greasy in appearance and there was some slight alopecia, probably due to irritation. The "specific" skin symptoms, however, did not appear earlier and were no more severe. Death occurred somewhat earlier in the case of the negative control rat, and a greater length of time was required to induce a cure.

Itter and co-workers[9] have correlated alopecia with the absence of the sulfhydryl group and found that feeding of *cysteine*-HCl led to cures. Prunty and Roscoe[10] showed that purified casein might be deficient in cystine, with the result that the growth rate was lessened. They could not cure the "specific" dermatitis with cystine, however. Whether or not our treatment of casein would lower the cystine content to such an extent as to be a factor in our results is being investigated.

It may be concluded that the "specific" dermatitis and cessation of growth can be readily induced in rats receiving a diet containing cornstarch, provided a highly purified and potent vitamin B_1 concentrate is used and the casein is so extracted as to remove the B_2 factors. Whether this dermatitis is identical with that induced by Hogan and Richardson remains to be determined.

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Veterinary Staphylo-Fibrinolysin.*

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In an examination of 132 local strains of staphylococci† it was found that 80% of all strains originally isolated from internal human lesions are capable of liquefying human fibrin.[1] Approximately 90% of all strains isolated from superficial human infec-

[9] Itter, S., Orent, E. R., and McCollum, E. V., *J. Biol. Chem.*, 1935, **108**, 585.

[10] Prunty, F. T. G., and Roscoe, M. H., *Biochem. J.*, 1935, **29**, 2491.

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

[1] Madison, R. R., PROC. SOC. EXP. BIOL. AND MED., 1935, **33**, 209.

tions, however, and all strains isolated from veterinary lesions are non-lytic by the same *in vitro* technic.

We have retested these and 9 additional local strains of *Staphylococcus aureus* or *albus* with fibrins isolated from lower animals. Data thus obtained are summarized in Table I.

TABLE I.

Effect of Staphylo-lysins on Lower Animal Fibrins.

24-hour veal-infusion broth cultures *of Staphylococcus aureus or albus* tested against serum-free fibrins by the method of Tillett and Garner.[2] +++, complete liquefaction of the fibrinogen-thrombin complex within 30 minutes. ++, complete liquefaction within 2 hours. +, partial liquefaction by the end of 2 hours. 0, no demonstrable softening of the fibrin-clot by the end of 2 hours.

No. of strains	Lysis of serum-free fibrin-clot from: Monkey	Man	Rabbit	Horse	Sheep	Other animals*
Group A (multivalent)						
1 strain	+++	++	++	0	++	0
12 "	+++	++	++	0	0	0
1 "	+++	+	+	0	0	0
4 "	++	++	++	0	0	0
Group B (bivalent)						
1 strain	+++	0	++	0	0	0
1 "	+++	+	0	0	0	0
7 "	++	++	0	0	0	0
1 "	++	0	+	0	0	0
1 "	0	++	++	0	0	0
Group C (univalent)						
11 strains	+++	0	0	0	0	0
19 "	++	0	0	0	0	0
1 "	+	0	0	0	0	0
1 "	0	++	0	0	0	0
1 "	0	+	0	0	0	0
2 "	0	0	0	++	0	0
Group D						
77 strains	0	0	0	0	0	0

*Rat, hog, cow, domestic fowl.

The table suggests that there are at least 5 different fibrinolytic factors formed by pathogenic staphylococci. Each lytic factor is apparently specific for the fibrin of one animal species. At least 3 of these species-specific fibrinolysins are apparently independent variables in staphylococci.

Of particular interest are the 2 monovalent horse-lytic strains (Group C). These 2 strains were originally isolated by the Cutter Laboratory from "abscesses" in horses. With these 2 exceptions all fibrinolytic staphylococci thus far tested by us were of human origin, all other local veterinary strains being included in Group D.

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