TABLE 1.

Average Values on the Gastrocnemius and Soleus Muscles of rats. D indicates denervated muscle and C signifies non-denervated contralateral control muscle. The *in vitro* O_2 consumption was measured on the solei. All other values refer to gastrocnemii.

Condition	No. of rats	Nerve lesion: Type	Nerve lesion: Days	Body wt, g: Initial	Body wt, g: Final	Gms tension per g muscle: D	Gms tension per g muscle: C	Muscle wt* / Final body wt: D	Muscle wt* / Final body wt: C	Muscle tension (g) / Final body wt (g) stimulated through Muscle: D	Muscle tension (g) / Final body wt (g) stimulated through Muscle: C	Muscle tension (g) / Final body wt (g) stimulated through Nerve: D	Muscle tension (g) / Final body wt (g) stimulated through Nerve: C	Muscle creatine mg/100 g: D	Muscle creatine mg/100 g: C	QO_2: D	QO_2: C	% atrophy†
Control	13	cut	14	183	188	965	1857	.358	.588	3.34	10.62			344	453	2.7	2.5	39.1
Atropine	10	cut	14	186	181	957	1879	.342	.533	3.25	9.37			321	459	2.5	2.4	35.2
Control	7	crush	21	255	243	1269	1830	.378	.595	4.22	10.82	2.35	10.75	392	483			36.3
Atropine	5	crush	21	230	198	1078	1557	.354	.528	3.82	8.27	2.23	7.89	383	469			32.4

* × 100.

† Calculated from the differences in weight between denervated and non-denervated control muscle.

or degree of regeneration under conditions in which control muscle undergoes appreciable changes in weight during the period of experimentation is obvious.

Summary. The daily administration of large doses of atropine to rats failed to retard the rate of atrophy of denervated muscle and did not enhance the rate of neuromuscular regeneration. Atropine did not significantly affect the creatine concentration and *in vitro* oxygen utilization of denervated and control muscle. When expressed as a fraction of the final body weight the strength and weight of the control muscles of the atropine-treated rats were found to be inferior to those of untreated control muscles. The change in weight of the contralateral control muscle following atropine administration invalidates any estimation of the extent and rate of atrophy or regeneration based solely on the differences between the terminal weights of the control and experimental muscle.

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Reproduction of Bacteria from the Large Bodies of *Streptobacillus moniliformis.**†

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In most strains of *Streptobacillus moniliformis* the bacteria develop by gradual swelling into large round forms. These forms transplanted under appropriate conditions germinate by the growth of small soft granules and diphtheroid-like forms and produce a pleuropneumonia-like, L1, colony.[1] Similar enlargement of bacteria and development to pleuropneumonia-like colonies was observed in the cultures of *B. funduliformis, B. coli,* and *B. influenzæ.*[1] In a culture of *B. coli*[2] and in the cultures of several strains of

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1 Dienes, L., *T. Bact.*, 1942, **44**, 37.

2 Dienes, L., PROC. SOC. EXP. BIOL. AND MED., 1939, **42**, 773.

3 Dienes, L., and Smith, W. E., PROC. SOC. EXP. BIOL. AND MED., 1942, **51**, 297.

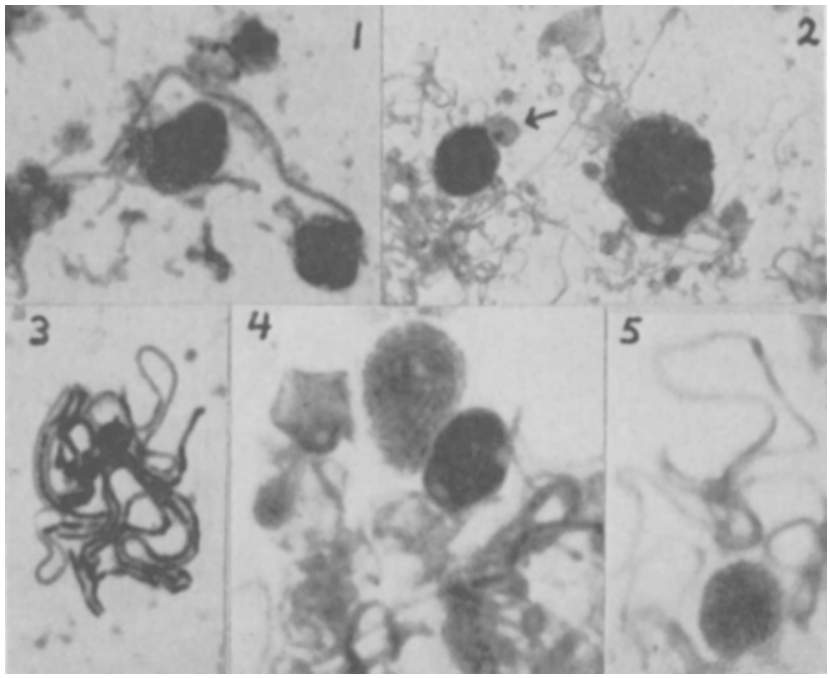

FIG. 1.

