THE BIOCHEMICAL MANIFESTATIONS OF RADIATION INDUCED DAMAGE IN CULTURED MAMMALIAN CELLS

БИОХИМИЧЕСКАЯ МАНИФЕСТАЦИЯ ПОВРЕЖДЕНИЯ ВЫЗВАННОГО В КУЛЬТИВИРОВАННЫХ КЛЕТКАХ МЛЕКОПИТАЮЩИХ ОБЛУЧЕНИЕМ

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The UV induced lesion of pre-eminence is the pyrimidine dimer. Repair of this lesion may take place by three routes: (i) photoreactivation, which is not present in the placental mammals, (ii) excision repair, where a region of DNA containing the dimer is removed and the resulting gap is filled by DNA synthesis outside the S phase of the cell cycle. This synthesis may be measured as repair replication by Caesium chloride gradient centrifugation techniques or autoradiographically as unscheduled DNA synthesis. Primate cells excise dimers efficiently but rodent cells to a limited extent or not at all. Since the amount of repair replication is small, rebanding of gradients is often necessary in order to display this phenomenon. If synchronized material is used, uniquivocable evidence for repair replication and unscheduled DNA synthesis can be shown for 3 cells lines of the Chinese hamster, (iii) post-replication repair; this mechanism is demonstrated by the recovery of control molecular weight as measured by alkaline sucrose gradient sedimentation. Following a treatment with UV DNA of low molecular weight is produced, the newly synthesised DNA containing gaps opposite dimers. The restoration of the molecular weight occurs by filling these gaps, in the case of mouse lymphoma cells this occurs by de novo synthesis not by a recombination process.

A study is in progress of a parental and UV sensitive line of the Chinese hamster. A number of lines of evidence suggest that the difference in sensitivity may have origin in the absence of excision repair. This assumption is supported by the biochemical data.