nutrient agar, suspended in saline, washed repeatedly, autoclaved 40 minutes at 125°C., and added to the basal diet for assay as described elsewhere.[3]

The results given in Table I demonstrate that an antihemorrhagic factor is present in certain species of bacteria. It is evident that this factor is a product of the bacterial metabolism.

Dried bacteria of some species show from 5 to 8 times the antihemorrhagic activity of dried alfalfa. In contrast, preparations of *Pseudomonas aeruginosa* had no demonstrable activity, and other species of bacteria may also prove to be poor sources. Yeast is another microörganism which contains little or none of the vitamin.

The antihemorrhagic factor from bacteria is also extractable by fat-solvents, but its further similarity to the vitamin-K from alfalfa[4] remains to be established. The distribution of this factor, the manner of its production, and its significance in bacterial metabolism are matters for further investigation.

9845 P

Effect of Pregnancy on the Growth of Rat Sarcoma.*

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In previous publications we reviewed the influence of pregnancy on tumor growth (Emge,[1] Emge and Wulff[2]) and showed that in transplantable mammary rat adenofibroma, fibroma, and sarcoma developed in our laboratory the growth rate either was not influenced or was at times only slightly retarded. From a more recent study[3] we learned that rapidly repeated pregnancy does not affect the adenomatous component of our transplantable mammary rat adenofibroma 5-B 1 beyond that expected in a single pregnancy,

[4] Almquist, H. J., *J. Biol. Chem.*, 1937, **117**, 517; 1937, **120**, 635.

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[1] Emge, L. A., *Am. J. Obst. and Gynec.*, 1934, **28**, 682.

[2] Emge, L. A., and Wulff, L. M. R., *Western J. Surg. Obst. Gynec.*, 1934, **42**, 45.

[3] Emge, L. A., and Murphy, K. M., PROC. SOC. EXP. BIOL. AND MED., 1938, **37**, 620.

and that wide variations in the daily weight gain of these tumors during pregnancy are not unlike those occurring in control animals.

In order to obtain further information on the influence of pregnancy on tumor types developed by us we undertook a study of the behavior of certain strains of sarcoma in relation to the number of days exposed to pregnancy.

Of several hundred mature animals implanted with rat sarcoma E, developed by us from transplantable mammary adenofibroma, 31 became pregnant during the growth of the original implant and 48 during the growth of a recurrent tumor. The growth rate was studied in relation to the percentage of time that each animal was pregnant.

TABLE I.

	Cases	Mean daily rate of growth in gm./day
Original implants	31	0.95
First recurrences	48	1.29
	79	Diff. 0.34

Since there are approximately 25 chances in 100 that such a difference of ±0.34 gm./day would occur simply as a result of sampling error, it was considered proper to combine both categories into a single group.

Pearson's coefficient of correlation (r) is −0.1889,† suggesting an inverse correlation between the growth rate and the percentage of time pregnant. The negative correlation, however, may be due to the use of ratios. The standard error of r (on the assumption that its true value is zero) is $1/\sqrt{N-1} = 1/8.8318 = 0.1132$. Thus the relative deviate (x/σ) equals 1.6687.

The probability that an absolute value of r as great or greater than that obtained would arise from a sample of this size by chance alone is 0.095, or roughly, 1 in 10, which is statistically of no significance. Therefore, there is no reason to believe that the growth rate of this rat sarcoma is influenced by pregnancy.

In 2 additional experiments we studied the growth rate of implanted mammary rat sarcomas‡ E-2 and E-5 during pregnancy and during a similar period of recurrence after littering.

Implant with Sarcoma E-2. Eighteen pregnant animals and 9 female controls 90 to 103 days of age were implanted. In the preg-

† The calculations were made on ungrouped data.

‡ It is emphasized that the sarcoma strains used in these experiments are morphologically similar and derived from the same stem.

nant group tumors were removed on the day of littering (from the 17th to the 20th day of tumor growth). In the control group all tumors were removed 20 days after implant. Because of the relatively faster growth of the recurrent tumors in the puerperal group (although this difference was later found to be of no significance), they were removed 13 to 16 days after littering. The experimental results with the calculated value of P§ are shown in Table II.

TABLE II.

	Cases	Av. gain in gm./day: Original Implant	Av. gain in gm./day: Recurrent Tumor	Diff.	P§
Pregnant	18	1.79	2.06	.27	.245
Control	9	2.44	2.58	.14	.788
Diff.		.65	.52		
P		<.01	.302		

Implant with Sarcoma E-5. Six pregnant animals and 5 female controls 107 to 120 days of age were implanted. Tumors were removed immediately after littering and on the 21st day following implantation, respectively. Recurrent tumors were removed at the end of a period equalling the growth period of the original tumor. The experimental results and P values are shown in Table III.

TABLE III.

	Cases	Av. gain in gm./day: Original Implant	Av. gain in gm./day: Recurrent Tumor	Diff.	P
Pregnant	6	.95	1.50	.55	.113
Control	5	1.69	1.69	0	1.000
Diff.		.74	.19		
P		.029	.731		

The results are comparable with those of the previous experiment.

Conclusions. 1. In the 3 types of sarcomas, E, E-2, and E-5, studied here, no significant difference was observed between the growth rate of the original implant and the recurrent tumor. This applies to controls as well as to pregnant animals. 2. The slight correlation existing between the growth rate of sarcoma E and the percentage of time the tumor was acted upon by pregnancy is not

§ P = probability of a difference as great or greater than that obtained occurring by chance alone. By accepted standards, values of .05 or less (*i. e.*, 5 per hundred or under) are considered significant.

significant. 3. The original implants of sarcomas E-2 and E-5, when implanted into pregnant animals, showed a significantly lower growth rate than those implanted into controls. No significant differences were observed, however, in the recurrent tumors.

9846 P

Phosphorus Fractions in Human Heart Muscle.

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In pursuance of our studies of the myocardial chemical changes that accompany congestive heart failure, we have attempted to determine the various phosphorus compounds in the left ventricles of hearts obtained at autopsy. These have been divided into two groups, those with and those without myocardial failure. It is well known that many hearts that have failed give no hint of their lack of functional capacity when examined histologically and previous studies[1] indicate that changes in the chemical compounds participating in the energy exchanges of muscular contraction accompany, and may play a causative rôle in the precipitation of congestive failure.

The total myocardial phosphorus is composed of that present in those compounds which are soluble in 5% trichloracetic acid, namely, phosphocreatine, hexose phosphates, nucleotid phosphoric acid, and inorganic or orthophosphate, plus that residual portion which is not acid-soluble. The residual phosphorus has been identified with the phospholipid fraction by Sorg[2] and Wassermeyer.[3]

It has been previously shown that a decrease in total phosphorus accompanies myocardial failure.[4] The hypothesis has been advanced that the endurance of a muscle parallels its phospholipid content,[5] and in agreement with this idea, Kutchera-Aichenberger[6] reported phospholipid decreases in the heart in congestive failure and in ex-

1 Herrmann, G., and Decherd, G., *Trans. Assn. Am. Phys.*, 1936, **51**, 295.

2 Sorg, K., *Z. f. physiol. Chem.*, 1929, **182**, 97.

3 Wassermeyer, H., *Deut. Arch. klin. Med.*, 1934, **177**, 573.

4 Wilkins, W. E., and Cullen, G. E., *J. Clin. Invest.*, 1933, **12**, 1063.

5 Lehnartz, E., *Erg. Physiol.*, 1933, **35**, 874.

6 Kutchera-Aichenberger, H., *Wien. Arch. f. inn. Med.*, 1929, **18**, 209.