Researchers find sudden cardiac death is associated with thin placenta at birth

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Researchers studying the origins of sudden cardiac death have found that in both men and women a thin placenta at birth was associated with sudden cardiac death. A thin placenta may result in a reduced flow of nutrients from the mother to the foetus. The authors suggest that sudden cardiac death may be initiated by impaired development of the autonomic nervous system in the womb, as a result of foetal malnutrition. The new study, published today in the *International Journal of Epidemiology*, also found that sudden death was associated independently with poor educational attainment. However, sudden cardiac death was not associated with maternal body size, foetal size at birth, or the length of gestation.

Professor David Barker and colleagues examined sudden death within the Helsinki Birth Cohort, which consists of 6075 men and 6370 women who were born in the city during 1934-1944 and attended child welfare clinics. Detailed information was recorded on the birth records of the group including the placental weight and the length and breadth of the placental surface as well as the child's weight, head circumference, and length.

Professor Barker, of the University of Southampton, comments that 'There is currently a growing body of research that shows that coronary heart disease is associated with alterations in prenatal growth and this has led to the hypothesis that coronary heart disease originates in the womb as a consequence of foetal malnutrition. In research recently carried out by myself and colleagues, we found that coronary heart disease among

men was associated with altered shape and size of the placenta.'

'Our new research published today continues the investigation of the relationship between cardiac death and development within the womb. Our new findings suggest that sudden death may be initiated by the impaired development of the autonomic nervous system in the womb, due to placental thinness. A thin placenta may result in foetal malnutrition, due to a shallow invasion of the spiral arteries in the placenta which provide nutrients and blood to the baby. There is evidence that people who experience foetal malnutrition and who are small at birth have an increased sympopathoadrenal response to acute stress, which is known to be linked to death from cardiac arrest.'

Among women, sudden death was associated with a large placental area in comparison to the baby's weight. Placenta expansion in sheep is well documented and the findings in this study suggest that the placenta attempted to compensate for a thin surface by expanding the area of the surface. Professor Barker notes that 'there is evidence that compensatory placental expansion occurs in human and that this expansion may be beneficial in some circumstances. However, if the compensation is inadequate and the foetus continues to be under nourished then the need to share its nutrients with an enlarged placenta may become a metabolic burden and the quality of foetal development may be harmed. We believe that the female foetuses in our study compensated for placental thinness by expanding the placental surface.

Professor Barker and his team also found that sudden cardiac death was strongly associated with low socio-economic status and with low education attainment. He suggests that 'poor educational results may be due to a poor cognitive ability or other issues such as the inability to concentrate or maintain attention. We suggest that the association we have found between sudden death and poor educational attainment results from impaired prenatal development of the autonomic nervous

system. These findings build upon a body of research that has consistently reported associations between sudden cardiac death and low socio-economic status.'

In the study, a total of one hundred and eighty-seven men (2.7%) and forty seven women (0.7%) suffered from sudden unexplained cardiac death outside of the hospital. The rate of sudden cardiac death among men and women increased with placental thinness, with a hazard ratio of 1.47. The authors restricted their analysis to deaths that occurred outside of hospital and were certified as coronary heart disease, with men and women who had never been admitted to hospital with coronary heart disease.

Professor Barker and his team do acknowledge that there are some limitations to the study. The placental measurements were made during routing obstetric practice 70 years ago and the quality of these measurements was not routinely checked and neither were other clinical measurements, such as blood pressure. The mean placental weight in this study was also more than the median recorded in a recent series of deliveries in Europe.

More information: The Placental Origins of Sudden Cardiac Death, by Barker, Larsen, Osmond, Thornburg, Kajantie and Eriksson, *International Journal of Epidemiology*, 2012, 1-6, DOI: 0.1093/ije/dys116