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Chronodisruption in healthcare workers

Oxidative damage is the main threat to the integrity of the genome in most living organisms and is therefore considered an essential factor in the process of carcinogenesis, although direct causal connections are still lacking. The molecular mechanisms underlying the process of tumor transformation induced by oxidative damage are not yet completely clarified.

Recent studies point out that shift work can compromise the ability of DNA to repair the damage of natural oxidative processes. Furthermore, night work seems to be a risk factor for some neoplastic diseases such as breast and prostate cancer.

In the present study, the urinary concentrations of 8-hydroxy-deoxyguanosine (8-OH-dG) and 6-sulfatoxymelatonin (aMT6) were determined in a group of operators who also performed night work. The 8-OH-dG allows the evaluation of oxidative damage; through the concentration of aMT6, it is possible to determine the concentration of secreted melatonin.

Twenty healthcare shift workers (HCSWs) and 20 non-shift workers (HCNSWs) were recruited. The study was proposed as part of a periodic health surveillance visit. Each was asked to collect the 24h urine. The values of 8-OH-dG and aMT6 were corrected for urinary creatinine concentration (CREU).

The study participants were all women, with a mean age of 46.5 ± 7.8 and 47.4 ± 6.4, respectively in HCSWs and HCNSWs. The average number of night shifts per month was 3.7 for HCSWs. The urinary 8-OH-dG concentration was significantly ($p < 0.05$) greater in HCSWs (10.3 ± 4.1 nmol/24hr) than in OSNs (6.3 ± 3.7 nmol/24hr). The mean values of aMT6 (expressed in ng/mgCREU) were significantly ($p < 0.05$) lower in HCSWs (20.5 ± 7.7) than in HCNSWs (44.5 ± 10.8).

In HCSWs, the detection of high urinary concentrations of 8-OH-dG, a by-product responsible for repairing oxidative stress damage, would appear to be due to the low amount of aMT6 found. Melatonin, a hormone that regulates the sleep-wake cycle, is one of the promoters of the nucleotide excision DNA repair mechanism. Therefore, it appears that at low melatonin levels, the repair mechanism does not go into action, and free radicals are not countered, generating altered levels of 8-OH-dG.