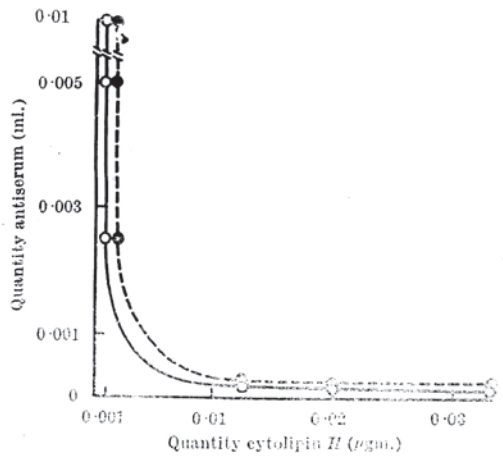

Fig. 1. Isofixation curves determined with cytolipin *H* and antisera to cervix carcinoma (broken line) and reticulum cell sarcoma (solid line). Three 50 per cent hæmolytic units (0·00375 ml. of guinea pig serum); 2 hr. incubation at 20° C. (ref. 9)

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Effect of Certain Polyphenylated Aliphatic Hydrocarbons on the Phagocytic Activity of the Reticulo-Endothelial System

In a recent communication to *Nature*, Nicol *et al.*[1] reported the effect of various stilbene compounds on the phagocytic activity of the reticulo-endothelial system, intimating that stimulation was associated with certain aspects in the chemical structure, and that the strongest stimulants possessed high œstrogenicity. The present communication reports the effect on reticulo-endothelial phagocytic activity of some polyphenylated aliphatic hydrocarbons related to the synthetic œstrogens of the diphenylmethane series.

The present experiments were carried out on male white mice (*T.O.* Swiss strain) of 20–30 g body-weight. Five of the animals were used for assessing the effect of each compound on the phagocytic activity of the reticulo-endothelial system, each animal receiving one subcutaneous injection of 0·5 mgm. of each substance in 0·05 ml. of arachis oil daily for six days. The phagocytic activity of the reticulo-endothelial system was measured on the eighth day, as described in the previous communication[1].

Twenty-five animals were used as controls. Each received one subcutaneous injection of 0·05 ml. arachis oil daily for six days and then showed an average phagocytic index or *K* value of 13 ± 2·4.

Table 1. Effect of Certain Polyphenylated Aliphatic Hydrocarbons, containing at least one *para*-Hydroxyl Group, on the Phagocytic Activity of the Reticulo-Endothelial System

	Compound used	Phagocytic index (*K* value)	Œstrogenic activity (I.U. (mgm.)
A	3 : 3-Di-(*p*-Hydroxyphenyl)-*n*-pentane	16 ± 2·2	5
B	3-(*p*-Hydroxyphenyl)-3-phenyl-*n*-pentane	11 ± 1·1	16
C	*p*-Hydroxyphenyldiphenylmethane	13 ± 1·7	25
D	1-(*p*-Hydroxyphenyl)-1 : 1-diphenylpropane	10 ± 0·6	100 (inactive)
E	*p*-Hydroxyphenyltriphenylmethane	8 ± 1·6	100 (inactive)
	Control values for 25 animals	13 ± 2·4	

Table 1 shows the relative *K* values of five polyphenylated aliphatic hydrocarbons investigated together with their known levels of œstrogenicity. The results indicate that compound *E* has a profound depressant effect on phagocytosis only hitherto found following the administration of cortisone[2] in which the phagocytic index was reduced to $K = 7$. Compound *E* is the only non-steroid found by us to cause such marked depression of phagocytic activity. Compounds *B* and *D* show only a minor depressant action; compound *C* causes no alteration; while compound *A*, which is œstrogenic in the rat following doses of 5 mgm., has a slight stimulatory effect.

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Species Difference in Pyridine Nucleotide Synthesis by Erythrocytes

Leder and Handler[1] have described the synthesis of pyridine nucleotide from nicotinamide and glucose by human erythrocyte homolysates in amounts as much as ten times the normal content and they identified the synthesized material as nicotinamide mononucleotide. These authors also suggested that fructose diphosphate and adenosine triphosphate

Table 1

Species	No. of observations	γ-Pyridine nucleotide (as diphosphopyridine nucleotide per c.c.): whole blood	γ-Pyridine nucleotide (as diphosphopyridine nucleotide per c.c.): erythrocytes	Synthesis of pyridine nucleotide by erythrocytes from nicotinamide + glucose (γ-pyridine nucleotide formed/c.c. red blood cells)	γ Ribose/c.c. of whole blood	γ Ribose synthesized/c.c. red blood cells/hr.
[illegible]	8	30·6	62·5	314·7	—	—
[illegible]key	6	63·1	145·0	nil	—	—
[illegible]	6	94·2	205·0	nil	186·0	82·3
[illegible]ise	2	194·1	407·5	nil	—	—
[illegible]inea pig	6	37·8	77·1	346·9	147·2	75·3
[illegible]bbit	2	59·5	131·8	nil	—	—
[illegible]eep	3	—	192·1	nil	—	—

[illegible]e required for optimal synthesis of nicotinamide [illegible]onucleotide. While synthesis of pyridine nucleo-[illegible]is thus known to occur *in vitro* by washed [illegible]an erythrocytes, its presence and its synthesis [illegible]other mammalian species have not yet been [illegible]mined.

