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in a mouse model of spermatogenic impairment.

Progressive loss of spermatogenesis is a major cause of male infertility. In humans, reduced rates of spermatogenesis are detected in approximately 20% of infertile men. Although the etiology of the majority of cases of male infertility remains unknown, genetic factors are known to influence spermatogenesis. The purpose of this study was to identify the genes involved in sexual development and

spermatogenesis that may be useful for developing animal models of spermatogenic impairment. We have analyzed the gene expression profiles of mouse testes at several different stages of spermatogenesis using two-color cDNA microarrays. The expression profiles of testes were compared with those of testes from a model mouse strain that shows spermatogenic arrest at the pachytene stage of meiotic prophase I. Male mice of strain C57BL/6J, which show

spermatogenic arrest at the pachytene stage of meiotic prophase I, and female mice of strain C57BL/6J were generated by crossing male heterozygotes and female homozygotes. The gene expression profile of the testis of the C57BL/6J mice was compared with that of the C57BL/6J x CBA/J F1 mice, and genes with a statistically significant difference in expression level were identified.

We suggest that 19 of the 722 genes, which showed a statistically significant difference in expression

level between C57BL/6J and C57BL/6J x CBA/J F1 testes, could be considered to be associated with spermatogenic impairment. Furthermore, in the genes showing a statistically significant difference in expression level between C57BL/6J and C57BL/6J x CBA/J F1 testes, 20 of the 71 genes were identified that showed a significant difference in expression level with castration. These gene expressions were observed in C57BL/6J mice, but not in CBA/J mice. The genes were related to the progression of

spermatogenesis and the maintenance of the histone-to-protamine transition in the nucleus. The results indicated that a large number of genes in the testes were expressed in a sexually dimorphic manner. We suggest that the genes,

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