By using a device which consists of a scintillation probe mounted against a 1-mm. slit in a mass of lead, and by moving the centrifuge tube (25 mm. in diameter) containing the supernatant fluid upwards and downwards, some indication has been found that there are more radioactive myelin forms in the upper than in the lower layers of the supernatant fluids.

The tiny fragments and myelin forms in the supernatant fluids can be demonstrated excellently with the interference microscope (AO-Baker). The refractive index of the myelin forms is about 1·48, which is about the same as that of lipids, and their water content must be small. The measurements, however, are difficult to make because of the smallness of the objects and because they are in active Brownian movement, so these results are given as approximations only.

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Effect of Thyroxine on the Swelling of Mitochondria isolated from Various Tissues of the Rat

SEVERAL recent investigations[1-4] have suggested that an important function of thyroxine may be an 'uncoupling' of oxidative phosphorylation. However, studies with a multi-enzyme system (isolated from digitonin extracts of mitochondria[5]) capable of performing oxidative phosphorylation have indicated[6] that thyroxine and its analogues have no direct effect on this process. Concurrently[7], it has been observed that these compounds alter the morphology of rat liver mitochondria *in vitro*. In concentrations as low as 10^{-6} *M*, both thyroxine and triiodothyronine produce a prompt and marked swelling of the mitochondria. In contrast, 2,4-dinitrophenol, in concentrations which completely 'uncouple' oxidative phosphorylation, provides protection against swelling. It has therefore been postulated[6] that the uncoupling of oxidative phosphorylation produced by thyroxine in intact mitochondria is secondary to an alteration in the morphology of the mitochondria and not the result of a direct interaction of the hormone with the enzymes of oxidative phosphorylation.

Not all tissues respond in the same degree to the *in vivo* administration of thyroxine. Gordon and Heming[8] found that the oxygen consumption of slices of liver, kidney, diaphragm and heart from thyrotoxic rats was significantly greater than normal, while that of slices of spleen, brain and testis was not increased. Gross and Leblond[9] investigated the distribution of injected iodine-131 thyroxine in the rat and found that high concentrations occurred in plasma, liver and kidney and considerably smaller or negligible amounts in all other tissues tested. Likewise, we have found differences among the various tissues in the degree of mitochondrial swelling produced *in vitro* by thyroxine and its analogues.

Table 1 illustrates the markedly different susceptibilities of mitochondria from several organs. Mitochondria were isolated from the liver, kidney, heart, spleen and testis of adult male Wistar rats and the swelling assay conducted spectrophotometrically in a medium containing 0·3 *M* sucrose and 0·02 *M* *tris*-(hydroxymethyl)aminomethane at *p*H 7·4, as previously described[7]. Diaphragm mitochondria were isolated in a similar fashion following homogenization in a Waring blendor. Brain mitochondria were isolated by method I of Brody and Bain[10]. Each value in Table 1 represents the percentage decrease in optical density of a suspension containing the compound tested (3×10^{-5} *M*) less that observed in a control sample without the added compound. These values are taken as a measure of the swelling produced by the compound. The relative effects of thyroxine and triiodothyronine varied with different samples of these substances; one lot of thyroxine was more effective than triiodothyronine and one less so. Table 1 demonstrates that the swelling produced is striking with mitochondria from liver and kidney, while that with mitochondria from diaphragm and heart is much less marked. In accordance with failure of slices of spleen, brain and testis to show any change in oxygen consumption in thyrotoxic rats[8], essentially no swelling was produced by thyroxine or its analogues with the mitochondria from these tissues.

Table 1. DEGREE OF MITOCHONDRIAL SWELLING PRODUCED *in vitro* BY THYROXINE AND VARIOUS ANALOGUES

	L-thyroxine	D-thyroxine	L-triiodothyronine	D-triiodothyronine	Tetraiodothyroacetic acid	Triiodothyroacetic acid
Liver	30	33	47	41	25	17
Kidney	12	10	18	14	20	12
Diaphragm	5	4	7	7	5	5
Heart	2	2	9	5	9	9
Spleen	0	0	0	0	0	0
Brain	4	1	1	0	4	2
Testis	1	2	2	2	4	3

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Amœbicidal Action of Azaserine

IN 1954, a group of workers reported on a new tumour-inhibitory substance[1]. This substance, called azaserine, also possessed antibacterial activity against numerous microbial species[2]. Chemically, this substance is O-diazoacetyl-L-serine[3]. It has been reported that azaserine is a profound inhibitor of formate incorporation into the nucleic acids[4]. Since the role