Inequality

The IFS Deaton Review

Individuals as active co-creators of their environments: implications for prevention of inequalities

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Individuals as active co-creators of their environments: implications for prevention of inequalities

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Introduction

The chapter by Cattan et al. (2022) in the IFS Deaton Review of Inequalities documents stark inequalities in early human development in the UK, and their long-reaching shadow. The authors' careful scrutiny of longitudinal cohort data is particularly pertinent at a time when reports of inequality feature regularly in the press[2] and when the COVID-19 pandemic has further highlighted the troubling differences in risks and opportunities between different parts of the population.[3]

Cattan et al. (2022) present convincing and systematic data, which indicate that tackling inequalities in the child's environment may be as, or even more, important than tackling specific skills aimed at reducing later inequalities. Particular attention is paid to parenting practices and behaviours that are critical during the early years and beyond – including relationship quality and attachment, cognitive stimulation, and family rituals. The chapter highlights that some children are disadvantaged on multiple fronts and that this requires that several factors must be addressed simultaneously for such children/families. The authors also point out that although there is a strong imperative for intervening early, it is also clear that the effects of some early interventions attenuate over time. The focus on early intervention should therefore not detract from the importance of investments throughout childhood and adolescence.

Although the extant longitudinal data point to seemingly obvious targets for preventative and intervention efforts, there remain challenges that we want to consider in this commentary. Even in contexts where there is universal access to health care, education and income support (e.g. Nordic countries), considerable differences between people in mental and physical health outcomes, as well as educational achievement, still remain and cannot be accounted for solely by lack of access to health care, education and income support (Paananen et al., 2013; Hegelund et al., 2018). Here we outline three important issues that warrant further consideration and investigation in the context of prevention and intervention of inequalities.[4]

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[2] See, for example, the online articles '"Bright but poor" pupils years behind better-off peers, study claims', 9 February 2017, https://www.theguardian.com/education/2017/feb/09/bright-but-poor-students-uk-years-behind-better-off-peers-study-claims, and 'Life expectancy gap between rich and poor widens', 15 February 2018, https://www.bbc.co.uk/news/health-43058394.

[3] See, for example, the online articles 'UK wealth gap widens in pandemic as richest get £50,000 windfall', 12 July 2021, https://www.theguardian.com/business/2021/jul/12/uk-wealth-gap-widens-in-pandemic-as-richest-get-50000-windfall, 'Fears over rising inequality as majority of Britons believe Covid pandemic has widened gap between richest and poorest', 11 March 2021, https://www.dailymail.co.uk/news/article-9348849/Majority-Britons-believe-Covid-pandemic-widened-gap-richest-poorest.html, and '"Jaw-dropping" fall in life expectancy in poor areas of England, report finds', 30 June 2021, https://www.theguardian.com/uk-news/2021/jun/30/life-expectancy-key-to-success-of-levelling-up-in-uks-poorer-areas-covid-pandemic.

[4] Focus on the issues outlined in this commentary naturally does not preclude the need to focus on the impact of socio-economic and opportunity inequalities that we know matter for child development (Marmot, 2017).

Gene–environment correlation

Studies of twins, adoption and molecular genetics have demonstrated that many established social risk factors associated with poor mental health and educational outcomes, which we know contribute to inequalities, partly reflect genetic risk (Moffitt, 2005). For example, a child's inherited temperament may put them at risk of poor self-regulation, which in turn can evoke frustration and anger in parents. The same genetic risk for poor self-regulation in a parent may compromise their ability to sensitively respond to a temperamentally challenging child. Together, these parent–child genetic endowments can contribute to dysregulated child–caregiver interactions. Research has shown that harsh and inconsistent discipline is associated with higher levels of conduct problems and ADHD, but this in part reflects shared genetic vulnerabilities between parents and children (Moffitt, 2005; Harold et al., 2013). In other words, the observed association between a variable that we tend to think of as 'environmental', such as parenting, is in part due to genetic factors that increase the likelihood of parental characteristics, which result in harsh and inconsistent parenting, as well as child characteristics, which increase the risk of displaying disruptive behaviours (this phenomenon is known as passive gene–environment correlation). The association between harsh and inconsistent discipline and higher levels of disruptive behaviours also partly reflects the reactions that a child with difficult behaviour evokes in parents (this phenomenon is known as evocative gene–environment correlation). Similar 'genetic confounding' is observed in relation to cognitive ability and educational achievement (Plomin and Deary, 2015; Belsky et al., 2016; Allegrini et al., 2020).

