Articles of Significant Interest Selected from This Issue by the Editors

More Than One Role for Diatom Silicon Transporters

Diatom silicon transporters (SITs) are involved in silicon uptake when Si is scarce in the external environment. Shrestha and Hildebrand (p. 29–40) developed methodology to examine the response of individual SITs to different extracellular silicon concentrations, demonstrating an inverse relationship between SIT protein abundance and cellular needs for silicon. In knockdown lines, lipid accumulation under silicon starvation occurred far earlier than usual, suggesting an artificial sensing of silicon limitation. The data suggest that the SITs' transport role is relatively minor under sufficient silicic acid. Their primary role is to sense silicic acid levels to evaluate whether the cell can proceed with its cell wall formation and division processes.

DNA Methylation Depends on a Noncanonical Role of CUL4 in *Neurospora*

Methylation of lysine 9 of histone H3 (H3K9) directs DNA methylation in *Neurospora crassa*. It was known that all components of the cullin-4 (CUL4) complex DCDC (DIM-5/-7/-9/CUL4/DDB1 complex) are required for methylation of H3K9. DCDC and its analogue in fission yeast, CLRC, resemble classic cullin RING E3 ubiquitin ligases, leading to the assumption that they are involved in ubiquitination. Now, Adhvaryu and Gessaman et al. (p. 25–28) provide evidence that this model is incorrect. Key features of *Neurospora* CUL4, including the neddylation site and a segment that normally interacts with the RING domain protein RBX1, the E2 ligase, CAND-1, and CSN, are dispensable for heterochromatin formation but not for the role of CUL4 in DNA repair. These data provide evidence for the ubiquitination-independent function of CUL4 in the establishment of heterochromatin.

Plasmodium Infection Is Host Cell Cycle Independent

Intracellular pathogens create a niche for themselves to replicate inside their host cells, often altering fundamental cellular processes during infection. Hanson et al. (p. 96–103) demonstrate that liver stage malaria parasites provoke clear changes in host cell cycle progression in a hepatoma cell infection model, but neither murine infection with *Plasmodium berghei* or *P. yoelii* nor infection of human primary hepatocytes with *P. falciparum* induces any change in the cell cycle status of the host cell. Hepatoma cells that are unable to escape pharmacologically induced S-phase arrest also support complete *P. berghei* liver stage development, demonstrating that infection may induce profound changes in the host cell which are nonadaptive for the parasite.

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