Literature evidence in support of the driver nodes of the EMT network: their dysregulation frequently occurs in and may be causative of cancer

TGF\(\beta\) is upregulated in 40\% of patients with heptocellular carcinoma [1], [2]. A tendency towards GLI overexpression in human breast cancer has been shown [3]. The Raf kinase inhibitor protein (RKIP) is known to be a prognostic marker in prostate cancer [4], [5]. GSK3\(\beta\) activity is known to be dysregulated and SNAI1 activity is known to be upregulated during the development and progression of human breast cancer [6], [5]. Wnt and betacatenin_nuc upregulation has been associated with cancer cell growth and maintenance [7], [6]. Upregulation and overexpression of DVL-1, the human counterpart of the DSH gene, in primary breast cancer has been shown [8]. A significant subset of children with medulloblastoma have been reported to carry germline and somatic mutations in SUFU [9]. B-RAF gene mutations have been reported in 66\% of malignant melanomas [10], over 60\% of which show elevated kinase activity and signal to ERK [11]. Aberrant NOTCH signalling has been linked to tumorigenesis and cancer [12]. Experimental data has suggested SMO activity to be a major mediator of breast tumor cell growth [13]. Upregulation of Frizzled-7 in human gastric cancer has been reported [14].

References


(gli) 1 in human breast cancer is associated with unfavourable overall survival,” *BMC cancer*, vol. 9, no. 1, p. 1, 2009.


