

**Public care during childhood and biomedical risk factors in middle-age: the 1970 birth cohort study**

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Prospective and retrospective cohort studies conducted over several decades have repeatedly demonstrated associations of childhood socioeconomic disadvantage with adult mortality and cardiovascular disease.<sup>1,2</sup> Such early life adversity has recently been more broadly defined to not only comprise economic hardship but also physical and emotional abuse, sibling loss, parental separation, and out-of-home care, amongst other characteristics.<sup>3</sup> Of these, out-of-home care is receiving increasing research attention. Also known as public care or being looked-after, the majority of these children have been removed from their biological families in order to safeguard their wellbeing following parental abuse and/or neglect. Although the underlying principle of assignment to public care is to deliver a more nurturing and protective environment than that provided via their family of origin, there is some evidence that childhood out-of-home care is linked to unfavorable outcomes in the short- and longer-term. Thus, adults who are exposed to public care in early life experience as much as a tripling of the risk of all-cause mortality relative to the general population.<sup>4</sup> That this gradient appears to be robust to the adjustment of early life confounding factors<sup>4</sup> raises the issue of how childhood public care might be embodied. In follow-up of a birth cohort study, children with experience of care had markedly less favourable levels of educational, social, and psychological outcomes in early middle-age.<sup>5</sup> In contrast, links between care and established biomarkers for mortality have been little-investigated.<sup>6</sup> We hypothesised associations between public care and biomarkers implicated in chronic stress pathways, including inflammation, glucose metabolism, and lipids.<sup>7</sup>

We used data from the 1970 British Cohort Study which is based on a representative sample of people born in England, Scotland and Wales in a single week of 1970.<sup>8</sup> An on-going investigation, since their birth, there have been nine surveys of study members. Out-of-home

care was reported by the parents of study members when they were aged 5, 10, and 16 years, and, for the purposes of the present analyses, exposure was denoted by a positive response to any of these enquiries. During the age 10 survey, information was also captured about the demographic and social circumstances of the parents (occupational social class, maternal age at birth of study member) and the pre-adult health of the study member (hospital visits, behavioural problems<sup>9</sup>). These data were used as covariates. Between 2016 and 2018 (ages 46-48 years), for the first time, cohort members participated in a comprehensive, nurse-administered, home-based clinical examination (N=8,581; 51.8% of the original sample).<sup>10</sup> Relative to non-participants, those who took part were somewhat more likely to be female (44.3 vs. 51.6%), had a lower likelihood of being in care (6.4 vs. 4.7%), had better health in early life based on visits to hospital (24.5 vs. 22.4%) and behavioral problems (Rutter score  $\geq$  95<sup>th</sup> centile: 6.3 vs. 3.9%), and were less likely to have a parent in a lower social group (18.9 vs. 14.3%) (all p-values for difference  $\leq$  0.004).

The medical examination included an assessment of blood pressure using the automated Omron HEM-907 device, standard measurement of height and weight, and the drawing of non-fasting blood samples for analysis of blood lipids, C-reactive protein, and glycated haemoglobin. We used linear regression to generate beta coefficients and accompanying 95% confidence intervals to summarise the relationship between childhood care status and adult biomarkers.

Of the 8,581 men and women who took part in the mid-life survey, 371 (4.3%) had been in care at some point by 16 years of age. Relative to children with a conventional home background, in early life, cared for children were three times more likely to have behavioral problems ( $\geq$ 95<sup>th</sup> centile 3.4 vs. 10.4%), while there were also some marginal differences in hospitalisation (22.1

vs. 25.6%), parental manual social class (13.6 vs. 15.8%), and maternal age (mean age 20.9 vs. 19.5 years).

In table 1 we show the associations between out-of-home care in childhood and biomarkers in middle-age. Mean levels of body mass index, systolic blood pressure, high density lipoprotein cholesterol, glycated haemoglobin, C-reactive protein, and triglycerides, were typically less favourable in adults who, as children, had been looked-after, however, these differences were slight. Any differences were lost after controlling for early life characteristics. Next, we separately examined the influence, if any, of care at each of the three childhood surveys to ascertain if there were relationships with adult biomarkers but there was no such suggestion of sensitive periods of exposure. Owing to missing data, the sample size varied across statistical models, however, analyses of a non-missing dataset did not change these conclusions. Lastly, in sensitivity analyses, in study members who were in care (80 of 3639 people at age 16 years), we tested if the number of different placements, a proxy for housing instability, was related to the biological outcomes, and there was no evidence that this was the case.

