

into the fallopian tubes arrested in metaphase of meiosis II. They remain arrested for 12–24 hours. If not fertilized during that time they degenerate. Mouse oocytes placed into culture just after ovulation show increasing levels of chromatid separation with time, an example of cohesion fatigue occurring during the normal physiological metaphase arrest of oocytes in meiosis II.

**Does cohesion fatigue play a role in the ‘maternal age effect’?** The frequency of aneuploid conceptions in humans increases strikingly with maternal age, approaching 35% for women in their 40s. Remarkably, if studies of mouse oocytes are applicable to humans, it appears that the actual cohesin molecules that bind chromatids together in the oocytes of women may be decades old. In mammals, S phase in oocytes occurs only during fetal life when all of a female’s oocytes are formed. These arrest in prophase of meiosis I, where they remain for decades in humans, until ovulation when meiosis resumes and the chromosome segregation events take place. Because the active cohesin complexes between sister chromatids are only formed in S phase, those cohesin molecules must then also persist from fetal life until ovulation. In other studies of mouse oocytes, loss or degeneration of cohesins and meiotic segregation errors increase with age. Thus, cohesion fatigue may exploit the age-associated defects in oocyte cohesin and contribute to the increased incidence of aneuploid conceptions in older women.

**What are the important future questions?** There are many basic facts we need to explore about the causes and potential consequences of cohesion fatigue in mitosis and meiosis. What genetic or environmental triggers lead to sustained metaphase and cohesion fatigue? How commonly does fatigue occur? Does cohesion fatigue contribute to chromosome instability in oncogenesis? Does it contribute to aneuploidy in birth defects and infertility? What is the mechanism by which the cohesin complex is breached by spindle pulling forces? How is the

cohesin complex retained on the chromatids that separate during cohesion fatigue? What are the long-term fates of chromatids that undergo cohesion fatigue? Does cohesion fatigue lead to chromosome breakage and fusion cycles? How does cohesion fatigue impact cell-cycle checkpoints, including the spindle checkpoint and DNA repair checkpoints? What are the consequences for cells in which only a few chromosomes undergo cohesion fatigue? Does partial cohesion fatigue, in which the centromeres are split but the arms remain tethered, result in chromosome instability? Could massive chromosome missegregation induced by drugs that cause metaphase arrest and cohesion fatigue be harnessed as an anti-cancer strategy? The answers to these and other questions promise to open a new chapter in understanding all the complex mechanisms regulating chromosome segregation in mitosis and meiosis.

#### **Where can I find out more?**

- Chiang, T., Duncan, F.E., Schindler, K., Schultz, R.M., and Lampson, M.A. (2010). Evidence that weakened centromere cohesion is a leading cause of age-related aneuploidy in oocytes. *Curr. Biol.* *20*, 1522–1528.
- Daum, J.R., Potapova, T.A., Sivakumar, S., Daniel, J.J., Flynn, J.N., Rankin, S., and Gorbsky, G.J. (2011). Cohesion fatigue induces chromatid separation in cells delayed at metaphase. *Curr. Biol.* *21*, 1018–1024.
- Hodges, C.A., Revenkova, E., Jessberger, R., Hassold, T.J., and Hunt, P.A. (2005). SMC1beta-deficient female mice provide evidence that cohesins are a missing link in age-related nondisjunction. *Nat. Genet.* *37*, 1351–1355.
- Lara-Gonzalez, P., and Taylor, S.S. (2012). Cohesion fatigue explains why pharmacological inhibition of the APC/C induces a spindle checkpoint-dependent mitotic arrest. *PLoS One* *7*, e49041.
- Lister, L.M., Kouznetsova, A., Hyslop, L.A., Kalleas, D., Pace, S.L., Barel, J.C., Nathan, A., Floros, V., Adelfalk, C., Watanabe, Y., et al. (2010). Age-related meiotic segregation errors in Mammalian oocytes are preceded by depletion of cohesin and Sgo2. *Curr. Biol.* *20*, 1511–1521.
- Mailhes, J.B., Young, D., and London, S.N. (1998). Postovulatory ageing of mouse oocytes in vivo and premature centromere separation and aneuploidy. *Biol. Reprod.* *58*, 1206–1210.
- Stevens, D., Gassmann, R., Oegema, K., and Desai, A. (2011). Uncoordinated loss of chromatid cohesion is a common outcome of extended metaphase arrest. *PLoS One* *6*, e22969.

