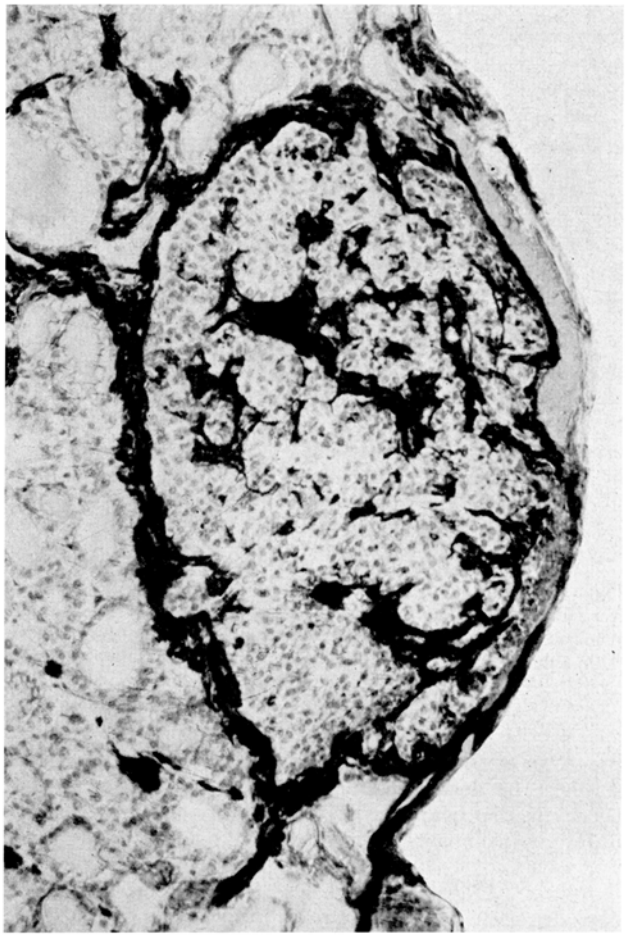

Fig. 2. Histologic aspect of the calcified parathyroid, showing selective deposition of calcium salts in the stroma of the parathyroid, with slight spreading into the adjacent thyroid stroma. The parenchymal cells are unaffected (von Kóssa × 150).

only two parathyroids which are embedded near the posterior margin of the thyroid. Normally, it is difficult to distinguish them from the surrounding thyroid tissue. Neither DHT alone (Group 1) nor the chromium salts alone (Group 2 and 3) altered the appearance of the glands but after treatment with $CrCl_2$ or $CrCl_3$ following DHT sensitization (Group 4 and 5), the parathyroids could easily be distinguished from the surrounding reddish-brown thyroid tissue as brilliant white nodules. On defatted specimens stained with the $AgNO_3$ technique, these parathyroids were selectively blackened, thereby suggesting that the calcium was presumably deposited in the form of silver reducing phosphates (Figure 1). Previous observations had shown that in various organs (e.g. pancreas, salivary glands), calcium deposition induced by calciphylaxis is almost invariably limited to the stroma[4]. This proved to be the case also in the present experiments. The parathyroid parenchyme remained free of calcium but the stroma was heavily impregnated with v. Kóssa positive material which tended to spread even into the stroma of the surrounding thyroid (Figure 2).

In groups 4 and 5, varying degrees of connective tissue calcification were also noted within the salivary and Brunner's glands, pancreas, renal cortex, diaphragma and pulmonary septa. The calcification was most intense and most selectively limited to the parathyroids in group 5. However, this fact is no proof of a fundamental difference in the action of the two chromium salts since $CrCl_3$ makes a perfect solution, while $CrCl_2$ forms a poorly tolerated suspension at the dose level used and hence could not be given at the same dose level as the $CrCl_3$.

Résumé. Une calcification des glandes parathyroïdes a été réalisée chez le rat par sensibilisation avec une dose orale unique de dihydrotachystérol suivie, le lendemain, d'une seule injection intraveineuse de $CrCl_2$ ou $CrCl_3$.

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Free Radicals in Animal Tissues

The recent work of Commoner, Lippincott, and Passoneau[1], on free radicals associated with enzyme systems, and Gordy, Ard, and Shields[2] on irradiation of compounds of biochemical significance, represents two approaches to the analysis of free radicals in biological materials using electron spin resonance techniques.

