showing that 12- to 13-year-old boys who matured significantly reaped socio-emotional benefits—*Differential Susceptibility* thinking was required to properly interpret our findings. In addition, as noted above, one reason why the association between pubertal tempo and depressive symptoms in boys is not reported as clearly as in girls in some studies may be that it is “hidden” because it does not

consider individual differences in *Environmental Sensitivity* to internal stimuli.

Why was no moderating effect of sensitivity observed during later puberty (14–15 years) in boys? One possible reason is that because 15-year-old boys are more matured (i.e., Tanner's stage 4–5; see Berenbaum et al., 2015), their physical condition may have been relatively stable. In other words, the significant interaction effect of sensitivity and pubertal tempo may have become so small as to be undetectable because the stimulus from physical growth became weaker with age. Furthermore, this discussion could be related to the finding that no moderating effect of sensitivity was detected in girls, who mature about one to two years earlier than boys.

In girls, our data did not show a significant Sensitivity x Pubertal Tempo interaction across ages 12–15 (see Tables 3, 4, and 5), and each variable was uncorrelated or weakly correlated with depressive symptoms (see Table 2). Personality trait of *Environmental Sensitivity* is a *plasticity* factor, not a *vulnerability* factor (Pluess et al., 2018), yet some studies have reported a very weak positive correlation ($r < .30$) with depressive symptoms (Liss et al., 2008; Liss et al., 2005). In addition, a previous study targeted girls in the same age group as our study and indicated that sensitivity was uncorrelated with depressive symptoms (Pluess & Boniwell, 2015). Our findings showed weak positive correlations ($r < .31$) or no correlations with depressive symptoms or its changes, partially replicating the findings of this previous study. In addition, our results show that, when we observe the entire period between ages 12–15, accelerated physical maturation in girls did not predict depressive symptoms *linearly* (Table 5), which is consistent with several previous works. For example, a study using large longitudinal twin data (most participants were Caucasian) reported that linear pubertal tempo across ages 9 to 15 was not associated with externalizing and internalizing problems, depression, or conduct disorder (Beltz et al., 2014). Nevertheless, as shown in Tables 3 and 4, examining our results in more detail, in girls, depressive symptoms decreased

with accelerated maturation from 12 to 13 years of age, while depressive symptoms increased with accelerated maturation from 14 to 15 years of age. These curvilinear relationships were similar to the findings by Keenan et al. (2014). Whether these consistencies and inconsistencies among the evidence are due to differences in race, analysis methods, or the age of the children is unclear and requires further investigation in future studies.

Limitations

Our study is the first to propose a reconsideration of the relationship between pubertal tempo and depressive symptoms from the perspective of the *Differential Susceptibility Theory*. Although this attempt is expected to advance our understanding of depressive symptoms in adolescents, the present study has some limitations. First, our findings did not consider the influence of other covariates previously reported to affect depressive symptoms in adolescents. For example, research on pubertal development and sensitivity suggested that stressful life events and parent–child relationships could interact with individual differences in pubertal growth and sensitivity to predict socio-emotional well-being (e.g., Ellis et al., 2011; Ge et al., 2001a). Given that the developmental mechanisms of depression in adolescents can have complex interactions such as Sensitivity $\times$ Pubertal Tempo $\times$ Stressful Life Events $\times$ Child-Parent Relationship, this study focused on only a part of them. Second, this study had a high dropout rate after Time 1 due to an unexpected lack of research funding. To make the results more robust, a replicability study should be conducted. Third, although other studies using Japanese adolescent samples have shown sufficient internal consistency of the HSC scale (Iimura, 2021; Iimura & Kibe, 2020; Kibe et al., 2020), it was somewhat poor in this sample. Finally, our findings were based on a limited sample size of Japanese adolescents and their mothers. Hence, it is necessary to verify whether our findings can be replicated in a study using a population-representative sample of Japanese adolescents. Furthermore, an international comparison is required to determine whether the consistency or

inconsistency with findings from Western countries is due to racial or cultural differences.

