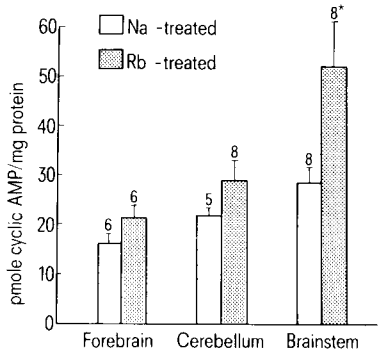
separated into brainstem, cerebellum and forebrain[9]. Cyclic AMP was assayed per procedure of Gilman[10], as modified by Weller et al.[11]. A Perkin-Elmer atomic absorption spectrophotometer (model 403) equipped with a 3-slot nebulizer burner was used for the determination of rubidium, sodium, potassium, calcium and magnesium ions[12].

Results. Rubidium-treated rats appeared more alert and aggressive relative to the sodium-treated controls. Hyperactivity was observed starting 2 weeks after rubidium ingestion. The activity meter reading (mean±SE) for rubidium-treated rats was 121±10 cpm as compared to 63±5 cpm for controls. Rubidium ingestion hat no effect on sodium, calcium and magnesium concentrations in the brain. However, rubidium was found to replace brain potassium approximately on a molar basis (table). The figure shows that ingestion of 50 mM rubidium chloride for 1 month lead to almost a doubling of the cyclic AMP concentration in the brainstem. There was also a general but statistically insignificant ($p > 0.05$) increase of cyclic AMP levels in the forebrains and cerebellums of rubidium-treated rats.

Discussion. The literature report that rubidium treatment increased locomotor activity in the rat[6] was also observed in this laboratory. In addition, this study demonstrated the replacement of brain potassium by rubidium in the rat. This may result in a less negative resting membrane potential[13], and rubidium-treated rats would be more responsive to depolarizing influences. It is therefore reasonable to predict that rubidium-treated rats may have a higher neuronal firing rate, which in turn releases more catecholamines at the nerve ending. This can explain the findings of Stolk et al.[3] that greater amount of neuronal stored norepinephrine were released to central adrenergic receptors in the rat after rubidium injections.

Cyclic AMP has been established to be the intracellular messenger mediating the biological effects of norepinephrine[14]. Increased levels of cyclic AMP would result in the activation of phosphorylase kinase in the synaptic region and phosphorylation of proteins in the plasma membrane[15,16]. Altered membrane permeability may then facilitate synaptic transmission[17], which could be expressed as hyperactivity in the affected animal.



Effect of ingestion of 50 mM rubidium chloride for 1 month on cyclic AMP levels in the rat; * $p < 0.05$.

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Radioprotective effect of a protein free parathyroid extract on the mitotic index of rat bone marrow cells

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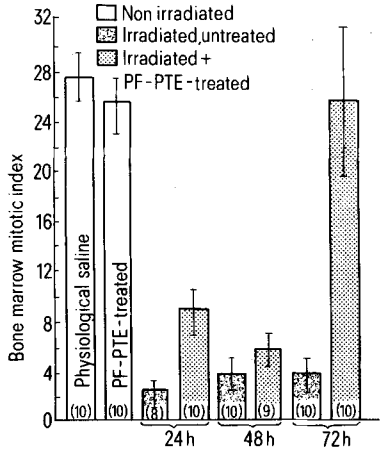
Summary. The protective efficacy of an orally administered bovine protein-free parathyroid extract (PF-PTE) was studied on rat bone marrow cells in vivo with the mitotic index after 850 R irradiation. A remarkable decrease was found in the mitotic activity of bone marrow cells after irradiation in the non-protected animals. However, in the animals treated with PF-PTE after irradiation, a significantly smaller decrease and a faster recovery were found in the mitotic activity of the bone marrow cells.

We have demonstrated recently[1] that a bovine protein-free parathyroid extract (PF-PTE) increased the survival of 850 R and 1000 R whole-body X-ray irradiated rats. It was assumed that the radioprotective effect of the protein-free, and consequently calcium-inactive extract, was mainly due to a hitherto unknown water-soluble bioactive agent. The aim of this study was to investigate the mode of action of this agent. The preparation of PF-PTE was described previously[2]. The experiment was carried out in male CFE rats weighing 150–250 g. The whole-body irradiations were exposed by a Super Liliput 200 X-ray apparatus (Medicor, Budapest) 180 kV, 4 mA, filtered with 0.5 mm Cu, the focus-target distance: 50 cm, with a dose-rate of 7.8 R per min. The mitotic activity of bone marrow was studied after 850 R whole-body irradiation. 1 group of the irradiated animals ($n = 30$) received 0.1 ml physiological saline i.p. as the solvent of PF-PTE, on the day of X-ray exposure and on the 3 successive days thereafter. Another group of

