Orchids and dandelions: How some children are more susceptible to environmental influences for better or worse and the implications for child development

Boyce and colleagues (1995) first put forward their theory of “Biological Sensitivity to Context” based on the findings of two prospective studies focusing on children’s propensity to develop respiratory illness. It was noted that young children who appeared more biologically “reactive” (e.g. high cardiovascular or immune reactivity to stressors) were most likely to develop asthma when reared in conditions of adversity but unexpectedly, these same biologically reactive children were the ones least likely to develop the illness when reared in low risk supportive environments, even compared to children with low biological reactivity reared in similarly supportive environments. This finding was replicated in a second study showing that children characterized by a heightened susceptibility to develop respiratory illness when in a high stress environment were those least likely to develop illness when in a low stress environment. Two years later Jay Belsky independently developed “Differential Susceptibility Theory,” arguing that a proportion of children with underlying genetic and temperamental vulnerability were prone to the best or worst developmental outcomes depending on the quality of the early child rearing environment (Belsky, 1997).

This idea that a group of children can be identified with underlying neurobiological sensitivity that can lead to bidirectional outcomes for “better or worse” challenges the more prevalent “dual risk” or “stress diathesis” paradigm (i.e. that vulnerability and stress interact to produce adverse outcomes). Whereas the majority of children might be conceptualized as “Dandelion” children, relatively hardy and able to withstand the vicissitudes of a range of environments, such neurobiologically sensitive and reactive children are designated “Orchid” children in their capacity to develop beautifully given sensitive nurturance alongside their tendency to develop adverse developmental outcomes in less than optimal environmental conditions (Boyce & Ellis, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenberg, & Van IJzendoorn, 2011).

Increasingly, these theories of biological sensitivity to context and differential susceptibility have been tested in empirical research and there is mounting evidence of the implications for understanding child development as well as targeting interventions at those children most likely to benefit. Examples of recent research include that of Knafo, Israel, and Ebstein (2011) whereby children with the 7 repeat allele of the DRD4 dopamine gene were the least social children in the absence of positive parenting but the most prosocial in the presence of such parenting, and a study by Pleuss and Belsky (2009) showing that children with difficult temperaments as infants exhibited more behavior problems when provided with low quality childcare care and fewer when experiencing high quality childcare than children with easy temperaments.

The 7 repeat allele of the DRD4 gene has attracted particular attention, with children with this gene variant showing more positive and negative outcomes with regard to externalizing problems and attachment security depending on the quality of maternal caregiving (Backermans-Kranenburg & Van IJzendoorn, 2007; Backermans-Kranenburg, van IJzendoorn, Pijlman, Mesman, & Juffer,
2008). This is leading to a reformulation of the role of this gene variant in heightening susceptibility to both risk promoting and development enhancing environments rather than being solely a risk factor for poor outcomes (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007).

Even more robust evidence is now emerging from a small number of randomized trials designed specifically to test for differential susceptibility. This has included studies showing that highly negatively reactive infants appear to benefit most from interventions developed to enhance parenting in relation to outcomes such as attachment security (Cassidy, Woodhouse, Sherman, Stupica, & Lejuez, 2011; Klein Velderman, Bakermans-Kranenburg, Juffer, & van IJzendoorn, 2006) and externalizing behavior problems (Backermans-Kranenburg et al., 2008). Research of this kind offers an important alternative perspective compared with much existing developmental research that focuses disproportionately on vulnerability to risk-promoting environments and adverse outcomes.

The underlying origins of neurobiological sensitivity to the environment have been further elucidated and influences from both “nature” and “nurture” appear to be important. Environmental stressors in the pre-natal environment may result in pre-natal programming of developmental plasticity (Pleuss & Belsky, 2011), alleles of certain dopamine, serotonin and monoamine oxidase genes appear to render individuals more susceptible to environmental influences and additionally factors such as biological reactivity to stress and negative emotional reactivity also seem to make a contribution (Ellis et al., 2011). Research on epigenetic effects illustrates how environmental factors impact on the genome through e.g. methylation, hence altering gene expression and thus providing a model of how individual experiences can interface with genetic expression and alter vulnerability to environmental influences (van IJzendoorn, Bakermans-Kranenberg, & Ebstein, 2011).

As further research emerges, the neat categorical distinction between “orchid” and “dandelion” children is unlikely to remain, with most children understood to be on a continuum of biological vulnerability, more or less susceptible to the beneficial impact of interventions and supportive environments. However, as a metaphor the orchid/dandelion distinction remains very powerful, not least in reminding us that in human development processes are often more complex than they initially seem, but also most importantly that it can be all too easy to lose sight of the “upside,” the potential for those considered most at risk to do best and benefit most when offered optimal care (Dobbs, 2012).

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References


