precipitin production coupled with a marked decrease in the yield of specific agglutinins, is conclusive evidence of the existence of at least 2 different physiological mechanisms for specific-antibody production.

A plausible explanation would be furnished by the hypothesis that there are 2 competitive defensive mechanisms operative in humoral immunity: (i) an extracellular synthesizing process mainly operative against relatively non-toxic alien proteins and responsible for specific-precipitin production, and (ii) an intracellular lytic process mainly operative against phagocyted microörganisms or microbic fragments. Vitamin-C activation of tissue enzymes, therefore, might conceivably increase precipitin-production while reducing the yield of specific agglutinins by causing a more rapid intracellular destruction of phagocyted antigens.

If this is true, vitamin C therapy would have a predictable clinical value in the treatment of specific infectious diseases, even though it is of no apparent clinical promise as an adjuvant in vaccine therapy. Recent reported successes of vitamin C therapy in the treatment of experimental poliomyelitis and tuberculosis are in line with this prediction.

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Cerebrospinal Pressure, Hydrocephalus and Blood Pressure in the Cat Following Intracisternal Injection of Colloidal Kaolin.

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Certain animals develop an increased intracranial pressure associated with vascular hypertension following the intracisternal injection of colloidal kaolin. This syndrome was first described in the dog by Heller[1] and his associates, and recently confirmed by Jeffers, Lindauer and Lukens.[2] A similar response in the white rat

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1 Dixon, W. E., and Heller, H., *Arch. f. exp. path. u. pharm.*, 1932, **166**, 265.

2 Jeffers, W. A., Lindauer, M. A., and Lukens, F. D. W., PROC. SOC. EXP. BIOL. AND MED., 1937, **37**, 260.

has been described by Griffith, Jeffers, and Lindauer,[3, 4] and Griffith and Roberts.[5] They found that the cerebrospinal pressure, normally less that 100 mm of water, was increased to about 260 mm, while the blood pressure, normally below 140 mm of mercury, ranged between 150 and 300 mm. At necropsy internal hydrocephalus was found in rats that had survived a month or more. The present report deals with similar studies in cats.

The method for measuring blood pressure was that described by Griffith and Collins[6] for man and by Griffith[7] for the rat. It consists in encircling the upper part of a limb with a blood pressure cuff constructed of appropriate size, while blood flow is observed through the microscope in a more distal cutaneous area of this same limb. This area usually has to be prepared in advance by shaving. When the pressure in the cuff exceeds systolic pressure the flow in the capillaries stops, to start again when the pressure in the cuff is lowered below systolic pressure. Thus systolic pressure alone is measured.

Blood pressure measurements were made on about 24 normal cats. Ether anesthesia was used throughout. Cisternal puncture was successfully performed on 15 cats. In approximately half of these the pressure of cerebrospinal fluid was measured directly by permitting the fluid to pass into a graduated capillary tube, so narrow that the loss of fluid was only 0.2 cc per 100 mm rise. Then 0.5 cc of cerebrospinal fluid was withdrawn, and a kaolin suspension slightly less in volume was injected. The kaolin suspension consisted of 25% by volume of kaolin boiled up in distilled water and permitted to cool before injecting.

These 15 cats had repeated blood pressure measurements at approximately weekly intervals. Three animals had measurements of cerebrospinal pressure made 3-5 weeks after the injection of kaolin. Six animals were killed and formalin injected into the internal carotid artery immediately after death. The brain was thus fixed *in situ* and subsequently removed.

In the normal cat under ether anesthesia the systolic blood pressure by this method ranges between 85 and 130 mm of mercury, while the cerebrospinal pressure does not exceed 100 mm of water.

3 Griffith, J. Q., Jr., Jeffers, W. A., and Lindauer, M. A., *Am. J. Physiol.*, 1935, **113**, 285.

4 Griffith, J. Q., Jr., Jeffers, W. A., and Lindauer, M. A., *Am. J. Physiol.*, 1937, **118**, 1.

5 Griffith, J. Q., Jr., and Roberts, E., *Am. J. Physiol.*, 1938, **124**, 86.

6 Griffith, J. Q., Jr., and Collins, L. H., Jr., *Am. Heart J.*, 1922, **8**, 671.

7 Griffith, J. Q., Jr., PROC. SOC. EXP. BIOL. AND MED., 1934, **32**, 394.

Following the injection of kaolin into the cistern the blood pressure, as a rule, remained within normal limits. Three readings were obtained ranging between 155 and 165 mm on different animals, but this trifling hypertension had returned to normal by the next week in 2 animals, while the third died before a subsequent measurement had been made.

The cerebrospinal pressure was definitely increased, being 140, 158, and 175 mm of water in 3 animals with consistently normal blood pressures. All animals autopsied showed marked internal hydrocephalus, this being present 4 or more weeks after the injection of kaolin.

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Enzymes in Orthopteran Ontogenesis. VI. Autocatalytic Nature of *in vivo* Formation of Protyrosinase.*

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Northrop[1] considered the autocatalytic formation of pepsin and trypsin from their zymogens as being an instance of protein synthesis. Therefore, since Wrinch's[2] theory described strata of two-dimensional, cyclol-structured lamina, Langmuir and Schaefer[3] point out that protein growth, as with crystal formation, is a determined one and should reasonably be autocatalytic. In the light of these observations, it should be of some general interest to describe the *in vivo* formation of an enzyme precursor. The present paper is concerned with the reporting of the autocatalytic nature of the formation of protyrosinase within eggs of the grasshopper, *Melanoplus differentialis*.

The preparation and activation of grasshopper egg protyrosinase has been dealt with in a series of papers (Bodine and Boell,[4] Bodine, Allen, and Boell,[5] Bodine and Allen[6, 7]) so that here a brief statement

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1 Northrop, J. H., *Physiol. Rev.*, 1937, **17**, 144.

2 Wrinch, D. M., *Roy. Soc.*, 1937, A **160**, 59.

3 Langmuir, I., and Schaefer, U. J., *J. Am. Chem. Soc.*, 1938, **60**, 1351.

4 Bodine, J. H., and Boell, E. J., *J. Cell. and Comp. Physiol.*, 1935, **6**, 263.

5 Bodine, J. H., Allen, T. H., and Boell, E. J., PROC. SOC. EXP. BIOL. AND MED., 1937, **37**, 450.

6 Bodine, J. H., and Allen, T. H., *J. Cell. and Comp. Physiol.*, 1938a, **11**, 409.

7 Bodine, J. H., and Allen, T. H., *J. Cell. and Comp. Physiol.*, 1938b, **12**, 71.