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Temperature Changes in the Chick During the Course of Fowl Typhoid*

A. E. BELL

Many diseases are characterized by the production of a fever in the infected host. Contrary to early beliefs a moderate fever is now thought to be one of the defense mechanisms used by the body to combat some infections. High temperatures alone are enough to destroy some invading organisms. Carpenter and others (1933) showed that the causative organism in gonorrhea can be destroyed in *vitro* by temperatures such as those induced in artificial fever. Ellingson and Clark (1942) observed increasing phagocytosis with rising temperatures. Others have reported increased antibody production under the influence of fever.

Since the classical demonstration by Pasteur that the fowl's immunity to anthrax is due to its high body temperature, the relatively high resistance of birds to bacterial infection has been attributed to this factor. Scholes and Hutt (1942) published data indicating that body temperature of the chick is of major importance in resistance to *Salmonella pullorum*. They found that resistant strains consistently maintained a higher temperature during the first 10 days of life, which is the period of greatest susceptibility. Severens, Roberts and Card (1944) concluded from their studies on resistant and susceptible strains of chicks that body temperature has little significance in regard to genetic resistance to pullorum disease. However, their resistant chicks did maintain a slightly higher temperature for the first 10 days. Cook and Dearstyne (1934) in studies on fowl typhoid in adult birds reported a temperature rise in three to six days after inoculation. The nature of their studies did not permit them to correlate this rise with resistance or susceptibility. To test further the role of body temperature in resistance, studies were made on fowl typhoid. A progress report on these studies is presented here.

Materials and Methods

In order to study adequately a problem of this nature it is necessary to establish genetically resistant and susceptible host strains. For the study of fowl typhoid such strains of chicks have been developed and are maintained by the Genetics Section at Iowa State College. The resistant strains were of the White Leghorn breed. They were developed over a number of years by rigid selection and inbreeding. The susceptible strain used was of the Rhode Island Red breed. Chicks were brooded at approximately 33°C. and were fed a standard chick mash.

*This report is one of a series of investigations directed by Dr. John W. Gowen at the Genetic Laboratory of Iowa State College. The work has been aided by a grant from the Rockefeller Foundation.

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Temperatures of chicks were determined rectally by means of a specially constructed thermo-couple. In order to exclude diurnal fluctuations, all temperatures were taken between 7 and 9 p. m. Daily temperatures of the chicks were recorded from the eighth to the thirty-first day of life.

A virulent strain of the fowl typhoid organism, *Shigella gallinarum,* was used. The resistant chicks were inoculated intraperitoneally at 10 days of age with 200,000 live organisms suspended in sterile physiological salt solution containing 0.05% bacto-peptone, while the susceptible strain received only 200 organisms. Concentration of the bacterial suspension was estimated by means of a Gates nephelometer and checked by poured agar dilution plates.

Experimental Results

In order to differentiate body temperature changes occurring normally from those associated with the disease, temperatures of non-inoculated resistant and susceptible chicks were also determined. Temperatures were taken for two days before inoculation, so that any normal variation in the inoculated groups might be noted. Twenty chicks were studied in each inoculated group and ten chicks in each control group.

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Table 1. Body Temperature Rise in Chicks due to Fowl Typhoid.

