KEYNOTE LECTURES

Monday, 1 July 1996
Auditorium 1

08:30-09:00

001. Evidence-based reproductive medicine

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As clinicians, we are confronted by a growing body of medical information, the quality of which varies from mostly poor to occasionally excellent. This information base is often called upon to help us answer questions regarding efficacy, the accuracy of diagnostic tests, prognosis, causation and cost-effectiveness. Unfortunately, the demands of a busy practice make it increasingly difficult to keep abreast of developments in the field of reproductive medicine. Consequently, our management decisions are usually based on our original clinical training, which quickly becomes out of date, or on unsystematic observations from our clinical experiences with individual patients. A new paradigm called evidence-based medicine has emerged, which places less emphasis on intuition and the clinical experiences obtained in a non-systematic manner as being sufficient grounds for making clinical decisions. This new approach focuses on tackling clinical problems using existing research findings which have to be sought and evaluated using formal rules for the critical appraisal of evidence. The best of the relevant studies in the literature are selected after subjecting each to a validity assessment. The clinical data are extracted and summarized so that a succinct solution can be provided for the clinical problem at hand. The interest in evidence-based medicine has led to the establishment of several resources that can be quickly accessed by the busy practitioner (e.g. Evidence-Based Medicine, Cochrane Database of Systematic Reviews), which have a special section for subfertility, and the presently being developed Journal Club in Human Reproduction. We have entered a new era of medical practice that requires us to learn how to critically appraise the literature so that we can better serve our patients with reproductive disorders.

09:00-09:45

002. Adam and Eve

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Recent research has revealed some very important differences between men and women. Studies in Sweden and the USA on the spontaneous mutation rate of the X-linked gene for haemophilia have shown that these mutations are up to 11 times more likely to occur in the germ cells of the testis than in those of the ovary. If the testis is the principal site of mutation for all somatic genes, then it holds the key to evolution. Perhaps scrotal descent and testicular cooling are important ways of lowering the metabolic activity of the testis, hence keeping the mutation rate in check. The ovary may be protected from mutagenesis when the female germ cells are metabolically at rest in dictyate. The genes most at risk of mutation are probably those located on the Y chromosome. Not only is the Y chromosome effectively confined to the testis for the whole of its evolutionary history, but it cannot repair defects in its DNA by recombination with the X chromosome, as there is no homologous pairing outside the pseudoautosomal region. The high mutation rate of Y-linked genes is reflected in the lack of homology of the testis-determining gene, SRY, between closely related species. Because the Y chromosome also contains one or more genes responsible for normal spermatogenesis, these are also likely to be particularly prone to mutation. Perhaps this is why unexplained male infertility is much more common and harder to treat than female infertility.