

The impact of latent CMV infection on NK-cell mobilization and expression of KLRG1 and CD57 in response to acute exercise.

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Natural killer (NK) cells are cytotoxic effectors of the innate immune system that are able to distinguish healthy autologous cells from tumors and virally infected cells. NK-cells kill the targeted cells by releasing cytotoxic proteins, a process that is governed by inhibitory surface receptors, such as KLRG1. Additionally, activated NK-cells are able to proliferate in response to immunological stimuli, a process that is inhibited in NK-cells expressing the senescence marker CD57. Acute bouts of exercise are known to mobilize NK cells into the blood compartment, which could alter immunity; however, whether or not exercise alters NK-cell KLRG1 and CD57 expression has not been fully elucidated. Furthermore, as latent CMV infection is associated with an increased frequency of inhibitory NK cells, it is not known if CMV status influences NK-cell mobilization in response to acute exercise. **PURPOSE:** To examine the impact of latent CMV infection on the mobilization of NK-cells and their expression of KLRG1 and CD57 in response to acute exercise. **METHODS:** Otherwise healthy CMV seropositive (CMV+) and CMV seronegative (CMV-) males (age 23-35 years) completed a 30-min cycling protocol at 85% of maximum power. Lymphocytes isolated from whole blood before, immediately after, and one hour after exercise were surface-stained with monoclonal antibodies against CD3, CD56, KLRG1 and CD57 and analyzed by 4-color flow cytometry. **RESULTS:** Preliminary analysis of the data show a prodigious increase in the number of CD56 dim (mature, highly cytotoxic subset) NK-cells immediately after exercise in all subjects, which subsequently fell below pre-exercise values 1 hour later. In CMV- subjects, the proportion of CD56 bright (immature, mildly cytotoxic) NK cells was considerably higher 1 hour post-exercise than before exercise, but the number of cells changed very little suggesting that the increased proportion was due merely to the egress of CD56 dim NK cells. Interestingly, CMV seropositivity was associated with a near complete absence of CD56 bright NK cells that was unaffected by exercise. Neither exercise nor CMV status influenced the proportion of NK-cells expressing KLRG1 or CD57. **CONCLUSION:** Preliminary analysis of this data indicates that acute exercise preferentially mobilizes CD56 dim NK cells without altering KLRG1 and CD57 expression. Latent CMV infection is associated with a lowered proportion of CD56 bright NK-cells; however, the NK-cell response to exercise was not influenced by CMV status. Future work will examine the role of aging on NK-cell response to exercise and CMV status.