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## Pollen

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**What is pollen, and is it haploid or diploid?** Pollen is a crucial stage of the plant life cycle — without pollen there will be no seed. When someone says “Think of a plant,” the plant you think of (whether it’s a tree, a tomato plant, or a geranium) is a sporophyte. Most land plants are sporophytes (usually diploids), and the sporophyte stage of the life cycle alternates with the gametophyte stage (haploids). The gametophytes are much smaller than sporophytes and are formed within structures on the sporophyte. A pollen grain is a male gametophyte, and pollen grains are formed in anthers, the male parts of flowers. Meiosis occurs in the anthers. Cells called pollen mother cells undergo meiosis. If a plant is diploid, each haploid product of meiosis (unicellular microspore) divides mitotically, but asymmetrically, to give two haploid cells (bicellular pollen grain). Amazingly, the smaller of these two cells is essentially engulfed into the cytoplasm of the larger cells, and then the smaller cell divides again to give two haploid sperm cells, so that the final pollen grain is composed of three cells: one haploid vegetative cell that will extend to form the pollen tube (a structure that transports the sperm cells to the ovule), and two haploid sperm cells carried inside the vegetative cell. Of course, in the case that the plant is tetraploid, then the pollen grains will be diploid.

**How about the pollen wall?** The outer wall of the pollen grain (the exine, sometimes called the pollen coat) is largely composed of a substance called sporopollenin, which is incredibly resistant to degradation, explaining why pollen grains are found in the fossil record. The exine has ornamentations (spines, ridges, undulations, etc.) that can be used to identify the plant species (Figure 1), which is why pollen sometimes has a role (although not starring) in television detective shows. Pollen of wind-pollinated species, such as maize, tends to have smoother surfaces, while non-wind pollinated species,

such as cucumber, have very ornate surface patterns. The inner wall of the pollen grain is called the intine. Most, if not all, of the components of the exine derive from the sporophytic part of the anther (i.e., from the tapetum, a cell layer lining the anther) and are deposited on the pollen surface, while most components of the intine derive from the gametophyte itself. Pollen grains have one or more apertures where the exine is thinner or absent, and the intine protrudes from one of these apertures to initiate the pollen tube. Thus, the exine is left behind when pollen tubes grow.

**What about pollen tubes?** Pollen tube growth is an excellent model system for studying cell polarity, being one of two cell types in plants (the other is root hairs) that grow by tip growth. Pollen tube growth can be amazingly fast — for example, a 100 micron diameter maize pollen grain extends a tube that *in vivo* can grow 1 cm/h, and some of the target ovules (at the base of ear) are 20 cm away from the starting point. As the pollen tube grows it periodically blocks off the old cell wall, so that the cytoplasm is maintained only near the tip. Pollen tubes normally grow through the female tissue in order to deliver the sperm cells to their fusion partners, the egg and the central cell (Figure 2). However, it is also possible to grow pollen tubes *in vitro*, in a simple medium, which facilitates live cell imaging. Pollen tube growth involves extensive endocytosis and exocytosis and rapid remodeling of the actin cytoskeleton. Numerous signaling pathways have been elucidated using this model. Directional cues from the female help the pollen tube find its way, and these signals are presumed to be detected by receptor kinases and processed by downstream effectors in the pollen tube cytoplasm.

**Why does pollen make allergens?** Pollen grains are not vindictive! In many cases the allergenic proteins are needed for pollen function. For example, Amb a 1, the major allergen from ragweed, is a pectate lyase. Pectate lyases are enzymes that de-esterify pectin, which is a major component of the intine and pollen tube wall. Profilin, an actin-binding protein, is another common pollen allergen. The group-1 allergens from

