Dr Dimitra Hartas, Associate Professor, Centre for Education Studies, University of Warwick: **Response to 'The Problem with ACEs'. Edwards et al.'s submission to the House of Commons Science and Technology Select Committee Inquiry into the evidence-base for early years intervention (EY10039). 12 December 2017.**

This submission aims to critically review three key papers on Adverse Childhood Experiences (ACE) and adult outcomes that subsequent studies refer to.

The original ACE Study was conducted by Felitti et al (1998) at Kaiser Permanente from 1995 to 1997 with two waves of data collection. 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 ACE 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 in Blackburn with Darwin (Bellis et al, 2014a). Subsequently, a national ACE study was undertaken in England in 2013 (Bellis et al, 2014bc).

There are a number of theoretical and methodological concerns to note in these studies.

1. There is lack of clarity in the papers by Felitti et al (1998) and Bellis et al (2014) about the **nature of the relationship** between 'childhood exposures' and 'risk factors' in adulthood.

The relationship is interchangeably referred to as an 'association' and a 'cause', with Felitti and colleagues stating that if adverse childhood experiences 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' (p 246). Similarly, in the study by Bellis et al, it was stated that 'links between such behaviors 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' (p. 9). The data and correlational analyses in Bellis' studies cannot support such statements. The design in both papers is not experimental and thus we cannot infer causality, and, certainly, we cannot state that ACEs

'can lead to structural and functional changes in the brain' in that there was no examination of brain functioning taking place in either study.

2. Due care is needed when reporting findings from regression analyses.

It is important to make a distinction between the odds for the occurrence of an event and the actual frequency or rate with which an event occurs. A logistic regression produces odd ratios that show an increased or decreased likelihood of an event happening (eg, an increase in the odds of obesity happening and not an increase in the rates of obesity per se). Turning the likelihood of risk into actual behaviour is mediated by a constellation of factors that are not discussed in these studies namely, family / social support, neighbourhood safety, housing and food security, employment opportunities to name but a few. Also, it is important to differentiate between an increase in the odds of an event occurring and actual'impact', especially if public policy is to be based upon. From a scientific point of view, impact should be examined within randomised experiments only whereby confounding variables are controlled through random assignment of persons to experimental and control groups. The findings from the original ACE study and the studies by Bellis and colleagues uncovered associations between predictor and outcome variables and thus impact cannot be inferred.

3. All nine ACEs were included in the regression analyses in the studies by Felitti et al and Bellis et al to calculate their cumulative contribution to the outcome variables.

This assumes that the nine ACEs, ranging from childhood abuse to family dysfunction, have comparable effects and are underpinned by common mechanisms. However, we do not know if this is the case considering that the study was observational and retrospective information about ACEs was obtained from a survey offering limited knowledge on the context within which ACEs occurred.

4. Across these studies, there is a significant variation in the prevalence rates of childhood abuse.

Regarding the prevalence of child abuse (physical, psychological and sexual), Power et al (2015) examined data from the 1958 birth cohort in the UK (18,000 individuals followed from birth to mid-life). The findings showed that most of the population (78%) were not identified as having experienced neglect or abuse during childhood, but about 10% were identified for neglect and also for psychological abuse, 6% for physical abuse and 1.5% for sexual abuse (Power et al 2015). In the study by Bellis et al (a nationally representative survey of English residents aged 18 to 69, n = 3,885) the prevalence of childhood sexual, physical, and verbal abuse was 6.3%, 14.8%, and 18.2% respectively. These findings show a significant variation, raising questions about sampling criteria and representativeness.

5. There is lack of conceptual clarity on the interplay between ACEs and socioeconomic factors

Although Bellis et al acknowledged that socioeconomic gradients are strong predictors of risk factors in adulthood, 'additional factors' in the form of childhood abuse and family dysfunction are required to 'explain the resilience and susceptibility of individuals' (p 2). However, measures of childhood abuse and household dysfunction are not 'additional factors' but mainly manifestations of poverty and deprivation, thus untangling their separate contributions is nearly impossible (even if statistically the analysis 'accounts for' poverty). Likewise in the study by Felitti et al the effects of sex, race and educational outcomes were 'accounted for' statistically. Although both papers accept that there are strong associations between socioeconomic and risk factors, they 'adjusted for' rather than examining the relative contribution of socioeconomic factors to outcome variables.

Most importantly, limited research and debate on the direct effects of financial inequality on family and children's life has contributed to a confused intellectual landscape. A recent study by Biglan and colleagues (2017) also acknowledged that societal conditions (eg, increasing financial inequality in the US marked by a steep decline in the proportion of families on middle class income and the fact that 1 in 5 children now live in poverty) contribute to ACEs. But then the focus of the study shifted on family intervention to support parenting to offer nurturance to children as a way of countering the toxic effects of widespread inequality. As the authors stated 'given the influence of poverty, discrimination, and economic inequality on ACEs, an

exhaustive public health approach to preventing ACEs needs to address these problems and try to reach individual families and children with evidence-based programs'.

They 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' as if lack of nurturance was the key contributor to ACEs (p 153). Not examining structural constraints in the form of poverty and disadvantage could mean that public policy is based on a potentially misleading and narrow evidence base.

References

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