pletely cured by this treatment, and for comparative purposes a control group of female rats which had been raised on a stock diet was included in the summary of data presented in Table I. The data for this added control group were taken from a previous publication.[5]

No significant difference exists between the fat deficient and the "cured" animals in the rate of entrance of labeled acids into the acetone insoluble fraction of the liver lipids. Furthermore, the small difference between the two experimental groups and the controls on stock diet is probably well within experimental error.

It is still possible that fatty acid turnover in some tissue phospholipids is affected by essential fatty acid deficiency. Hevesy and Smedley-MacLean[2] found that rats receiving a fat deficient diet showed the same relative turnover of radioactive phosphorus in the kidney and liver phospholipids but greater in muscle phospholipids than did rats which had been cured of the deficiency with linoleic or arachidonic acids. Barnes, Miller, and Burr[3] found a decreased exchange of labeled fatty acids in the intestinal mucosa phospholipids of fat-deficient rats. Until more is known about the metabolic role of this fatty acid exchange phenomenon in phospholipids, the significance of small deviations from normal must be a matter of conjecture.

Summary. Rats suffering from a severe deficiency of the essential fatty acids show the same rate of incorporation of labeled fatty acids into the liver phospholipids as do rats which have been cured of this deficiency by the administration of 3 to 4 drops of corn oil for 1 month.

The rate of incorporation of labeled acid into the liver phospholipids is the same for rats raised on the fat deficient diet as has been previously observed for rats receiving a regular mixed stock diet.

13913 P

Production of the Shwartzman Phenomenon with a Sulfonamide Conjugate of a Bacterial Filtrate.*

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A bacterial filtrate* of meningococcus, previously shown to be capable of producing the Shwartzman phenomenon,[1] was coupled with paraaminobenzenesulfonylacetylimide† by means of a modification of the original Landsteiner method.[2] The resulting conjugate was a highly colored substance, russet and soluble in alkalis, yellow and insoluble in acids. The conjugate was purified by 10 reprecipitations from sodium carbonate solution using dilute acetic acid as the precipitating agent. The final precipitate was suspended in normal saline and dissolved by neutralization with normal sodium carbonate.

Eight experiments were performed on rabbits. In 4 instances the skin was prepared by an intradermal injection of 0.5 cc of the original bacterial filtrate and in the other 4 by the conjugate. A single intravenous injection of 1.0 cc of either the filtrate or the conjugate was given 24 hours later in each instance. Two hours later a hemorrhagic reaction was observed at the site of skin

* Courtesy of Dr. Gregory Shwartzman, Department of Bacteriology, Mount Sinai Hospital, New York City.

† We are indebted to the Medical Research Division, Schering Corporation, Bloomfield, for this compound.

1 Shwartzman, G., *The Phenomenon of Local Tissue Reactivity,* Paul B. Hoeber, Inc., Med. Book Dept., Harper and Brothers, New York, 1937.

2 Landsteiner, L., and Lampl, H., *Z. Immunitaet. Forsch.,* 1917, **26**, 293.

preparation, reaching a maximum intensity in 4 hr. Either the bacterial filtrate or its conjugate could be used for skin preparation or intravenous injection, interchangeably, for production of the phenomenon. Apparently chemical treatment of the bacterial filtrate in the synthesis of the conjugate did not affect its ability to produce the Shwartzman phenomenon.

The property of the bacterial filtrates to combine with chemicals is comparable with that of animal proteins, horse serum, egg albumen, human serum. Further studies are indicated to isolate the specific components in the filtrate responsible for this combination. These might serve as a further link for the understanding of the factors responsible for human and animal sensitization to protein and bacterial substances.

13914 P

Sodium Sulfathiazole Resistant *Shigella paradysenteriae* Flexner and Sonne.

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Numerous reports have appeared regarding the development of sulfonamide resistance by pneumococci *in vitro* and *in vivo*, and by gonococci. We have not seen a similar report regarding Shigella paradysènteriae.

In the course of our *in vitro* studies we have developed 2 sodium sulfathiazole resistant strains of Shigella paradysenteriae, one each of the Flexner and Sonne types. Sodium sulfathiazole was bactericidal for the parent Flexner strain in a concentration of 80 mg % and for the sulfonamide resistant sub-strain in a concentration of 600 mg %. Sodium sulfathiazole was bactericidal for the parent Sonne strain in a concentration of 300 mg % and for the sulfonamide resistant sub-strain in a concentration of 1000 mg %.

The sulfonamide resistant Flexner strain retained its virulence for white mice (1 M.F.D. being 1.0 cc of 10^{-5} in 3% mucin), but was non-resistant to sodium sulfathiazole *in vivo*. The resistant Sonne strain became non-virulent.

Further studies are planned with these parent and sulfonamide resistant strains and other sulfonamide compounds.