[illegible]n our recent studies on the metabolism of pyridine [illegible]leotide it was observed that the washed red blood [illegible]s of the rat were unable to synthesize that sub-[illegible]ce under conditions similar to those employed by [illegible]er and Handler[2] in human erythrocyte studies. [illegible]s finding induced us to examine further the be-[illegible]iour of different species of animals, namely, mouse, guinea pig, rabbit, sheep, monkey and man, as [illegible]rds synthesis of pyridine nucleotide by erythro-[illegible]es.

[illegible]he total pyridine nucleotide content of the whole [illegible]od was estimated by the method of Levitas *et al.*[3]. [illegible] capacity of fresh erythrocytes, washed four times [illegible]3° C. with four volumes of glucose–Ringer phos-[illegible]te, *p*H 7·2, to synthesize nicotinamide mono-[illegible]leotide was estimated by the method of Leder [illegible] Handler[2]. In some experiments with rats and [illegible]e, the incubation mixture contained fructose-[illegible]-diphosphate, adenosine triphosphate and D-ribose, [illegible]ddition to nicotinamide and glucose.

[illegible]he results presented in Table 1 demonstrate that, [illegible]he seven species studied, only man and guinea [illegible]erythrocytes were capable of synthesizing pyridine [illegible]leotide under the conditions of the experiment. [illegible]wever, all the species that were unable to syn-[illegible]size it had a higher pyridine nucleotide content [illegible]blood than guinea pig or man. This suggests [illegible]t the major site for synthesis of nicotinamide [illegible]nonucleotide in these species may be a tissue [illegible]er than blood.

[illegible]lthough it is uncertain whether the formation of [illegible]otinamide mononucleotide in blood cells occurs [illegible]the direct phosphorylation of nicotinamide riboside [illegible]adenosine triphosphate as shown in liver prepara-[illegible]s by Rowen and Kornberg[4], or by the reaction [illegible]ween the base and ribose-5-phosphate or 5-phos-[illegible]ribosyl-1-pyrophosphate[5], it is reasonable to [illegible]me that the conversion of glucose-6-phosphate [illegible]ibose phosphate by way of a hexose monophosphate [illegible]nt may be an essential step in the synthesis of [illegible]otinamide mononucleotide from nicotinamide and [illegible]cose. My results demonstrate that the formation [illegible]ribose phosphate from glucose-6-phosphate in rat [illegible]l guinea pig erythrocytes, as well as the ribose [illegible]tent of the whole blood of these two species, [illegible]e almost equal. It was evident, therefore, that the [illegible]se phosphate did not seem to be a limiting [illegible]tor in synthesis of pyridine nucleotide in rat [illegible]throcytes. It is hoped that experiments with [illegible]throcyte homolysates and liver preparations now [illegible]progress may provide some information regarding [illegible]comparative role of these tissues in synthesis of pyridine nucleotide in different species of animals.

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Investigations into Temperature Changes on the Surface of Human Tooth Enamel

In one of the first observations made with the aid of the microscope, Leeuwenhoek[1,2] found that he could detect fewer organisms in scrapings taken from teeth after drinking hot fluids than in scrapings taken before the drink. Although many investigations have been made into the physical effect of thermal changes upon enamel and into the effects of conducted heat upon the dental pulp, there is no evidence in the literature of Leeuwenhoek's observation having been pursued.

In the present series of experiments, investigations are being carried out from the aspect of the effects of temperature changes upon chemical or bacterial activity taking place upon the surfaces of human teeth. To ascertain the range of such changes on enamel surfaces during meals, pilot experiments were performed using copper-constantan thermocouples attached to the surface of enamel and in areas between teeth. Meals were eaten at a serving temperature of 60–70° C., during which thermocouple readings were taken every 30 sec.

It was found that maximum temperatures of 50–60° C. were reached and temperatures of 40–45° C. were maintained for up to ten minutes at a time. In areas between the teeth, the temperature on the enamel surface took, on average, thirty minutes after the finish of the meal to return to 37° C.

When measuring surface temperatures it is necessary to know the thermal conductivity of the material the surface of which is under examination, and since only a rough estimate of the thermal conductivity of enamel has been made[3] it was thought that as a preliminary investigation the thermal conductivity of enamel should be determined. The apparatus in use for this is a modification of that used by Phillips, Johnson and Phillips[4] for the determination of the thermal conductivity of dentine and dental cements. Results indicate that the value of the thermal conductivity of enamel is of the order 0·002 c.g.s. units, and that variations occur with the age of the