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Molecular Mechanisms and Therapies of Colorectal Cancer 2.0

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Colorectal cancer (CRC) is currently the third leading cause of cancer-related mortality. Transforming growth factor beta (TGF-β) signaling has been associated with CRC growth and metastasis due to its involvement in proliferation, epithelial-to-mesenchymal transition (EMT), and angiogenesis. The TGF-β superfamily contains over forty members, including TGF-βs, Nodal, Activin, and bone morphogenetic proteins (BMPs). Three types of TGF-β receptors (TGFβRs) have been identified: types 1, 2, and 3. After ligand binding, TGF-βR2 recruits and phosphorylates TGF-βR1, which, in turn, phosphorylates downstream SMAD (small mother against decapentaplegic) proteins. Phosphorylated SMAD4 translocates into the nucleus, where it activates the transcription of numerous target genes (including *SERPINE1, LTBP2, CDKN1A, ARID3B, ATXN1, PTPRK, RAB6A, SMAD7, EHBP1* , etc.), acting predominantly as a tumor suppressor gene. Interestingly, alterations in *SMAD4* are frequent in metastatic CRC and, together with *TGF-βR2* gene mutations, have been reported as late events able to promote CRC progression. The study of the TGF-β pathway in metastatic CRC is challenging because of the great genetic heterogeneity of CRC. However, the increasing availability of targeted and whole-exome DNA sequencing techniques makes it possible to identify genetic mutations in complex, dynamic, and heterogeneous clinical contexts and make correlations with clinical outcomes.

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