ferric ion. However, it was found that this treatment was insufficient for the removal of all the ferric ion from the resin. It seems that in the naphthazarin state the resin functions as an ion exchanger. It was necessary to treat the resin with 3 normal hydrochloric acid to effect complete removal of ferric ion. This cycle was repeated five times in all for the oxidation of ferrous ion. In the same manner, the oxidation of iodide ion and ferrocyanide ion was proved.

The reducing properties of the resin were shown in a similar way. Approximately 1 g of resin was treated with excess sodium hydrosulfite and was then washed free of excess reductant. A solution of ferric chloride was added to the reduced polymer. The centrifugate showed the absence of ferric ion by test with thiocyanate. Addition of hydrogen peroxide to this test mixture gave a deep red color, showing the presence of ferrous ion. The polymer was regenerated by treatment with 3 normal hydrochloric acid and successive water washes. This cycle was also repeated five times in all. The reduction of ferricyanide and iodide ions was similarly demonstrated.

In view of the fact that ferric ion was picked up by the resin when it was presumably in the naphthazarin oxidation state, several other ions were tested for exchangeability. It was found that Ni^{2+}, Cu^{2+}, and Co^{2+} were readily picked up and eluted.

References and Notes

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Alterations in Serum Properdin Levels Following Injection of Zymosan

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The discovery and isolation of properdin, a new serum protein that participates in many immune reactions, has been described recently (*1*). Properdin acts in conjunction with complement or complement-like substances and requires Mg^{++} for its activity (*1*). Available evidence suggests that this system plays an important role in natural immunity.

Zymosan (*2*), the insoluble cell-wall residue from yeast, combines with properdin *in vitro*. This provided the basis for the isolation of properdin (*1*). Experiments directed toward a better understanding of the role of the properdin system in experimental infections and other disease states have now shown that the injection of zymosan causes marked alterations in the serum properdin levels of laboratory animals (*3*).

A series of experiments has been conducted in which healthy 12- to 16-g CF1 female mice, and 175 to 200-g Wistar rats were injected intravenously or intraperitoneally with various doses of a boiled saline suspension of zymosan (*4*). Changes in the properdin content of the blood were followed by doing titrations (*1*) on pools of at least six serums obtained from groups of animals sacrificed at various times before and after injection. The serum samples were frozen and stored at −70°C in a mechanical deep freeze until the last sample had been obtained. Properdin titrations were then done on all the samples with the same reagents on the same day. Some of the samples were also titrated for complement and for C'3.

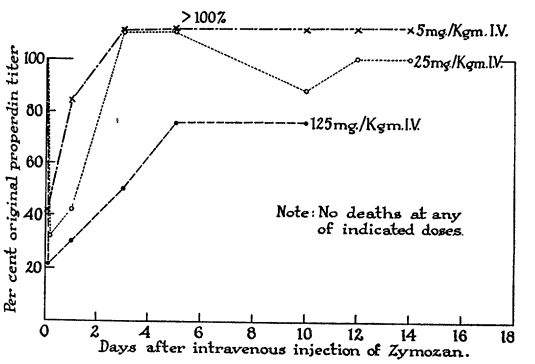

Fig. 1. The serum properdin levels in mice following intravenous injection of zymosan.

The results of a typical experiment with mice (Fig. 1) show that the intravenous injection of small doses of zymosan caused a rapid fall in properdin titer within 1 to 2 hr, followed, after 2 to 14 days, by a marked rise in titer 200 to 300 percent above normal level. Large doses of zymosan caused a greater fall in the properdin levels, which slowly returned to 75 percent of the normal level after 6 to 10 days. It is, therefore, possible either to decrease or increase the properdin concentration in the serums of mice by injection of suitable doses of zymosan. The intraperitoneal injection of zymosan produced similar, although slower and more prolonged, changes in the properdin titers. A more detailed report of these experiments is in preparation. Rats and rabbits behaved similarly to mice in their reaction to zymosan. In contrast to the marked changes in properdin following zymosan injection, no changes were observed in C'3 titers. This result is the opposite of that expected on the basis of *in vitro* experiments which showed that the zymosan-properdin complex inactivates C'3 at 37°C (*1*).

