



susceptible to small numbers of environmental opportunists such as E. coli in inhaled dust. Birds then developed aerosacculitis followed by pericarditis, with associated electrocardiogram changes, sometimes followed by death associated with fever and reduced blood pressure. Losses were greatly reduced by reducing the incidence of Mycoplasmal infection through eradication by breeders.

Within the gut pathogenic serotypes of *E. coli* cause no disease. The only primary *E. coli* diseases are yolk sac infection and embryo death due to soiled eggs. We found that secondary *E. coli* infections were responsible for salpingitis, peritonitis, panophthalmitis, and synovitis.

Research on physiological effects of *E. coli* infection, especially the monitoring of air sac air pressure changes and a rooster that crowed, led to investigating voice production by birds (Physics Dept helped). This resulted in the development of surgical procedures for devocalizing chickens and peafowl.

Some puzzling 2 peaked distributions of test results led to the finding that high levels of stress were associated with increased resistance to: bacterial infections, northern fowl mite, coccidiosis, toxins and the induction of cell mediated immunity as well as increased adaptability. Low levels of stress were associated with resistance to viral, Mycoplasmal and avian tuberculosis infections, tumors and increased levels of antibody response as well as the effectiveness of cell mediated immunity.

Both high and low levels of social stress are detrimental to chickens. An intermediate optimal level of stress results in increased feed efficiency and a much greater expression of genetic traits. Resistance to bacterial infections is near its peak. The response to a single stress begins at 18 hours/ peaks 20 hrs, and reaches normal levels in about 30 hours. Stress during the first 2 weeks of life results in long term effects on subsequent responses. Most are detrimental. Chickens become habituated to all stressors except a disease in progress, social interactions and harsh humans at a major cost of resources.

At an optimal, stress dependent, feed level of deoxycorticosterone or corticosterone there was a large increase in resistance to bacterial infections which is additive to the effects of antibacterial drugs.

Levels of plasmal corticosterone did not correlate well any of our experimental findings. This is because birds differ in their rate of absorption of corticosterone from the blood. This led to the development of the heterophil/lymphocyte (H/L) ratio. As stress or corticosterone levels increase, numbers of heterophils increase while numbers of lymphocytes decrease. Group, but not individual, H/L ratios correlate well with experimental results.

With W. E. C. Moore the anaerobic bacterial pathogenesis of liver granulomas in turkeys was determined. With L. D. Smith the pathogenicity of the various types of botulism toxin was determined for chickens.

With C. H. Domermuth the pathogenesis, spleen pathology and etiology of hemorrhagic enteritis of turkeys was determined. This research led to the development of an injected antibody treatment and to a live virus vaccine as well as a live virus vaccine for marble spleen disease of pheasants.

High levels of stress were found to be a major factor in the development of Marek's disease. The administration of adrenal blocking chemicals resulted in a rapid (less than 10 days) destruction of the tumors by greatly increasing the effectiveness of cell mediated immunity which is inhibited by stress. The mean post treatment time of death for untreated controls was 6 days.

In cooperation with P. B. Siegel lines of leghorn type chickens were selected for high (HC) or low (LC) corticosterone response to social stress, high or low persistence of the antibody response and for high (HA) or low (LA) 6 day antibody response. Among HC and LC lines the stress effects of social interactions was increased or decreased. The response of the lines was similar for all other stressors. This indicated that there is a genetic basis for the severity of the stress response. Lines selected for a low persistence of antibody levels were inferior for extended immunity, susceptibility to *E. coli*, mycoplasma and mites as well as adaptability to new environments. Chickens from the HA line as compared to the LA line had increased immunity and were resistant to mycoplasma and mites. They had reduced body weight and adaptability. This indicated that the production of antibody has a high resource cost.

Chickens which are socialized to their human associates have increased: feed efficiency, resistance to environmental stressors, resistance to all infectious diseases, immunity, and they are much easier to handle.

Under low levels of stress a narrow optimal level of ascorbic acid results in a major increase in the phagocytic defense. At higher levels of stress the optimal dose of ascorbic acid is an excellent blocker of the stress response in the adrenals resulting in increased lymphoid defense and increased resistance to excessive heat and to respiratory infection.

All body functions require resources. The allocation of resources is the result of genetic-environment interactions. Chickens tend to hold some resources in reserve to the detriment of defense. During defense against bacterial infections added resources in the feed such as animal protein can be very helpful.

For all responses the effects of genetics and environment are interdependent. The response of any chicken to any factor are dependent on it's genetics, environment during the first 2 weeks, all subsequent environments, the current environment, nutrition and their relationship to their handlers. Dose of infectious agents and antigens is a major factor in the precision of experiments.

Doing this research was a lot of fun.

*Editors addendum:*

*As Bernie Gross has not included many dates or personal milestones in his statement, a few pertinent items extracted from his cv are included here. He received a DVM from The Ohio State University in 1946, and an MS and PhD from the University of Minnesota in 1952 and 1956, respectively. He served in the Army of the United States 1946-1947. He was a field inspector, USDA, stationed in Mexico 1947-1948. He joined the faculty of Virginia Polytechnic Institute and State University in 1949, serving as Assoc. Prof. of the Department of Biology (1949-1960), Professor, Department of Veterinary Science (1960-1978), and Professor in the Virginia-Maryland Regional College of Veterinary Medicine until his retirement in 1991. He received several awards for research excellence, including the AAAP Research Award (1967), VPI Alumni Award for Research (1977), Upjohn Award for Research in Avian Medicine (1984) and the Beecham Award for Research (1985).*

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*Biography solicited by the Committee on the History of Avian Medicine, American Association of Avian Pathologists.*

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