Corrigendum

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Ming Q, Gonzalez-Perez D and Luca VC. Molecular engineering strategies for visualizing low-affinity protein complexes. *Exp Biol M*. Epub ahead of print: DOI: 10.1177/1535370219855401.

In this article, the peptide in the crystal structure was incorrectly labelled in the legend for Figure 3. The correct legend is provided below:

Sortase and SpyCatcher ligation for tethering protein complexes. (a) Schematic describing the sortase ligation reaction. The sortase transpeptidase enzyme covalently links peptides harboring a C-terminal LPXTG motif (where X is any amino acid) with N-terminal polyglycine sequences. (b) Crystal structure of the peptide:MHCII:DM "peptide selection" complex, PDB ID: 4GBX. A disulfide bond was introduced between the P6 position of a truncated HA peptide (GKQNCLKLAT) and residue 65 of the MHCII a-chain to prevent dissociation of the peptide (orange, stick representation) from the MHCII molecule (blue) and sortase ligation was used to enforce low-affinity interactions between the ECDs of MHCII and DM (red). To facilitate the sortase reaction, an LPATG sequence was fused to the C-terminus of the MHCII b-chain and a peptide with a polyglycine N-terminus was conjugated to the C-terminus of the DM b-chain. (c) Crystal structure of PlexinC1 (blue) bound to human Rap1B-GDP (brown), stabilized through sortase ligation. In this case, an LPTEG motif was attached to Rap-GDP C-terminus and a GG sequence was introduced at the N-terminus of PlexinC1, PDB ID: 4M8N. (d) Schematic describing the formation of an isopeptide bond between a Lys residue of SpyCatcher Partner (green) and an Asp residue of SpyTag (magenta), PDB ID: 4MLI. (e) The successful reconstitution of asymmetric WT:mutant zTRAP1 heterodimers was achieved using SpyCatcher. WT protomers (orange) with C-terminal SpyTags (magenta) were mixed with R417A mutant protomers (grey) with C-terminal SpyCatcher partners (green) to stabilize the WT:R417A zTRAP1 complex for co-crystallization PDB ID: 5TTH.

On page 6, the peptide in the crystal structure was incorrectly labelled in the following paragraph:

Reconstitution of a stable MHCII:CLIP: DM complex for structural studies is difficult for two reasons: (1) the binding of DM to MHCII induces dissociation of CLIP and (2) the soluble extracellular domains (ECDs) of DM and MCHII bind weakly in the absence of their transmembrane domains. To solve the first problem, the authors used disulfide trapping to prevent CLIP dissociation from the MHCII peptide-binding groove. To solve the second problem, the authors developed a sortase-based method to link the C-termini MHCII and DM, thereby mimicking their parallel orientation in the membrane. This atypical C-to-C linkage was generated by fusing a C-terminal MHCII sortase tag to the N-terminus of a polyglycine peptide that was conjugated to DM (Figure 3(b)). Following covalent stabilization, MHCII:CLIP:DM was crystallized and the structure revealed how DM-induced rearrangements drive peptide release and stabilize the empty MHCII groove.

The corrected paragraph is provided below:

Reconstitution of a stable peptide:MHCII:DM complex for structural studies is difficult for two reasons: (1) the binding of DM to MHCII destabilizes peptide binding and (2) the soluble extracellular domains (ECDs) of DM and MCHII bind weakly in the absence of their transmembrane domains. To solve the first problem, the authors used disulfide trapping to prevent a partial HA peptide from dissociating from the MHCII peptide-binding groove. To solve the second problem, the authors developed a sortase-based method to link the C-termini MHCII and DM, thereby mimicking their parallel orientation in the membrane. This atypical C-to-C linkage was generated by fusing a C-terminal MHCII sortase tag to the N-terminus of a polyglycine peptide that was conjugated to DM (Figure 3(b)). Following covalent stabilization, the peptide:MHCII:DM complex was crystallized and the structure revealed how DM-induced rearrangements drive peptide release and stabilize the empty MHCII groove.