Age of chicks (days)	Susceptible Chicks			Resistant Chicks		
	Mean body temperature (°C)		Difference	Mean body temperature (°C)		Difference
	Control	Inoculated	(degrees)	Control	Inoculated	(degrees)
8	40.1 ± .17	40.3 ± .05	+0.2	40.2 ± .06	40.2 ± .09	0
9	40.2 ± .08	40.3 ± .11	+0.1	40.2 ± .12	40.2 ± .08	0
		Inoculated			Inoculated	
10	40.4 ± .07	40.6 ± .05	+0.2	40.2 ± .09	40.6 ± .07	+0.4**
11	40.4 ± .07	40.5 ± .04	+0.1	40.3 ± .09	40.4 ± .07	+0.1
12	40.9 ± .06	41.0 ± .05	+0.1	40.8 ± .14	40.8 ± .07	0
13	40.7 ± .06	41.0 ± .11	+0.3*	40.7 ± .08	40.7 ± .06	0
14	40.6 ± .06	41.3 ± .11	+0.7**	40.7 ± .08	40.7 ± .07	0
15	41.0 ± .05	41.8 ± .12	+0.8**	40.9 ± .09	41.1 ± .06	+0.2
16	40.8 ± .09	41.4 ± .14	+0.6**	40.7 ± .09	40.7 ± .07	0
17	40.8 ± .05	41.6 ± .13	+0.8**	40.7 ± .08	40.9 ± .08	+0.2
18	41.3 ± .04	42.0 ± .24	+0.7*	41.3 ± .04	41.3 ± .09	0
19	41.1 ± .06	41.6 ± .16	+0.5*	41.1 ± .10	41.0 ± .08	−0.1
20	41.0 ± .11	41.7 ± .09	+0.7**	40.8 ± .06	40.8 ± .08	0
21	41.1 ± .11	41.6 ± .09	+0.5**	41.0 ± .14	40.9 ± .10	−0.1
22	41.0 ± .09	41.5 ± .09	+0.5**	41.1 ± .10	40.8 ± .10	−0.3
23	41.4 ± .10	41.6 ± .20	+0.2	41.3 ± .10	41.0 ± .09	−0.3*
24	41.3 ± .07	41.8 ± .18	+0.5*	41.5 ± .07	41.3 ± .06	−0.2*
25	41.3 ± .06	41.8 ± .10	+0.5**	41.6 ± .16	41.1 ± .08	−0.5**
26	41.1 ± .06	41.6 ± .33	+0.5	41.2 ± .05	41.1 ± .07	−0.1
27	41.3 ± .07	41.8 ± .37	+0.5	41.3 ± .12	41.4 ± .09	+0.1
28	41.1 ± .09	41.4 ± .21	+0.3	41.1 ± .10	40.8 ± .07	−0.3*
29	41.2 ± .10	41.6 ± .20	+0.4	41.2 ± .10	41.0 ± .07	−0.2
30	41.7 ± .11	42.2 ± .16	+0.5	41.5 ± .14	41.4 ± .08	−0.1
31	41.5 ± .08	41.6 ± .35	+0.1	41.4 ± .09	41.4 ± .07	0
Average daily mean temp.	40.97	41.40		40.95	40.90	

Table 1 shows the mean body temperatures and standard errors for successive days. Differences between means were tested for significance by the t test. Corrections for heterogeneity of variance were made when the corresponding variances were greatly different.

