



Cell Citation Style (in RefWorks it is called VertPhys2)

IN-TEXT CITATIONS: The following paragraph is an example of in-text citations. Cite no more than two authors of an article (see **Bachelder**). If more than two, cite only one and represent others by et al. (see **Falcioni**).

Studies of in vitro models suggest that the $\alpha 6 \beta 4$ integrin—a component of hemidesmosomes—contributes to oncogenesis by sustaining RTK signaling. $\beta 4$ integrin signaling proceeds through Src family kinase (SFK) mediated phosphorylation of the cytoplasmic domain of $\beta 4$, recruitment of Shc, and activation of Ras and PI-3K (Mainiero et al., 1997; Shaw et al., 1997). The RTKs ErbB2, EGF-R, and Met associate with $\alpha 6 \beta 4$, and there is evidence suggesting that they promote invasive signaling through phosphorylation of $\beta 4$ (Falcioni et al., 1997; Mariotti et al., 2001; Trusolino et al., 2001). Accordingly, wildtype, but not signaling-defective, $\beta 4$ causes a gain in invasive ability in a breast carcinoma cell line expressing Met (Shaw et al., 1997). In spite of this body of work, the hypothesis that $\alpha 6 \beta 4$ has a protumorigenic function remains controversial. Expression of wild-type, but not signaling defective, $\beta 4$ activates p53 and induces cell-cycle arrest and apoptosis in rectal carcinoma cells (Bachelder and Mercurio, 1999).

REFERENCES: List no more than 10 authors in an article. If more than 10, list the first 10 and represent others by et al. (see Mainiero).

Bachelder, R.E., and Mercurio, A.M. (1999). Activation of p53 function in carcinoma cells by the $\alpha 6 \beta 4$ integrin. *J. Biol. Chem.* 274, 20733–20737.

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