Assessing the Foundational Studies on Adverse Childhood Experiences

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This article critically reviews the foundational studies carried out by Felitti in the US and Bellis in the UK and their colleagues examining the relationship between Adverse Childhood Experiences (ACEs) and adult health and morbidity. These studies have paved the way for much research on childhood adversity and its impact on child development and brain functioning at a family level. ACEs have gained traction in the UK in terms of policy targeting dysfunctional families through early intervention to stop the intergenerational effects of adverse childhood experiences. This article questions the foundational research that argues for family-level, parent-based intervention, especially in light of substantial evidence about the biological embedding of poverty and the direct links between disadvantage and child development. It also hopes to raise awareness about the contested nature of ACEs and their growing influence on family policy.

Keywords: ACEs, retrospective recall, social policy, family intervention.

Introduction

This article aims to critically review four key papers, namely Felitti *et al*. (1998) and Bellis *et al*. (2014a, 2014b, 2014c) on the relationship between Adverse Childhood Experiences (ACEs), in the form of childhood abuse and household dysfunction, and adult outcomes. The

original ACE study was conducted by Felitti and colleagues (1998) at Kaiser Permanente from 1995 to 1997. Over 17,000 Health Maintenance Organization members from Southern California receiving physical exams completed confidential surveys regarding their childhood experiences and current health status and behaviours. In the UK, similar research on ACEs was initially conducted by Bellis and colleagues in 2012 and 2014. In 2012, the Centre for Public Health ran the first UK study using internationally validated ACE tools (Bellis *et al*., 2014a). Subsequently, a national ACEs study was undertaken in England in 2013 (Bellis *et al*., 2014b, 2014c). These foundational studies paved the way for other studies making claims about the intergenerational consequences of ACEs thought to be transmitted from mother to offspring (McDonnell and Valentino, 2016) and the impact of ACEs on children's developing brain (e.g. Hughes *et al*., 2017). They also propelled social policy promoting surveillance and intervention based on ACEs (Austin and Herrick, 2014; Center for Youth Wellness, 2014).

In critically reviewing the foundational studies, a number of theoretical and methodological shortcomings are noted. The conceptual and methodological caveats identified refer to the nature of the relationship (causal or association) between 'childhood exposures' and 'risk factors' in adulthood and notions of impact; the context, definitions, measurement and prevalence of childhood adversity; the longitudinal claims made with cross section designs and the validity of retrospective recall; the regression analyses and assumptions about comparable effects and common mechanisms underpinning ACEs; the uni-directional nature of the relationship between ACEs and disadvantage; and the policy implications from ACEs research.

ACEs and adult outcomes: association or causality?

In the studies by Felitti *et al*. (1998) and Bellis *et al*. (2014a, 2014b, 2014c), notions of association, causality and impact were used interchangeably. The relationship between

'childhood exposures' and 'risk factors' in adulthood was referred to as an 'association' and 'cause'. Felitti and colleagues accepted that if ACEs contribute to the development of risk factors in adulthood 'then these childhood exposures should be recognized as the basic causes of morbidity and mortality in adult life' (1998: 246). They considered health behaviours and lifestyle factors to be the leading causes of morbidity and mortality in the United States and extrapolated that if ACEs contribute to these factors then ACEs should be recognised as the 'basic causes' of morbidity in adult life. At the same time, the authors acknowledged that ACEs data were based on retrospective self-reports that could only demonstrate associations between ACEs and health risk behaviours in adulthood. As such, it cannot be deduced that 'the impact of these adverse childhood experiences on adult health status is strong and cumulative' (1998: 251), considering that other risk (e.g. poverty) and protective (extended family support, community ties) factors may explain this association.

Similarly, the findings from the studies by Bellis and colleagues cannot support claims about 'links between such behaviours and childhood circumstances are likely to operate through the impact of ACEs on the developing brain. Thus, early life trauma can lead to structural and functional changes in the brain and its stress regulatory systems, which affect factors such as emotional regulation and fear response' (2014c: 9). We cannot infer causality and state that ACEs 'can lead to structural and functional changes in the brain' considering that the research design was not experimental and child brain functioning was not examined. Also, identifying a control group (adults with 0 ACEs) retrospectively rather than at the time of experiencing risk and abuse as children is problematic when making causal inferences. Finally, to establish causality, the order of the occurrence of events matters. Thus, retrospective recall data have substantial limitations due to possible inaccuracies in the recall of time sequences (Hardt and Rutter, 2004).

