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Title	STUDIES ON ANTI-TUMOR IMMUNITY IN MAREK'S DISEASE AND VACCINAL IMMUNITY CAUSED BY HERPESVIRUS OF TURKEY IN CHICKENS
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by acetylcholine ($5\times10^{-8}\sim5\times10^{-7}$ M). The inhibitory effects increased with increasing the concentrations, thus, the concentration producing 50% inhibition was 10^{-7} M for adrenaline and clonidine, 10^{-6} M for noradrenaline, and 5×10^{-5} M for dopamine.

3) Phenylephrine poorly inhibited the response to stimulation of the vagus nerve at 2.5×10^{-6} M.

4) Isoproterenol ($5\times10^{-8}\sim5\times10^{-7}$ M) inhibited both the responses induced by stimulation of the vagus nerve and acetylcholine.

5) Clonidine failed to inhibit the response induced by stimulation of the vagus nerve with a higher frequency, such as 8 or 16 Hz.

6) The inhibitory effects of adrenaline, noradrenaline and clonidine were blocked by phentolamine (2.7×10^{-6} M) but not affected by 5-(3-tert-Butylamino-2-hydroxy) propoxy-3, 4-dihydrocarbostyril hydrochloride (OPC 1085), which was effective in blocking the effect of isoproterenol.

7. Clonidine also inhibited the response to transmural stimulation at 0.5 Hz in the presence of hexamethonium. However, the rate of inhibition was smaller than that of the response induced by stimulation of the vagus nerve.

These results suggest that presynaptic α-receptors are present in the myenteric plexus of the chick proventriculus and play an important role in controlling the cholinergic transmission. It seems likely that adrenaline, noradrenaline, and clonidine may inhibit the acetylcholine output induced by the vagal and transmural stimulation via the activation of the α-receptor, and may result in the inhibition of the contraction.

STUDIES ON ANTI-TUMOR IMMUNITY IN MAREK'S DISEASE AND VACCINAL IMMUNITY CAUSED BY HERPESVIRUS OF TURKEY IN CHICKENS

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These experiments were made to clarify the anti-tumor immunity in Marek's disease (MD) and the vaccinal immunity caused by herpesvirus of turkey (HVT) in chickens. The cytotoxic effects of peripheral blood lymphocytes (PBL) and sera from chickens infected with Marek's disease virus (MDV) or vaccinated with HVT against MD derived lymphoblastoid cell line (MSB-1) cells were studied.

The cytotoxic effects of anti-MSB-1 chicken sera from chickens hyper-immunized with inactivated MSB-1 cells were also tested. In the complement dependent antibody cytotoxicity (CDAC) test, the complement activities of sera from several animal species were compared. Furthermore, antisera to MD derived cell line (MSB-1 and RPL-1) cells were prepared in rabbit and tested for their specificities and cross-reactivities to the surface antigens of both cell line cells.

The results obtained were as follows:

1) The cytotoxic effect of PBL from MDV infected chickens against MSB-1 cells was detected by the lymphocyte cytotoxicity (LC) test.
2) The cytotoxic effect of PBL from HVT vaccinated chickens against the MSB-1 cells was not detected by the LC test.
3) In the CDAC and antibody dependent cell-mediated cytotoxicity (ADCC) tests, the cytotoxic effects of sera from MDV infected or HVT vaccinated chickens against MSB-1 cells were not detected.
4) The cytotoxic effects of anti-MSB-1 chicken sera against MSB-1 cells were detected by both the CDAC and ADCC tests.
5) In the CDAC test using anti-MSB-1 chicken serum and MSB-1 cells, the duck complement showed satisfactory activity for the test when it was used alone or together with the rabbit complement.
6) In the CDAC and membrane fluorescent antibody tests, anti-MSB-1 and anti-RPL-1 rabbit sera reacted specifically with the corresponding MSB-1 and RPL-1 cells, respectively; however, these antisera did not react with the heterologous cell line cells.

STUDIES ON THE ANTIGENIC DETERMINANT OF SEROVAR-SPECIFIC ANTIGEN OF *LEPTOSPIRA INTERROGANS* SEROVAR *COPENHAGENI* STRAIN SHIBAURA

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Nondialyzable delipidized type-specific main antigen (NDTM antigen) was prepared from the organisms of *Leptospira interrogans* serovar *copenhageni* strain Shibaura.

The NDTM antigen of *copenhageni* Shibaura showed an inhibition of the complement fixation between the type-specific main (TM) antigen of strain