Introduction
There is an unprecedented surge of population growth in economically backward countries, those least able to provide for basic needs and create opportunities. At the same time, 61 countries are seeing fertility at or below replacement level, and their populations could decline over the long-term (UNFPA: state of world population 1999). As fertility falls in more countries, this phenomenon could affect countries with as many as two-thirds of the world’s people. Hence, development of acceptable male contraceptives and treatment for infertility, both, demand a great attention and concern.

Up to 50% of cases of male infertility are idiopathic in nature. This is mainly due to the fact that most of such defects are homed in the testis and our knowledge about the intra-seminiferous tubular regulation of spermatogenesis is extremely poor. All this necessitates a detailed understanding of the hormonal regulation of spermatogenesis within the seminiferous tubule. The Sertoli cell (Sc) surround the germ cells (Gc) at all stages of their development and provide necessary factors including nutrients, metal binding proteins, proteases, androgen binding proteins and growth factors. Because of the blood-testis barrier created by the Sertoli- Sertoli cell tight junctions, blood borne substances fail to reach the adluminal (inner) compartment of seminiferous tubules (Sharpe, 1994b). Hence Gc development and differentiation is solely dependant on the milieu exclusively generated by the Sc. The absolute necessity of Sc functions in spermatogenesis is further highlighted by the lack of any reported conditions where testes contain Gc but no Sc (Griswold, 1993). Lack of sufficient knowledge regarding Sc mediated regulation of spermatogenesis has severely hampered the diagnosis and treatment of infertility due to seminiferous tubule dysgenesis (Winters, 1990). Although, procedures for isolation and culture of Sc from immature rats (18±2 days old) were developed three decades ago, scarcity of primate testes and lack of established procedures for isolation and culture of primate Sc were the proximate causes underlying our meager knowledge about primate Sc functions. Similarly, difficulties encountered in obtaining substantial yield of Sc from adult testis (even from rats) have limited researchers from determining exact regulatory role of adult Sc in spermatogenesis.

Lutenizing hormone (LH) and follicle stimulating hormone (FSH) from the pituitary are the main endocrine regulators of testicular functions. LH stimulates testosterone (T) production by the Leydig cells (Lc). The responses to both FSH and LH
(via T) converge onto Sc, which are the only cells bearing receptors for both FSH (FSHR) as well as T (AR) in the testis (Baccetti et al., 1998). Hence, interaction of FSH and T with Sc has a crucial role in the initiation and maintenance of spermatogenesis. The endocrine activity of the hypothalamic-pituitary-testicular axis, as reflected by the circulating levels of LH, FSH and T, in infant rats (9-11 days old) and monkeys (3-4 months old) is similar to that found in adults. In spite of adult-like concentrations of LH, FSH and T in infant male monkeys and rats, activity of germinal epithelium is limited to proliferation of undifferentiated Type A spermatogonia; noticeable number of primary spermatocytes cannot be detected until puberty in monkeys (3-3½ years) and day 15 of age in rats (Dym et al., 1995; Plant, 1994). Lack of initiation of spermatogenesis in the face of adequate hormones in infants is a situation similar to that found in certain categories of male infertility and demands a physiological justification. Thus, the infant testis can be used as a surrogate for research related to infertility primarily due to testicular failure. One hypothesis for this testicular quiescence, despite a robust hormonal drive, could be that the infant Sc fail to transduce the hormonal (FSH and T) signals necessary for the occurrence of spermatogenesis. In response to FSH and T stimulation, adult Sc are known to create a favorable environment inside the seminiferous tubule and produce factors necessary for the development and differentiation of spermatogonia into spermatozoa. Hence, a comparative evaluation of FSH and androgen signal transduction pathway in Sc from adult testis (containing advanced Gc) and infant testis (containing only spermatogonia) might unfold developmental deficits, if any, in FSH and androgen signaling pathways of infant Sc which might be responsible for the spermatogenic quiescence at this stage of development.

Culture of Sc from a spermatogenetically active testis is an essential pre-requisite to define the role of Sc in spermatogenesis, because this process occurs only in the mature testis. However, culture of Sc from adults has posed serious methodological problems with regards to the isolation of pure population and yield of Sc (Majumdar et al., 1995b; Russell and Steinberger, 1989). The major difficulty is encountered due to the presence of large number of sperms and other advanced Gc in adult testis. Also, adult Sc do not attach readily to the substratum and fail to multiply restricting their numbers, unlike infant or juvenile Sc. One of the most likely solutions for this problem is to isolate
and culture Sc from testis at the onset of spermatogenesis. During this phase, Sc are mature enough to initiate spermatogenesis in response to hormones but there are less numbers of advanced Gc in the testis. Moreover, such pubertal Sc can potentially proliferate (unlike adult Sc) to provide substantial numbers, in vitro.

Sertoli cells isolated from such stage of development may be used as a yardstick of mature ‘hormonally responsive Sc’ and their functional comparison with infant Sc may help us in resolving the deficits, if any, residing in the signal transductional pathways of the infant Sc.

Such a comparative evaluation would increase our understanding about idiopathic male infertility (for which the non-spermatogenic infants are surrogates of choice) and help in the diagnosis and treatment of such infertility. This would also provide an opportunity to attempt induction of such (infant-like) natural contraceptive state in normal males with a viewpoint of developing a safe and reversible contraceptive.