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Tired of looking for an online movie title, or not knowing which video player to use. and may represent a novel therapeutic target. The common molecular pathway for TGFβ-induced cancer cell migration is activation of MAPK, which is downstream of multiple cellular-signaling pathways. Although there are many distinct TGFβ receptors, TβRI appears to be the primary mediator of TGFβ signaling and has been linked to TGFβ-induced EMT and pericellular proteolytic remodeling in various types of cancer. Our results suggest that the interference with TβRI/Smad2/3 signaling may be an option to counteract cancer cell migration, invasion, and metastasis. The present study indicates that TβRI signaling is essential for TGFβ2-induced cancer cell migration. TβRI inhibition by metenolone or SB431542 prevents cell migration in various cell lines, without interfering with cell viability, cell proliferation, or cell cycle progression. We found that TβRI inhibition was effective to prevent cancer cell migration and TGFβ2-induced α-SMA expression. It is possible that TGFβ2 may induce Smad2/3 by a TβRI-independent pathway, but this is unlikely. We observed a Smad2/3-independent TGFβ2-induced Smad4 phosphorylation (at S467), which was inhibited by metenolone or SB431542 treatment. There was also significant reduction of p-Smad3 in cell lines with Smad2/3 knockdown. These results suggest that metenolone and SB431542 may inhibit TGFβ-induced cancer cell migration in an Smad2/3-independent manner. However, we cannot completely rule out this possibility because the effects of metenolone and SB431542 on Smad4 phosphorylation (at S467) and p-Smad3 were blocked by Smad4 knockdown. Furthermore, we performed experiments with Smad3 knockdown to support this hypothesis. However, we have no data on Smad3 knockdown because no siRNA targeting human Smad3 has been commercially available. We are also interested in knowing whether the TβRI inhibitor can be effective for other types of cancer cells. In addition to tumor cells, we have confirmed that metenolone and SB431542

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antigens by B and T cells in an organism
requires the activation of antigen-specific
receptors, the binding of which to their
specific ligands leads to the activation of the
functional receptors. In the case of B cells,
differentiation into antibody producing cells
is triggered by binding of B cells to
multivalent antigens. Furthermore,
activation of B cells is mediated by
interactions between the Ag receptors and
their specific ligands, TcR and BcR, for which
binding of soluble peptides or proteins would

seem the more relevant mode of action. In addition, BCR binding of ligands of defined structure has allowed the identification of so-called 'superagonists' which activate B cells more efficiently than even the closest relative of the peptide/protein/superagonist itself. These 'superagonists' provide a unique tool to study the signal transduction pathway for BCR activation, and are particularly important to test the specificity of anti-IgM for triggering B cell maturation and proliferation. This would be difficult in vivo, since the peptide/protein/superagonist may only be present in pathogens which can give rise to a restricted set of B cell responses. The activation of T cell receptors (TCR) is critical for the control of infections by various pathogens. The key to TCR

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