of the activator, TFIIB adopts a conformation that inefficiently supports further assembly of the preinitiation complex. In this conformation the binding sites for TFIIF (N-terminal) and RNA polymerase II (C-terminal[12]) interact with each other, blocking access to TFIIF–RNA polymerase II.

An acidic activator binds directly to TFIIB, disrupts the intramolecular interaction and exposes the binding sites for TFIIF and RNA polymerase II. Thus, an acidic activator affects TFIIB assembly, both quantitatively, by recruiting TFIIB into the preinitiation complex[5,8,9], and qualitatively, by altering TFIIB conformation in a manner that drives preinitiation complex assembly forward. This latter function of the activator–TFIIB interaction explains why raising the concentration of TFIIB *in vitro*[8,19,20], or overexpressing TFIIB *in vivo*[21], does not overcome the requirement for an activator.

Activators function during at least two steps of preinitiation complex assembly[8,9,22], first to recruit TFIIB to the promoter and second to recruit GTFs that assemble after TFIIB. Interestingly, the TBP-associated factors (TAFs) participate in this second step. Thus, one function of TAFs may be to facilitate the activator-induced conformational change of TFIIB, perhaps by a direct TAF–TFIIB interaction. Consistent with this possibility, it has been shown that *Drosophila* $TAF_{II}40$ interacts directly with TFIIB[23]. Future studies using purified preinitiation complexes[5,8] will test this idea and other aspects of our model. □

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ERRATUM

Crystal structure of the extracellular region of human tissue factor

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