

## Letter to the Editor

### Resonance assignments of the 34 kD rabbitpox vCCI:human MIP-1 $\beta$ complex

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Chemokines play important roles in both the immune response and in inflammation processes. The pox-virus encoded viral CC chemokine inhibitor (vCCI) tightly binds many members of the CC chemokine subfamily, effectively inhibiting host chemokine actions. A protein–protein complex was formed between rabbitpox vCCI and the  $^{45}\text{AASA}^{48}$  mutant of the human CC chemokine MIP-1 $\beta$ . Here we report the  $^1\text{H}$ ,  $^{13}\text{C}$ ,  $^{15}\text{N}$ , CO, and  $\text{C}^{\alpha\beta}$  chemical shift assignments for vCCI, and complete  $^1\text{H}$ ,  $^{13}\text{C}$ ,  $^{15}\text{N}$  chemical shift assignments for MIP-1 $\beta$ – $^{45}\text{AASA}^{48}$  when both proteins are bound to each other. The residues with no assignment information available correspond to loop regions and/or show conformational flexibility, namely residue 1 for MIP-1 $\beta$ – $^{45}\text{AASA}^{48}$  and residues 1–16, 29, 109–110, 126, 147, 152, 171, 207, 231, 235 for vCCI. For this work, [ $^2\text{H}/^{13}\text{C}/^{15}\text{N}$ ]-labeled vCCI and [ $^{13}\text{C}/^{15}\text{N}$ ]-labeled MIP-1 $\beta$ – $^{45}\text{AASA}^{48}$  were used to form complexes with their unlabeled binding partners.

The NMR chemical shifts were deposited in the BMRB (<http://www.bmrb.wisc.edu>) under the Accession Code 7024.

References: Seet BT and McFadden G (2002) *J. Leukocyte Biol.* **72**(1), 24–34

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