The context, definitions and prevalence of childhood adversity

What constitutes childhood adversity requires conceptual clarity and justification. Little information is offered in the foundational studies about the decision to include certain types of childhood abuse and household dysfunction (i.e. physical, verbal, and sexual abuse; parental separation; exposure to domestic violence; and growing up in a household with mental illness, alcohol abuse, drug abuse, or incarceration) and not others such as food insecurity, inadequate housing, societal marginalisation and stigma, or neighbourhood violence. The types of childhood adversity excluded are as important as those included. In terms of its psychometric properties, the ACE retrospective recall questionnaire was developed by adapting questions from published surveys. For example, questions about psychological and physical abuse during childhood and violence against the respondent's mother were taken from the Conflicts Tactics Scale (Straus, 1990) and questions about exposure to alcohol or drug abuse during childhood were adapted from the 1988 National Health Interview Survey. The original surveys have shown good reliability and construct validity; however, because the scales used to assess emotional and physical neglect have shown lower internal consistency compared to those that evaluate major conflict or abuse (Straus, 1990), issues related to reliability and validity of the ACE questionnaire remain unclear.

Furthermore, there is a limited contextual information about the multitude, frequency and duration of childhood adversity; the age of a child at the onset of abuse or the first instance of a traumatic event; the presence of other risk (e.g. poverty) or protective factors (access to extended family support) in the child's environment; sources of validation of the occurrence of a traumatic event (e.g. hospital / school records); and cultural influences and interpretations of childhood experiences to understand their impact on children's lives. The intergenerational divide in recalling adverse childhood experiences (60 per cent of the over

sixty-five reported zero ACEs in the study by Felitti and colleagues and 59.5 per cent in the 2014a study by Bellis and colleagues) highlights generational differences in parenting and family interactions. What could have been described as 'good enough' parenting three to four decades ago, in an era of intensive parenting may be seen as parental negligence. Furthermore, the low prevalence of ACEs in Asian groups (1.7 per cent of Asian reporting four or more ACEs compared to Hispanic 11 per cent in Felitti *et al.* study and the also 5.2 per cent of Asian, compared to 8.3 per cent White and 12.1 per cent 'Other', in the 2014a study by Bellis *et al*.), points to cultural and ethnic influences on perceptions of parenting and childhood experiences. Culture is the framework within which parents and parenting happen; culture influences and also is influenced by parental beliefs and practices (Bornstein, 2012). Different cultural groups possess distinct beliefs and behave in unique ways with respect to their parenting. These beliefs and behaviours shape how parents care for and interact with their children.

A significant variation in the prevalence rates of childhood abuse is noted between the foundational and other cohort studies. In the study by Bellis and colleagues, the prevalence of childhood sexual, physical, and verbal abuse was found to be 6.2 per cent, 14.3 per cent, and 17.3 per cent respectively (2014a). However, data on the prevalence of child abuse (physical, psychological and sexual) from the 1958 birth cohort in the UK showed about 10 per cent were identified for neglect and also for psychological abuse, 6 per cent for physical abuse and 1.5 per cent for sexual abuse (Power *et al*., 2015). Such variation raises questions about sampling criteria and representativeness but also what counts as childhood adversity.

Cross-sectional designs and retrospective recall

The foundational studies by Felitti and Bellis and their colleagues are cross- sectional relying on between- group comparison rather than within- individual change to test retrospectively

recalled ACEs and adult health outcomes. Cross-section designs with retrospective recall are vulnerable to possible biases associated with attrition as well as recall biases that might be relevant to the causal hypothesis being tested (Hardt and Rutter, 2004). Longitudinal designs are more likely to be valid because they examine both within- individual change and case-control comparisons through the collection of multiple data over time to deal effectively with missing data. They also separate person effects on the environment from environmental effects on the person and assess attrition bias (Hardt and Rutter, 2004).

The reliability and validity of retrospective recall of a traumatic experience could also be threatened by inconsistencies in early memories; awareness of the experience at the time of its occurrence; capacity to recall time sequences (e.g. whether an event occurred before or after a traumatic experience); infantile amnesia during the first two or three years or so of life (Lewis, 1995); the degree to which an experience can be objectively verifiable; the frequency of recall and memory consolidation of an event; and individual's mood / personality style or presence of disease at the time of retrospective reporting (Lewinsohn and Rosenbaum, 1987; McFarland and Buehler, 1998). Ferguson *et al*. (2000) examined the temporal stability of retrospective reports of physical and sexual abuse by asking 1,000 young adults at the ages of eighteen and twenty-one years. The consistency of recalling having been abused was low (correlation coefficient about .50) whereas the recall consistency of not having been abused was high (correlation coefficient about .95). In other words, agreement was higher for events that did not happen than for events that did.

