Correction to "Ca^{2+} regulation of inositol 1,4,5-trisphosphate receptors: can Ca^{2+} function without calmodulin?"

In the above article [Rossi AM and Taylor CW (2004) *Mol Pharmacol* **66:**199–203], Fig. 1 was inadvertently printed in black white. The color figure appears below. The online version has been corrected in departure from the print version.

We regret this error and apologize for any confusion or inconvenience it may have caused.

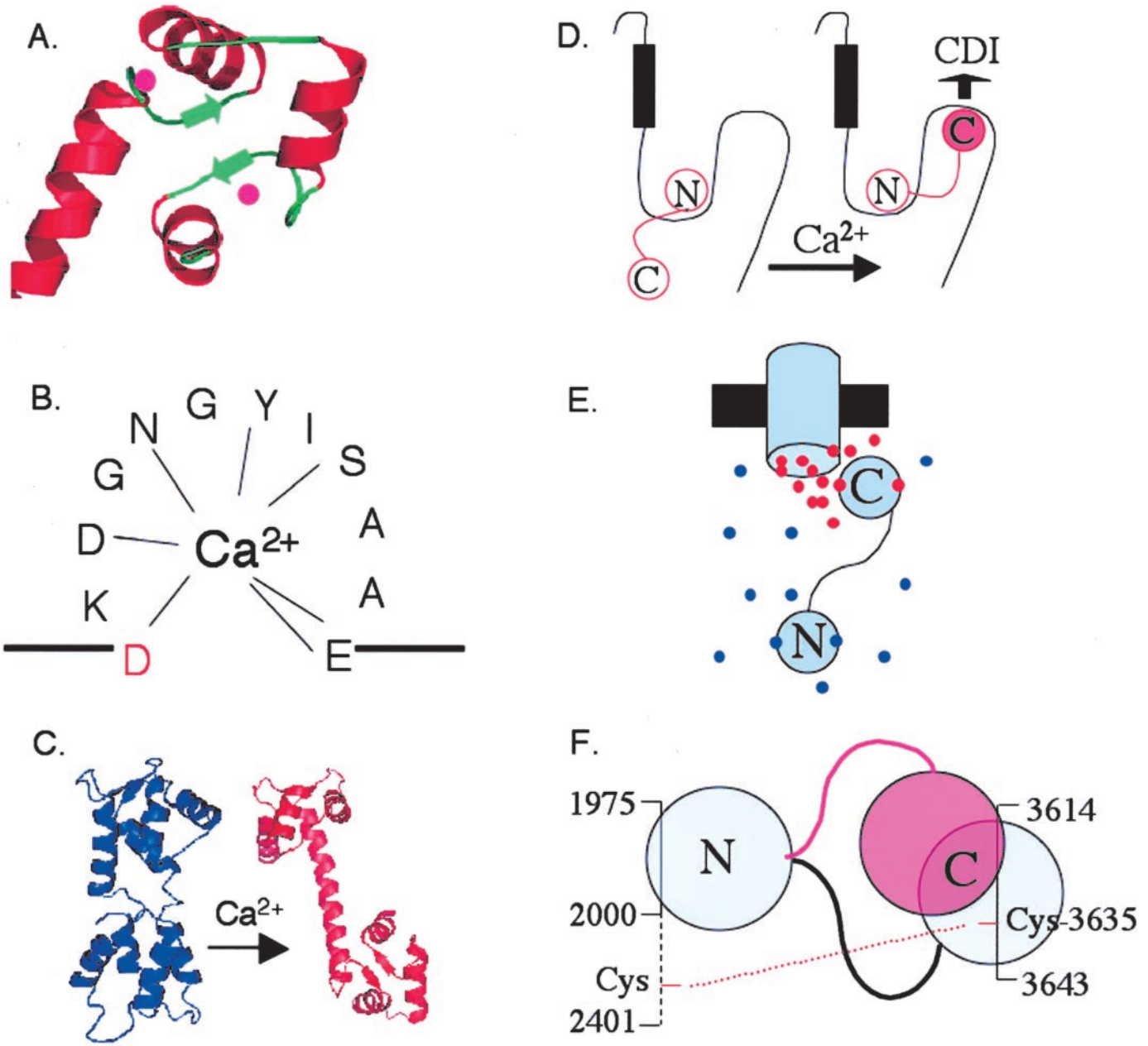

Fig. 1. A–C, Ca^{2+} binding to CaM. A, structure of the C-terminal lobe of CaM with Ca^{2+} bound (PDB code 4CLN). B, Ca^{2+}-binding loop of the third EF hand of CaM; the residue mutated to produce CaM with much reduced affinity for Ca^{2+} is shown in red. C, structures of apoCaM (blue; PDB code 1CFC) and Ca^{2+}-CaM (red; PDB code 3CLN). D–F, regulation of Ca^{2+} channels by CaM. D, CaM tethered in the C-terminal tail of the L-type Ca^{2+} channels binds Ca^{2+} and thereby acquires the ability to interact with a second CaM-binding site through which CDI is initiated. E, for non–L-type Ca^{2+} channels the two lobes of Ca^{2+} are positioned to respond to different Ca^{2+} signals, the C-lobe preferentially detects Ca^{2+} (red) passing through the channel, whereas the N-lobe responds to global Ca^{2+} signals (blue). F, in RyR1, the tethered C-lobe of CaM moves toward the N-terminal of a CaM-binding region when it binds Ca^{2+} (pink), whereas the N-lobe interacts with a site on a neighboring subunit. The C-lobe provides the essential Ca^{2+} sensor and its movement leads to channel inhibition, possibly by causing rearrangement of the interactions between the subunits. The Cys residues that also mediate cross-linking of subunits are also shown.