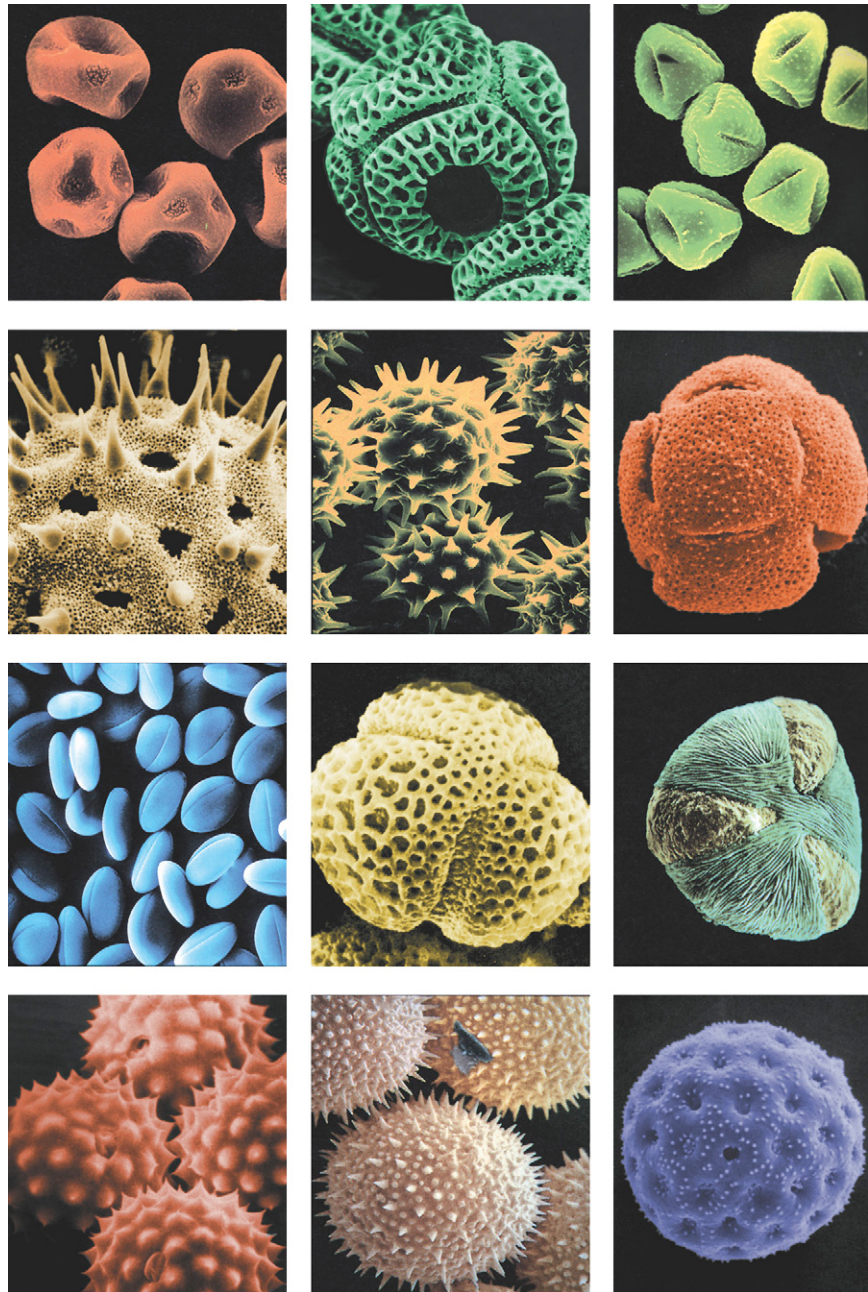


Figure 1. Pollen. False-colored scanning electron micrographs show the diverse ornamentation patterns on the surfaces of pollen from different species. From Chapter 19 “Reproductive Development”, by J. Derek Bewley, Frederick D. Hempel, Sheila McCormick and Patricia Zambryski, in *Biochemistry & Molecular Biology of Plants*, edited by Bob Buchanan, Wilhelm Gruissem and Russell Jones, published by American Society of Plant Biologists (2000).

grass pollen are  $\beta$ -expansins, and are thought to be needed for cell wall formation. The major allergen from perennial ryegrass is Lol p I, which is a small cysteine-rich secreted protein likely involved in pollen tube growth, since it is homologous to LAT52, a ligand for receptor kinases involved in pollen tube growth. Researchers have made transgenic plants with

reduced levels of ryegrass pollen allergens, but because of the variety of different pollen allergens, this approach is unlikely to replace antihistamines anytime soon.

**Why do plants make so much pollen?** Anyone who has ever parked their car under a tree in the spring must wonder about that. It

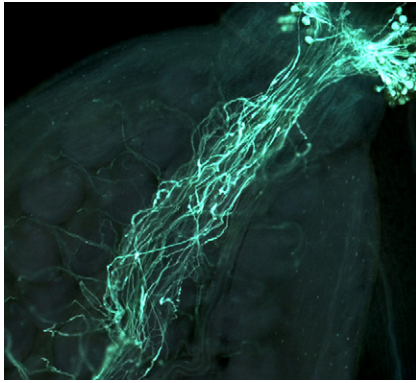


Figure 2. Pollen tubes.  
An *Arabidopsis* pistil with pollen tubes growing down towards the ovules. The pistil is stained with decolorized aniline blue to visualize the pollen tubes. (Photo: Dr. Yuan Chen.)

makes sense that wind-pollinated plants would make a lot of pollen, to increase the chance of finding a female, but even in self-pollinated plants, such as tomato, each anther makes many more pollen grains than are needed for the available females in that flower. Pollen competition (i.e. faster growing pollen tubes succeed) might explain this conundrum.