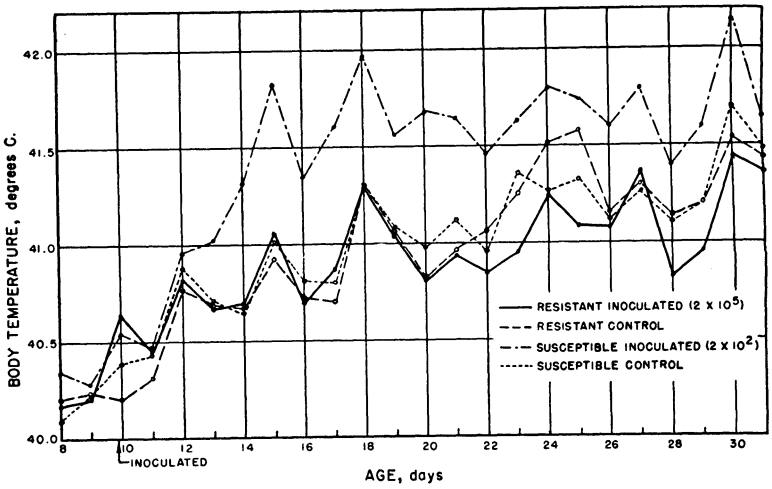

FIGURE 1. CHANGES IN BODY TEMPERATURE DURING FOWL TYPHOID

The graphs in Figure 1 facilitate the interpretation of these data. Before inoculation there were only slight differences between the four groups. Inoculations were made three hours before the temperatures were taken on the tenth day. The significant increase in temperature shown on the tenth day in the inoculated groups was probably due to local inflammation produced in the peritoneal cavity by the bacterial protein. Since the dosage for the resistant chicks was 1000 times that for the susceptible chicks, the higher temperature response shown would be expected. Throughout the rest of the experiment only slight fluctuations occur between the means of the resistant inoculated, resistant controls and susceptible controls. By the third day after inoculation a significant increase in body temperature was evidenced in the susceptible chicks. This fever of slightly less than 1°C. was maintained for a period of thirteen days before gradually declining. All the differences during this period, with one exception, were statistically significant or highly significant. This rise in body temperature was ineffective in combating the disease. During this period only 10% of the inoculated susceptible chicks survived. The variability in body temperature was much greater in this group than in the corresponding controls. This is revealed by the higher standard errors. This could be due to some chicks' responding to infection by developing high temperatures while others do not; or it could be due to a differential susceptibility within the group, whereby the infection develops more rapidly and produces a fever earlier in the more susceptible individuals.

Even though the inoculated resistant chicks were given 1000 times the number of organisms given the susceptible chicks, they failed to develop a corresponding fever. Of those inoculated, 85% survived the test. The resistant chicks were able to wipe out the pathogen without the aid of increased body temperature.

The significant increase in the temperature of the resistant controls during the latter part of the test over that of those inoculated could be rather extreme random fluctuations, or it could be due to some infection, possibly a contact infection with the typhoid organism. Two of the resistant control chicks died during this period; however, *Shigella gallinarum* could not be isolated from them, while it was easily isolated from all inoculated chicks that died.

The daily fluctuations shown by all groups were closely associated with changes in room temperature.

The averages of the daily mean body temperatures for the total period were: Resistant Control—40.95°C.; Resistant Inoculated—40.90°C.; Susceptible Control—40.97°C., and Susceptible Inoculated—41.4°C.—the inoculated susceptible chicks averaging .4 to .5°C. higher than the other groups.

Chicks that succumbed rapidly to the pathogen might be considered less resistant than those that survived for a longer period. Within the inoculated susceptible chicks it was noted that mortality occurred roughly during two periods. Twelve of the chicks died between the sixth and the tenth days after inoculation, while six individuals died between the thirteenth and eighteenth days. The average daily temperatures of the two groups are shown graphically in Figure 2.

