Translocation trumps receptor-binding in colicin entry into *E. coli*.

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Of the steps involved in the killing of *E. coli* by colicins, binding to a specific outer membrane receptor was the best-understood and earliest-characterized. Receptor binding was believed to be an indispensible step in colicin intoxication, coming before the less well-understood step of translocation across the outer membrane to present the killing domain to its target. In the process of identifying the translocator for colicin Ia, I created chimeric colicins, as well as a deletion missing the entire receptor-binding domain of colicin Ia. The normal pathway for colicin Ia killing was shown to require two copies of Cir—one that serves as the primary receptor and a second copy that serves as translocator. The novel Ia colicins retained the ability to kill *E. coli*, even in the absence of receptor binding, as long as they could translocate via their Cir translocator.

Experiments to determine whether colicin M uses a second copy of its receptor, FhuA, as its translocator were hampered by precipitation of colicin M chimeras in inclusion bodies. Nevertheless, I will show that receptor binding can be bypassed for killing, so long as a translocation pathway is maintained for colicin M. These experiments suggest that colicin M, unlike colicin Ia, may normally use a single copy of FhuA as both its receptor and its translocator.

Colicin E1 can kill in the absence of receptor binding, using translocation through TolC.