In This Issue

SUN proteins melt the nuclear envelope

Turgay et al. reveal that SUN proteins help disassemble the nuclear envelope at the start of mitosis and promote assembly of the mitotic spindle during metaphase.

Compared with a control cell (left), the nuclear envelope (green) remains associated with mitotic chromosomes (red) in a cell lacking SUN1 and SUN2 (right).

Dynamin loss bulks up endocytosis

Kasprowicz et al. reveal that, in addition to its role in vesicle fission, dynamin helps recruit clathrin to the plasma membrane during the earliest stages of synaptic vesicle formation.

Compared with control cells (left), the junctions (green) that connect Sertoli cells to apical spermatids are disorganized in tests lacking TAp73 (right).

p73 helps developing sperm stick to the right path

The transcription factor p73 promotes spermiogenesis by regulating the adhesions between developing sperm and their support cells, Holembowski et al. reveal.

The p73 family of transcription factors has an ancient and well-conserved function in protecting the germline. Mammalian p63, for example, promotes the death of male and female gametes that have sustained DNA damage, and female mice lacking p73 are infertile due to defects in oocyte development. Male mice lacking p73 are also infertile, but the reason for this is unknown. Holecmbowski et al. therefore studied the testes of p73-deficient rodents.

Sperm develop in the multilayered epithelia of seminiferous tubules. Basal stem cells give rise to precursors that undergo meiosis and differentiate as they move toward the apical lumen. This process

is enabled and organized by somatic Sertoli cells, which span the seminiferous epithelium and tightly envelop maturing germ cells at each stage of their development in “nursing pouches,” guiding their differentiation and movement toward the luminal surface, where they release mature spermatozoa. Developing spermatids lacking all forms of p73, or a specific isoform called TAp73, detached from the epithelium prematurely and underwent apoptosis. The p73-deficient germ cells showed altered expression levels of many proteins that regulate spermatids’ adhesion to Sertoli cells, including integrins, proteases, and protease inhibitors.

Sertoli cells don’t express p73, but they were also affected by the loss of germ cell adhesion in p73-null testes, losing their characteristic morphology as well as the inter-Sertoli cell adhesions that form the blood–testis barrier, which protects developing spermatids from circulating immune cells and toxins. Senior author Ute Moll now wants to investigate whether mutations in p73 can cause human infertility.

Holembowski, L., et al. 2014. J. Cell Biol. http://dx.doi.org/10.1083/jcb.201306066.