17 Isolation of UV resistant cells from Cockayne syndrome group A cell line by introduction of human expression cDNA library.

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To understand the molecular mechanism of the DNA repair system in human cells, it is necessary to isolate the repair genes in such cells. To isolate the repair gene defective in Cockayne syndrome group A (CS-A) cells, we constructed the human expression cDNA library containing the neomycin resistant gene. SV40 transformed CS-A cell line (CS20SSV) was transfected with the cDNA library by lipofection method. About 1.2 x 10^5 neomycin resistant colonies were selected by UV irradiation and several colonies survived. Further analysis of these survivors is in progress.

18 **Interstrand cross-linking, repair and lethal effects of cisplatin derivative DWA2114 in Fanconi's anemia cells**

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I investigated interstrand cross-linking, repair and lethal effects of a new cisplatin derivative, DWA2114R, which has two structural modifications with aminomethylpyrrolidine and cyclobutanedicarboxylic acid (CBDCA) in place for amino and chloro-ligands, respectively of cisplatin. Compared with cisplatin, an order of magnitude greater molar concentration of DWA2114R was required for induction of the equal number of interstrand cross-linking of human cell DNA and killing of human cells. XPA cells repaired normally (t1/2 = 7 h) the interstrand cross-links induced by cisplatin and DWA2114R. However, Fanconi's anemia (FA) cells failed in the repair of interstrand cross-links. XPA cells were unable to repair intrastrand cross-links, but FA cells were able. DWA2114R and carboplatin exerted the most effective killing of FA cells, while cisplatin did the equal killing of FA and XPA cells, suggesting more effective production of interstrand cross-links by DWA2114R and carboplatin than by cisplatin

19 **Establishment of cell line derived from the patient with Nijmegen breakage syndrome and the genetic complementation with Ataxia telangiectasia disease.**

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Fibroblasts from patients with Nijmegen breakage syndrome(NBS) were transformed by infection of SV40 virus. The cells used were 0816J, 0823H, 1129W, 1022Q, 1217G(each Czech), GM07166(USA), 2239, 2240(each German). Although all virus-infected fibroblasts showed the prolonged life span, only GM07166SV clone was immortalized. Southern blot analysis indicated the SV40 virus genome present at this clone. The GM07166SV clone was high sensitive to ionizing radiation-induced cell killing, similar to parental GM07166 cells. The hybrid clones obtained between GM07166SV(gpt) and AT5BIVA(neo) showed the restoration of radiation sensitivity, implying the different mutations of both diseases. We are also trying to determine the chromosome carrying NBS gene, by introduction of a single chromosome via micro-cell fusion technique.