Future Research

Although the cultural significance of the pubertal transition may vary based on ethnic or social background (e.g., Deardorff et al., 2019), our understanding lacks empirical findings, especially for adolescents living in Asian countries. Therefore, our findings from adolescents living in Japan, a country with low ethnic diversity compared to Western countries (e.g., the United States), may contribute to the discussion of the relationship between puberty and ethnic/cultural factors. However, as pointed out in a recent review article (Deardorff et al., 2019), future studies may further advance our understanding by examining within-culture variation and between-culture variation in further detail. For example, including psychological responses such as whether menarche is an embarrassing event, an event that needs to be hidden, or to be celebrated (e.g., in old Japan, when a girl experienced menarche, her family would celebrate by preparing a special meal called “seki-han”) may be helpful in understanding the cultural significance of pubertal transition.

We also expect that these psychological responses to puberty will likely affect self- and parent-reported assessments of pubertal status. However, our understanding of this is quite limited. Therefore, the validity of questionnaire-based assessments of pubertal status could be improved by simultaneously measuring the contextual and cultural factors and social norms that are expected to affect it.

Conclusion

Despite several limitations, we have provided the first evidence for understanding the relationship between pubertal tempo and psychopathology from the perspective of the *Differential Susceptibility Theory*. We have demonstrated that boys aged 12 to 13 who experienced accelerated physical maturation showed different expressions of depressive symptoms depending on individual differences in their susceptibility to internal or external

stimuli. Specifically, highly sensitive boys showed decreased depressive symptoms with greater physical changes, and increased depressive symptoms with slow progression. In contrast, for boys with low sensitivity, pubertal tempo was not clearly associated with depressive symptoms. Such an interaction between sensitivity and pubertal tempo was consistent not only with the *Diathesis-Stress Model*, which views sensitivity as a *vulnerability* factor, but also with the *Differential Susceptibility Theory*, which views sensitivity as a *susceptibility* or *plasticity* factor. Beyond the *Diathesis-Stress* paradigm, *Differential Susceptibility* thinking might advance current understanding of socio-emotional adjustment mechanisms in early adolescence.

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Table 1*Descriptive Statistics of Boys (n = 98) and Girls (n = 111).*

	Boy			Girl		
	<i>M</i>	<i>SD</i>	α	<i>M</i>	<i>SD</i>	α
Depressive symptoms						
T1 (12-13 years old)	1.55	0.40	0.81	1.58	0.42	0.84
T2 (13-14 years old)	1.53	0.45	0.78	1.69	0.49	0.80
T3 (14-15 years old)	1.56	0.45	0.84	1.63	0.49	0.81
$\Delta T1 \rightarrow T2$	-0.01	0.38	—	0.11	0.44	—
$\Delta T2 \rightarrow T3$	0.00	0.35	—	0.00	0.36	—
$\Delta T1 \rightarrow T3$	-0.01	0.34	—	0.10	0.42	—
Pubertal development						
status T1 (12-13 years old)	2.08	0.66	0.84	2.34	0.54	0.61
status T2 (13-14 years old)	2.38	0.80	0.85	2.65	0.72	0.70
status T3 (14-15 years old)	2.88	0.64	0.71	3.07	0.70	0.75
tempo $\Delta T1 \rightarrow T2$	0.23	0.50	—	0.31	0.49	—
tempo $\Delta T2 \rightarrow T3$	0.31	0.53	—	0.51	0.58	—
tempo $\Delta T1 \rightarrow T3$	0.54	0.47	—	0.81	0.55	—
Sensitivity						
T3 (14-15 years old)	4.57	0.69	0.54	4.66	0.68	0.67

Table 2*Bivariate Correlations for Boys (Above the Diagonal) and for Girls (Below the Diagonal).*

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Depression T1	—	.63 **	.64 **	-.26 **	-.07	-.35 **	.03	.26 **	.18 †	.30 **	-.12	.18 †	.15
2. Depression T2	.54 **	—	.70 **	.59 **	-.50 **	.15	.12	.30 **	.30 **	.23 *	-.02	.22 **	.22 **
3. Depression T3	.55 **	.72 **	—	.20 *	.26 **	.49 **	.01	.23 *	.18 †	.29 **	-.07	.22 *	.18 †
4. ΔDepression T1→T2	-.34 **	.60 **	.28 **	—	-.55 **	.54 **	.11	.10	.19 †	-.02	.09	.08	.12
5. ΔDepression T2→T3	-.02	-.42 **	.33 **	-.45 **	—	.40 **	-.15	-.12	-.18	.04	-.06	-.03	-.07
6. ΔDepression T1→T3	-.37 **	.26 **	.57 **	.65 **	.39 **	—	-.03	-.02	.02	.01	.05	.07	.05
7. Pubertal status T1	.04	.11	.09	.08	-.03	.06	—	.70 **	.71 **	-.38 **	-.05	-.46 **	.12
8. Pubertal status T2	.10	.23 *	.21 *	.17 †	-.04	.14	.65 **	—	.64 **	.40 **	-.50 **	-.15	.25 *
9. Pubertal status T3	.15	.30 **	.25 **	.20 *	-.08	.14	.53 **	.54 **	—	-.09	.35 **	.30 **	.14
10. ΔPubertal tempo T1→T2	.08	.18 †	.17 †	.12	-.02	.11	-.28 **	.54 **	.10	—	-.58 **	.40 **	.17 †
11. ΔPubertal tempo T2→T3	.04	.06	.03	.02	-.04	-.01	-.17 †	-.53 **	.43 **	-.48 **	—	.51 **	-.15
12. ΔPubertal tempo T1→T3	.12	.22 *	.18 †	.13	-.06	.09	-.43 **	-.07	.54 **	.38 **	.63 **	—	.01
13. Sensitivity T3	.27 **	.09	.31 **	-.16	.28 **	.08	.27 **	.40 **	.18 †	.21 *	-.26 **	-.08	—

Note. Boys: $n = 98$. Girls: $n = 111$.** $p < .01$, * $p < .05$ † $p < .10$

Table 3

Regression Model for Change from Time 1 to Time 2 by Gender (Sensitivity x Pubertal Tempo → Changes in Depressive Symptoms).

Parameter	Boy				Girl			
	Model 1: Main effects		Model 2: Interaction		Model 1: Main effects		Model 2: Interaction	
	<i>b</i> (SE)	<i>p</i>	<i>b</i> (SE)	<i>p</i>	<i>b</i> (SE)	<i>p</i>	<i>b</i> (SE)	<i>p</i>
B_0 (Intercept)	-0.30 (0.25)	.229	-0.42 (0.25)	.096	0.65 (0.28)	.020	0.63 (0.28)	.023
B_1 (Δ Pubertal tempo T1→T2)	0.07 (0.06)	.234	0.09 (0.06)	.093	-0.12 (0.06)	.048	-0.11 (0.06)	.061
B_2 (Sensitivity)	-0.03 (0.08)	.637	0.83 (0.40)	.041	0.15 (0.09)	.089	0.98 (0.64)	.128
B_3 (Interaction: $B_1 \times B_2$)			-0.19 (0.08)	.031			-0.17 (0.13)	.192
BIC	103.82		103.45		143.69		133.08	
AIC	93.47		90.57		132.86		146.63	
R^2	0.02		0.06		0.05		0.07	
F (df)	0.74 (2, 95)	.481	2.12 (3, 94)	.103	2.87 (2, 108)	.006	2.50 (1, 107)	.063
ΔR^2			0.04				0.02	
F (df)			4.83 (1, 94)	.031			1.73 (1, 107)	.192

Note. BIC = Bayesian Information Criterion. AIC = Akaike Information Criterion. Boys: $n = 98$. Girls: $n = 111$.

Table 4*Regression Model for Change from Time 2 to Time 3 by Gender (Sensitivity x Pubertal Tempo → Changes in Depressive Symptoms).*

