deposition with oral and intraperitoneal DTPA treatment were observed by Smith[4]; however, plutonium in liver was not removed to the extent observed in this investigation.

The 6 m*M*/kgm. oral dosage of TTHA was somewhat more toxic than DTPA and resulted in severe diarrhœa and the death of one animal during the 4-day course of the experiment. At a higher dosage of 7·5 m*M*/kgm. severe diarrhœa was observed with both chelating agents and 50 per cent mortality occurred in the TTHA animals by the fourth day. Toxicity was not grossly apparent at the lower oral dosage of 2·8 m*M*/kgm. It seems quite probable that slight alterations in the manner of administration of the TTHA might significantly reduce its toxicity.

While not strikingly superior to DTPA when administered intraperitoneally, TTHA would appear to offer substantial advantages as an orally effective agent. It is, in fact, the first substance to show promise of a practical degree of oral effectiveness in removing plutonium. Further studies are planned to more clearly define the limits of toxicity and therapeutic effectiveness as a function of dosage-level.

This work was performed under contract No. *AT*(45-1)-1350 between the Atomic Energy Commission and the General Electric Company.

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Alkoxyglycerols in Irradiation Treatment

It has previously been shown that the alkoxyglycerol esters to a certain extent prevent leuco- and thrombo-cytopenia[1]. During 1955–56 about 300 patients, suffering from cancer of the uterine cervix, were given alkoxyglycerol esters for the whole period of treatment with radium and X-rays. The white cell and the thrombocyte counts were higher for the 'prophylactic group', that is, patients treated with alkoxyglycerol esters, than for the controls, that is, the group treated with irradiation only, both during the treatment and after its termination.

Further experiments[2] on irradiated rats have shown that the alkoxyglycerols and their esters prohibit to some extent the decrease both of megakaryocytes and of nucleated cells in the bone marrow. The alkoxyglycerols thus seem to act as a protective agent of the bone marrow.

Finally[3], it was found that the alkoxyglycerols promote the growth of rats and that they act as growth-stimulating substances for *Lactobacilli*.

For the major group of patients suffering from cancer of the uterine cervix, to which alkoxyglycerols were administered during the radiation therapy, 5–6 years have now elapsed. Thus it is possible to make a comparison between the times of survival for the group treated with alkoxyglycerols and for the group treated with radiation only.

Table 1 gives numbers of 1- and 5-year survivals. The patients are grouped in stages I–IV, the most severe one being IV. The time of survival for the control group agrees with the results quoted in literature[4]. Table 1 shows that particularly for stage II the time of survival is markedly longer for the treated group than for the controls.

Table 1. Patients suffering from Cancer of the Uterine Cervix treated in 1955 and 1956 (Alkoxyglycerol Esters administered to 'Prophylactic Group')

		Total patients	1-yr. survivals No.	1-yr. survivals Per cent	5-yr. survivals No.	5-yr. survivals Per cent
Stage I	Prophylactic group	66	64	97·0	59	89·4
	Control group	120	118	98·3	100	83·3
Stage II*A*	Prophylactic group	88	83	94·3	66	75·0
	Control group	132	123	93·2	79	59·8
Stage II*B*	Prophylactic group	85	72	84·7	49	57·6
	Control group	135	111	82·2	56	41·5
Stages II*A* and II*B*	Prophylactic group	173	155	89·6	115	66·5
	Control group	267	234	87·6	135	50·6
Stage III	Prophylactic group	37	27	73·0	9	24·3
	Control group	106	67	63·2	26	24·5
Stage IV	Prophylactic group	8	4	(50·0)	2	(25·0)
	Control group	32	11	34·4	4	12·5

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BIOLOGY

Mechanism of Hair-Growth

Functional activity of the hair follicle is known to be a cyclical phenomenon in many species[1,2], involving periods of active growth and periods of apparent inactivity. The mechanism controlling this cycle is cutaneous, since hair-growth occurs *in vitro*[3–5]. The nature of this control mechanism is unknown. Chase[1,6–8] has suggested the growth-cycle is caused by the rise and fall in the activity of an 'inhibitor'. This proposal, however, contains certain limitations. Although Chase claims his hypothesis does not exclude the participation of a 'stimulator', its presence is an essential premise of his argument. The wane of 'inhibitor' activity would, in itself, be incapable of initiating a growth-cycle, for within the follicle there must exist a propensity to grow. Presumably this is manifested when the activity of the hypothetical 'inhibitor' declines to a threshold-level. This growth propensity necessarily requires a cause, in effect the action of a 'stimulator'.

A cyclical change in 'stimulator' activity alone could cause the growth-cycle. According to this scheme, anagen would commence above a threshold of 'stimulator' activity and terminate when activity had sufficiently declined. 'Stimulator' activity may decline without involving the action of an 'inhibitor', as by depletion through utilization, or loss in the blood. This is a simpler hypothesis than those also involving 'inhibitors'; it is therefore more desirable at the present state of knowledge.

During the life of an animal a follicle may grow many hairs. Cyclical changes in 'stimulator' or 'inhibitor' activity are inadequate to explain the occurrence of a sequence of growth-cycles, for the wax and wane of such hair-growth factors are them-