In order to correlate free radical concentrations with the metabolic activity of tissues under different conditions, we have examined the free radical concentration of various whole tissues. As lyophilization has been shown to cause the production of unpaired electrons[3], liquid nitrogen temperatures were used to trap and stabilize free radicals in tissue samples. The tissues were thus frozen but kept hydrated.

Fresh tissues were removed as quickly as possible from freshly killed Norway Rats, and packed into silica glass tubes with an internal diameter of 4 mm, and immediately cooled to 77 K. Absorption of microwave power was analysed at a carrier wave frequency of 9000 Mc/s and a magnetic field of 3000 Gs. The concentration of free radicals was determined by comparison with the absorption curves obtained from a standard carbon sample. Figure 1 shows the pattern of signals obtained from heart,

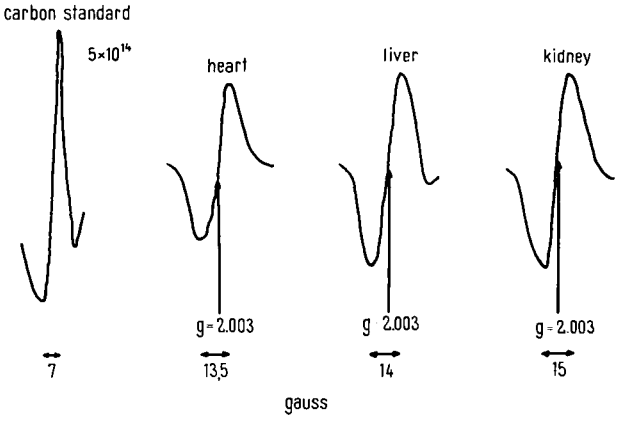

Fig. 1. The signals obtained from a carbon standard (5.1×10^{14}), rat heart muscle; rat liver, and rat kidney.

[1] B. Commoner, B. B. Lippincott, and J. V. Passoneau, Proc. Nat. Acad. Sci. *44*, 1099 (1958).
[2] W. Gordy, W. B. Ard, and H. Shields, Proc. Nat. Acad. Sci. *41*, 983 (1955).
[3] F. K. Truby and J. W. Goldzeiher, Nature *182*, 1371 (1958).

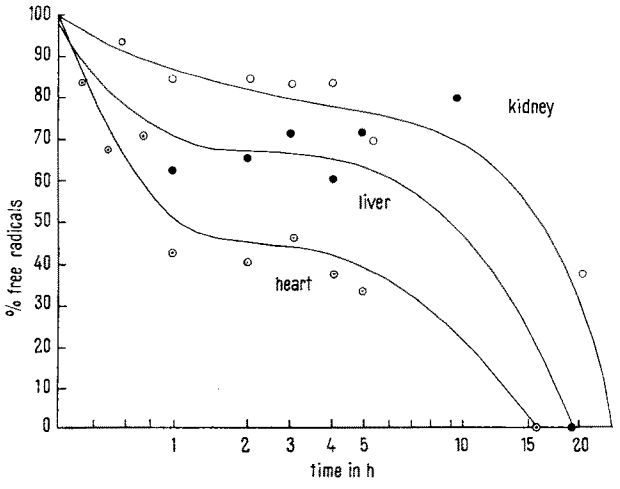

Fig. 2. Decay of free radical concentration at room temperature. The % of free radicals is taken as 100% at the first sample, after rapid removal from the animal. They are then left to decay at room temperature and the % of free radicals determined at specific time intervals.

