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The role of macrophages and mast cells in testicular fibrosis seen in Klinefelter men

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**Introduction**

Klinefelter syndrome (KS) affects 1-2 in 1000 newborn males. Men suffering from this genetic disorder have at least one supplementary X-chromosome. KS is mostly diagnosed at adult age when consulting a centre for reproductive medicine since 95% of these men are infertile. During testicular development, a loss of spermatogonial stem cells can be detected. From puberty onwards, testicular fibrosis is detectable. How this germ cell loss and fibrotic process occur, remains unknown.

**Aim of research**

The general aim of this study was to characterize the fibrotic remodelling in Klinefelter testes through the identification of key players in the initiation of testicular fibrosis. We hypothesized that macrophages and/or mast cells and their secretory products were involved in the initiation of the fibrotic process observed in testicular tissue of KS men, since previous research revealed an increase in these cells in other men with impaired spermatogenesis.

**Material & Methods**

**Patient samples:** KS, Sertoli cell only (SCO) syndrome, testis atrophy and control testicular tissue from adult, peripubertal and prepubertal patients

**Techniques:** Immunohistochemistry, RT-qPCR and RNA sequencing

**Genes/proteins included:** CD68, tryptase, TNF-α and decorin

**Results**

**Conclusion**

This study is the first report revealing an increase in macrophage and mast cell number, as well as an increase in decorin expression in adult testicular tissue of Klinefelter men. In conclusion, we assume that macrophages and mast cells are not solely responsible for the fibrotic process in KS. However, these cells and their secretory products definitely play a role in the fibrotic process, leading to infertility. In future research, other possible key players, which will be identified through differential gene-expression analysis, will be studied for their capacity to initiate testicular fibrosis.

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