Ontologies Classes Object Properties Data Properties Annotation Properties Individuals Datatypes Clouds

Class: Biological_Effects_LC

Annotations (1)

- rdfs:comment ""“EPITHELIAL–MESENCHYMAL TRANSITION EMT has been initially described as a process in embryonic development. EMT is composed of a developmental shift from a polarized, epithelial phenotype to a highly motile fibroblastoid or mesenchymal phenotype (43). In addition to embryonic development, EMT has been implicated in chronic inflammation, fibrosis, and cancer development (44–47). In normal development, EMT is a tightly regulated process (47). In contrast, in cancer development and progression, EMT is unregulated, with selective elements of the process amplified while other aspects are circumvented (48). A variety of pathways are now appreciated to impact EMT in cancer. For example, the TGF-β pathway, PI3K/Akt, ROS, receptor tyrosine kinase/Ras signaling, and Wnt pathways have been among those implicated (43, 44, 49). Thus, EMT is operative in a variety of malignancies (50), including lung cancer (48). The connection between inflammation and EMT progression in lung cancer development and resistance to therapy has recently been emphasized (15, 51). For example, IL-1β and PGE2 have the capacity to decrease E-cadherin expression and promote EMT. These inflammatory mediators have the capacity to up-regulate the zinc-finger E-box–binding transcriptional repressors of E-cadherin, including Zeb1, Snail, and Slug, thus leading to EMT progression (15, 52). Recent work from Robert Weinberg's laboratory suggests a direct link between EMT and gain of epithelial stem cell properties (53). Thus, inflammation may impact stem cell properties via EMT-dependent events in the pathogenesis of lung cancer. While EMT-induced alterations have been widely implicated in the epithelial malignancy metastatic process, the work of Mani and colleagues suggests that the EMT genetic program may also regulate early events in carcinogenesis, therefore implicating the inflammatory pulmonary environment in both lung cancer initiation and progression. The fact that tobacco and tobacco-specific carcinogens may be involved by directly or indirectly promoting EMT adds additional importance to these relationships. For example, Yoshino and coworkers (54) found that benzo[a]pyrene induced EMT-related genes in lung cancer cells; while fibronectin and Twist were induced, E-cadherin expression was deceased. In support of these findings, and in the context of another tobacco-induced malignancy, Fondrevelle and colleagues (55) found that the expression of Twist was influenced by smoking status in patients with bladder cancer. Tobacco-specific carcinogen 4-(n-methyl-n-nitrosamino)-1-(3-pyridyl)-1-butanone (NNK) has also been found to promote EMT via induction of E-cadherin transcriptional repressors in human bronchial epithelial cells (56).”"(xsd:string)

Superclasses (1)

- Tobacco_Smoking

OWL HTML inside