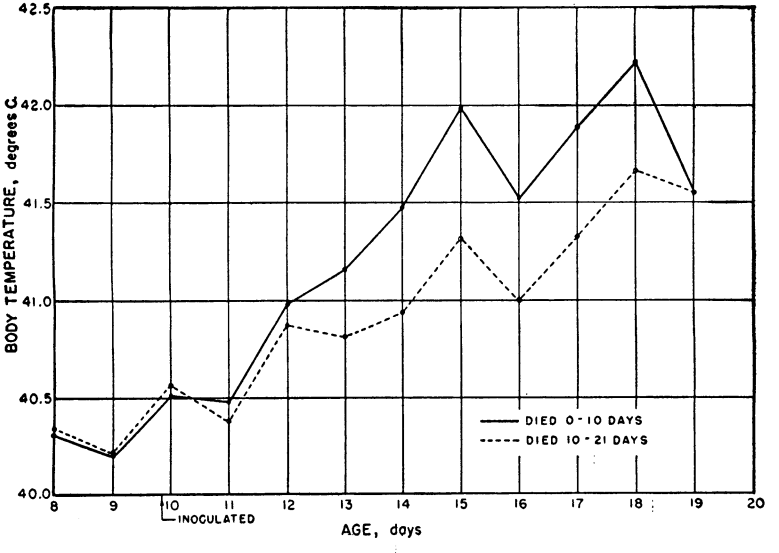

FIGURE 2. COMPARISON OF SUSCEPTIBLE CHICKS DYING WITHIN 10 DAYS AFTER INOCULATION WITH THOSE DYING AFTER 10 DAYS

It can be seen that no difference exists between the means of the two groups until the third day after inoculation. At that time a rise in temperature is evidenced in the early mortality group. The differences exhibited on the fourteenth, fifteenth, and seventeenth days of age are statistically significant. The rise in temperature appears to be associated with the severity of the infection. Two of the susceptible chicks survived the test. They maintained a slightly higher temperature throughout the experiment than those that succumbed; however, the importance of this difference cannot be evaluated until larger numbers are obtained.

Within the inoculated resistant group three chicks died during the period studied. No temperature differences were evidenced between these and the survivors until the onset of the disease. At that time the susceptible individuals showed a marked rise in body temperature, but due to the small number of these individuals the increase in temperature was not statistically significant.

Summary

This paper presents a preliminary study on temperature changes during the course of fowl typhoid, *Shigella gallinarum,* in resistant and susceptible strains of chicks.

The normal temperature of the chick under the conditions of this experiment rises from approximately 40.2°C. at eight days of age to about 41.5°C. at 31 days of age. Its temperature control mechanism is easily influenced by environmental temperature fluctuations.

The normal body temperatures of resistant and susceptible strains of chicks do not differ from the eighth to the thirty-first day of age. During the course of the disease susceptible chicks develop a significant fever, while resistant chicks do not.

The highly efficient mechanism utilized by the resistant strain of chicks in combating the pathogen is not associated with a higher body temperature. If this mechanism is absent in the susceptible strain, the fever produced could be of a defensive nature; however, it is ineffective in controlling the etiological agent of fowl typhoid.

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Abstract: A spectrophotometric study of a developing egg (Orthoptera)with especial reference to riboflavin and its derivatives. (lantern) J. H. Bodine and L. R. Fitzgerald, Zoology Laboratory, State University of Iowa, Iowa City, Iowa.

Spectrophotometric studies of the developing egg of the grasshopper (*Melanoplus differentialis*) show, (a) the presence of riboflavin in the newly-laid egg; (b) no changes in the riboflavin of the developing egg occur until late in prediapause (15th to 19th day of development at 25°C.); (c) differences in the transmittance of the acid (pH 4.5) and alkaline (pH 10) diffusible fractions indicate changes in the original riboflavin content of the eggs; (d) during the diapause or resting developmental period no further changes in the riboflavin are noted; (e) in active postdiapause development changes in the riboflavin increase up to the time of hatching; (f) these changes in riboflavin are thought to be due to the formation of other fluorescing compounds, probably pterines; (g) the fluorescent fraction of the riboflavin is thought to act as a "prosthetic group" for the pterines thus formed.

Abstract: Lethal Effect of some Stylet Cercariae on Various Fishes. F. G. Brooks, Cornell College, Wisconsin Conservation Department, University of Wisconsin.

Incidental to a study being conducted on the cercariae infesting the snails of Carrol Lake, Oneida County, Wisconsin, it has been found that certain Xiphidiocercariae, when in high concentrations, will kill fish of various species. Controlled experiments using five cercariae of the stylet group indicated that the cercariae to which the provisional designations D. S. and Eta have been given pending publication of their descriptions were lethal to sunfish, perch, and large-mouth bass when they were placed in battery jars with from three to forty-eight infested snails, while *C. Gamma* and the cercaria of *Plagiorchis ameiurensis,* McCoy, 1928 were not. The fish were killed in periods of time ranging from three and one-half hours to three and a fraction days. The species of cercaria, the species of fish, and the concentration of the cercariae were factors influencing the time element. *Cercaria D* was found to have the most lethal effect of the three killers. As many as 46,875 *Metacercaria D* were recovered by the digest method from a single sunfish. Either no metacercariae or only small numbers of them were recovered by digest from the other two lethal species, though large numbers could be recovered by dissection. The fish killed by *Cercaria D* showed hemorrhage of the fins, gills, and lips.

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