Normally, the BCR-ABL protein, that fusion protein, functions in a cancer cell to phosphorylate a substrate or a target protein. And the way it does that is it takes a phosphate group from ATP, which binds to it and transfers that phosphate group to its target substrate protein. The target substrate protein then changes its shape and goes on to stimulate cell growth of the leukemia cells. This abnormal chromosome, Philadelphia chromosome, is found in leukemia cells. In a particular form of leukemia called CML. Now Gleevec, a drug which many of you may have heard of, mimics ATP. It binds to the site within the BCR-ABL that's normally bound by ATP, and it prevents ATP from binding. It thereby prevents phosphorylation of the substrate target protein and prevents cell growth.