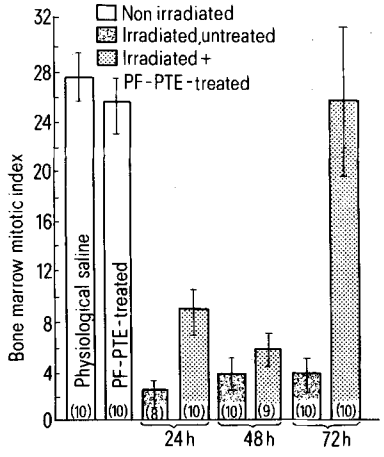
irradiated rats (n=30) was treated with 0.1 mg per 100 g b.wt PF-PTE i.p. also on the day of irradiation, immediately after the exposure, and on the 3 following days. A 3rd group of animals (n=30) without irradiation obtained as above 0.1 ml physiological saline, and a 4th group (n=30) was treated with 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

290 synapses were counted in each cat. Details of the procedure have been previously described[14]. No corrections were made for shrinkage and Holmes effects.

In order to calculate the number of synapses per unit volume of molecular layer (N_v) in the absence of an accurate determination for form and size distribution of synapses, we considered that the synaptic contacts could be compared to flat circular surfaces[13]. This assumption renders the use of the average length of the synaptic contacts in the calculation of the area of the referred synaptic circles ($A=\pi(0.5\ L_{sin})^2$) possible. Serial sections were used to measure the greatest tangential diameter of 30 synapses at a final magnification of $\times 60{,}000$. The value thus obtained was compared with the average length of synaptic contacts, calculated according to the method of Vrensen and De Groot[13], in order to evaluate the underestimation caused by this method.

Assuming that there is no overlap between adjoining dendritic trees[5,8,15], multiplication of synaptic surface and number of synapses per unit volume of molecular layer by the volume of molecular layer occupied by each dendritic tree (540,000 μm^3, according to Eccles et al.[5]), gives the synaptic surface and the number of molecular layer synapses per Purkinje cell (S_p and N_p, respectively).

Results and discussion. The results obtained are summarized in the table. The average length ($\pm$SD) of synapses was 0.316 ± 0.150 µm (1153 synapses were counted in the 4 cats). In order to calculate the number of parallel fibre-Purkinje dendrite synapses from the number of molecular layer synapses per Purkinje cell that we obtained (table), 2 kinds of corrections appeared necessary. 1. We used an underestimated value for the synaptic circle; from the value that we obtained in serial tangential sections (0.397 ± 0.062 µm), we estimated such error to be about 37%. 2. It must be considered that we counted all the synapses in the molecular layer, thus including other than parallel fibre-Purkinje dendrite synapses. According to Palkovits et al.[6], this procedure gives an error of about 6%. The number of cat parallel fibre-Purkinje dendrite synapses is slightly over 200,000 (202,000), when those corrections are made. This number is in keeping with Eccles et al.[5] who state that 209,000 parallel fibres cross and establish synaptic contacts with each cat Purkinje cell dendritic tree, and is much greater than that found by Palkovits et al.[6], who estimated that only 80,000 of the 400,000 parallel fibres which cross each cat Purkinje cell dendritic tree establish synaptic contacts with dendritic spines.

Our results support Palay and Chan-Palay[8] criticism regarding the accuracy of techniques currently employed in calculating the number of cerebellar synapses, and point to the usefulness of morphometric methods for such calculations. In fact, ultrastructural morphometry not only facilitates direct counting of synapses, but also allows the evaluation of their number per unit volume of tissue.

Ultrastructural morphometric results of molecular layer synaptic contacts

Synaptic surface per unit volume of molecular layer* S_v ($\mu m^2/1000\ \mu m^3$)	Synapses per unit volume of molecular layer* N_v (No./$1000\ \mu m^3$)	Synaptic surface per Purkinje cell** S_P (μm^2)	No. of synapses per Purkinje cell** N_P
49.2 ± 4.7	628 ± 60	26,568	339,120

* Results are expressed in mean$\pm$SE (80 micrographs were analyzed in 4 cats). ** The volume of cat Purkinje cell dendritic tree was assumed to be 540,000 μm^3, according to the values reported by Eccles et al.[5].

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Thoughts about high chlorine peaks in X-ray microanalysis

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Summary. In X-ray microanalysis of biological tissues, high Cl-peaks are constantly observable. Higher Cl-peaks in the tissue than in the epon environment suggest that most Cl originates from the tissue. Therefore rough regional estimations of the Cl level could be possible.

The amazingly high level of the very unstable element chlorine (Cl) found by X-ray microanalysis is well known and has been mentioned in most papers dealing with this subject. Not only conventionally prepared tissues for morphological investigations in the transmission electron microscope - having the chance to acquire their Cl from the epon impregnation - show abundant Cl, but also other methods like freeze-drying, for example, yields high Cl-peaks.

Our results too have always provided evidence for a 4-8-fold higher yield of Cl compared with the other elements analyzed in epon-embedded vascular tissues from different organs[1]. Comparing control animals with rats, exposed to experimental metabolic disorder by overdoses of sodium