A Possible Link Between the Pulmonary and Urinary Tract IgA Response*

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Using as a model ascending pyelonephritis in rats caused by E. coli O6, we investigated the antibody response in serum as well as in bronchial lavage.

Nine rats immunized orally with live E. coli O6 two weeks before injection of the same bacteria into the urinary bladder gave rise to anti-O6 antibodies of the IgG, IgM and IgA classes in both serum and bronchial washings one week after the injection. Only two of eight animals not pre-immunized orally gave rise to IgM and IgA antibodies in serum and bronchial washings after bladder injection. The preimmunized rats showed a higher anti-O6 IgA titer in the bronchial washings than the not pre-immunized ones.

The anti-O6 antibody levels in serum and bronchial lavage were determined in infected rats killed two to four months after the introduction of bacteria into their bladders. A significant correlation was found for IgG and IgM antibodies (P < 0.02 and < 0.001) but not for the IgA antibodies (P < 0.1) comparing serum and bronchial washings. The IgG anti-O6/IgA anti-O6 ratios were higher in serum than in bronchial lavage. This indicates the possibility that some of the IgA antibodies appearing in the lung may be produced locally or that serum IgA antibodies more readily diffuse into the air spaces of the lung than IgG or IgM antibodies.

These observations suggest that IgA antibodies in the lung may be part of a local antibody response transferred via a "homing" mechanism for IgA producing lymphocytes. A transport of E. coli antigens via the blood to the pulmonary lymphoid tissue seems less likely. Further experiments are required to substantiate this extension of the homing mechanism hypothesis which would agree with the findings of Rudzik et al and Bienenstock et al in that lymphoid cells from various sources as BALT, GALT and mesenteric lymph nodes are "homing" to various mucosal sites.

REFERENCES


DISCUSSION

Dr. Richerson: How did you compare serum antibody levels to those in bronchoalveolar lavage fluids?

Dr. Mattsby: We compared the relation between antibody levels of IgA, IgG and IgM classes in serum to the respective antibody levels in bronchoalveolar fluid. We also compared the ratios of the antibody titres of IgG class to the antibody titres of IgA or IgM in serum with the same ratios in bronchoalveolar fluid.

Dr. Richerson: Dr. Mattsby, where are the urinary antibodies being produced and how do they get into the bronchial fluid without being detected in the blood?

Dr. Mattsby: The urinary antibodies are probably mainly of local origin, and we have found antibodies of all three Ig classes in the urine of rats. The local formation may perhaps partly be attributed to the lymphoid cells and cell aggregates observed in the submucosa of the rat urinary bladder. The submucosal lymphoid cells may also function in a similar mode as has been reported for lymphoid cells from Peyer’s patches or BAL in that the cells may migrate to other submucosal sites of the body giving rise to preferentially IgA producing lymphocytes. Intestinal colonization of pregnant women with a bacterial strain give rise to antibodies in their milk but not in serum, maybe also indicating migration of antigen-stimulated lymphoid cells. IgA antibodies could not be detected in serum at the dilution used as compared to our reference sera.