Ovastacin cuts off sperm binding

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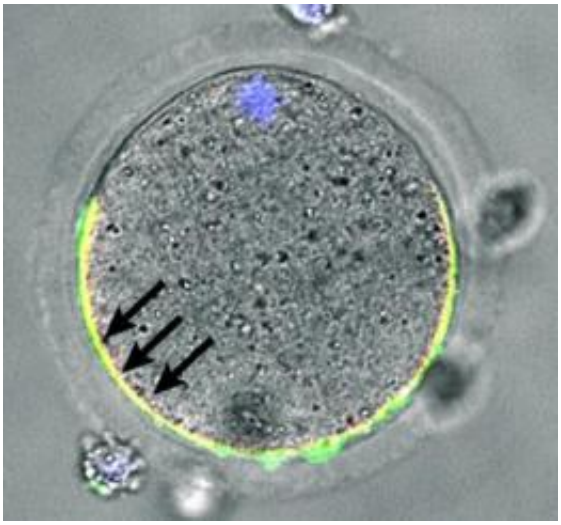

A study in the *Journal of Cell Biology* describes how ovastacin helps egg cells avoid being fertilized by more than one sperm. In this image, ovastacin (red) localizes to secretory granules (green) in the cortex of an unfertilized egg. Credit: Burkart, A.D., et al. 2012. J. Cell Biol. http://dx.doi.org/10.1083/jcb.201112094.

A study in *The Journal of Cell Biology* describes how a secreted enzyme helps egg cells avoid being fertilized by more than one sperm.

Because polyspermy disrupts embryonic development, oocytes take several steps to ensure they only fuse with a single sperm. One key step is to prevent additional sperm from binding to the surface of an already-fertilized egg, a blockade that involves the release of secretory granules and cleavage of a protein called ZP2, a component of the zona pellucida matrix that surrounds eggs. ZP2 is cleaved at a site targeted by the

astacin family of metalloendoproteases – enzymes that cut proteins into smaller fragments. Researchers from the NIH's National Institute of Diabetes and Digestive and Kidney Diseases therefore investigated the function of ovastacin, an astacin family member expressed in oocytes.

Ovastacin localized to cortical granules that were exocytosed after fertilization, and recombinant ovastacin cleaved ZP2 when added to zonae pellucidae. Mice lacking ovastacin failed to cleave ZP2 after fertilization, allowing sperm to continue to bind to the surface of early embryos. Female mice lacking ovastacin had slightly fewer offspring than wild-type animals but otherwise appeared normal.

The researchers found that ovastacin targeted several sites in ZP2. Senior author Jurrien Dean now wants to investigate how this proteolysis blocks sperm binding, a critical question because the molecular interactions between sperm and egg cells remain unknown. He also wants to examine how ovastacin is packaged into oocyte cortical granules and to identify other components of these secretory organelles.

More information: Burkart, A.D., et al. 2012. *J. Cell Biol.* dx.doi.org/10.1083/jcb.201112094

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