The observation that serum properdin levels in animals may be increased or decreased by injection of zymosan under controlled conditions now offers a means for studying the role of the properdin system in experimental infections and other disease states. Indeed, Derrick Rowley, at the Wright-Fleming Institute of Microbiology, London (personal communication), recently found that the injection of zymosan

into mice caused marked changes in their susceptibility to experimental *Escherichia coli* infection. During the first hour after zymosan injection, the mice became highly susceptible to infection with an *E. coli* strain that was avirulent for normal mice. Moreover, during the period between 2 and 5 days after injection of zymosan, the mice were highly resistant to a different *E. coli* strain that was fully virulent for normal mice. In addition, Rowley found that rats which are naturally resistant to *E. coli* infection were killed by this organism if zymosan had been injected with 1 hr preceding challenge.

These experiments suggest that the initial fall and secondary rise in blood properdin that we have found to follow zymosan administration may influence the increase and decrease in natural resistance of the animals in Rowley's experiments. Experiments to be published in detail elsewhere (*5*) also show that the injection of zymosan into mice or rats can markedly increase or decrease their susceptibility to the effects of lethal doses of total-body irradiation depending on the dose and the time of injection.

References and Notes

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Observations on Growth Responses to Antibiotics and Arsonic Acids in Poultry Feeds

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During the past few years the relative growth responses that we have obtained when low levels of antibiotics and arsonic acids were added to poultry feeds have been progressively decreasing. Recent tests have sometimes shown a total lack of response.

Several different antibiotics, arsonic acids, basal rations, and breeds of chickens have been used in our experiments (*1*). However, equipment, management, and sanitation practices were reasonably uniform from year to year. The facilities for growing chickens have been in use almost continuously for many years, so that the environment may be considered "old" rather than "new."

Birds were started at 1 day of age and were carried for 3 to 6 wk on wire floors in batteries or up to 10 wk on corncob litter in a brooder house with heated floors. Feeds and water were offered *ad libitum,* and lights were used for 24 hr a day in the battery room and 14 hr a day in the brooder house.

Table 1 gives the yearly percentage growth response of birds fed rations containing antibiotics or arsonic acids over those fed control rations without these additives. Although the data for arsonic acids are not as extensive as those for antibiotics, both substances showed a similar trend—a progressive percentage decrease in response. Inasmuch as these chemicals exert their influence on nutrition in the same manner (*2*), their data are combined in column 3. This table contains data on 3900 chicks in 146 comparisons between groups of birds that were simultaneously fed the control rations or these same rations containing dietary levels of antibiotics or arsenicals.

It is seen that during the first year these substances considerably improved the early growth of chicks. This occurred on all basal diets. The response to antibiotics and arsonic acids, however, has since been progressively decreasing. Waibel (*3*) recently noted this in the case of dietary penicillin and Aureomycin during a 3-yr period.

At first sight this decreasing response appeared to be in accord with the idea that antibiotics may lose their effectiveness with continued usage, because of the gradual establishment of a microflora resistant to the antibiotics (*4*). If this were the case, the value of such additives would decrease in the course of time with prolonged use in the same place.

However, before this inference is drawn from the data presented, the growth rate of birds that were not fed antibiotics or arsonic acids should be noted. As is

Table 1. Growth responses from dietary antibiotics and arsonic acids and the mortality by years. The figures in parentheses indicate number of comparisons with control birds.

Periods	Increase in weight with respect to birds fed the control ration (%)			Increase in weight of control birds with respect to 1950–51 control birds (%)	Mortality to 6 wk of age (%)
	Birds fed antibiotics	Birds fed arsonic acids	Average		
1950–51	19.0 (75)	16.0 (2)	18.9	0	8.5
1951–52	12.8 (32)	(0)	12.8	7.4	8.2
1952–53	10.0 (4)	3.8 (5)	6.5	15.3	4.6
1953–54	3.3 (19)	3.2 (9)	3.2	19.1	2.8