

Supplementary Online Content

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eTable. Bivariate Correlations Across Predictors, Covariates, and Outcomes

eAppendix. Details on the Fulfillment of the 5 Steps for Determining a Genuine Differential Susceptibility Interaction Effect

This supplementary material has been provided by the authors to give readers additional information about their work.

eTable. Bivariate Correlations Across Predictors, Covariates, and Outcomes

	Age	Eth	Sex	SES	Gene	SFold
Age						
Ethnic Minority	.031					
Sex	-.082	-.030				
SES	.070	.179*	.033			
Genotype	.033	.036	.075	-.045		
SkinFold	.140	-.026	-.286**	-.073	.104	
BMlz	.074	-.148*	.011	-.117	.108	.671**

Note: * = $p < .05$, ** = $p < .01$, *** = $p < .0001$; Age= age at anthropometric exam (BMI and skinfold measures); SES = socioeconomic status; Genotype= A-allele carrier versus GG genotype; Sfold = skinfold thickness; BMlz = body mass index age- and sex-adjusted CDC-derived z-score; Males , Caucasians, and A allele-Carriers were all coded "0".

eAppendix. Details on the Fulfillment of the 5 Steps for Determining a Genuine Differential Susceptibility Interaction Effect

Following the steps proposed by Belsky et al. (1) for identifying a genuine differential susceptibility interaction, we first identified a genuine interaction effect, in that SES was related to BMIz only for children with the A allele, such that A-carriers exhibited the highest BMIz and skinfold thickness in low SES environments but the lowest BMIz and skinfold thickness in high SES environments. Steps two and three of the test proposed by Belsky et al. require testing for independence of the susceptibility factor and predictor (OT and SES) and then testing for independence of the susceptibility factor and the outcome (OT and BMIz; OT and skinfold thickness). Independence was established in both models, as evidenced by the nonsignificant associations found in Table 2. Step four requires comparing graphical versions of the data with the prototype graphs presented in Belsky et al.; the graphs generated in this study are most consistent with the differential susceptibility graph. Finally, we tested the specificity of the model by replacing the susceptibility factors with two other genes of interest (DRD4 and 5HT polymorphisms) and outcomes (cortisol levels, behavioral outcomes), which rendered the model insignificant. As such, this study meets the stated criteria and provides strong evidence that the oxytocin receptor polymorphism confers differential susceptibility to the effects of SES on childhood obesity risk.

Reference

1. J. Belsky, M. J. Bakermans-Kranenburg, M. H. Van IJzendoorn, For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science* 16, 300–304 (2007).