AKR1C3 has the capability of regulating the transactivation of various nuclear receptors including androgen receptor, estrogen receptor, and peroxisome proliferator activated receptor (PPARG) by regulating the ligand availability for the nuclear receptors.

**Homology**

A member of the AKR1C family proteins; AKR1C1, AKR1C2, AKR1C3, AKR1C4 in human, and AKR1C9 in rat.

**Mutations**

Note: Mutation of AKR1C3 has not been identified.

**Implicated in**

**Various cancers**

Note: Elevated levels of AKR1C3 expression are implicated in leukemia cell differentiation, prostate cancer (in both androgen-dependent and androgen-independent prostate cancer), and endometrial cancer. Expression of AKR1C3 was detected in a patient with myelodysplastic syndrome (MDS, refractory anemia) with progression to acute myelogenous leukemia. Overexpression of AKR1C3 in a human promyelocytic leukemia cell line, HL-60, rendered cells more resistant to all-trans retinoic acid (ATRA) and 1a,25-dihydroxyvitamin D3 induced cell differentiation.

**Prostate cancer**

**Disease**

Immunohistochemical staining of human prostate tissues detected negative or low levels of AKR1C3 expression in normal prostate epithelial cells. Strong positive AKR1C3 immunoreactivity was demonstrated in primary and androgen-independent prostate cancers.
Variable increases in AKR1C3 expression were also demonstrated in non-neoplastic changes in the prostate including chronic inflammation, atrophy, and urothelial cell metaplasia.

**Endometrial cancer**

**Disease**

Quantitative transcriptome analysis using real-time polymerase chain reaction, AKR1C3 mRNA expression was shown to be elevated in endometrial cancer versus adjacent normal endometrium.

**Breast tumor**

**Disease**

Expression of AKR1C3 mRNA was reduced in breast tumor as compared to adjacent normal breast tissue. Immunohistochemistry revealed that the ductal epithelial cells and stromal cells of the breast express AKR1C3. In myoepithelial cells of the breast, immunoreactive AKR1C3 was absent in normal tissues, whereas strong AKR1C3 staining was apparent in cells surrounding the neoplastic epithelium of ductal carcinoma in situ.

**References**


This article should be referenced as such: