do not show the same behavior in their ability to form lactic acid.[2] As a method of approach to the study of this difference and its possible regulation by the nucleotide level of the tissue, a study of the quantitative distribution of nucleotide nitrogen among the various tissues has been carried out.

TABLE I.

Tissue	Nucleotide Nitrogen per 100 gm. Tissue			
	Dog		Rabbit	
	Arithmetical Mean	Extreme Variation	Arithmetical Mean	Extreme Variation
	mg.	mg.	mg.	mg.
Whole Blood	3.4	3.2- 3.9	6.6	6.4- 6.7
Whole Brain	13.3	12.5-15.5	19.3	17.7-20.7
Intestine	13.3	8.7-18.1	—	—
Pancreas	24.5	19.2-27.7	—	—
Kidney	21.8	17.6-25.5	30.9	28.0-33.6
Spleen	22.6	21.3-24.5	—	—
Liver	27.2	25.8-28.3	47.8	46.2-49.0
Whole Heart	28.9	26.0-31.0	35.0	32.0-37.9
Muscle	50.8	46.5-56.9	60.9	54.1-64.4

Table I contains the results of the analyses of certain tissues of the dog and rabbit for their nucleotide nitrogen content. The quantitative method of Kerr and Blish[3] was used. All results were obtained from duplicate check samples. A minimum of 5 animals was used for each tissue determination.

8311 C

Fibrinolytic Staphylococci.*

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Aoi[1] found that 88% of all Staphylococcus strains isolated from "pusturating foci" are capable of dissolving Congo-red-fibrin. Ap-

[2] Warburg, O., *Biochem. Z.*, 1927, **184**, 484.

[3] Kerr, S. E., and Blish, M. E., *J. Biol. Chem.*, 1932, **98**, 193.

* Work supported in part by the Eli Lilly and Co. Streptococcus Research Fellowship of Stanford University and in part by the Rockefeller Fluid Research Fund of the Stanford Medical School.

[1] Aoi, F., *Kitasato Arch. Exp. Med.*, 1932, **9**, 171.

plying their routine plasma-clot technic, however, Tillett and Garner[2] found only an "occasional" strain that was thrombolytic. Moreover, these few strains were of very low fibrinolytic titer, requiring approximately 24 hours to cause demonstrable softening of the plasma-clot.

We have tried to harmonize the 2 sets of data by titrating 145 local strains of *S. aureus* or *albus*† for fibrinolysin, using the more delicate isolated-fibrin technic.[2] Data thus obtained are summarized in Table I.

TABLE I.

Fibrinolytic Function of Staphylococci.

Serial dilutions of 24-hour broth cultures of each of the 145 strains were added to the routine serum-free human fibrinogen-thrombin complex, volumes, dilutions, temperatures, etc., being identical with those used by Tillett and Garner.[2]. The highest serial dilution liquefying the resulting fibrin-clot by the end of 2 hours, was assumed to contain one lytic unit. From this, the approximate number of lytic units per cc. was calculated.

Cultures giving no lysis by this technic were re-titrated by the 10-fold enzyme-concentration method,[3] this giving the approximate number of decifibrinolytic units per cc.

The table is based on the average of 3 titrations. Each strain is assigned to the nearest recorded serial dilution, strains giving titers from 4 to 7, for example, being recorded under titer 5.

Staphylococcus aureus or *albus* isolated from:	No. and % of lytic and non-lytic strains in each group — Fibrinolytic units per cc. of broth culture							Total No. of strain in each group
	10	5	2.5	1	0.05	0.1	0	
Group A	3	8	5	8	1	2	3	30
Internal human tissues	*37%*		*43%*		*10%*		*10%*	*100%*
Group B	1	4	—	3	4	6	60	78
Superficial human tissues	*6%*		*4%*		*13%*		*77%*	*100%*
Group C	—	—	—	—	—	—	24	24
Veterinary tissues	*0%*		*0%*		*0%*		*100%*	*100%*

The table shows that 77% of all local strains of Staphylococcus isolated from superficial human lesions (acne, boils, nasal sinus, etc.) are without demonstrable fibrinolytic function, even when tested by the 10-fold enzyme-concentration method. In contrast, 90% of all local strains isolated from internal human lesions (septicemia, osteomyelitis, empyemia, cellulitis, etc.) are fibrinolytic, 37% of them yielding as many as 5 to 10 lytic units per cc. of broth culture.

None of the 24 local veterinary strains (horse, cow, dog, swine) is capable of liquifying human fibrin.

[2] Tillett, W. S., and Garner, R. L., *J. Exp. Med.*, 1933, **58**, 485.

† The strains and clinical histories used in this survey were kindly furnished by the various hospitals, clinics, diagnostic laboratories and veterinary institutions of the San Francisco Bay Region.

[3] Madison, R. R., PROC. SOC. EXP. BIOL. AND MED., 1934, **32**, 445.

The fibrinolytic factor formed or secreted by the Staphylococcus is not of the same immunologic specificity as the streptofibrinolysin. Streptococcus antiserum‡ containing as many as 1000 antifibrinolytic units per cc. does not neutralize the staphylofibrinolysin.

8312 C

Survival of Two Depancreatized Dogs Treated with Insulin.*

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Soon after the discovery of insulin, attempts were made to maintain completely depancreatized dogs with insulin. Macleod[1, 2, 3] found that dogs receiving insulin and a diet of meat and sucrose survived for 8 months, whereas the addition of raw pancreas to their diets permitted the survival of 2 completely depancreatized dogs for about 4 years. Although the diets employed were deficient, Macleod concluded that raw pancreas was essential for the survival of the depancreatized dogs for periods longer than 8 months. More recently Hershey[4] reported that lecithin was of value in this connection. Hershey and Soskin[5] state that the ingestion of lecithin supplements enables depancreatized dogs to live indefinitely and that it cures the hepatic insufficiency that appears from 6 weeks to 11 months after pancreatectomy. However, the longest period of survival reported by these workers was 1 year and 3 months. Later, Best and Hershey[6] and Best, Ferguson and Hershey[7] found that the

‡ The antistreptococcus serums used in these and other tests were kindly furnished by: Eli Lilly and Co.; The Cutter Laboratory; Parke, Davis and Co.; Lederle Laboratories; and E. R. Squibb and Sons.

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1 Allen, F. N., Bowie, D. J., Macleod, J. J. R., and Robinson, W. L., *Brit. J. Exp. Path.*, 1924, **5**, 75.

2 Macleod, J. J. R., Carbohydrate metabolism and insulin. Longmans, Green and Co., Ltd., New York, 1926.

3 Macleod, J. J. R., *Lancet*, 1930, **219**, 383.

4 Hershey, J. M., *Am. J. Physiol.*, 1930, **93**, 657.

5 Hershey, J. M., and Soskin, S., *Am. J. Physiol.*, 1931, **98**, 74.

6 Best, C. H., and Hershey, J. M., *J. Physiol.*, 1932, **75**, 49.

7 Best, C. H., Ferguson, G. C., and Hershey, J. M., *J. Physiol.*, 1933, **79**, 94.