

Mannes M, Dopler A, Zolk O, et al. Complement inhibition at the level of C3 or C5: mechanistic reasons for ongoing terminal pathway activity. *Blood*. 2021;137(4):443-455.

In [Figure 1A](#) on page 445, an error was introduced during the publication process. The green label "AP" should be "CP." The corrected [Figure 1A](#) is shown below.

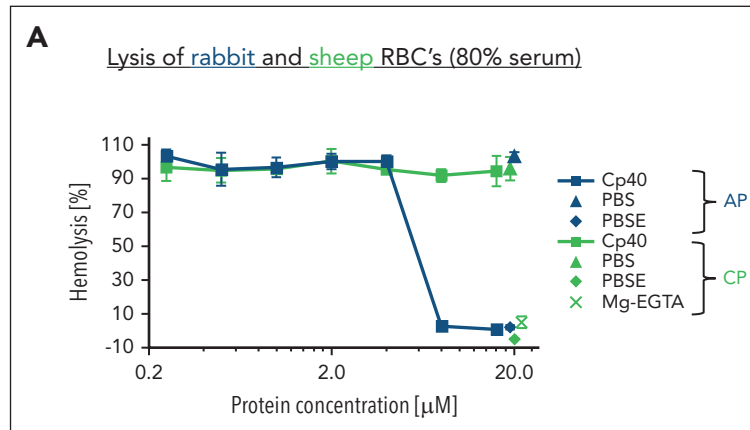


Figure 1. Strong CP activation leads to hemolysis despite C3 peptide inhibitor Cp40. (A) CP- and AP-mediated lysis in presence of Cp40. A total of 80% NHS naturally containing antibodies against the Forssman antigen (which is present on sRBCs) was mixed with Cp40 to obtain final Cp40 concentrations of 0.25 to 16 μ M, as indicated. AP activity was determined by mixing rabbit erythrocytes with 80% NHS supplemented with 5 mM Mg-EGTA in the presence of the same range of Cp40 concentrations. A serum mix with phosphate-buffered saline (PBS) instead of Cp40 served as positive control, whereas 5 mM EDTA and 5 mM Mg-EGTA dissolved in PBS (in case of CP; for each final concentration) served as negative controls (PBSE, PBS containing EDTA). Released hemoglobin was measured as a marker of hemolysis (average of 3 independent assays with standard deviation [SD] is shown).

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