Parameter	Boy				Girl			
	Model 1: Main effects		Model 2: Interaction		Model 1: Main effects		Model 2: Interaction	
	<i>b</i> (<i>SE</i>)	<i>p</i>	<i>b</i> (<i>SE</i>)	<i>p</i>	<i>b</i> (<i>SE</i>)	<i>p</i>	<i>b</i> (<i>SE</i>)	<i>p</i>
B_0 (Intercept)	0.20 (0.24)	.405	0.08 (0.27)	.771	-0.70 (0.24)	.004	-0.63 (0.31)	.041
B_1 (Δ Pubertal tempo T2→T3)	-0.04 (0.05)	.423	-0.02 (0.06)	.784	0.15 (0.05)	.003	0.14 (0.06)	.032
B_2 (Sensitivity)	-0.04 (0.07)	.511	0.32 (0.43)	.462	0.02 (0.06)	.726	-0.11 (0.40)	.785
B_3 (Interaction: $B_1 \times B_2$)			-0.08 (0.09)	.396			0.03 (0.08)	.743
BIC	86.65		90.49		97.20		101.79	
AIC	76.31		77.56		86.36		88.25	
R^2	0.01		0.02		0.08		0.08	
F (<i>df</i>)	0.47 (2, 95)	.624	0.56 (3, 94)	.645	4.83 (2, 108)	.010	3.23 (3, 107)	.025
ΔR^2			0.01				0.00	
F (<i>df</i>)			0.73 (1, 94)	.396			0.12 (1, 107)	.743

Note. BIC = Bayesian Information Criterion. AIC = Akaike Information Criterion. Boys: $n = 98$. Girls: $n = 111$.

Table 5*Regression Model for Change from Time 1 to Time 3 by Gender (Sensitivity x Pubertal Tempo → Changes in Depressive Symptoms).*

Parameter	Boy				Girl			
	Model 1: Main effects		Model 2: Interaction		Model 1: Main effects		Model 2: Interaction	
	<i>b</i> (SE)	<i>p</i>	<i>b</i> (SE)	<i>p</i>	<i>b</i> (SE)	<i>p</i>	<i>b</i> (SE)	<i>p</i>
B_0 (Intercept)	-0.16 (0.23)	.496	0.06 (0.35)	.872	-0.20 (0.28)	.468	-0.08 (0.48)	.873
B_1 (Δ Pubertal tempo T1→T3)	0.03 (0.05)	.598	-0.02 (0.08)	.779	0.05 (0.06)	.358	0.05 (0.06)	.357
B_2 (Sensitivity)	0.04 (0.08)	.520	-0.42 (0.58)	.474	0.07 (0.07)	.324	-0.08 (0.49)	.869
B_3 (Interaction: $B_1 \times B_2$)			0.10 (0.13)	.421			0.03 (0.10)	.751
BIC	85.49		76.47		140.56		145.17	
AIC	75.15		89.40		129.72		131.62	
R^2	0.01		0.01		0.02		0.02	
F (df)	0.35 (2, 95)	.704	0.45 (3, 94)	.716	0.84 (2, 108)	.431	0.60 (3, 107)	.620
ΔR^2			0.00				0.00	
F (df)			0.65 (1, 94)	.421			0.10 (1, 107)	.751

Note. BIC = Bayesian Information Criterion. AIC = Akaike Information Criterion. Boys: $n = 98$. Girls: $n = 111$.

Figure 1.

Three Models Depicting a Typical Shape of Person $\times$ Environment Interaction.

Note. Model A represents *Diathesis-Stress*, which in this framework explains that susceptible individuals are more likely to be affected to adversarial influences from a negative environment. Model B represents *Differential Susceptibility*, which in this framework describes highly sensitive individuals as being more susceptible, depending on the quality of their environment, for better and for worse. Model C represents *Vantage Sensitivity*, which in this framework explains that susceptible individuals tend to benefit more from a positive environment.

Figure 2.

Graphical Illustration of a Simple Slope Test Predicting the Change in Depressive Symptoms from Time 1 to Time 2 by Sensitivity at Time 3 x Pubertal Tempo from Time 1 to Time 2 Interaction in Boys.

Note. The slope of students with high sensitivity ($M+1SD$, i.e., $Z = 1$) was $b = -0.21$ ($p = .036$). The slope of students with low sensitivity ($M-1SD$, i.e., $Z = -1$) was $b = 0.17$ ($p = .064$). The multiple lines drawn around the slope of the high-sensitivity students indicate upper threshold and lower threshold for the Regions of Significance on Z $[-1.15, 0.80]$, where values of Z inside this region are significant at $p < .05$. The Proportion of Interaction, shown in the area between the two regression lines, was 0.46 ($=$ better area / better area + worse area).

Appendix

Figure A1.
Proportion of Interaction