

The Graduate School of Medical Sciences
Training and Educational Program for Eradication of AIDS Course
- Practice on AIDS VI -

Center for AIDS Research Seminar

Friday, 2nd June 2017, 15:00-17:00
2F Seminar Room, Center for AIDS Research

Dr. Maureen Patricia Martin

Talk 1, 15:00-16:00

Senior Scientist, Leidos Biomedical Research, Inc., NCI-Frederick, USA

Partnership of HLA-B*57 and KIR3DL1 in control of HIV

HLA-B*57 is well known to associate with control of HIV, largely due to enhanced CD8+ T cell responses to infected cells. There is, however, extensive heterogeneity in control of HIV among B*57+ individuals. We sought to identify genetic modifiers of B*57 using whole genome sequencing. A valine to isoleucine substitution at amino acid 47 (I47V) in the KIR3DL1 gene was the only variant to reach genome wide significance, with valine conferring protection. This was confirmed in an independent cohort and replicated across multiple outcome measures. These data implicate KIR3DL1 in modifying the effect of B*57 in control of HIV.

Dr. Xiaojiang Gao

Talk 2, 16:00-17:00

Senior Scientist, Leidos Biomedical Research, Inc., NCI-Frederick, USA

HLA/KIR Association with NPC in Southern China and Taiwan

Nasopharyngeal carcinoma (NPC) is an epithelial malignancy that is strongly associated with EBV infection and is known to be associated with HLA. We have shown that the previously described protective effect of HLA*A11 on NPC is entirely attributed to A*11:01, whereas the less common A*11:02 shows no association. The two A*11 allotypes share identical amino acid sequences except for a single difference at position 19, a location well outside of the peptide binding region. Studies have shown that A*11:02 binds more efficiently to the inhibitory KIR3DL2 and the activating KIR2DS4 than does A*11:01. We show here that differential binding of A*11:01 and A*11:02 to KIR may explain their disparate associations with NPC risk.

Organized by

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