known potency, and with this have been able to treat a number of roaches with standardized dosages. The penicillin was dissolved in sterile water in such dilution that the dose desired in each case was contained in 0.2 cc, which is a convenient volume for injection. These injections were repeated, as shown by the appended table, where the effects of the several concentrations is also indicated. It will be noted that the tolerance of the roaches is many times greater on the basis of body weight than the therapeutic dosages employed in human medical practice.

COCKROACHES (*Blaberus cranifer*) TREATED WITH PENICILLIN

Lot No.	Dosage in Oxford Units	Results
A. (2♀)	200 units × 4 doses Total—800 units	After 28 days, one killed and fixed. Separated clumps of symbionts present. Other roach perfectly healthy.
B. (2♀)	200 units × 5 doses Total—1,000 units	After 4 days, one killed and fixed. Symbionts apparently in good condition. After 8 days, another killed and fixed. Symbionts apparently in good condition. Both were lively when killed.
C. (3♀)	400 units × 4 doses Total—1,600 units	After 24 hours, one killed and fixed. No symbionts visible, although the large cells formerly containing them evident. After 26 days, one almost dead, killed and fixed. Symbionts in scattered cells. Fat frothy and almost completely liquefied. After 27 days, third specimen killed and fixed. Condition as in the previous one.
D. (3♀)	800 units × 4 doses Total—3,200 units	After 24 hours, one dead. After 24 hours, one killed and fixed. No symbionts in larger cells, but a very few in smaller ones. After 27 days, the third specimen killed and fixed. Symbionts present in scattered cells.
E. (2♀)	2,000 units × 3 doses Total—6,000 units	After 6 days, one killed and fixed. No symbionts visible. After 12 days, second specimen died.

These results show very clearly that the bacteroids are affected by penicillin when this is administered at high concentrations, although the roaches give no immediate indication of any toxic effect from the drug,[2] even when dosages as great at 200 times the therapeutic dose is injected. The condition of the fat cells and their contained bacteroids were examined after fixation in formaldehyde, sectioning in paraffin and a heavy staining in haematoxylin.

From the data here presented, it is apparent that the administration of penicillin at the lower concentrations very greatly reduces the number of bacteroids, but after the insects are allowed to live normally for a time, the bacteroids gradually increase in numbers. Meanwhile, the roaches show no ill effects from the treatments. If, however, sufficient penicillin is administered to destroy the bacteroids, or to reduce their numbers beyond a very low level, the roaches finally die after some days have elapsed. Since they do not succumb immediately, their death can not be attributed to a direct toxic effect of penicillin, but rather to a lack of something supplied by the bacteroids. We can not regard the present results as conclusive evidence that the bacteroids are necessary for the continued life of the cockroaches, but they make it appear very probable that such is the case and that they are symbiotic, and not parasitic, microorganisms.

[2] Occasionally roaches die shortly after having received an "intraperitoneal" injection, due probably to perforation of the alimentary canal by the hypodermic needle.

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THE OCCURRENCE OF NORMAL SERUM GAMMA-GLOBULIN IN HUMAN LYMPHOCYTES

DESPITE the interest and importance of the problem, there is little information concerning the origin of serum globulins. Since the human serum γ-globulin fraction contains almost all the antibody activity of pooled plasmas,[1] and since numerous attempts to demonstrate antigenic[2,3] and physical[4] differences between immune and non-immune globulins have been uniformly unsuccessful, there would seem to be little doubt that antibodies are modified serum globulins, and that in man, at least, they are chiefly modified γ-globulins.

Recent demonstrations of the occurrence of antibody in lymphocytes of rabbits[5] and mice[6] substantiate earlier indications of the role of lymphoid tissue in the development of immunity.[7] It follows that if antibody is synthesized in lymphocytes, normal serum γ-globulin should also be present.

We have, therefore, prepared rabbit antisera to highly purified γ-globulin fractionated from pooled normal human plasmas, and have found that extracts of lymphocytes of human origin react specifically with these antisera.

The γ-globulin used for immunization[8] was further

[1] J. F. Enders, *Jour. Clin. Invest.*, 23: 510, 1944.
[2] H. P. Treffers and M. Heidelberger, *Jour. Exp. Med.*, 73: 293, 1941.
[3] E. H. Kass, M. Scherago and R. H. Weaver, *Jour. Immunol.*, 45: 87, 1942.
[4] M. L. Petermann and A. M. Pappenheimer, Jr., *Jour. Phys. Chem.*, 45: 1, 1941.
[5] T. N. Harris, E. Grimm, E. Mertens and W. E. Ehrich, *Jour. Exp. Med.*, 81: 73, 1945.
[6] T. F. Dougherty, J. H. Chase and A. White, *Proc. Soc. Exp. Biol. and Med.*, 57: 295, 1944.
[7] C. H. Bunting, *Wisconsin Med. Jour*, 24: 305, 1925.
[8] Kindly supplied by Dr. J. L. Oncley of the Department of Physical Chemistry, Harvard Medical School. See E. J. Cohn, J. L. Oncley, L. E. Strong, W. L. Hughes, Jr., and S. H. Armstrong, Jr., *Jour. Clin. Invest.*, 23: 417, 1944.

purified by the author and found to be 98 to 100 per cent. pure γ-globulin by electrophoretic examination. This degree of purity probably represents the limit of accuracy of the Tiselius apparatus, hence no more precise figures for purity can be given.

