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.

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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.