TABLE I.
Respiratory Quotients after Depression of Oxygen Consumption.

Depressant	cmm. O_2/dry mg./hr.		Respiratory Quotient	
	Without depressant	With depressant	Without depressant	With depressant
Ethyl urethane 3%	19.3	13.5	.814	.922
" "	19.3	10.6	.904	.885
" "	22.7	11.8	.863	.934
" "	15.2	9.2	.812	.883
" "	17.5	13.0	.760	.833
Thymol .012%	22.8	13.0	.878	.929
" .014%	19.8	11.7	.872	.919
" .012%	20.2	17.4	.894	.919
" .010%	25.0	17.9	.908	.888
After previous anaerobic exposure	19.7	11.3	.894	.932
"	19.2	7.5	801	.863

TABLE II.
Selective Action of Depressants on Oxidation of Fat and Carbohydrate.

Depressant	No. of experiments	Average depression of O_2 cons.	Average effect on fat	Average effect on carbohydrate
		%	%	%
Ethyl urethane 3%	5	—38	—57	+7
Thymol .010 to .014%	4	—30	—52	—19
Anaerobiosis	2	—59	—68	—33

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Observations on the Pathogenesis of Pulmonary Suppuration in the Albino Rat.*

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The well recognized syndrome of pulmonary suppuration in the albino rat is, as in many human affections, an advanced stage of the condition. The earlier manifestations of the disease are imperfectly understood and the common appellation "pneumonia" for the well developed lesion is a reflection of this lack of knowledge of the fundamental nature of the process. The lesions of the lungs vary greatly according to the stage and extent of the process but the

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essential nature of the completely developed disease is that of chronic lung abscesses.

We have been particularly impressed with the frequent occurrence of atelectasis in white rats which are apparently in perfect health. These observations lead us to suggest that atelectasis subsequent to bronchial obstruction is a primary factor in the development of pulmonary infection in the white rat. To substantiate this hypothesis it is necessary to show that obstructive atelectasis occurs in white rats showing no evidence of pulmonary infection and that organisms may gain an entrance to the lungs and selectively localize in the collapsed lobes. The probable pathway of infection has been indicated by Jones,[1] who isolated organisms occurring in the environmental atmosphere, from the lungs of several laboratory animals. The regularity with which the cultures were positive varied directly with the amount of dust in the atmosphere. It is a well known fact that after bacteria have once gained entrance into the body their ability to proliferate and invade the host is greatly increased by an obstruction to the part.

Our own observations have clearly shown that an obstructive atelectasis is a very common finding in white rats. This condition has been observed many times in animals showing no gross or microscopic evidence of pulmonary infection. The atelectatic areas vary in size from a minute portion at the tip of a lobe to a massive collapse of the entire right side and the infra-cardiac lobe. In many instances we have demonstrated occlusive mucus plugs in the bronchi leading to collapsed lobes. The points of occlusion have been demonstrated by the injection of air and fluid into the trachea and by careful dissection of the bronchi leading to collapsed lobes. The objection that the occlusion may develop as the result of infection can be readily answered as we have frequently observed obstructive atelectasis in animals showing no evidence of pulmonary infection. If the obstruction persists, small abscesses are frequently seen within the collapsed portions of the lung. As the infection progresses the abscesses become larger and all evidence of a pre-existing atelectasis is obscured.

On the basis of these observations we suggest that the sequence of events in the pathogenesis of pulmonary infection in the white rat is an obstructive atelectasis followed by the growth of organisms and the development of pulmonary suppuration.

This report is based on incidental observations made during the course of experiments on the effect of dietary protein on the re-

[1] Jones, F. S., *J. Exp. Med.*, 1922, xxxvi, 317.

maining kidneys of rats after a unilateral nephrectomy. More than 200 animals have been studied and the lesions described have occurred independently of the diets fed.

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Relation of Vitamins B (B_1, F) and G (B_2) to Renal Enlargement in Rats Fed High Concentrations of Protein.*

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That vitamins B and G play a unique and important rôle in the metabolism of proteins is believed by some investigators. Reader and Drummond[1] have stated that there is little hypertrophy of the kidneys of rats fed on diets rich both in protein and in vitamin B complex, and that the required vitamin B complex is quantitatively related to the amount of protein in the diet. As this conclusion was not in accord with certain of our own observations we planned experiments to test the thesis that renal enlargement produced by high protein diets is lessened by increased ingestion of vitamins B, G and the vitamin B complex.

Groups of 12 to 14 animals (30 days old), consisting of intact rats as well as those from which one kidney had been removed, were fed diets of 18 and 90% protein respectively for 56 days. The groups were fed 3 different levels of yeast and in addition the highest level of yeast was supplemented with (1) factor G, using autoclaved yeast or (2) factor B, in the form of tikitiki extract.† Additional experiments are in progress to test the effect of an excess of the heat-stable (vitamin G) and heat-labile (vitamin B) factors fed separately without yeast. At autopsy hearts and kidneys were weighed and renal enlargement calculated on the basis of the weights of these organs as published by Donaldson.[2]

In the accompanying table are presented experimental data which show that: 1. Renal enlargement occurs in both nephrectomized

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[1] Reader, V., and Drummond, J. C., *Biochem. J.*, 1926, xx, 1256.

† Furnished through the kindness of Dr. Brown of the Philippine Bureau of Science.

[2] Donaldson, H. H., "The Rat," 1924, 2d ed., Philadelphia, Penn.