

Scientists identify potential cause for 40 per cent of pre-term births

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Scientists from Queen Mary University of London (QMUL) and UCL (University College London) have identified what they believe could be a cause of pre-term premature rupture of the fetal membrane (PPROM), which accounts for 40 per cent of pre-term births, and is the main reason for infant death world-wide.

The researchers, whose work was funded by the charity Wellbeing of Women, used bioengineering techniques to test the effect of repetitive stretch on tissues of the [amniotic membrane](#) which surrounds and protects the baby prior to birth.

They found that stretching of the amniotic membrane leads to the overproduction of prostaglandin E2 (PGE2) which is damaging to both the cells, and mechanical structure, of the tissue. This overproduction activates the stretch-sensitive protein connexin 43 (Cx43) and reduces the mechanical properties of the membrane, potentially leading to rupture and pre-term birth.

The research, published today (Monday) by the journal Placenta, is the first study to investigate the role of Cx43 in causing PPRM.

The team are now researching possible treatments that would allow the amniotic membrane to be repaired, through successful funding by the Rosetrees Trust.

Co-author of the research, Dr Tina Chowdhury from the School of

Engineering and Material Sciences at Queen Mary University of London, said:

"To have potentially found a way to reduce pre-term births and prevent early deaths of young babies worldwide is incredibly exciting. The unique bioengineering tools at QMUL have allowed us to test the tissue in a way that has never been done before. This gives us an understanding of both the mechanical as well as biological mechanisms involved and will help us to develop therapies that will reduce the number of pre-term births."

Dr Anna David, a consultant in obstetrics and pre-term birth from the UCL Institute for Women's Health and a co-author of the paper, said:

"Our findings have provided a new understanding of why pregnant women who have pre-term contractions go on to rupture their membranes early. The new project funded by the Rosetrees Trust could lead to a therapy that will heal the amniotic membrane and reduce preterm births. This has the potential to save many lives worldwide and improve the health and well-being of women during pregnancy and their families after birth."

Sam Howard, CEO at Rosetrees Trust, said:

"The findings are extremely encouraging and show the value of conducting early stage research into [biological mechanisms](#). This piece in the puzzle may lead to novel treatment strategies to reduce pre-term birth, the main cause of [infant death](#) world-wide. Rosetrees Trust is delighted to be able to support such cutting edge research carried out by outstanding researchers such as Dr Tina Chowdhury and Dr Anna David, which has a direct human impact."

Liz Campbell, Director at Wellbeing of Women, said:

"Wellbeing of Women is committed to funding research which looks at the possible causes of premature birth and offers safe and effective solutions. Premature birth can be very dangerous for mother and baby; and is particularly devastating when babies die. Almost half of premature births occur as a result of waters breaking too early and up until now doctors have not been able to say why this happens.

"This study, co funded by Wellbeing of Women, helps explain why some amniotic sacs, which protect the baby as it grows inside the womb, are more prone to breaking too early - which can lead to [premature birth](#). Going forward, we can now focus on new treatments to address this weakness in the womb tissue and help women who have had previous preterm births feel more confident about their pregnancies and their ability to carry a pregnancy to full term."

More information: 'Tensile strain increased COX-2 expression and PGE2 release leading to weakening of the human amniotic membrane' will be published by the journal '*Placenta*' on Monday 13th October 2014. [DOI: 10.1016/j.placenta.2014.09.006](https://doi.org/10.1016/j.placenta.2014.09.006)

Provided by Queen Mary, University of London

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