THE LACK OF ASSOCIATION OF $\theta$ STATUS AND MURINE LEUKAEMIA VIRUS CONTENT IN THE AKR

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Summary.—Two AKR sublines appear atypical in possessing $\theta^{\text{C}3\text{H}}$. One of these two sublines—AKR/FuA—is notably resistant to lymphomata and is also characterized by reduced levels of the group specific murine leukaemia viral (MuLV) antigen. This suggested a possible association between $\theta$ status tumour susceptibility and viral content. Results here show no reduction in viral antigen titres in the other $\theta^{\text{C}3\text{H}}$ tumour susceptible subline AKR/Cum, thus eliminating the possible association of $\theta$ status with the extent of MuLV replication.

Acton and his colleagues initially drew attention to the existence of 2 AKR sublines possessing $\theta^{\text{C}3\text{H}}$ rather than the normally characteristic $\theta^{\text{AKR}}$ (Acton et al., 1973). These two sublines, namely AKR/FuA and AKR/Cum, also appeared atypical in other aspects since both were alleged to be relatively resistant to lymphomata (Acton et al., 1973). Recently we showed, however, that this was not the case in the AKR/Cum which has an incidence of lymphomata comparable with the susceptible AKR/J (Barnes, unpublished data). Therefore, having upon the basis of this finding dismissed the possible association between $\theta$ status and lymphoma susceptibility, we have sought to learn if there was any direct association between $\theta$ status and viral content in the AKR/Cum since Acton (Acton et al., 1973) had earlier noted that levels of the group specific murine leukaemia viral (MuLV) antigen were notably less in the $\theta^{\text{C}3\text{H}}$ tumour resistant AKR/FuA.

AKR/Crc has been described previously (Barnes et al., 1975), and preliminary findings suggest that the AKR/Cum is equally susceptible. The differing $\theta$ status of the 2 sublines has also been confirmed earlier (Barnes unpublished data).

Investigation.—Various tissues were obtained from the mice at different ages and MuLV/gs titration was performed on soluble extracts according to the technique of Hilgers (Hilgers et al., 1972). This technique involves indirect immunofluorescent absorption and is performed in 2 stages. In the first stage the specific anti-MuLV-gs serum is titrated against target AKR-A lymphoma cells (Woods et al., 1970). The second titration of the same antiserum is then performed after absorption with soluble antigens obtained in the case of solid tissues following ultrasonic disintegration. The gs-antigen titre was then expressed as the reciprocal of the reduction in antibody titre following absorption.

DISCUSSION

The incidence of "spontaneous" lymphomata is known to vary in the AKR and this remains unexplained. The recent description of 2 sublines possessing $\theta^{\text{C}3\text{H}}$ and the fact that both were allegedly lymphoma resistant (Acton et al., 1973) led us to question the possible association

MATERIALS AND METHODS

Mice.—AKR/Cum mice were obtained directly from Cumberland Farms and compared with our AKR/Crc which were originally derived from AKR/J.

The incidence of lymphomata in the
between lymphoma susceptibility and $\theta$ status. Since the AKR/Cum are not resistant to lymphomata this rules out this possible association (Barnes, unpublished data).

The fact that the titres of viral antigen in the AKR/FuA are notably less than in the AKR/J (Acton et al., 1973) also raised the possible direct association between viral replication and $\theta$ status. This association is ruled out by the findings here in the $\theta^{c}^{AH}$ AKR/Cum, leaving the possibility that another factor influences viral replication and that this in turn might effect lymphoma susceptibility.

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