SUMMARY AND CONCLUSION

AMH, a member of the TGF-β superfamily, is a Sertoli cell-specific product. This gonadal glycoprotein is responsible for the regression of the Müllerian duct in the male fetus. The AMH also possesses another functions include inhibition of spermatogonium maturation, differentiation of gonads, regulation of fetal lung development, suppression of tumor growth, and many functions still unknown.

By the end the 5th embryonic week, Sertoli cells synthesize AMH. The initiation of AMH secretion is independent of pituitary function. Afterwards, fetal FSH regulates Sertoli cell activity. Serum AMH declines substantially during puberty, as an early sign of local testicular testosterone activity and spermatogenic development.

AMH is secreted by Sertoli cells both apically and basally. In prepuberty, the secretion is mainly basally determining a high serum concentration. After puberty the secretion is apically resulting in higher seminal AMH concentration compared to serum AMH concentration. AMH concentration in seminal plasma in obstructive azoospermia is lower than in normal men suggesting that AMH is also a marker of Sertoli cell function.

In female, AMH expression begins in the 3rd trimester of gestation and it has an important role in early follicular development. AMH

secretion increases from barely detectable levels at birth to high levels after puberty. Then AMH remain stable until age 30 yr and declining more steeply thereafter. AMH is only expressed by the ovaries. AMH has many functions include inhibitory effect on primordial follicles recruitment, responsiveness of growing follicles to FSH, diagnosis of premature ovarian failure, diagnosis of PCOS, estimation of ovarian reserve, predicting the outcome of in vitro fertilization and predicting age of menopause.

Delayed puberty is defined as the lake of development of secondary sex characteristics by 14 years of age or failure to complete sexual maturation within 4.5-5 years after its onset. CDGP is defined as: "constitutional delay of growth occurring in otherwise healthy adolescents with stature reduced for chronological age, but generally appropriate for bone age and stage of pubertal development, both of which are usually delayed". Also CDGP is defined as lack of any signs of puberty at an age 2.0 SD above the mean chronological age for puberty onset.

This study was done to evaluate the level of AMH in different aged groups of healthy men since delivery till senility, and to evaluate the AMH level in a group of boys complaining of constitutional delayed puberty.

The study included one hundred and twenty males were selected from those attending the Dermatology and Andrology clinic, **Benha**

University Hospital in the period from January 2009 to January 2011. They were classified into groups. Seven groups (from Group 1 to group 7) included 15 normal male representing different ages from birth to seventy years. Group 8 included 15 boys complained of CDGP.

All males in the first seven groups were subjected to through history taking, complete physical and genital examination (determine the Tanner stage of puberty. Any male with any medical problem that may affect the AMH level as chronic diseases, drug intake, operations and genital anomalies were excluded.

The boys in group 8 were subjected to the measures in addition tom Left wrist bone X ray were done to determine the bone age. Boys with elevation of gonadotrophins level were excluded.

Blood samples were withdrawn from an antecubital vein and were clotted, centrifuged, and serum stored at minus 20 Cº until hormone analyses were performed to all males in this study. AMH were measured by **DSL-10-14400 ACTIVE® MIS/AMH ELISA**.

The AMH levels in different groups were statistically analyzed. AMH levels were elevated in prepubertal boys. The AMH showed marked decrease in boys who showed signs of puberty clinically. The signs of puberty were determined according to Tanner staging and testicular volume was estimated by using Prader's orchidometer.

The AMH levels were nearly stable in healthy males from twenty to sixty years who were included in groups (group 3 to group 6). The AMH level showed another sharp decline in group 7 which included healthy men older than sixty years. These results may be explained by the decline in Sertoli cell number and function by age. These results cannot be explained by the probable decline in testosterone in aged males.

The AMH levels in CDGP group were nearly equal that of high prepubertal levels and there was a highly significant difference when compared to AMH levels of age matched normal pubertal controls.

In conclusion, detectable AMH serum levels were found at all ages, with the highest measured levels during childhood. At the time of puberty, AMH concentrations declined and remained relatively stable throughout adulthood, and declined again in aged men. Patients with constitutional delay of puberty showed nearly equal the high level measured during childhood.

To the best of our knowledge, this study is the second longitudinal study focusing on the evolution of AMH secretion through life in healthy males. And this study is the first one that focused on AMH level in old aged men.