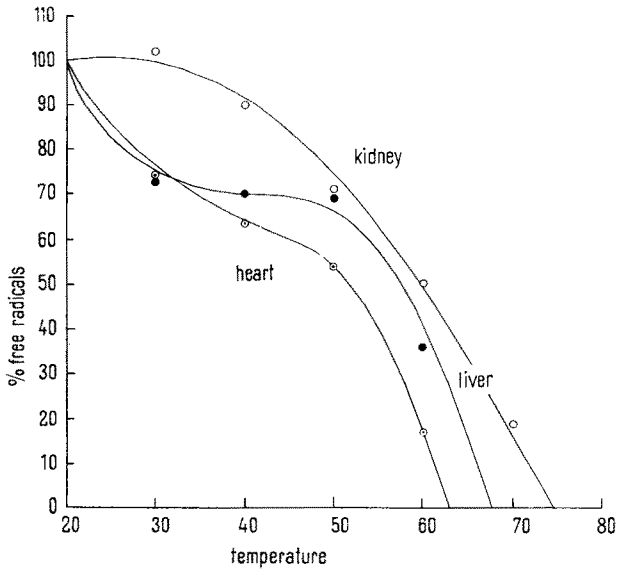

Fig. 3. The effect of temperature on the development and decay of free radicals. The samples are removed from the animal and kept at a specified temperature for 15 min. The concentration of free radicals is then determined, and plotted here with the 20°C taken as 100%.

liver, and kidney, together with a carbon sample containing 5.1×10^{14} free radicals. The signals obtained from the three tissues are clearly much alike. They all have g values of 2003, and d for heart is 13.5, whilst that for liver is 14.0, and kidney 15.0.

The rate of decay of the signals of specimens kept at room temperature (18°C) varied according to the tissue used. The signal from heart decayed rapidly over the first hour after removal from the animal, and thereafter persisted for up to 16 h. Free radicals were detectable for up to 24 h in kidney and 20 h in liver (Figure 2).

In a study of the effect of temperature on the initial free radical concentrations, each tissue was kept at a constant temperature for 15 min and then immersed in liquid nitrogen. There was an optimum free radical production at 20–30°C and the radicals in the kidney persisted up to 75°C. No signal was detectable from heart or liver above 65°C (Figure 3). The effect of temperature on the development and decay of free radical concentrations appears to be characteristic and specific for a given system. Though the shape of the radicals in all three systems appears to be similar, the decay and development rate indicates that those in the heart, liver and kidney are probably in different systems in the tissues[4].

Résumé. L'auteur décrit les radicaux libres présents dans des tissus congélés du rein, du foie et du cœur du rat. Les taux de décomposition et les effets de la température sur la stabilité des indices montrent qu'il y a probablement dans ces tissus plusieurs systèmes de radicaux libres.

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[4] We are indebted to the Medical Research Council for financial aid in this research.

Cysteamin-Sauerstoff-Antagonismus bei der Strahlenwirkung auf T2-Bakteriophagen

Obwohl in den letzten Jahren zahlreiche Untersuchungen über die Schutzwirkung von Cystein[1–4] oder Cysteamin[4–6] gegenüber sogenannten direkten Röntgenstrahleneffekten auf suspendierte Bakteriophagen durchgeführt wurden, war bisher keine einheitliche Deutung der Resultate erkennbar. So fanden MARCOVICH[5] und HOWARD-FLANDERS[6] beim Phagen T2 nach Cysteaminzugabe *keinen* Schutzeffekt, wenn Sauerstoff während der Bestrahlung anwesend war. Dies schien im Widerspruch zu den Befunden von HOTZ und MÜLLER[4] zu stehen, die sowohl unter aeroben als auch anaeroben Bedingungen den gleichen Schutzfaktor beobachten konnten. Die hier mitgeteilten Ergebnisse ermöglichen es nun, eine einfache Erklärung für diese nicht ohne weiteres verständlichen Unterschiede zu geben.

Die in früher[7] beschriebener Weise gereinigten und titrierten Konzentrate des Coliphagen T2 (wild) enthielten zwischen 10^{10} und 10^{12} plaquebildende Einheiten pro ml Phagenpuffer. Diese Stammsuspensionen wurden zum

[1] A. H. DOERMANN, zitiert bei J. D. WATSON, J. Bacteriol. *63*, 473 (1952).
[2] H. T. EPSTEIN und D. SCHARDL, Nature *179*, 100 (1957).
[3] G. HOTZ und A. MÜLLER, Z. Naturforschg. *15b*, 450 (1960).
[4] G. HOTZ und A. MÜLLER, Z. Naturforschg. *16b*, 282 (1961).
[5] H. MARCOVICH, Radiation Res. *9*, 149 (1958).
[6] P. HOWARD-FLANDERS, Nature *186*, 485 (1960).
[7] A. MÜLLER, G. HOTZ und K. G. ZIMMER, Z. Naturforschg., im Druck.