Retrospective reports of adverse experiences are sufficiently valid when applied to experiences and events objectively verifiable such as the death of parent, divorce or homelessness. The recall of subtle aspects of child–parent interactions (e.g. emotional closeness to parent) or experiences that are culture specific (e.g. corporal punishment, displays of parental affection) have not been found to have satisfactory validity (Hardt and

Rutter, 2004; Offer *et al*., 2000). In a study by Offer and colleagues (2000), accounts of parent-child interactions and family life (e.g. questions such as 'Was the discipline you received unfair?' 'Was love withheld as a punishment?') at age fourteen showed little agreement with those retrospectively recalled at age forty-eight.

Using both prospective and retrospective measures of ACEs in a longitudinal study, Rueben *et al*. (2016) found that, in comparison to prospective ACE measures, retrospective ACE measures may downplay the influence of negative childhood experiences on 'objective' adult outcomes and exaggerate it on 'subjective' outcomes. This is consistent with criticisms of the validity of retrospective recall of negative childhood experiences, in that mental health problems in adulthood have been found to associate with retrospective self- reports than with other sources of information about childhood adversity. The foundational studies by Felitti and Bellis and their colleagues relied only on adults' retrospective recall of ACEs without including any prospective measures (e.g. reports from siblings, official records from schools, hospitals) for comparison. Cross- sectional studies with retrospective recall are limited in their examination of long-term impact of abuse and household dysfunction on adult outcomes (Hardt and Rutter, 2004).

Regression analyses, comparable effects and interpretations

In the foundational studies, ACEs are assumed to have comparable effects although their origin and context may be distinctly different. As psychological constructs, they are multi-dimensional which leads to greater measurement error. Furthermore, certain ACEs – for example, physical, psychological or sexual abuse – are likely to strongly correlate with each other. In regression analyses, this refers to multicollinearity, which makes it difficult to separate the individual contribution of each ACE to adult outcomes.

Furthermore, a number of binary regressions were run examining associations between dichotomous predictors (i.e. Had/caused unintentional pregnancy < eighteen years; had sex < sixteen years with Yes/ No responses) and outcome variables (in Bellis *et al*., 2014b). These two dichotomous variables could be thought of as dimensions of the construct 'sexual behaviour' so they could have been analysed within a multinomial logistic regression simultaneously instead of running separate binary logistic regressions: which are likely to increase Type 1 error, and falsely uncover statistically significant relationships between ACEs and outcome variables. Also, in the studies by Bellis *et al*. (2014b), logistic regressions were run with essentially one predictor variable: ACE with five categories (0,1,2,3,4+). It is not clear why a regression analysis was used if the intent was to examine bivariate associations – the idea behind regression is to examine multiple predictor variables (that do not correlate strongly with each other) to delineate their unique and cumulative contribution to the outcome variable. Finally, the analyses did not include different types of ACEs (only ACE counts) to examine their relative individual contribution, nor any protective factors, which affect not only recall but also how adverse the experience was understood to be in the first place.

In interpreting regression results, a distinction should be made between the odds for occurrence and actual occurrence of an event. Odd ratios show an increased or decreased likelihood of an event happening. Interpreting the likelihood of risk as actual behaviour is erroneous. Likewise, a slippage of language is observed when impact and odd ratios are discussed interchangeably. Impact should be examined within randomised experiments only whereby confounding variables are controlled through random assignment of persons to experimental and control groups.

ACEs, inequality and disadvantage

The foundational papers claim a unidirectional causal trajectory of ACEs and reduced social mobility in families. Bellis and colleagues argued that ACEs pose obstacles to social movement and contribute to intergenerational disadvantage, stating 'those with 4+ ACEs were more likely to live in deprived areas, be unemployed/on long-term sickness and have no qualifications'. However, considering the study's correlational design, we cannot deduce that ACEs cause poverty, disadvantage and reduced social mobility – in that a causal relationship between ACEs and adult deprivation and poverty was not examined. Rather, the findings showed that individuals who live in deprived areas were more likely to retrospectively recall four or more ACEs. Claims were also made about cycles of influence whereby individuals who reported ACEs were thought to 'take sexual risks, become parents early and raise their children in environments where risks of ACEs are again high' (Bellis *et al*., 2014b: 5). Parents and families are held into account for 'exposing their own children to ACEs' – essentially being blamed for systemic inequality. The authors also stated 'ACE counts correlate with worse health, criminal justice, employment and educational outcomes over the life course. The impacts of ACEs on criminality, violence, early unplanned pregnancy and retention in poverty means those with ACEs are more likely to propagate a cycle that exposes their own children to ACEs' (Bellis *et al*., 2014b: 5). The findings however cannot support that ACEs cause criminality, unplanned pregnancies, retention in poverty and intergenerational disadvantage.

