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 Chabner, Bruce A.; Longo, Dan L.  
 Lippincott Williams & Wilkins, 2019  
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← Chapter 2: Target Ident... →

antitumor uses, but also to toxicities (see Table 2.1). Since there is high homology of the ATP binding sites of subset of receptor tyrosine kinases (Fig. 2.6), toxicities to (Print pagebreak 24) (Print pagebreak 25) (Print pagebreak 26) skin and GI epithelium are common side effects for compounds of this class. However, it has been possible to synthesize drugs highly specific for a single, specific target unique to cancer cells, as for example osimertinib,<sup>48</sup> a highly active and specific inhibitor of the T790M mutant EGFR gene. It is 100-fold more potent against the mutant enzyme as compared to the wild-type EGFR.

Inactive Conformation of CSF1R with Imatinib

A

Inactive Conformation of CSF1R with PLX3397

B

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(Fig. 2.6) Internal Book Link

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Disruption of **imatinib binding** by the T315I mutation. The bulky isoleucine side chain causes steric hindrance, preventing drug access to

However, while **imatinib binding** (A) prevents the further conformational binding of a juxtamembrane (JM domain) into the pocket, the more

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