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The most pervasive mechanism for specificity in biology is structural complementarity. It was long ago postulated in enzyme-substrate, antibody-antigen, DNA-RNA, DNA-transcription factor binding, and in many protein-protein recognition events. Though these processes are highly specific, paradoxically the recognition motifs are usually small and highly redundant. This is also the case for the recognition of proteins for regulated degradation by the ubiquitin proteasome system in all eukaryotic cells, a multicomponent dissipative pathway of protein modification. We have developed single molecule imagining techniques to watch the addition and removal of ubiquitin on substrates catalyzed by the ubiquitin ligase, which controls much of the cell cycle, the Anaphase Promoting Factor. Many details can now be resolved for this complex process but the most broadly interesting is to understand how simple recognition motifs present widely in the genome are interpreted narrowly, based on other features distributed on the target. As a result, specificity is greatly enhanced. This requires energy investment and repeated sampling; the kinetic scheme has some relationship to kinetic proofreading, but is more efficient in several aspects. I will discuss more generally why this approach may be widespread in biology and why evolution has chosen this route over simply extending the domains of complementarity to achieve higher specificity.