Accumulating data indicate that although risk for psychopathology or low cognitive ability is heritable, the genetic risk does not operate in a disorder- or domain-specific manner (Haworth et al., 2009; Plomin and Deary, 2015; Caspi and Moffitt, 2018; Selzam et al., 2018). Instead, 'generalist genes' have been associated with a broad range of outcomes (Plomin and Deary, 2015; Belsky et al., 2016; Wertz et al., 2018). In line with this, risk mechanisms are shared by a number of different conditions, and co-morbidities are the norm (Kovas et al., 2007; Chu, Temkin and Toffey, 2016; Caspi et al., 2020). It therefore follows that children who have had a particularly unlucky roll of the genetic dice, and inherited multiple genes that increase risk for psychopathology and/or low cognitive ability, will be at most risk for facing inequalities. These children are also more likely to have biological parents who may be ill equipped to respond to their complex needs because they often share some or all of the very same vulnerabilities. Data from different cohort studies convincingly show that a small proportion of high-need individuals and families (10%–20%) account for up to 80%–90% of the outcomes that are related to sustained inequalities – including poor health and incarceration (e.g. Richmond-Rakerd et al., 2020). High psychopathology and low cognitive ability cluster among these individuals and their families.

We argue that it is important to be mindful of gene–environment correlation when planning and evaluating prevention and intervention programmes designed to reduce economic inequalities, improve education provision and improve parent–child interaction. There is often apprehension regarding genetically informative data, with fears of reductionist or deterministic approaches. However, we risk failing those who are most vulnerable to poor outcomes if we ignore the implications of genetically influenced individual differences. Such differences do not mean that we should start genotyping all families or focus on biological interventions. Instead, they suggest that we need to become better at understanding how biological endowments affect the functioning of social environments and what that means for intervention planning and delivery.

Latent vulnerability following early adversity

Child maltreatment can have profound negative and long-lasting consequences on children's mental health (Gilbert et al., 2009). We do not yet have a clear mechanistic understanding of how maltreatment increases risk of mental health problems or why some children with maltreatment experience are more vulnerable to developing mental health problems than others. However, a growing body of work is delineating how extreme childhood adversity can lead to a variety of neurodevelopmental adaptations that may confer latent vulnerability to subsequent maladaptive development (McCrory and Viding, 2015). In effect, the brain may adapt to an adverse environment in ways that may be helpful in the short

term, but mean that the child is less well equipped to function in more normative environments (McCrory and Viding, 2015).

Neuroimaging research has documented changes in a range of neurocognitive systems, including the threat, reward and autobiographical memory systems (McCrory, Gerin and Viding, 2017). For example, children who have experienced maltreatment in the past show heightened reactivity to threat (McCrory et al., 2011; Hein and Monk, 2017). Being alert to potential threat will clearly serve a purpose in an environment that is not safe. However, it may result in threat reactive aggression in response to perceived threat, even if threat was not intended, causing problems in school (e.g. increasing the likelihood of conflictual interactions). We have argued that this 'mismatch' – where brain systems calibrated for an adverse environment function less well in a more normative environment – may increase mental health vulnerability (i) as a function of altered neurocognitive processing, and (ii) as a function of how a child shapes and experiences the social world over time. For example, in respect of the former, in a large community sample of adolescents with varying degrees of childhood maltreatment, Hanson, Hariri and Williamson (2015) found that severity of emotional neglect was associated with reduced development of striatal neural response during reward processing. This blunted neural response to reward was found to partially mediate the association between a history of neglect and depressive symptomatology two years post-baseline. In relation to socially mediated vulnerability, we have postulated that altered neurocognitive functioning may contribute to stress generation (the generation of new stressful interpersonal events; Gerin et al., 2019), and social thinning (a pattern of attenuated social capital relative to peers over time, including the extent and quality of social relationships; McCrory, 2020). We have found that, for example, maltreatment experience in childhood is associated with increases in stress generation in young adults, which in turn contribute to an increased risk of future internalising symptoms and, plausibly, externalising symptoms (Gerin et al., 2019).

