Introduction

This session took a fresh look at puberty from two different angles. First, Allan E. Herbison reviewed the genetic basis of puberty, followed by Sabine M.P.F. de Muinck Keizer-Schrama, who presented the therapeutic options in induction and management of puberty in girls.

Allan Herbison, Professor of Physiology and Director of the Centre for Neuroendocrinology at the University of Otago, Dunedin, New Zealand, introduced the audience to the many genes now known to be involved in the activation of pubertal development in rodents and primates, including man. He pointed out the high degree of redundancy that had developed within the participating neural and hormonal networks in the course of evolution. His special emphasis was on the different genes involved in the variants of the Kallman syndrome. Defects of these genes interfere with the migration of gonadotropin-releasing hormone-producing neurons into the hypothalamus. He also discussed evidence that a recently discovered peptide, Kisspeptin, is a key regulator of puberty and reproduction. Kisspeptin, encoded by the *KiSS-1* gene, activates the G protein-coupled receptor GPR54. He showed that inactivating GPR54 mutations cause normosmic hypogonadotropic hypogonadism in humans.

Professor Sabine M.F.P. de Muinck Keizer-Schrama from the Division of Endocrinology at the Sophia Children's Hospital in the Erasmus Medical Center Rotterdam, The Netherlands, reviewed the options for inducing puberty in hypogonadal females with special emphasis on Turner syndrome. Most centers prefer oral 17 β -estra-

diol, starting with a low dose of 0.2 mg/day. Professor de Muinck Keizer-Schrama predicted an increasing role for transdermal estradiol formulations that avoid the 'first-pass' effect in the liver. She pointed out that estrogens have a biphasic action on longitudinal growth, with low doses enhancing growth, and high doses interfering with the attainment of normal adult height. Professor de Muinck Keizer-Schrama provides compelling evidence that low-dose estradiol therapy, instituted at a normal age for puberty in girls, does not interfere, but is rather synergistic with growth-promoting therapy (GH treatment). With this in mind, she ended her presentation with a plea to the audience not to withhold estrogen replacement therapy from hypogonadal girls being at an appropriate age for puberty.

Disclosure Statement

The author has a relevant financial relationship with a commercial interest. He is a teacher/speaker at Pfizer, Novo Nordisk and Lilly, a member of the Advisory Committee/Review Panel of Pfizer Germany, and an Advisory Board member of Pfizer. Consulting fees and honoraria have been received from Pfizer.

Berthold P. Hauffa
University Children's Hospital
Pediatric Endocrinology
Essen, Germany