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35. Vulnerable and resilient children after disasters and gene–environment interplay

by
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The World Health Organization (WHO) estimates that disaster doubles the occurrence of mental distress. Yet certain children show huge resilience, despite losing their homes and parents, while others suffer enormous mental distress. Gene–environment interdependence plays a crucial role in children's different reactions: experience of disasters is genetically influenced, and may influence the rest of a victim's life.

Disasters affect a large share of the world's population, but hit some regions more than others. In the past decade, about 40% of natural disasters took place in the Asia-Pacific region, bringing untold damage, loss of life and hardship, especially to countries with less well-developed infrastructures and weak rescue systems. Beyond physical and infrastructural devastation, disasters and their aftermath have psychological consequences related to the loss of family and friends, property, environment and personal injury, as well as many other stressors.

According to World Health Organization (WHO) estimates, a disaster doubles the prevalence of mental distress. Research syntheses on children and youth (Furr et al., 2010) have demonstrated associations between exposure to disaster of various kinds (proximity, perceived threat, distress at the time) and broad indices of psychopathology, particularly post-traumatic stress symptoms and disorder (PTSD). The specific mechanisms by which such stressors impact human behaviour and development have so far been attributed to the breakdown of the family, local communities and other social mechanisms. Such a breakdown makes it hard to satisfy the basic emotional needs of children and adolescents, which is necessary for their healthy development toward a balanced and productive adulthood. Skills that are fundamental for adequate social relationships and the regulation of impulses are especially likely to be underdeveloped when such stress is prevalent (Norris et al., 2002).

Looking at the impact of such disasters on young people, scientists and practitioners have long wondered about the great range of responses to such misfortune. Individuals can exhibit anything from devastating psychopathologies to almost intact functioning,

or resilience, despite a seemingly equal level of exposure to disaster-related stressors. Now recent cross-disciplinary research on the heterogeneity of response demonstrates pathways of behavioural, brain-related and genome activity that may shed new light on the various ways in which humans respond to disasters, and especially on the risk of lasting adverse psychosocial conditions or the ability to survive such disasters in a resilient way (Masten and Osofsky, 2010).

At the core of this new research are three concepts of the interdependence between genes and the environment that play a crucial role in normative or psychopathological development (for an overview, see Rutter, 2012).

The first is the gene–environment correlation, which addresses the various environmental risk factors that ultimately derive from human behaviour mediated by genetics. This means that the experience of disasters is itself influenced in part by genetics.

The second is the gene–environment interaction, which means that genes moderate environmental effects, making people more or less susceptible to negative or positive environmental effects. Interest in this kind of interaction in disaster research was prompted not only by the limited prevalence of PTSD following exposure, but also by the fact that it runs in families. It is now well known, for instance, that genes related to serotonin production (5HTTLPR, a contributor to feelings of anxiety and depression) interact with particular early environments, such as child maltreatment. More specifically, some less effective polymorphisms of the gene (those with short alleles) promote the development of lasting clinical depression in later life if individuals are exposed to maltreatment (Caspi et al., 2003). Likewise, early exposure to child abuse in interaction with polymorphisms on the FKBP5 gene – an important regulator of the stress hormone system – increases adults' vulnerability to PTSD in response to disaster. It may not be the initial event so much as its consequences, perhaps involving displacement promoting physical and emotional neglect, that imply aggravated risks for genetically vulnerable children.

The third new strand of research on gene–environment interdependence – and maybe the most relevant for human response to disaster – refers to the modulation of gene expression at the molecular level through environmental stressors. These so-called "epigenetic" processes do not represent a change of the structural DNA sequence, but instead concern biochemical changes, such as DNA methylation, which alter the expression of particular DNA segments, or their "readability", in the regulation of protein and enzyme production. Recent research with animal and plant models shows that these changes, induced by environmental forces, are reversible but can be transmitted to future generations (Yehuda and Bierer, 2009).

With regard to disasters, the best example is probably the following pathway: turmoil at the aggregate level of a disaster-affected region is translated into a range of particular adversities experienced by the victims in their own contexts, such as the breakdown of established and secure family relationships and routines. The subsequent trauma experienced by parents may result in a sharp decline in the quality of parenting and even atypical, neglectful parental behaviours that are damaging to the child.

Such experiences, especially concerning maternal care and attachment relationships during the first few years of life, lead to individual differences in the expression of genes involved in the regulation of the cortisol levels in the brain and body – such as FKBP5 – which may provoke differences in habitual stress response. More specifically, drastic changes in parent–infant interaction may modify epigenetic markers or regions of DNA that regulate

the HPA axis response to stress, with enduring effects on biological, psychological and social development. Recent research has gone beyond earlier animal models, and has shown that differences in DNA methylation in FKBP5 or 5HTTLPR resulting from early trauma (such as child maltreatment) may have a persistent influence on PTSD and even on propensity to suicide (van Ijzendoorn, Bakermans-Kranenburg and Ebstein, 2011).

Such processes offer new explanations for the role of family history of PTSD, the cumulative effects of exposure to disasters, and intergenerational effects in general. The core pathway seems to be triggered by deficits in maternal care. This has an enduring effect on gene expression that underlies individual differences in endocrine functioning and ultimately how offspring respond to environmental challenges, including disasters.

Questions for further research relate to which particular environmental influences bring about the largest epigenetic changes, in which body tissues, and at what stage of development. Thus far, the effects of some adverse events and treatments concerning small children have been studied on the HPA axis with cortisol as its product, but other pathways can be imagined, for example using the dopamine system. The reason we focus on stress is because several models of individual consequences of negative societal change, including disasters, have put the experience of adverse conditions and coping with ensuing stress in the foreground (Meaney, 2010).

It is not new for genetic endowment and environmental processes to work interdependently in human development. But now, for the first time, the biochemical processes which translate experiences into modifications of physiological and brain processes can be addressed specifically. This means that we are able to create a full picture, from the objective environment, via psychological experience and the biochemical modifications of the genes involved in the production and transfer of major neurotransmitters and hormones, to behaviour. From a basic science perspective, this brings psychology and its allied disciplines back to the middle of recent progress in the natural sciences. From an applied point of view, many years of talking about the ecology of human behaviour and development have led to a specific focus on where and how to intervene early in the chain of processes leading to maladjustment (Silbereisen, Ritchie and Overmier, 2010).

In spite of their biochemical nature, adverse DNA methylation and similar processes may be influenced by changing a specific environmental trigger, such as disaster-induced inept parenting. More specifically, it has been shown that it is possible to reprogramme methylation through later positive experiences, at least in animal models. It is even imaginable that in the distant future, protective medication will be able to prevent biochemical modification. Further, as the three facets of gene–environment interdependence do not work in isolation, their interaction can be used for prevention and intervention. Exposure to potentially damaging experience by particular genetically influenced behaviours might be reduced at the beginning of the process. Further, knowledge about genetic susceptibility to environmental effects may be used to reduce risks, for instance, by offering positive alternative environments with less risk potential.

This exciting new research on gene–environment interdependence should be the start of a new collaboration between the various fields of social and behavioural science, especially with the aim of improving mental health and the adaptive development of competence under extremely adverse conditions. It will be a point of departure for more research on how the environment, with its challenges and opportunities, leaves traces on human

behaviour and development. This research will provide a new scientific underpinning of disaster response guidelines that will demand priority in nurturing adaptive systems for human development, and restoring the secure base of attachment relationships.

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