Mitosis is the process by which cells divide their genome after DNA replication to give rise to two genetically identical cells. The decision to enter and exit mitosis is critical for genome stability and the control of tissue homeostasis, perturbation of which has been linked to cancer. While we have obtained a deep understanding of the molecular networks that drive cells into mitosis, the molecular mechanism that triggers mitotic exit remains elusive. According to a “clock” model, Cyclin B1 degradation under control of the spindle assembly checkpoint leads to Cdk1 inactivation just as cells enter anaphase and sets the time for chromosome decondensation and nuclear envelope reformation (NER), which define mitotic exit. However, chromosome decondensation and NER were recently shown to be a function of chromosome separation during anaphase, mediated by a midzone Aurora B phosphorylation gradient - the “ruler” model. Because anaphase duration is responsive to the extent of chromosome separation, mitotic exit cannot simply be explained as a byproduct of Cdk1 inactivation after complete degradation of Cyclin B1 at the metaphase-anaphase transition. Thus, a crosstalk between molecular “rulers” and “clocks” must take place during anaphase, but the underlying mechanism is unknown. Here I will discuss our recent progress on this front. Our findings support a model in which a feedback between molecular “rulers” and “clocks” licenses mitotic exit only after proper chromosome separation. These findings uncover an unexpected level of regulation at the end of mitosis to promote chromosome segregation fidelity.