MON-029: Exposure to Fine Inhalable Particulate Matter of Air Pollution (pm2) of the City of Sao Paulo Causes Tubular Degeneration and Altered Mitochondrial-Related Gene Expression in Mice Testis

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Background: Air pollution in industrial and urban centers is related to negative effects in living organisms, including alterations in male fertility. The fine inhalable particulate matter is the most deleterious fraction of air pollution and consists of a complex mixture of particles sized 2.5 μm or less of aerodynamic diameter (PM2.5). Experimental studies had already showed a deleterious effect of air pollution of Sao Paulo city on fertility in male mice. The hypothesis of its toxicity is based on activation of oxidative stress and inflammatory via as well as DNA adducts, leading to epigenetic alterations.

Aim: To evaluate, under different conditions, the effects of the exposure to air pollution of city of Sao Paulo on mice testis.

Methods: A group of mice was exposed during gestational period and after birth, from the weaning day until adulthood (prenatal and post-weaning group - PNPWG); another group of mice was exposed to PM2.5 during gestational period only (prenatal group - PNG); a third group of mice was exposed to PM2.5 after birth, from the weaning day until adulthood only (post-weaning group - PWG); and finally, a fourth group of mice was exposed to filtered air during gestational period and from post-weaning day until adulthood (control group - CG). Analyzes were performed on testis from adult animals. Stereological techniques were used to analyze structures of testis and Johnsen's score to evaluate spermatogenesis in a qualitative way. DNA microarray were used to evaluate gene expression.

Results: Testis of all the exposed groups showed tubular degeneration characteristics. In comparison with CG, the testis of PNPWG showed higher weight and volume, surface area of the seminiferous tubules, volume of the seminiferous epithelium and epididymis weight. Quality of spermatogenesis in PNPWG was also significantly worse in comparison with CG. PWG had similar results when compared with CG, however the only parameters with significantly difference were the volume of the seminiferous epithelium and the quality of spermatogenesis. Mean volume of the seminiferous epithelium from PNG was significantly lower when compared to CG. Gene expression analysis exhibited differential expression of genes mostly related to mitochondrial tRNA in group-to-group comparison. The mt-Ty (mitochondrially encoded tRNA tyrosine) gene was hyperexpressed in PNPWG in comparison with other groups.

Conclusion: We demonstrated for the first time that the exposure to PM2.5 of the city of Sao Paulo causes tubular degeneration with decreased or absent spermatogenesis associated with differential expression of genes related to mitochondrial function in mice testis. In addition, we demonstrated that post-natal exposure seems to be more deleterious for the testicular function.

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