occurrence of lymphosarcoma (13 of 16). Two separate control series were also run, involving the implantation of *R. pipiens* kidney into the right forelimbs of *T. viridescens* from uninfected populations, but no lymphosarcomas were obtained.

We consider these results to support the view that we have expressed previously[8], namely, that an individuation field, as represented by a regeneration-competent amphibian limb, is not capable of controlling cancer developing within it, if the cells forming the cancer do not possess limb tissue-forming capacities. That circulating and wandering cells find themselves within the limb does not necessarily mean that they are subject to control by the individuation field[12].

Résumé. Du rein de *Rana pipiens* adultes a été transplanté dans les membres antérieurs de vingt *Triturus viridescens* qui étaient en train de développer des lymphosarcomes. Parmi les 12 pattes analysées, six étaient infiltrées par le cancer et avaient répondu à la présence des greffes xénogéniques en formant des structures accessoires de la patte. Nous en avons conclu que le champ de régénération n'est pas capable de contrôler le développement de ce cancer.

L. N. Ruben, M. Balls, and J. Stevens

The Reed Institute, Portland (Oregon, USA), January 14, 1966.

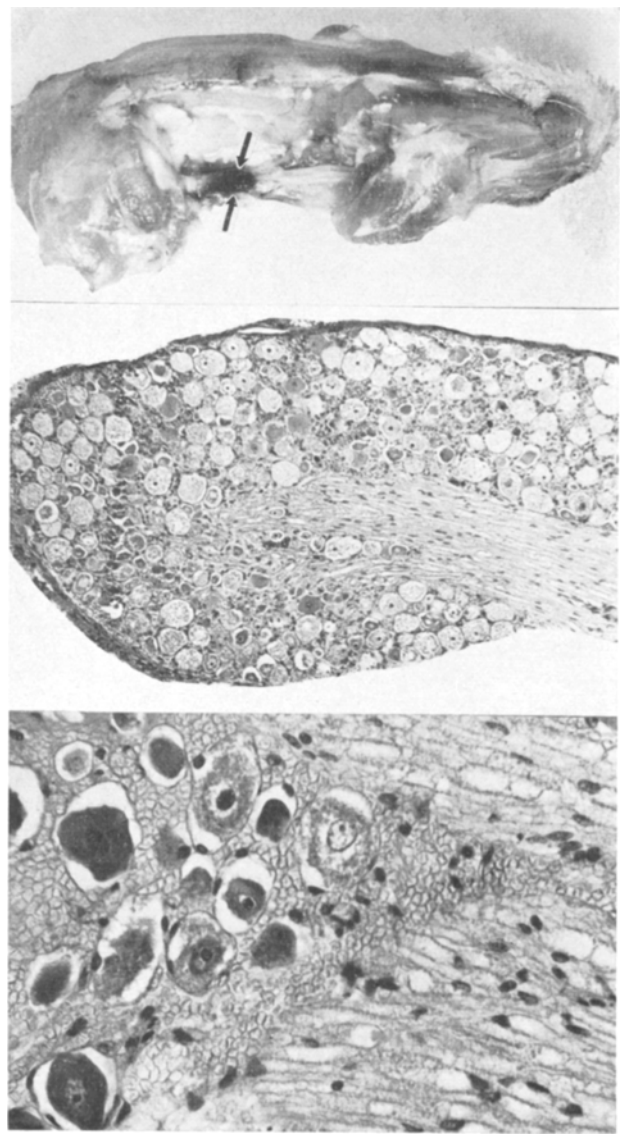
[12] This investigation has been aided by grants from the National Cancer Institute, National Institutes of Health, Bethesda, Maryland, USA (No. CAO-8268) and the Jane Coffin Childs Memorial Fund for Medical Research.

Action of Cadmium Chloride on Sensory Ganglia[1]

Cadmium is known as a highly toxic metal which is employed for several industrial purposes. While studying the pharmacology of certain metallic compounds, we noted that, in the rat, the subcutaneous or intravenous administration of cadmium chloride produced severe lesions of the Gasserian ganglion and of the spinal sensory ganglia. Here we would like to report the work performed with a view to investigating this phenomenon more thoroughly.

In the first experiment 80 Sprague-Dawley rats (40 males and 40 females) with a mean body weight of 189 g (range 180–198 g) were divided into 4 equal groups and given different amounts of cadmium chloride ($CdCl_2$, Fisher Scientific Co., Fairlawn, N.Y., U.S.A.) subcutaneously in the back, always in 1 ml of water at the doses indicated in the Table. All animals were killed three days after the injections. For the second experiment, we injected 25 female rats of 190 g (range 182–201 g) with 3.5 mg of cadmium chloride in 1 ml of water subcutaneously in the back and killed 5 animals 30 min, 1, 3, 5, and 24 h respectively after the injections to study the chronological development of the lesion.

