

Bear bile chemical could help keep hearts in rhythm

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A synthesised compound which is also found in bear bile could help prevent disturbances in the heart's normal rhythm, according to research published today in the journal *Hepatology* by a team from Imperial College London.

Ursodeoxycholic acid (UDCA) is manufactured as a drug to decrease production of cholesterol in the body and to dissolve gallstones. It is also present in many traditional Chinese medicines made from bear bile.

The new study suggests it could also potentially treat [abnormal heart rhythm](#) or arrhythmia, both in the fetus and in people who have suffered a [heart](#) attack. Laboratory tests suggested that UDCA acts on non-beating pathological [heart cells](#) called [myofibroblasts](#), which interfere with how electrical signals travel across the heart.

UDCA is already used to treat a condition called obstetric cholestasis, which affects around one in 200 pregnant women in the UK and is linked to a higher risk of arrhythmia and sudden death in the fetus. UDCA lowers the levels of harmful bile acids which build up in the mother's blood in the disease and can pass into the infant through the placenta.

The study published today demonstrates for the first time that UDCA can prevent arrhythmia by altering the electrical properties of myofibroblasts. These cells are found in the fetal heart but disappear shortly after birth. However, they reappear in patients that have had a

heart attack, when they are involved in laying down [scar tissue](#). The study found that these cells disrupt the transmission of electrical signals that control the heart's rhythm.

The study is the result of a long-term collaboration between two Imperial research groups, headed by Dr Juila Gorelik, at the National Heart and Lung Institute and Professor Catherine Williamson at the Institute of Reproductive and Developmental Biology.

"These findings are exciting because the treatments we have now are largely ineffective at preventing arrhythmia in patients who develop an abnormal heart rhythm after a heart attack," said Dr Julia Gorelik, the study's senior author.

"Our results from the lab suggest that UDCA could help the [heart muscle](#) conduct electrical signals more normally. We're hoping to set up a clinical trial to test whether these results translate to patients with heart failure."

The researchers became interested in the role of myofibroblasts after observing that they appear in the heart tissue in the second and third trimesters of gestation, when sudden death of the infant is most common in pregnancies affected by obstetric cholestasis.

The researchers grew myofibroblasts on top of heart muscle cells in the laboratory to create a model for studying the fetal heart. Then they used a number of specialised microscopic techniques to study how the cells communicate to relay electrical signals.

Exposing the cells to high level of bile acid, as found in the mother's and fetal blood in obstetric cholestasis, caused the cells to conduct electrical signals more slowly and increased the likelihood of arrhythmia. This effect was not seen when there were no myofibroblasts present among

the heart muscle cells, as in the healthy adult heart.

When the cells were exposed to [bile acids](#) and UDCA, it altered the electrical properties of the myofibroblasts, and the electrical signals propagated across the cell culture more regularly.

"Myofibroblasts affect the propagation of [electrical signals](#) that coordinate the pumping function of the heart," said Dr Michele Miragoli, first author of the study. "Complications from obstetric cholestasis occur most commonly in the last trimester of pregnancy, when the density of myofibroblasts is highest in the [fetal heart](#)."

"Our study suggests that it is the appearance of myofibroblast cells that make the fetus vulnerable to arrhythmia in obstetric cholestasis. We think that targeting these cells could be an important new approach for preventing abnormal heart rhythm, not just in the fetus, but also in people who have had a [heart attack](#)."

More information: M. Miragoli et al. 'A protective anti-arrhythmic role of ursodeoxycholic acid in an in-vitro rat model of the cholestatic fetal heart.' *Hepatology*, 2011

Provided by Imperial College London

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