Introduction
Human population growth is in an exponential surge due to enormous advancements in medical sciences and massive increase in agricultural productivity in the last century. However, about 15% couples worldwide are childless due to infertility (Reviewed by Matzuk and Lamb, 2008). 61 countries are facing fertility related problems at or below the replacement level (UNFPA: state of world population, 1999). 50% of such infertility is because of the male factor and of which half is idiopathic in nature (Irvin, 1998). Development of an acceptable treatment for this kind of male infertility demands a sincere concern. This necessitates a detailed understanding of the regulation of spermatogenesis within the testis.

Testis plays a major role in the development of male reproductive system and provides the site of spermatogenesis. Spermatogenesis is a highly ordered process by which germ cell lineage gives rise to functional gametes under the influence of a complex interplay between endocrine and paracrine signals. Lutenizing hormone (LH) and Follicle stimulating hormone (FSH) from the pituitary are the main endocrine regulators of testicular physiology. LH acts on testicular Leydig cells (Lc) to produce Testosterone (T). Sertoli cells (Sc), present in the seminiferous tubular compartment of the testis, posses both the receptors for FSH and T. Germ cells (gc) development occur under the structural support and milieu provided exclusively by the Sc. Sc surround Gc at all stages of development and provide necessary factors including nutrients, metal binding proteins, proteases, androgen binding proteins and growth factors. Therefore, productive interaction of these hormones with Sc has a crucial role in the initiation and maintenance of spermatogenesis (de kretser and Kerr, 1994).

Rat is a well accepted model to study the endocrine regulation of testicular functions. Onset of puberty in male rats is well documented by the fluctuations in the levels of serum gonadotropins and androgens. First week of postnatal age is known as neonatal stage, followed by infancy upto day 21 after birth. Juvenile period is upto day 35-40 of age, and thereafter puberty starts and ends at around day 55-60 of age with the presence of mature sperm in vas deferens (Reviewed by Ojeda et al. 1980). However, unlike primates, the circulatory levels of LH, FSH and T in postnatal developmental phases of rats from birth to onset of puberty are not remarkably different (Dohler and Wuttke, 1975).
Rat Sc continue to proliferate under the influence of FSH and other various mitogens upto about day 15 of postnatal age (Clermont and Perey, 1957) to establish their final number inside the seminiferous tubules. The final number of Sc inside the seminiferous tubules determines the maximum sperm output in adult males (Orth et al. 1988) and constitutes the blood-testis barrier (BTB) by the formation of tight junctions between two adjacent Sc. After day 15 of age, Sc proliferation ceases and Sc undergo tremendous changes in terms of hormone mediated responsiveness in a process known as functional maturation or differentiation of Sc (Gondos and Berndston, 1993).

Gc population observed in the seminiferous tubules of neonatal and infant rats (upto 9 days of postnatal age) are predominantly spermatogonia A. Presence of Spermatogonia B becomes conspicuous only after day 11 of age (Dym et al. 1995). Therefore, it may be presumed that Sc matures in terms of responsiveness to FSH and T somewhere around 9 to 11 days of postnatal age which in turn might be the cause of the initiation of robust Gc differentiation. Hence, it is essential to find out the mechanism that triggers Sc differentiation in an age dependent manner to understand the cause of azoospermia during infancy in rats. Absence of robust spermatogenesis in spite of sufficient hormonal milieu during infancy is a situation similar to certain forms of idiopathic male infertility.

Primary culture of Sc is a suitable model system to study Sc physiology that has been established in 1970s and widely used till date (Welsh and Wiebe, 1976 and Steinberger et al. 1978). The studies include mainly the identification and characterization of the secretary products of Sc (Androgen binding protein, Transferrin, Ceruloplasmine, Sulphated glycol proteins, Lactate, Inhibin, Estradiol, Anti Mullerian Hormone etc.) under various endocrine stimuli, and expression profile of FSH and T responsive genes. Although behavior (biological response) of isolated Sc in vitro differs from that of the in vivo, cultured Sc remain hormone responsive and moreover seems to be the only existing technique available till date to evaluate of hormone responsiveness of Sc in terms of expression of genes and secretion of various products (Wood and Walker, 2009). Mechanism of Sc differentiation also has been investigated using Sc primary culture [Holsberger et al. (2003), Buzzard et al. (2003) and Wood and Walker, 2009].
A comparative signal transduction study for both FSH and T in Sc from spermatogenetically inactive testes (Sc isolation and culture from 5-days-old, or 9-days-old rats) and spermatogenetically active testes (Sc isolation and culture from 12-days-old or 18-days-old rats) would provide useful information to address the changes associated with the maturation of Sc in terms of hormone responsiveness and expression of various genes necessary for the induction of Gc differentiation. Identification of genes specifically expressed in immature and mature rat Sc would provide an opportunity to understand the basis of the regulation of the initiation of spermatogenesis and hence would indirectly address some issues related to idiopathic male infertility.