#### How do I find out more?

- Carlson, A.L., Fitz Gerald, J.N., Telligman, M., Roshanmanesh, J., and Swanson, R.J. (2011). Defining the genetic architecture underlying female- and male-mediated nonrandom mating and seed yield traits in *Arabidopsis*. *Plant Physiol.* 157, 1956–1964.
- Cheung, A.Y., and Wu, H.-M. (2008). Structural and signaling networks for the polar cell growth machinery in pollen tubes. *Annu. Rev. Plant Biol.* 59, 547–572.
- Dobritsa, A.A., and Coerper, D. (2012). The novel plant protein INAPERTURATE POLLEN1 marks distinct cellular domains and controls formation of apertures in the *Arabidopsis* pollen exine. *Plant Cell* 24, 4452–4464.
- Okuda, S., Tsutsui, H., Shiina, K., Sprunck, S., Takeuchi, H., Yui, R., Kasahara, R.D., Hamamura, Y., Mizukami, A., Susaki, D. *et al.* (2009). Defensin-like polypeptide LURES are pollen tube attractants secreted from synergid cells. *Nature* 458, 357–361.
- Petrovska, N., Wu, X., Donato, R., Wang, Z., Ong, E.-K., Jones, E., Forster, J., Emmerling, M., Sidoli, A., O’Hehir, R. and Spangenberg, G. (2004). Transgenic ryegrasses (*Lolium spp.*) with down-regulation of main pollen allergens. *Mol. Breed.* 14, 489–501.
- Qin, P. Ting, D., Shieh, A. and McCormick, S. (2012). Callose plug deposition patterns vary in pollen tubes of *Arabidopsis thaliana* ecotypes and tomato species. *BMC Plant Biol.* 12, 178.
- Tang, W., Ezcurra, I., Muschietti, J. and McCormick, S. (2002). A cysteine-rich extracellular protein, LAT52, interacts with the extracellular domain of the pollen receptor kinase LePRK2. *Plant Cell* 14, 2277–2287.

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## Primer

# Vision and the light environment

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Almost all animals, no matter how humble, possess eyes. Only those that live in total darkness, such as in a pitch-dark cave, may lack eyes entirely. Even at tremendous depths in the ocean — where the only lights that are ever seen are rare and fitful sparks of bioluminescence — most animals have eyes, and often surprisingly well-developed eyes. And despite their diversity (there are currently ten generally recognised optical types) all eyes have evolved in response to the remarkably varied light environments that are present in the habitats where animals live. Variations in the intensity of light, as well as in its direction, colour and dominant planes of polarisation, have all had dramatic effects on visual evolution. In the terrestrial habitats where we ourselves have most recently evolved, the light environment can vary quite markedly from day to night and from one location to another. In aquatic habitats, this variation can be orders of magnitude greater. Even though the ecologies and life histories of animals have played a major role in visual evolution, it is arguably the physical limitations imposed on photodetection by a given habitat and its light environment that have defined the basic selective pressures that have driven the evolution of eyes.

### Terrestrial light environments

The light experienced by day-active (diurnal) terrestrial animals is completely dominated by direct and indirect light from the sun, which behaves approximately as a celestial blackbody radiator. Like all blackbody radiators, the spectrum of light emitted by the sun characteristically depends on its surface temperature (around 5800 °K), although before reaching the Earth’s surface this broad spectrum is narrowed by absorption in the ultraviolet (UV) and the

infrared (by the filtering affects of the ozone layer, water vapour and other atmospheric constituents). As a result of atmospheric (Rayleigh) scattering of this sunlight — which is much greater at shorter wavelengths — the wide dome of the sky, whilst considerably dimmer (per unit area) than the sun, is also substantially bluer. Because of its much larger size compared to the disc of the sun, the blue sky contributes a significant fraction of the shorter wavelength light seen by diurnal animals (light in the 300–500 nm range) and affects the final measured spectrum of skylight irradiance (Figure 1A).

Scattered skylight is also linearly polarised, with the exact direction of each light ray’s electric field vector, and its degree of polarisation, varying systematically across the dome of the sky. Rayleigh scattering thus creates a distinct pattern of skylight polarisation, within which the electric field vectors are approximately arranged in concentric circles around the sun. The pattern has a symmetry plane defined by the solar meridian, the semicircular line that traverses the entire dome of the sky (from horizon to horizon) and contains both the sky’s zenith (the point directly above the observer) and the sun. This symmetry allows many terrestrial and shallow-living aquatic animals — particularly invertebrates — to use the polarisation pattern as a visual celestial compass during navigation.

As the sun’s elevation declines from its highest at midday (60–90°) to 0° at sunset, the daylight intensity drops approximately 100-fold, most of this drop occurring in the final 5°. By the time the sun has further sunk to 18° below the horizon (signalling the end of astronomical twilight and the onset of true night), light levels on a moonless night will have fallen a further 1–10 million times, although a night lit by a full moon will be around 100–1000 times brighter than this minimum. As we ourselves can attest, vision during the day and even at brighter twilight levels is reliable and of high quality. But at night our visual abilities are severely impaired by the paucity of light. We lose our ability to see colour (a loss likely shared by practically all other vertebrates) and