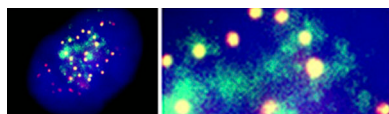


## Centromere stabilizer



**Rsf-1 (green) stabilizes CenP-A (red) in centromeric chromatin.**

to centromeres and stays there has been a mystery. [Perpelescu et al.](#) bring us one step closer to unraveling the mystery with their discovery that a chromatin remodeling protein called RSF stabilizes CenP-A (the human CenH3) at centromeres.

The expansive DNA strands of the genome are wrapped at regular intervals around nucleosomes to ensure orderly packaging. Everywhere besides centromeres, these nucleosomes contain

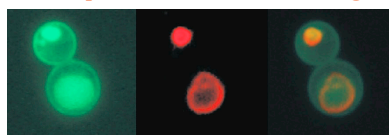
**T**he defining feature of centromeres is the presence of a protein called CenH3, but how this specialized histone gets

histone H3. At centromeres they contain the related protein CenH3. When DNA is replicated new nucleosomes must be incorporated into the daughter strands. H3 nucleosomes are added immediately, but the loading of CenH3 nucleosomes is delayed until after cell division.

During this incorporation interval, CenP-A and RSF colocalized at centromeres, the team showed. The arrival of RSF at centromeres followed the arrival of CenP-A very slightly, suggesting RSF does not chaperone CenP-A to its chromatin home. Instead RSF appears to stabilize CenP-A's incorporation—without RSF the amount of stably associated CenP-A in chromatin was decreased. The next step is to identify CenP-A's chaperone(s) and the signals that recruit RSF.

[Perpelescu, M., et al. 2009. \*J. Cell Biol.\* doi:10.1083/jcb.200903088.](#)

## Ubiquitin traffic signals



**Addition of a K63-linked chain of ubiquitins drives Gap1 permease (green) to the vacuole (red) via MVBs in yeast cells.**

of mammalian cells, is a disposal system for unwanted cellular components. According to [Lauwers et al.](#), two yeast proteins—one a disposer that resides in the vacuole, the other a disposee—are routed to this destructive organelle after being tagged with a special chain of ubiquitin molecules.

The addition of ubiquitin molecules—ubiquitylation—controls the function and cellular localization of a wide variety of proteins. This diversity seems to depend on the many

**L**[auwers et al.](#) have discovered how certain yeast membrane proteins end up in a cellular trash can.

The vacuole of yeast, like the lysosome

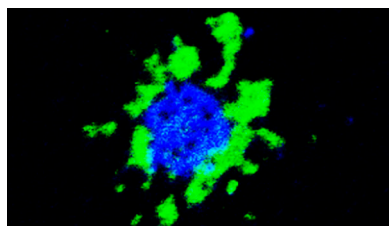
different forms that ubiquitylation can take. For example, addition of a string of ubiquitins called a K48-linked chain is well known for sending proteins to another type of cellular trash can called the proteasome.

[Lauwers et al.](#) showed that addition of a single ubiquitin molecule to a plasma membrane protein called Gap1 permease prompted the protein's endocytosis. Addition of a K63-linked chain of ubiquitins, however, sent Gap1 to its destruction in the vacuole—via intermediate membrane compartments called multivesicular bodies (MVBs). K63-linked ubiquitylation also prompted MVB sorting of a second protein, carboxypeptidase S, to the vacuolar lumen—this particular protein's workplace.

K63-linked modification has been reported for a number of mammalian proteins destined for the lysosome, indicating that this signal and the pathway that recognizes it are conserved.

[Lauwers, E., et al. 2009. \*J. Cell Biol.\* doi:10.1083/jcb.200810114.](#)

## How T cells shape their signals



**TCR (blue) and CD2 (green) form a bullseye and ring arrangement when they cluster at the T cell surface.**

vation. The synapse centers around antigen recognition by the T cell receptor (TCR), but other proteins on the cells' surfaces also partake in the union. One such pair of proteins is CD58 and CD2, which reside on the APC and T cell, respectively. Whether the coupling of this pair simply increases adhesion between the cells or also triggers a signaling cascade has been a point of debate.

[Kaizuka et al.](#) have now gotten to the bottom of the matter by observing the CD58–CD2 interaction independently of other

**K**[aizuka et al.](#) describe the shape of signaling during T cell activation.

When antigen-presenting cells (APCs) and T cells get together, the two become locked in a synaptic embrace that triggers T cell activation.

cell–cell interactions. Planar lipid bilayers, which mimic the APC's membrane but contain just one or two proteins of interest, were prepared on microscope coverslips. The team then followed the reactions of T cells to the bilayers, both visually and biochemically.

When presented with CD58, the CD2 receptors on the T cell's surface came together into small clusters. When presented with both CD58 and anti-TCR antibody, CD2 and TCR initially coalesced for several minutes, and then rearranged so that TCR formed a central bullseye surrounded by a ring of CD2.

As for signaling, CD58 or anti-TCR alone triggered the same initial kinase cascade in the T cell. But when presented together, the activation of these kinases was more robust, suggesting synergy. Active kinases were also clustered at the T cell's cortex reflecting the receptor clustering on the cell surface, and interestingly, when CD2 and TCR formed their bullseye and ring arrangement, the kinases were most abundant in the ring. As yet, however, the significance of these signal shapes remains to be determined.

[Kaizuka, Y., et al. 2009. \*J. Cell Biol.\* doi:10.1083/jcb.200809136.](#)