As can be seen in the Table, ganglionic lesions were found in all the groups treated with the different doses of cadmium chloride. The percentage of incidence was similar in males and females. Macroscopically, the first signs were observed 5 h after the injection and consisted of hemorrhagic spots under the capsule; 24 h later the affected organs appeared dark red because of massive hemorrhages sharply localized in the ganglionic tissue (Figure). Oc-



Hemorrhagic necrosis of sensory ganglia induced by cadmium chloride. Top: Macroscopic aspect of the Gasserian ganglion. The necrosis is sharply limited to the ganglionic tissue (arrows). Middle: Necrosis of the nerve cells in a spinal ganglion (Susa, PAS, × 120). Bottom: Pycnosis of nuclei and lysis of cytoplasm in the Gasserian ganglion cells which appear surrounded by hemorrhages (Susa, PAS, × 460).

[1] This work was supported by the John A. Hartford Foundation and the Medical Research Council of Canada.

casionally, however, hemorrhagic spots were observed in the peripheral nerves especially in the sciatic.

Histologically, the most striking change was in the ganglion cells which showed pycnosis of nuclei, karyorrhexis and subsequent lysis of the cytoplasm. Hemorrhagic suffusions were characteristically located around the damaged ganglion cells (Figure) and a small degree of leucocyte infiltration was also noted. Sympathetic ganglia appeared normal. Other morphological lesions present in the same animals were located respectively at the site of the subcutaneous injection, in the liver, kidney, heart, testes and ovaries.

Preliminary results indicate that the same ganglion lesion can be reproduced in the guinea-pig and the hamster. In similar experiments performed in the rat with the chlorides of mercury, thallium, lead and indium, the ganglionic changes described above were not observed.

Morphological lesions produced by cadmium salts have been described in organs such as the liver, kidney[2] and testes[3]. At present, in relation to our work we have found in the literature only one reference concerning the action of this ion on nervous conduction[4].

Cadmium is known as a strong inhibitor of sulfhydryl enzymes[5] and several symptoms of poisoning are consistent with the theory that its toxic effects are due to this property; it seems difficult, however, to explain the selective effect on nervous ganglia on this basis and further studies will be needed to clarify the mechanism of this toxic action.

Action of $CdCl_2$ on sensory ganglia

Group	Dose of $CdCl_2$ (mg)	% of animals with ganglion necrosis	% mortality
1	5.5	100	100
2	3.5	100	100
3	2.0	100	0
4	0.5	65	0

Résumé. Le chlorure de cadmium produit chez le rat, une nécrose hémorragique sélective du ganglion de Gasser et des ganglions sensitifs spinaux. Dans ces organes, on observe une pycnose des noyaux et une lyse du cytoplasme des cellules nerveuses qui sont entourées d'effusions hémorragiques.

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PRO EXPERIMENTIS

Infrared Emission Spectra of in vivo Human Skin

The recent observation of IR-emission spectra of various solid surfaces[1] suggested the extension of the emission techniques to the study of biological systems. Exploratory experiments were therefore made to observe the IR-emission spectra of human skin in vivo. As conventional spectrometers were unsuitable, because the amount of radiation emitted at body temperatures was small, an interference spectrometer was used. Such an instrument separates the component frequencies of incident radiation by interference rather than by dispersion. As narrow entrance slits are not required and all of the radiation of interest is incident on the detector simultaneously, the light-gathering power and the signal-to-noise ratio of the interference spectrometer are relatively high. This permits the observation of faint sources.

The Block Model I-4T interferometer spectrometer was used[2]. The instrument and its operation are described elsewhere[3-5]. The detector temperature was about 30 °C. The spectral region from 2000 cm^{-1} to 700 cm^{-1} was scanned repetitiously at a rate of 120 scans per min. The signal of each scan was added digitally and stored in the memory of a Block Coadder, a coherent information adding device. After the accumulation of the individual signals of 360 consecutive scans, the cumulative signal was fed from the Coadder to a wave analyzer coupled with a potentiometer recorder, to result in traces such as those shown in the Figures 1, 2, and 3. The resolution was 20 cm^{-1}, indicated in each Figure at R. The ordinates of relative spectral emission are arbitrary, and have been shifted to avoid overlapping of spectra. All subjects were fair-skinned Caucasians. The aperture of the spectrometer was situated 2–3 cm from the skin of each subject, so that a skin area of about 3 cm diameter was observed.

Plot A of Figure 1 shows the IR-emission of normal skin near the right elbow of a girl, 'as is', i.e. there was no attempt to clean or especially treat the patch of skin in some way. Spectrum B was then recorded after the same area of skin had been swabbed with acetone. Spectrum B of the whitened, fat-free skin is seen to show different detail than spectrum A. Spectra C–G were obtained from one area of skin near the left elbow of a man. Spectrum C was obtained from the untreated skin. A small amount of oleic acid was then applied, and spectrum D was recorded. The area was then swabbed with acetone, and the skin was abraded with sandpaper. Spectrum E of

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