It is not difficult to imagine how an increased experience of interpersonal conflict would create significant stress for a child. Poorer interpersonal functioning may also mean that social relationships are not cultivated and maintained in the same way as peers, and they may even be disrupted through experiences of exclusion, which over time contribute to social thinning. We know that maltreatment experience in childhood is associated with smaller social networks in adulthood as well as an increased experience of loneliness (Hanlon et al., 2020). Such continued adverse social relationships are likely to affect not only mental health, but also social and educational learning opportunities more generally. Cumulatively, these cascading effects of early adversity are likely to both potentiate the impact of any risk factors that the child continues to encounter, and indeed actively contribute to the accumulation of further risk. The fact that children who experience childhood maltreatment are at elevated risk of school exclusion and involvement in the criminal justice system starkly illustrates this point.

Dynamic developmental effects

A number of recent genetically informative longitudinal studies have demonstrated that in addition to those genetic and environmental factors that contribute to initial risk of maladaptive outcomes, there are novel genetic and environmental influences that come 'online' at later time points and affect the developmental course of the child's phenotype. In other words, although initial genetic and environmental risk factors are no doubt important, addressing these will be insufficient for those individuals where additional (new) risks become manifest during later childhood and adolescence. As an example, it has been shown that those genetic factors that increase early risk of developing conduct problems are largely independent of those genetic factors that explain subsequent changes in these behaviours (Pingault et al., 2015). A comparable pattern is seen in relation to development of empathy (Takahashi et al., 2020). The early and later environmental risk factors are not fully overlapping either, which is not surprising given the changing social contexts of an individual as they grow up. Parents are of key importance for infants and young children, but as children become adolescents, it is peers who exert most influence (Andrews, Ahmed and Blakemore, 2020; Viding, 2020).

We have speculated that those genes influencing the baseline level of conduct problems/empathy may be related to the temperamental make-up of the child, including genetic variants that influence emotional reactivity or ability to resonate with other people (Bird and Viding, 2014; Viding and McCrory, 2019). A second set of genetic factors influencing the child's developmental course may relate more specifically to traits and capacities that mature in childhood and adolescence, and which can affect the expression of behaviour over time. For example, the capacity to engage in complex, goal-oriented thinking substantially increases across childhood and adolescence (Crone and Steinbeis, 2017), as does sensitivity to what other people think (Foulkes and Blakemore, 2018). This capacity is linked to changes in adolescent brain structure and function (Crone and Steinbeis, 2017; Foulkes and Blakemore, 2018). Goal-oriented thinking and sensitivity to what others think may be important for assessing the best strategies for executing one's own goals, and adolescents' competencies in these domains influence how well they interact with adults and peers. Environmental risk and protective processes, in part those reflecting gene–environment correlation, will also vary according to developmental stage. We argue that consideration of developmentally dynamic effects is critical for refining interventions that address inequalities.

Implications for improving outcomes and reducing inequalities

Even the most successful current interventions, whether these are directed at the caregiver–child relationship or educational support, do not help everyone. Here we consider how gene–environment correlation, early adversity and developmentally dynamic effects might guide our thinking about interventions and how we may refine them.

Many interventions emphasise the ways in which adult behaviour or engagement can affect the child's outcome. However, children also have a key role in shaping and influencing the responses of adults around them, a notion in line with 'evocative' gene–environment correlation. If the child is particularly disruptive (for example), adults may experience difficulty in maintaining a positive relationship over time and constructively guiding the child's behaviour or education. Furthermore, as pointed out earlier, the parents might share some of the vulnerabilities with their child, augmenting the challenge of delivering an intervention. Helping parents, caregivers and teachers reframe a child's behaviour, in the context of a profile of dispositional strengths and weaknesses that the child presents with, could change how the adults around them experience and respond to a child's behaviour. Moreover, having systems in place to ensure that adults caring for the child receive support themselves, as a space to process their relationship with the child, is a prerequisite for them being able to, in turn, provide sustained and predictable caregiving and educational support. Genetic influences on behaviour and cognition are not immutable, but neither are children blank slates. Acknowledging dispositional differences, and the challenges and opportunities they present, is a key to refining preventative and targeted interventions to be more tailored to specific needs of children and families, which in turn will serve to reduce inequalities. Genetic effects are not deterministic, but this should not mean that individual differences in genetic endowments are ignored when preventative and intervention programmes are designed; see Harden (2021) for a thorough discussion of the importance of genetic information for advancing social equality. Addressing the challenges of vulnerable children and families early on, before income, attainment and physical and mental health gaps widen and consolidate, is likely to have particularly positive long-term impact at both individual and societal levels.