Our main finding was that a history of early life public care was essentially unrelated to a range of biomedical risk factors for mortality, suggesting that experience of care was not physiologically expressed, at least not in middle-aged men and women in the present study.

These analyses of data from the 1970 birth cohort confirm recent findings from another study of men and women born in the late 1950s.<sup>6</sup> Instead, socio-economic and mental health factors would appear to offer some insights into mediation in the public care–mortality relationship.<sup>11</sup>

As yet untested, health behaviours (smoking, heavy alcohol intake, etc) may also have mediatory capacity given their links with public care.<sup>5</sup>

Our study of course has its limitations. Parents may have provided a socially desirable response to an enquiry as revealing and sensitive as the public care history of their offspring. We are unaware, however, of any studies assessing the agreement of parental self-reported care with a gold standard such as administrative data. It is also the case that we were not able to examine the impact of duration or reason for out-of-home care placement in the present dataset. Lastly, around half of the original cohort members with out-of-home care data took part in the biomedical survey, with participation lower in individuals with such a history. However, provided there was still normal variation in exposure and outcomes in the remaining study members, attrition is unlikely to have impacted upon our findings.<sup>12-14</sup>

In conclusion, in the present study we found that a history of early life public care was not related to a range of biomedical risk factors for mortality. It may be care experience is more likely to be subsequently psychosocially expressed.

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**Table 1. Association of out-of-home care in childhood with biomarkers in middle-age in the 1970 British Cohort Study<sup>a</sup>**

| Biomarkers   | History of Public Care, mean (SD) |              | Analytical sample size | Sex-Adjusted        |               | Analytical sample size | Multiple-adjusted   |             |
|--|-----------------------------------|--------------|------------------------|---------------------|---------------|------------------------|---------------------|-------------|
|  | No                                | Yes          |                        | $\beta$ Coefficient | 95% CI        |                        | $\beta$ Coefficient | 95% CI      |
| Body mass index <sup>b</sup>                               | 28.4 (5.5)                        | 28.9 (5.8)   | 6830                   | 0.50                | -0.12, 1.12   | 4980                   | 0.28                | -0.46, 1.03 |
| Systolic blood pressure (mmHg) <sup>c</sup>                | 124.7 (15.6)                      | 125.0 (16.0) | 6934                   | 0.35                | -1.33, 2.03   | 5055                   | -0.04               | -2.06, 1.99 |
| High density lipoprotein cholesterol (mmol/l) <sup>d</sup> | 1.52 (0.44)                       | 1.46 (0.42)  | 5576                   | -0.06               | -0.11, -0.003 | 3847                   | -0.006              | -0.07, 0.06 |
| Triglycerides (mmol/l) <sup>e</sup>                        | 1.90 (1.49)                       | 1.88 (1.15)  | 3132                   | 0.02                | -0.25, 0.22   | 2283                   | -0.14               | -0.43, 0.15 |
| Glycated haemoglobin (mmol/mol) <sup>f</sup>               | 37.0 (9.1)                        | 38.2 (10.5)  | 5536                   | 1.18                | 0.02, 2.35    | 3817                   | 0.14                | -1.25, 1.53 |
| C-reactive protein (log units)                             | 0.92 (0.65)                       | 1.04 (0.73)  | 3097                   | 0.13                | 0.02, 0.23    | 2120                   | 0.12                | -0.02, 0.25 |

Abbreviations: SD standard deviation, HDL high density lipoprotein

<sup>a</sup>Multiply-adjusted coefficients are adjusted for sex plus hospital visits, behavioral problems, paternal social class, and maternal age at birth.

<sup>b</sup>Weight (kg)/height (m)<sup>2</sup>

In study members reporting relevant drug therapy, correction to biomarkers was made as follows:

<sup>c</sup>In study members reporting relevant drug therapy, correction to biomarkers was made as follows: +10mmHg for systolic blood pressure

<sup>d</sup>In study members reporting relevant drug therapy, correction to biomarkers was made as follows: -5% for HDL-cholesterol

<sup>e</sup>In study members reporting relevant drug therapy, correction to biomarkers was made as follows: +18% for triglycerides;

<sup>f</sup>In study members reporting relevant drug therapy, correction to biomarkers was made as follows: +11 mmol/mol for HbA1C.<sup>15</sup>