phenyl β -glucosidase activity of rat liver lysosomes is associated with the membrane fraction. I found this activity was fully available in fresh granules from *A*- or saline-injected rats. Such non-latency would result either if the enzyme was located on the cytoplasmic face of the lysosome membrane or if the lysosome membrane provided no barrier to substrate entry. The latter possibility was excluded by the finding that lysosomes from rats injected with *B* had six times the nitrophenyl β -glucosidase activity of those from saline- or *A*-injected rats, and that the additional activity was 90% latent.

Cellobiase activity, detected only in granules from rats injected with *B*, was almost wholly latent, a result consistent with the ability of cellobiose to afford osmotic protection to rat liver lysosomes (J. B. Lloyd, 1969).

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Lysosomal Uptake of Actinide Elements

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A number of metals are known to concentrate in lysosomes of liver and other cells (Witschi & Aldridge, 1968). Some of these metals are known to produce tumours at various sites and lysosomal involvement in carcinogenesis has been suggested (Allison, 1968). Colloidal plutonium has been shown to concentrate in rat liver lysosomes (Rahman & Lindenbaum, 1964), and this communication reports a similar lysosomal accumulation of plutonium, americium and actinium in rat liver and/or testes after injection of ^{239}Pu , ^{241}Am or ^{227}Ac in soluble form.

Male Marshall-August hybrid rats were injected intravenously with 0.5–2 μC of [^{239}Pu]plutonium citrate, [^{241}Am]americium citrate or [^{227}Ac]actinium nitrate and killed from 1 hr. to 90 days later. Immediately before injection the solutions were filtered through a 10nm.-pore-diameter filter to remove polymeric material (Turner & Taylor, 1968). The livers and testes were homogenized in

0.25M-sucrose and fractionated by differential centrifugation (Worwood & Taylor, 1969), or by centrifugation through a sucrose continuous density gradient.

At 1 hr. after injection most of the plutonium or americium in liver or testes was in the soluble fraction (105000g supernatant), but at longer time-intervals most of the plutonium or americium was in the lysosomes, as shown by the coincidence of the metal and arylsulphatase A and B distribution profiles. Similar results were obtained in more limited studies of actinium distribution in liver.

These results show that lysosomal uptake of plutonium, americium and actinium occurs in liver and/or testes within a relatively short time after injection of the metals in a soluble form. Retention of these elements is prolonged in both tissues and plutonium can induce testicular (Bensted, Taylor & Sowby, 1965) and hepatic tumours (Taylor, Dougherty, Shabestari & Dougherty, 1969).

The mechanisms concerned in the lysosomal uptake of these elements are not understood. However, for plutonium at least, it does not appear to be due entirely to endocytosis of colloidal plutonium formed in the blood or extracellular fluids. Plasma plutonium is bound to transferrin (Turner & Taylor, 1968) and during the first few hours after injection the plutonium in the soluble fraction of liver is bound to a protein of molecular weight about 300000 (Boocock, Popplewell & Taylor, 1968).

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