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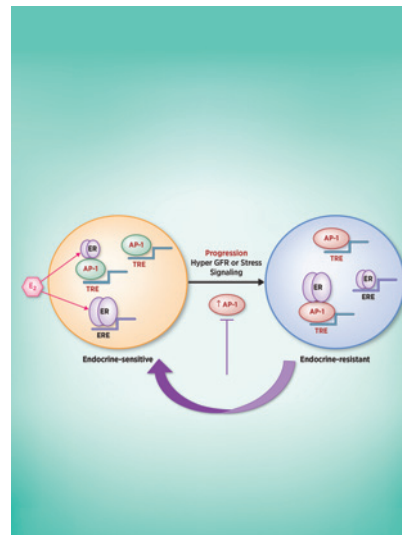
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ABOUT THE COVER

In this issue, a study by Malorni and colleagues (beginning page 470) demonstrates that the transcription factor AP-1 is a key determinant of endocrine resistance by, at least partly, mediating a global shift in the ER transcriptional program. The cover image shows the authors' proposed working model. In endocrine-sensitive cells (left side), estrogen triggers binding of ER to DNA at E2 responsive elements (ERE) to convey mitogenic and survival signals. At this stage, AP-1 activity by itself or as bound to ER may also partially contribute to tumor growth. Chronic blockade of ER by endocrine therapies activate adaptive responses or diverse escape pathways [such as growth factor receptors (GFR), or microenvironment and stress stimuli], which contribute to the development of endocrine resistance and disease progression (right). These multiple pathways converge on AP-1, which becomes a major determinant of global transcription, resulting in a molecular shift in the ER genomic network to an AP-1-dependent transcriptional program. Targeting AP-1 or its key downstream signaling components represents a new therapeutic strategy to enhance endocrine sensitivity and overcome resistance.



Molecular Cancer Research

14 (5)

Mol Cancer Res 2016;14:409-503.

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