Nicotine (Ni), present in cigarettes, reaches the maternal milk and crosses the placental membrane. It inhibits steroidogenesis, suppresses testosterone secretion in adult male rats, causes testicular atrophy and infertility. Previously, we observed that Ni, administered in pregnant and lactating rats, provokes, in offspring, late morphofunctional alterations of Leydig cell, body weight raise in adulthood (90 day postpartum-dpp) and an evident injury of the seminiferous epithelium. Currently, we studied whether the spermatogenic damage observed from pregnant and lactating Ni-exposed rats are maintained or whether it is worsen in older offspring after two complete periods of spermatogenesis (53 days). Pregnant and lactating rats were Ni-exposed (2mg/Kg/day) through an osmotic minipump implanted at first day of pregnancy and replaced after birth. Control (no minipump) and Sham (minipumps without Ni) groups were established. The offspring was killed at 90, 143 and 196dpp. The progenies did not show significant alterations of body weight, testicular and epididymary volume, also the testicular and epididymary stereological and morphometric measurements. Testicular concentration of 19 step spermatids, sperm diary production and transit time of the sperm of epididymis did not show significant changes. However, Ni group presented significant high frequency of morphologically abnormal sperm. Testicular and epididymary histological study, plasmatic and intratesticular levels of cholesterol and testosterone, sperm mitochondrial activity, motility and DNA fragmentation (Comet Assay) are being evaluated. These data indicate the occurrence of a late damaging effect on spermatogenesis evidenced by the high frequency of abnormal sperm and suggesting a low sperm quality in rats Ni-exposed.