Twenty mesenteric lymph nodes were obtained within one hour after death of a patient, a fifty-four year old white female who died of arteriosclerotic heart disease, with pulmonary edema and terminal bronchopneumonia. The nodes were dissected free of fat and connective tissue, washed in saline, and sliced into narrow sections (2–5 mm in thickness) with a razor blade. These slices were suspended in 25 ml of saline, shaken vigorously for 5 minutes, and filtered through cheesecloth. The filtrate, containing about 95 per cent. small lymphocytes and about 5 per cent. large lymphocytes, was centrifugalized and the cells washed twice in 25 ml and finally in 1 ml of saline. The supernate, following the last washing, failed to react with antiserum to γ-globulin. The cells were resuspended in 1 ml of saline and frozen and thawed three times; smears made at this time showed practically all cells to be lysed. The supernate, after centrifugalization, reacted with the antiserum to γ-globulin, and failed to react with normal rabbit serum. If diluted until it failed to precipitate with the antiserum, the lymphocytic extract, when mixed with the serum, inhibited the ability of the antiserum to precipitate purified human γ-globulin or to form a precipitate when overlaid with normal human serum. Extracts of washed slices of human liver failed to react with the antiserum.[9]

The occurrence of normal γ-globulin in lymphocytes, when coupled with the demonstration of lymphoid hyperplasia accompanying antibody production[10,11] and the failure of lymphocytes to adsorb antibody *in vitro*,[5] suggests that the specific alteration of γ-globulin to cause the molecule to become reactive toward a given antigen occurs within the lymphocyte.

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RETENTION OF THIAMINE, RIBOFLAVIN AND NIACIN IN DEEP FAT COOKING[1]

THE doughnut continues to increase in popularity as shown by the following records of sales in this country: 1929, 201 millions of dozens; 1937, 258 millions; 1939, 336 millions and estimated for 1943, 665 millions. A food material of this importance in the national dietary merits attention with respect to its contributions to the daily regimen. The present report indicates the losses in vitamins which occur in the process of cooking.

The doughnuts were made in a commercial machine. The dough ring is ejected into the hot fat (approximately 375° F.) and rises to the surface due to the immediate leavening action. Half the doughnut is immersed in the cooking fat for 45 seconds, then the doughnut is turned mechanically and for the rest of the 90 seconds total cooking time the other half is immersed.

Quantitative estimations were made of the concentration of thiamin, riboflavin, niacin and iron in the dry doughnut mix containing enriched flour and in the freshly cooked doughnut. The thiochrome method[2] was used for thiamine, the procedure of Snell and Strong[3] using *Lactobacillus casei* for riboflavin and the method of Krehl, Strong and Elvehjem[4] using *Lactobacillus arabinosus* for niacin. The composition of the finished doughnuts (30 gm each) was as follows:

Protein	1.84 gm
Fat	6.03 "
Carbohydrate	14.70 "
Calcium	13.40 mgm
Phosphorus	92.70 "
Iron	0.57 "
Thiamine	0.085 "
Riboflavin	0.066 "
Niacin	0.600 "
Moisture	6.84 gms
Calories	123.6

Making allowance for the addition of water to the dry mix in preparing the dough and also for the fat absorbed during frying, it was found that the average loss in thiamine during cooking was 22.9 per cent. and in niacin was 20 per cent., whereas there was no measurable loss in riboflavin or in iron.

The loss of thiamine in this type of food in the course of deep fat frying is within the limits observed by Melnick, Oser and Himes[5] in baking various kinds of cake made from enriched flour. They found that as the pH of the cake batters increased from 5.9 to 7.9 the losses of thiamine during baking generally increased. In the present study the pH of the doughnut batter was 6.1, a value not ordinarily con-

[9] Extraction by similar means of lymphocytes obtained from a case of visceral sporotrichosis gave similar results. The nodes in this case, however, could scarcely be called normal.

[10] W. E. Ehrich and T. N. Harris, *Jour. Exp. Med.*, 76: 335, 1942.

[11] A. Rich, *Proc. Soc. Exp. Biol. and Med.*, 32: 1395, 1935.

[1] This study was supported by a grant from the Doughnut Corporation of America.

[2] D. J. Hennessy, *Cereal Chemists Bul.*, 2: 1, 1942.

[3] E. E. Snell and F. M. Strong, *Ind. Eng. Chem., Anal. Ed.*, 11: 346, 1939.

[4] W. A. Krehl, F. M. Strong and C. A. Elvehjem, *Ind. Eng. Chem., Anal. Ed.*, 15: 471, 1943.

[5] D. Melnick, B. L. Oser and H. W. Himes, *Cereal Chemistry*, 20: 661, 1943.