The photographs were taken from Giemsa stained impression preparations following Klieneberger's agar fixation technic.[4]

1. Represents the surface before incubation of an agar block just inoculated with a 48-hour culture. Bacterial filaments, large bodies, and debris resulting from the autolysis of the culture are visible in the photographs. Bacteria cannot be seen in the large bodies. × 2000.

2. Surface of the agar after 8 hours incubation as seen with moderate magnification. (× 1000.) Two round dense bacterial colonies are visible in the picture. The bacterial filaments of the inoculum show no growth. The round body marked with an arrow is full of tiny bacilli.

3. A young colony resulting from the growth of the bacterial filaments of the inoculum (10 hours incubation). (× 2000.)

4-5. Show large bodies packed with bacillary forms (high magnification × 3000). Note the smallness of the bacillary forms in the large body in photograph 5.

B. funduliformis[3] the large bodies went through another type of development and produced bacteria of the usual shape and size. A similar process was observed recently in a strain of *Streptobacillus moniliformis*.

The strain which showed this phenomenon was received from Doctor Heilman. It was preserved without being transplanted in CO_2 refrigeration. The strain grew in small colonies which after 24 hours consisted of intertwined filaments with relatively few large bodies. After 48 hours the colonies consisted mostly of very large flat bodies (up to 20 microns in diameter) filled with vacuoles of various size. The bacterial filaments in these colonies were to a large extent autolyzed and stained only faintly. L1 colonies did not develop beneath the bacterial colonies but they developed in moderate numbers among the bacterial colonies in transplants. The bacterial growth in young cultures often was first noticeable in the form of very tight round colonies in contrast to the usual loose colonies of the streptobacillus. This observation suggested that the bacterial growth started from the large bodies.

To examine this supposition, agar blocks inoculated with 24- and 48-hour-old cultures were incubated on coverslips. The blocks after varying lengths of incubation were fixed

with Bouin solution.[4] The coverslips were stained with Giemsa and differentiated with tapwater and ascitic fluid. The blocks were incubated between 25-30°C. Bacterial forms were never visible in the large bodies in the original cultures or immediately after transplantation. After a few hours of incubation, however, many large bodies appeared tightly filled with bacteria and their growth produced the round dense colonies. Bacteria developed more often in the large bodies transplanted from 48-hour-old cultures than from 24-hour cultures. The appearance of the culture and the large bodies filled with bacteria are illustrated in the photographs (Fig. 1).

The bacteria according to all probability were produced inside the large bodies by transformation of the contents of the large bodies. They did not grow in from the outside. We mentioned above that bacteria were never visible in the large bodies in the original cultures. They appeared after as short incubation as 1½ hours and were quite numerous after 5 hours. The bacterial multiplication did not start during this period. The large bodies were either completely filled with bacterial forms or contained none. One never saw only a few bacteria or bacteria in vacuoles as occasionally occurs in bacterial contamination of cultures of L1. The bacteria inside the large bodies are much smaller and thinner than the bacteria which later start to grow outside of them. Bacteria are never visible in the autolyzed large bodies which do not stain well. They are always in those which are deeply stained and well maintained. The growth of bacteria from the content of large bodies was observed in 2 other bacterial species, which makes it easier to accept that a similar process occurs in *Streptobacillus moniliformis*.

Apparently in all species the large bodies possess a double potentiality to develop either into regular bacteria or into pleuropneumonia-like (L) forms. The L forms under appropriate conditions return to regular bacterial morphology. They might represent intermediate forms in the development of the bacteria inside the large bodies. These forms in certain cases progress immediately into regular bacterial forms; while in others they are able to grow in this intermediate form.

[4] Klieneberger, E., and Smiles, T., *J. Hygiene*, 1942, **42**, 110.

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A Toxic Factor Associated with the Agent of Lymphogranuloma Venereum.

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The production of a toxic factor during the development of the agent of lymphogranuloma venereum has been suspected for the following reasons: (A) The disease can occur in man in severe generalized form[1] with marked headache, chills, and other symptoms suggesting toxemia. (B) The cause of death of the chick embryo after yolk-sac infection is most readily explicable as due to a toxin, since the agent does not invade the embryo to any great extent[2] and disturbance of nutrition would appear unimportant since weight of embryos dead of the infection does not differ from that of uninfected embryos of the same age. (C) In mice, convulsive deaths occurring some 30 hours after intracerebral inoculation suggest toxemia and are not wholly accounted for by the lesions formed in the meninges. Bearing on this problem Gildermeister and Haagen

[1] Harrop, G. A., Rake, G., and Shaffer, M. F., *Trans. Am. Clin. and Clim. Assn.*, 1941, **56**, 154; *Trans. Assn. Am. Phys.*, 1941, **56**, 101.

[2] Rake, G., and Jones, H. P., *J. Exp. Med.*, 1942, **75**, 323.