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Cover Although human L1 elements are actively mobilized in many cancers, a role for somatic L1 retrotransposition in tumor initiation has not been conclusively demonstrated. In this issue, a new study shows that L1 can initiate colorectal cancer by evading somatic repression and then mutating the gatekeeper *APC* tumor suppressor gene in normal colon cells. The illustration depicts an L1 source element (labeled “L1”) generating a new somatic L1 insertion that disrupts the *APC* gene (arrow depicting mobilization). The highly active “hot” L1 source element that generated the insertion is found only in specific human populations, suggesting that some L1s can create a novel form of ancestry-specific cancer risk. (Cover illustration by Jennifer Fairman, © Fairman Studios, LLC, 2016. [For details, see Scott et al., pp. 745–755.]