

G2 checkpoint-dependent DNA repair and its response to catalase in Down syndrome and control lymphocyte cultures

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The amount of DNA lesions repaired in G2 and also G2 timing are controlled by the DNA damage-dependent checkpoint. Down syndrome (DS) lymphocytes showed twice as much constitutive DNA damage in G2 than control ones, when recording it as chromosomal aberrations in metaphase, after caffeine-induced checkpoint abrogation. During G2, DS lymphocytes repaired 1.5 times more DNA lesions than control ones. However the DS cells displayed a decreased threshold for checkpoint adaptation, as the spontaneous override of the G2 to mitosis transition block induced by the checkpoint took place in the DS cells when they had three times more DNA lesions than controls. Catalase addition to cultures scavenges hydrogen peroxide diffused from cells, resulting in subsequent intracellular depletion (Antunes and Cadenas, 2000). The intracellular H₂O₂ level seemed to regulate the G2 checkpoint. Thus, in controls, H₂O₂ depletion (induced by 3.2-50 μ g/mL catalase) prevented its functioning: chromosomal damage inc