Although Bellis and colleagues acknowledged that socioeconomic gradients are strong predictors of risk factors in adulthood, they consider childhood abuse and family dysfunction to be 'additional factors' to 'explain the resilience and susceptibility of individuals' (2014b: 82). Likewise, in the study by Felitti *et al*., the effects of race and educational outcomes were 'accounted for' statistically. However, measures of childhood abuse and household dysfunction are not simply 'additional factors' but mainly

manifestations of poverty, deprivation and gender inequality; thus untangling their separate contributions to adult outcomes is nearly impossible (even if statistically these factors are 'accounted for'). Although the foundational papers accept strong associations between socioeconomic and risk factors, they 'adjusted for' rather than examined their relative contribution (Hartas, 2018).

The assumed causal trajectory of ACEs and negative adult outcomes requires rethinking, especially in light of strong evidence on the 'biological embedding' of childhood poverty and the durability of socioeconomic differences in health and wellbeing across the life course (Elgar *et al*., 2016; RCPCH, 2017). The Royal College of Paediatrics and Child Health found that certain family and social indicators such as poverty impact upon children's health (2017). Consistently, in the US, Biglan and colleagues (2017) showed that societal conditions such as increasing financial inequality marked by a steep decline in the proportion of families on middle class income contribute to ACEs. Socioeconomic adversity in childhood has been found to associate with adverse health, developmental, cognitive, educational and long-term social and behavioural outcomes in adulthood (Dugravot *et al*., 2009; Fors *et al*., 2009; Horvat *et al*., 2014; Lyu and Burr, 2016). These findings are particularly relevant considering that, currently, one in five children in the UK live in conditions of poverty and this figure is projected to rise (Hood and Waters, 2017).

ACEs and policy implications

In public policy ACEs are invoked to indicate the possibility of risk in families and the need for intervention at an individual level. Increasingly, public health advocates accept childhood adversity to be a 'hidden health crisis' with 'far- reaching consequences' (Center for Youth Wellness, 2014: 1). In the US, with 12.5 per cent of children thought to have experienced 4 or more ACEs, exposure to ACEs has become a major public health concern. Many states now

monitor for childhood adversity among adults through an ACE module provided by the U.S. Centers for Disease Control in Behavioral Risk Factor Surveillance System surveys (Austin and Herrick, 2014).

With ACEs thought of as leading to poverty rather than being a manifestation or direct outcome of it, the foundational studies have contributed to shifting the policy focus on family intervention to support parents to 'nurture' their children as a means of countering the toxic effects of rising inequality. Bellis and colleagues advocated for better ways to 'identify risky or maladaptive family conditions and intervene with families as needed' and 'helping families become less coercive and more nurturing' (2014c: 453) as if lack of nurturance is what underpins structural inequality. A disproportionate focus on ACEs without examining structural constraints in the form of poverty and disadvantage could mean that public policy is based on a narrow and potentially misleading evidence base.

ACE-driven policy trends highlight a disjuncture between the aims and scope of epidemiological research and public service provision. Epidemiological research focuses on understanding the mechanisms underlying chronic non-communicable diseases in adulthood at the level of population probabilities, whilst service provision works at the level of individuals. There is little doubt that poor adult health is intrinsically related to health inequality as well as to other forms of inequality, such as inequality of opportunity. If the aim of public policy is to prevent health inequality at a population level, the effects of childhood inequality should be examined and accounted for. Instead, policy priorities have shifted towards tackling the intergenerational transmission of ACEs via developing and testing family-level interventions that support resilience (seen as being affected by temperament, self-esteem, emotional regulation and trauma) and promote healthy childhood experiences.

On a positive note there is a growing acknowledgement of worse health and poorer educational outcomes in children who grow up in low-income families. A number of recent

studies (Biglan *et al*., 2017; Bruner, 2017; Davis *et al*., 2017) published in a special issue of Academic Pediatrics have concluded that, to improve child health, there must be a reduction in health disparities by socioeconomic status through community-building efforts. Collectively these authors acknowledge the social context of childhood adversity shaped by poverty, racism and discrimination, arguing for community-level intervention to promote health and equalise opportunities. Davis and colleagues talked about the need to support children and families from the start to 'build a culture of health: where everyone has an equal opportunity to be healthy, regardless of who they are, where they live, or how much money they have' (2017: 4). This is a noble vision which, through public investment, could translate into steps towards reducing health inequalities seen as part of wider inequalities in children's life that link to poor health outcomes and social, educational and economic gaps that only get intensified as children move into adulthood.

Conclusion

By examining the nature of the relationship between ACEs and adult health outcomes, the validity of retrospective recall and the use of cross-sectional designs, and the claims of unidirectional links between disadvantage and ACEs, this article sheds light on the contested nature of ACEs and ACE-driven policy trends. It also raises questions about the influence of the foundational studies on shifting family policy on intervention, ultimately highlighting a disjuncture between epidemiological research and public service provision.

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