Swimming with sperm

A putative calcium channel specific to sperm tails may be the best target yet for a male contraceptive. The protein, dubbed CatSper, was discovered in a homology search by David Clapham and colleagues of Harvard Medical School, Boston, MA.

CatSper looks like a calcium channel, although Clapham could not detect a calcium current in transfected cells, probably because another component of the channel is missing. CatSper is, however, required for a calcium influx into sperm that is triggered by cyclic nucleotides. Such signaling events may be part of the process by which sperm either gain their initial motility in the epididymis, or augment that motility (in a process called hyperactivation) into the wall of the uterus. But then CRH, as detailed by George Chrousos (National Institutes of Health, Bethesda, MD) and colleagues have found that the cells that help an embryo implant also kill off some of the mother’s T cells—the ones that might otherwise reject the embryo as foreign.

The pathway begins with corticotropin-releasing hormone (CRH). As a stress hormone in the body, CRH is first proinflammatory (via mast cell degranulation) and then antiinflammatory (via effects in the brain that induce cortisol production). A similar sequence of events may take place during pregnancy. An inflammatory process is needed to initiate implantation of the embryo but for now he has established another explanation. Mutants lacking Swi6 maintain cohesion on chromosome arms during a mitotic block but lose cohesion at the centromeres thanks to the loss of a cohesin subunit. “We knew before that mutants in Swi6 lost chromosomes at a high rate and displayed a high frequency of lagging chromosomes on anaphase spindles,” says Allshire. But the explanation for what Swi6 and heterochromatin are doing at the centromere is first proinflammatory (via effects in the brain that induce cortisol production).

“The problem with going after a male contraceptive is that you have millions of sperm and you have to get them all,” he says. “Amazingly enough, when this channel was gone they got no fertilization. That makes it reasonably attractive as a drug target.”

Reference: Bernard, P., et al. 2001. Nature. 413: 603–609.

Killing for love

Embryos are at least half foreign to their mothers. Now, George Chrousos (National Institutes of Health, Bethesda, MD) and colleagues have found that the cells that help an embryo implant also kill off some of the mother’s T cells—the ones that might otherwise reject the embryo as foreign.

Blocking CRH (left) results in fewer pregnancies unless T cells are absent (bottom).

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