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[揭密心臟肥大基因 / 英國醫學研究委員會 \(2011\)](#)

中文翻譯 (由駐英科技組翻譯) :

揭密心臟肥大基因

資料來源：英國醫學研究委員會 2011年10月5日

英國醫學研究委員會 (MRC) 帶領歐美日科學家從事的一項跨國研究近來有了突破性的進展，從眾多動物基因中辨識出造成心臟肥大以及心臟衰竭的關鍵基因之一——Endog基因。

研究人員利用不同老鼠做實驗，發現老鼠體內的Endog基因不但影響心臟肌肉厚度、心臟收縮效益，更關係著心臟脂肪的囤積量。這項新發現預計能協助醫學界開發『治本』的心臟療法，而不再只是『頭痛醫頭，腳痛醫腳』的『治標療法』。

心臟肥大是許多心臟病的問題根源，不但嚴重影響個體日常生活品質，還可能因為心臟肌肉受損或僵硬無力而無法正常收縮輸送血液，最終導致心臟衰竭。心臟肥大還有很多其他原因，除了糖尿病或高血壓等疾病連帶影響造成之外，遺傳基因也是不可抹滅的重要因素。雖然過去研究已證實心臟肥大與人體多段基因密碼有密不可分的關係，但此新發現是科學界首次成功明確地辨識出單個基因於其中所扮演的角色。

研究人員解釋，不正常的Endog基因會干擾心肌細胞能量工廠『粒線體』(mitochondrion) 的正常運作，導致細胞無法製造足夠能量供應心臟運轉所需，心肌細胞為了彌補能量不足而必須加倍工作，加工過程中產生有毒副產物『反應性含氧物種』(reactive oxidative species, ROS)，刺激心臟肌肉增長，造成心臟肥大，影響心臟功能。

過去相信Endog基因與細胞死亡有關，此研究意外揭開其與粒線體和心臟肥大之間的關聯，引導學界從徹底不同的角度探視心臟問題，未來或可因此發展出新的心臟病療法——從粒線體及有毒分子ROS等問題著手。

英文原文：

New research found a genetic link for a “heavy heart”
(From the Medical Research Council, 5th Oct. 2011)

An international research team with joint forces from Europe, the US and Japan, led by the Medical Research Council (MRC) in the UK, has for the first time pinpointed a single gene - Endog gene - associated with one of the leading causes of heart thickening and failure.

Scientists have found that the Endog gene in rats and mice influences the thickness of the muscular heart wall, how well the heart pumps and how much fat accumulates inside the organ. It is believed that the findings bring the researchers one step closer to developing new treatments that target the underlying causes of heart conditions, rather than just treating the symptoms.

Enlargement of the heart is one of the many causes of heart problems. It doesn't only have serious impacts on people's daily life but also cause heart muscle to turn weak or stiff or become damaged, hence affecting the heart's ability in pumping blood, resulting in heart failure at the end. While some cases of enlarged heart are caused by other medical conditions, such as diabetes or high blood pressure, a person's genetic makeup can also play an important role. Previous studies have shown a link between heart wall thickening (cardiac hypertrophy) and several sections of the genetic code, but this is the first time researchers have isolated a single gene.

Researchers explained that malfunctioned Endog gene interferes the heart cells' energy source – the mitochondria. Like any other muscle in our body, the heart needs energy to keep in pumping. If the mitochondria don't work properly, the heart struggles to make enough energy and the cells produce toxic by-products, called reactive oxidative species, which increase thickening of the heart wall.

It was previously thought that the Endog gene was involved in cell death. This new research surprisingly unveiled its connection to the mitochondria and the enlargement of the heart, showing scientists a completely different angle to explore heart problems. It opens new ways to develop treatments which target the mitochondria and toxic oxidative molecules.

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