Latent vulnerability following early adversity means that a child is likely to develop altered patterns of processing across a range of domains relative to peers. These alterations are posited to reflect adaptation to harmful and unpredictable environments, and relate to changes in learning processes (both social and educational). Ellis et al. (2020) have argued that these changes do not always need to be detrimental, but may, under the right circumstances enhance skills for problem solving (what they call 'hidden talents'). They suggest that education and interventions could be tailored to account for the cognitive adaptations of children who have grown up in adverse circumstances. Traditionally, interventions have focused on bringing children from adverse circumstances more in line with their peers – in terms of both behaviour and educational achievement. Many programmes have focused on 'closing the gap' between children with experiences of adversity and their peers by increasing cognitive stimulation or focusing on intensively training particular cognitive skills or behaviours (e.g. Blair and

Raver, 2014; Yousafzai et al., 2016; Calarco, 2018; Rosen et al., 2020). Ellis et al. (2020) argue that these approaches should be complemented by approaches that focus on leveraging the adaptations that may have occurred following adversity. The logic behind this approach is grounded on learning theory, which posits that new information is acquired more readily when it builds on previous learning and abilities or replicates aspects of prior learning environments (Bransford, Brown and Cocking, 1999). For example, children who have grown up in adversity are less likely to have experienced quiet and orderly environments than their peers. Their past social circumstances also mean that they are less likely to be motivated by the same things as their peers. Preliminary data indicate that classroom environments that offer more opportunities for task-switching and problem-solving content that is more directly relevant for a child's circumstances can help close the attainment gap between children from high- versus low-adversity backgrounds (for a review, see Ellis et al., 2020). In this context, it is also important to underscore that children learn in trusting relationships, and trust is often what has been severely compromised following adversity (Gobin and Freyd, 2014). This is another area where we need systematic enquiry, as interventions supporting formation and maintenance of social relationships is likely to be of key importance for promoting social integration and educational opportunities – both of which will contribute to reducing inequalities following adversity.

Finally, research on developmentally dynamic genetic and environmental effects underscores one key message, already highlighted by Cattan et al. (2022). Early intervention is important, but it is not enough. Those children who are most vulnerable, will need the 'inoculation' offered by early intervention, but will also require 'booster shots' of later, developmentally appropriate preventative and targeted interventions. Different developmental vulnerabilities emerge at different ages. For example, early behavioural difficulties may be attenuated or exacerbated during adolescence, depending on the degree to which a child develops ability to regulate emotions and plan ahead (Viding and McCrory, 2020). Developmental research also points to considerable stochasticity in environmental risk and protective factors. Non-shared (i.e. individual-specific) environmental factors typically account for up to 50% of variance on any given trait, yet specific non-shared environmental influences that would account for a substantial proportion of variance on any given trait have been surprisingly hard to find (Asbury, Moran and Plomin, 2016). These findings suggest that when tackling inequalities, it is important to take a life-course approach that is sensitive to changing individual capacities and circumstances. To do this, we need to adopt statistical approaches that can model dynamic developmental effects in a multivariate space that might include, for example, genetic, epigenetic, brain, cognitive, social, physical environment and economic measures. Such approaches will allow researchers to examine how relationships between different variables develop and change over time, how a particular variable may be a 'gatekeeper' for later developmental milestones, or how the impact of a particular biological or environmental risk factor may only emerge at a later developmental stage, conditional on biological and/or environmental contexts relevant for that stage. If our research questions and statistical analyses remain focused on relatively simple associations, we are in danger of making simplistic assumptions regarding developmental processes that may or may not fit those assumptions.

In short, we argue that there is a need for a cross-disciplinary, integrated approach for preventing inequalities, which emphasises the importance of social development. Within this approach, individual dispositions and proximal environments are not viewed as separate things. Instead, researchers will endeavour to better understand individuals as active co-creators of their environments and will consider what this means for developing more sensitive and nuanced approaches for interventions.

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