Infertility is a widespread health problem, caused by the male factor in about half of all cases, and in about a half of the infertile men the cause is unknown. In a significant number of these men, genetic etiology is assumed. Current routine methods of laboratory diagnostics, which include karyotype examination, exclusion of mutations in the CFTR gene, and Y chromosome microdeletions, do not usually reveal the cause of infertility. That is why researchers' efforts aim at detecting mutations in other genes that are causing male infertility. In recent years, animal models have been used to identify many genes necessary for fertility. Based on these findings, 12 candidate genes have been selected (CAPZA3, CDC14B, CDC42, CNTROB, CSNK2A2, GOPC, HOOK1, HRB, OAZ3, ODF1, RIMBP3, SPATA16) that are essential for spermatogenesis. Mouse or rat mutants in these genes are primarily associated with oligoasthenoteratozoospermia, since they are involved in sperm morphogenesis. However, the phenotype spectrum may comprise also azoospermia. The purpose of the thesis was to determine the sequence of the afore mentioned genes in infertile men with impaired spermatogenesis and to reveal presence or absence of pathogenic mutations in these genes, using cDNA and genomic DNA from peripheral blood. The candidate genes were amplified by PCR and sequenced using next-generation sequencing on the GS Junior platform (Roche). 73 patients and 7 control samples of men with normozoospermia have been sequenced, uncovering 14 new and 63 known variants that have been classified according to their potential pathogenicity. None of the observed variants could be classified as causal mutations responsible for the infertility phenotype.