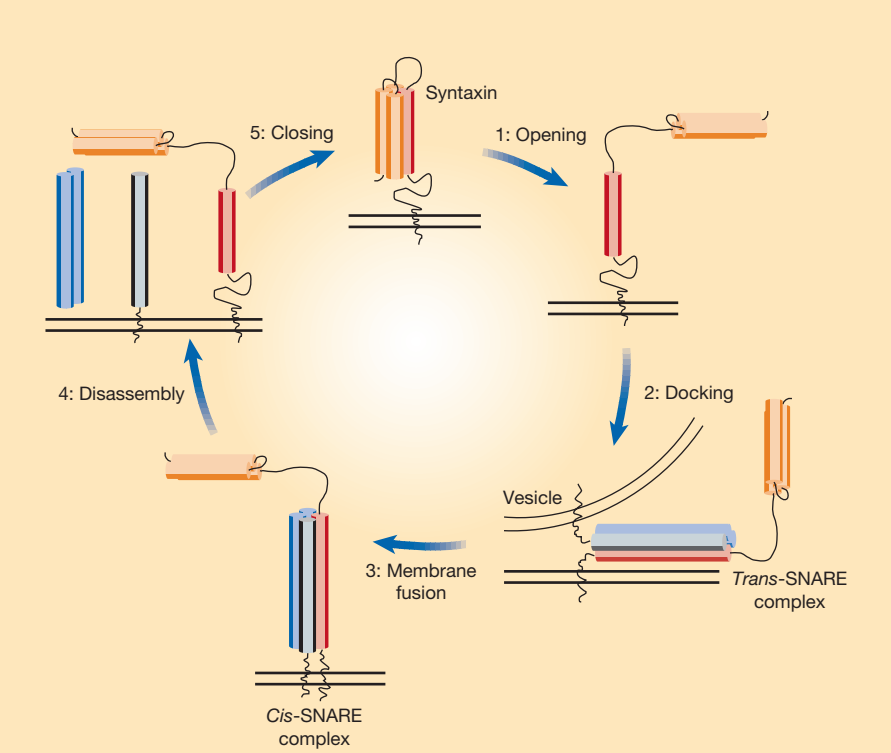

Figure 1 **A simplified model for membrane fusion, showing SNARE-complex assembly and conformational changes in syntaxin. Step 1, opening. Syntaxin, a SNARE protein on the target membrane, undergoes a structural change from a 'closed' to an 'open' form. In the open form, the inhibitory domain (orange) is dislodged from the H3 helix (red). Step 2, docking. The vesicle bears a vesicle-SNARE (grey), which assembles with syntaxin's H3 helix and another target-membrane SNARE (blue) to form a '*trans*'-SNARE complex, pinning the vesicle to the target membrane. Step 3, membrane fusion. After membrane fusion, the vesicle- and target-membrane SNAREs span the same membrane in a '*cis*'-SNARE complex. Step 4, disassembly. SNAREs are separated and recycled for further rounds of fusion. Step 5, closing. The inhibitory domain of syntaxin binds to its H3 helix to form the closed conformation. In this form, syntaxin cannot assemble into a SNARE complex. Sec1 (not shown here) is essential for membrane fusion, but the point at which it acts is unknown. Misura *et al.*[1], from their structure of neuronal Sec1 (nSec1) and syntaxin 1a, propose that nSec1 holds syntaxin 1a in an inactive conformation, and that the binding of other factors to nSec1 may induce it to change conformation itself, so releasing syntaxin 1a in an open form. nSec1 bound to syntaxin may also prevent the reassembly of *cis*-SNARE complexes after their disassembly.**

change its conformation and release open syntaxin 1a, as described above. Yeast syntaxin, however, may not require an extra inhibitor if its inhibitory domain is sufficient to favour the closed conformation. For this reason, yeast Sec1 may not pair with syntaxin as an inhibitor. Consistent with these ideas, SNARE-complex assembly is limited by the rate of syntaxin opening in both yeast and neurons; this rate appears to be at least an order of magnitude slower for the yeast syntaxin homologue[5] than for its neuronal counterpart[4] *in vitro*.

Although the details of the interaction between nSec1 and syntaxin 1a are now clear, the purpose of this partnership has not been resolved. Sec1's function in vesicle fusion has yet to be defined biochemically. But for now, ideas about Sec1 should take into consideration the evidence that Sec1 proteins can bind to the four-helix bundle of syntaxin and perhaps also to the four-helix bundle of an assembled SNARE complex. In the end, syntaxin may not be the only one that is changing dance partners. ■

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