47_3:b:1,5) proved the O antigen of *S. chersina* to be complex and composed of 47_1 and 47_3. Therefore, *S. chersina* shares the same O complex with *S. kaolack*, *S. phoenix*, and *S. lyon*, i.e., with the majority of the O-47 members. [*See* Kauffmann (Acta Pathol. Microbiol. Scand. **56**:343–351, 1962) for further information about the O group 47.]

When *S. chersina* was placed in tubes of semi-solid medium which contained z serum for the isolation of phase 2, migration through the medium was noted after 24 to 48 hr of incubation. The spreading growth was agglutinated by the H antiserum z_6. Antigen relations exist between the z_6 fraction of *S. chersina* and the Ha antigen.

S. chersina has the formula 47_1, 47_3:z:z_6. *S. chersina* was primarily resistant to: ampicillin (25 μg), cloxacillin (5 μg), oxacillin (5 μg), methicillin (10 μg), phenethicillin (5 μg), tetracycline phosphate (50 μg), novobiocin (30 μg), erythromycin (6 μg), matromycin (15 μg), and spiramycin (30 μg). It was sensitive to streptomycin (50 μg), chloramphenicol (20 μg), neomycin (50 μg), framycetin (100 μg), and kanamycin (30 μg). The antibiogram was carried out on paper discs with the amounts stated.

The O-1 phage disintegrates only about 30% *S. chersina* colonies. Therefore, *S. chersina* is only partially sensitive to the O-1 phage. Similar to *S. calvinia* (Brede, Zentr. Bakteriol. Parasitenk. Abt. I Orig. **188**:137, 1963), the O-1 phage resistance of *S. chersina* is even stronger than in the case of *S. zanzibar*, until the present the usual control species for O-1 phage tests. These facts were confirmed by H. Fry, Bern, Switzerland, to whom I express my thanks for doing control tests.

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ABSENCE OF LETHAL EFFECT OF PENICILLIN IN GERM-FREE GUINEA PIGS

WALTER L. NEWTON,[1] HARRY G. STEINMAN, AND MICHAEL W. BRANDRISS

Laboratory of Germfree Animal Research and Laboratory of Clinical Investigations, National Institute of Allergy and Infectious Diseases, National Institutes of Health, U.S. Public Health Service, Bethesda, Maryland

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The lethality of small doses of penicillin for the guinea pig is in marked contrast to its lack of toxicity in other animal species (Hamre et al., Am. J. Med. Sci. **206**:642, 1942). Earlier investigations seemed to indicate that the antibiotic was directly toxic to the guinea pig (Cormia et al., J. Invest. Dermatol. **7**:261, 1947). Later studies suggested, however, that death was the result of a harmful change in the intestinal flora (De Somer et al., Antibiot. Chemotherapy **5**:463, 1955). If the toxicity of small doses of penicillin were a secondary effect of a disturbance of the normal floral balance, then animals without a bacterial flora might be expected to be less affected. To test this conclusion, a comparison was made of the lethality of penicillin for both conventional and germ-free guinea pigs. Our results confirm those recently reported by Formal et al. (Nature **198**:712, 1963), who performed a similar experiment.

Both conventional and germ-free guinea pigs were from the National Institutes of Health general purpose colony and weighed from 250 to 350 g. Aqueous penicillin G (potassium salt) was given as a single intraperitoneal injection. In the first series of experiments, varied doses of penicillin were given to conventional animals only. In the second series of experiments, conventional and germ-free animals were given 120 mg/kg of penicillin.

[1] Present address: Division of Research Services, National Institutes of Health, Bethesda, Md.

TABLE 1. *Mortality of conventional 300-g guinea pigs given single injections of potassium penicillin G intraperitoneally*

Dosage (mg/kg of body wt)	No. of expt	No. of deaths/ no. injected
3.75–7.5	2	0/8
15	4	8/18
30	2	4/9
60	4	13/18
120	3	10/24
240	2	8/18

TABLE 2. *Comparative mortality of conventional and germ-free guinea pigs after a single injection of potassium penicillin G intraperitoneally at a dosage of 120 mg/kg of body weight*

Animal type	Expt no.	Mortality	Total
Conventional	1	2/12	
	2	6/10	
	3	10/10	
	4	4/10	22/42
Germ-free	1	0/11*	
	2	0/11	
	3	0/8	0/30

* Five were given 60 mg/kg; all others were given 120 mg/kg.

The results of the first series of experiments are seen in Table 1. At 15 mg/kg or more, there was no apparent relationship of dose to lethality. Overall mortality was 49%. In the second series of experiments (Table 2), the death rate varied considerably among different groups of conventional animals but the overall mortality was 52%, a result similar to that of the first experiment. Most deaths occurred between 3 and 5 days after penicillin injection. None occurred earlier than 3 days and only one as late as 8 days. Death was preceded by the appearance of listlessness, weight loss, weakness, and ruffled fur. Autopsy was performed on four conventional animals that died. A hemorrhagic colitis was seen in three. Cultures of blood as well as stains and cultures of tissue revealed no bacteria, except for one guinea pig from which *Escherichia coli* was recovered from heart blood.

In contrast, none of 30 germ-free animals died in an observation period of 2 weeks, and their outward appearance remained normal.

The absence of lethality of penicillin for germ-free guinea pigs is compatible with the hypothesis that the drug kills through some indirect effect on the intestinal flora. The lack of a typical dose effect on mortality and the fact that death did not occur until at least 3 days after a single injection, or long after the bulk of the drug would have been excreted, also suggest that the effect of the drug is not directly pharmacological.

It is of interest that not only penicillin but also a number of other antibiotics manifest an increased toxicity for guinea pigs as compared with other animals (Eyssen et al., Antibiot. Chemotherapy **7**:55, 1957). The mechanism of this indirect toxicity in the guinea pig appears to be complex. Although overgrowth of *E. coli* has been observed in moribund animals given antibiotics, death did not result when intestinal infection with *E. coli* was deliberately established in previously germ-free guinea pigs (Phillips et al., Am. J. Trop. Med. Hyg. **4**:675, 1955; Formal et al., J. Bacteriol. **82**:284, 1961). This observation suggests that additional factors may be involved. Pertinent perhaps are recent studies which indicate that the numbers and composition of the intestinal flora can effect the reaction of some animals to bacterial endotoxins (Jensen et al., Proc. Soc. Exptl. Biol. Med. **113**:710, 1963).

REDUCTION OF VITAMIN B_{12} BY *PSEUDOMONAS RUBESCENS*

J. B. HUFHAM, R. C. BURGUS, W. M. SCOTT, AND J. J. PFIFFNER

Department of Physiology and Pharmacology, Wayne State University College of Medicine, Detroit, Michigan

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While studying the degradation of vitamin B_{12} by microorganisms, we observed that the red color of the vitamin was initially converted to a yellow or yellow-brown product(s) by *Pseudo-*