

Scientists uncover a new understanding of male puberty

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Scientists from Monash University have uncovered a new understanding of how male puberty begins.

The key to their findings lies with a protein known as SMAD3 and the rate at which it is produced. Researchers, Associate Professor Kate Loveland and Dr Catherine Itman from the Faculty of Medicine, Nursing and Health Sciences have discovered through laboratory testing that half as much SMAD3 protein results in faster maturation than the norm, and an inability to create SMAD3 results in abnormal responses to testosterone.

"SMAD3 is a protein that translates signals from the environment outside the cell to the nucleus, where it switches genes on or off," Dr Itman said.

"We have been investigating how SMAD3 influences the growth of testis cells and their ability to respond to testosterone".

Puberty begins when the body starts to produce large amounts of the [hormone testosterone](#). Early, or precocious, puberty involves the onset of puberty before eight years of age and affects around 1 in 10,000 boys. On the other hand, puberty is delayed when testis cells cannot respond normally to testosterone. Altered timing of puberty has implications in adulthood, with precocious puberty linked to reduced adult height and delayed puberty associated with reduced [bone density](#).

Testosterone acts through specialized cells in the testis called Sertoli

cells. Before puberty, Sertoli cells multiply, allowing the testis to grow. At puberty, Sertoli cells must stop growing so they can support sperm [precursor cells](#) to develop into sperm.

Professor Loveland, Dr Itman and their colleagues have been investigating how Sertoli cells switch from a multiplying state, making the testis big enough to make sperm, to a mature state that sustains sperm production.

"We have discovered that this is not an "on-off" switch. Rather, it is the amount of the SMAD3 protein in the Sertoli cell that is different in the immature, multiplying Sertoli cell compared to the mature, adult cell". The research identified that it is the amount of SMAD3 present that controls Sertoli cell activity prior to, or after, puberty. When SMAD3 levels are reduced, sperm develop earlier. When SMAD3 is absent, Sertoli cells take longer to respond to testosterone.

Previous research on puberty suggests that pubertal development is delayed in boys exposed to endocrine disrupting compounds, chemicals which impair cell responses to hormones. These chemicals are widely used in industry and in the manufacture of everyday items, such as plastics, cosmetics, paints and detergents.

Dr Itman is supported by a National Health and Medical Research Council (NHMRC) Early Career Project Grant to investigate how these hormone-disrupting chemicals in the environment affect the growth and [maturation](#) of Sertoli cells around puberty, including changes to SMAD3 levels and activity.

"We hope that through our research, we will inform decisions about the influence of chemicals in our environment on the timing of [puberty](#) in boys and on the fertility of adult men" Dr Itman said.

More information: The findings were published in the international biomedical journal *Endocrinology* and can be downloaded at:
endo.endojournals.org/cgi/rapidpdf/en.2010-1453v1

Provided by Monash University

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