irradiated rats (n=30) was treated with 0.1 mg per 100 g b.wt PF-PTE i.p. also on the day of irradiation, immediately after the exposure, and on the 3 following days. A 3rd group of animals (n=30) without irradiation obtained as above 0.1 ml physiological saline, and a 4th group (n=30) was treated with a daily dose of 0.1 mg per 100 g b.wt PF-PTE according the above-mentioned program. The animals were sacrified 24, 48 and 72 h after the last injection, respectively. The femurs were removed and bone marrow smears were prepared and stained according to Giemsa. The mitotic index was calculated as described by De et al.[3]. Significance was calculated according to Student's t-test. The results the figure, show that the mitotic index in the bone marrow of the non-irradiated physiological saline or PF-PTE treated animals did not differ from each other significantly, either in the 24th, or in the 48th or 72th h sacrificed animals (figure). The mitotic index of the 850 R irradiated bone marrow of rats sacrificed in the 24th, 48th and 72th h was significantly lower ($p < 0.01$) compared to the non-irradiated animals. No difference was found between the 24, 48 and 72 h killed groups. The mitotic index of the irradiated and PF-PTE treated animals was also lower at the 24th h, but this difference was not strongly significant compared to the untreated rats ($p < 0.05$). At the 48th h, the difference between the mitotic activity of irradiated and irradiated plus PF-PTE treated animals was not significant either. However, in the 72th h, a striking difference was found between the irradiated PF-PTE treated and irradiated untreated animals ($p < 0.01$). The bone marrow of the PF-PTE treated irradiated rats shows a normal (same as the non-irradiated physiological saline treated, or only PF-PTE treated) mitotic activity. According to our results described above, the PF-PTE treatment applied immediately after the 850 R whole-body X-ray irradiation seems to reduce the developed bone marrow cell destruction. Furthermore the recovery of the haemopoietic cells is still significant 72 h after the last treatment, compared to the untreated animals where no sign of recovery was observed. It is interesting to note that PF-PTE did not stimulate the mitotic activity of the non-irradiated (intact) bone marrow cells, but only that of cells damaged by irradiation.



Bone marrow mitotic index: number of mitoses per 1000 nucleated cells. Non irradiated groups: Killing 72 h after treatment. Irradiated, untreated groups: 850 R X-ray irradiated, untreated. Irradiated + PF-PTE treated groups: 0.1 mg PF-PTE in 0.1 ml saline per 100 g b.wt injected i.p. immediately after 850 R X-ray exposure and on the three following days, and sacrificed at 24, 48 and 72 h respectively after treatment, $\bar{X} \pm$ SE. (n) = number of animals.

We tried to identify the cell types which have been mostly protected by the PF-PTE treatment. It was observed that after irradiation first the myeloblasts and myelocytes, and thereafter the erythroblasts, disappear. In the phase of recovery during the 72th h after irradiation, maturation of the marrow continues in a great number of myeloblasts, myelocytes and erythroblasts. It was not possible to identify 1 special cell type protected, because the 850 R X-ray exposure destroyed significantly all the mitotic cell types. Mitotic figures are seen in great numbers in the bone marrow during recovery both in the myeloid and erythroid cell lines.

It appears reasonable to conclude that the described prolongation of the survival time after irradiation[1] may be one of the consequences of stimulated haemopoietic first of all of leukopoietic recovery in the bone marrow.

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The number of parallel fibre-Purkinje dendrite synapses. A morphometric evaluation[1]

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Summary. Quantitative parameters concerning synapses were studied in the cerebellar molecular layer of 4 cats using ultrastructural morphometric methods. The number of parallel fibre-Purkinje dendrite synapses was estimated to be about 200,000.

Light and electron microscopic methods were used to evaluate, through indirect procedures, the number of parallel fibre-Purkinje dendrite synapses[3-8]. The morphometric calculation of such, using direct counting of synapses at the ultrastructural level however, has never been reported. In the present study, the number of parallel fibre-Purkinje dendrite synapses of the cat were calculated using an ultrastructural morphometric method.

Material and methods. Observations were made on the molecular layer of the cerebellar cortex of 4 adult cats *(Felis domestica)*. Tissue fragments of the cerebellar vermis, Larsell lobules 4-6[9], were obtained under ether anaesthesia. Blocks were fixed according to the Kanaseki and Kadota[10] method; details of the procedure have been described in a previous study[11].

5 tissue blocks from each cat were selected at random. A silver ultra-thin section of molecular layer chosen at random from each block was stained with uranyl acetate and lead citrate and used to calculate, at a final magnification of × 18,000, the synaptic surface per unit volume of molecular layer (S_v) and the average length of synaptic contacts (L)[12,13]; 20 microphotographs and approximately