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Abstract

Chronic wound fuid (CWF) from chronic venous leg ulcers has been shown to inhibit dermal fbroblast growth by interfering with cell-cycle progression from G1 to S phase. CWF was found to reduce the levels of hyperphosphorylated retinoblastoma tumor-suppressor gene (Rb) and cyclin D1, both of which are required for the cell cycle to enter the S phase. To better understand the efects of CWF, researchers looked into a Ras-mediated signalling pathway involving the mitogen-activated protein kinase kinase (MEK), which is known to modulate the expression of these cell-cycle-regulatory proteins [1-15]. The growth suppressive efects of CWF on hyperphosphorylated Rb (ppRb) and cyclin D1 were abolished by transient transfection of dermal fbroblasts with constitutively active Ras. in comparisonThe efects of CWF on these cell-cycle-regulatory proteins were mimicked by a MEK inhibitor, PD 98059. Concurrent administration of PD 98059 and CWF resulted in additive efects. These fndings suggest that CWF inhibits dermal fbroblast growth, at least in part, by decreasing the level of active Ras, which results in lower levels of ppRb and cyclin D1. As a result, a Ras-dependent signalling pathway may mediate the growth inhibitory efect of CWF, and restoring Ras activity may overcome this efect. Wound fuid is thought to play an important role in wound healing Acute wound fuid has been shown to stimulate fbroblast and endothelial cell growth induce chemotaxis and increase extracellular matrix production.In contrast, chronic wound fuid (CWF) has been shown to inhibit cellular proliferation contributing to the poor healing of chronic ulcers .CWF inhibits the proliferation of newborn dermal fbroblasts (NbFb) and DNA synthesis in human neonatal fbroblasts .and it halts the cell cycle in the G1 phase (Phillips et al, 1998). Fibroblast proliferation is critical to wound healing,McClain et and any disruption can signifcantly alter proper wound healing.

all eukaryotic cells (Lowy and Willumsen, 1993). Ras receives signals from a wide range of extracellular stimuli, and Ras mediates its efects by activating a cascade of protein kinases (acts as a molecular switch at the plasma membrane's inner leafet, and its activity is regulated by a guanosine mitogen-activated protein (MAP) kinase pathway Active Ras binds inactive Raf and translocates it to the plasma membrane where the Raf is activated (Many studies have established that cyclin D1 expression is induced by Ras through a Raf/MEK/MAP kinase-dependent pathway the ability of oncogenic Ras to shorten the G1 phase can be attributed to increased induction of cyclin D1. Furthermore, expression of dominant-negative Ras into cycling cells causes a decline in cyclin D1, accumulation of hypophosphorylated Rb and subsequent growth arrest in G1, which can be overcome with induction of Pathway of mitogen-activated protein (MAP) kinase Active Ras binds inactive Raf and transports it to the plasma membrane, where it activates Raf Many studies have shown that Ras induces cyclin D1 expression via a Raf/MEK/MAP kinase- and that the ability of oncogenic Ras to short.

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Acknowledgement

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Con ict of Interest

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