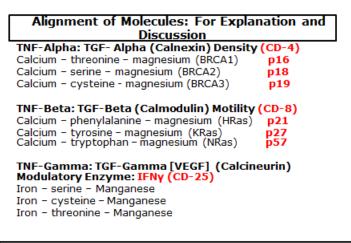Quantum biology has identified Importin 11 as being, with near certainty, an epigenetic signaling molecule for the motility of elements that can result in colorectal cancer.

Qualified computational biologists can use the following as a tool from which bioinformatics can be used to correlate IPO11 to calmodulin with a DIY process.

Alignment of Molecules: For Explanation and Discussion

TNF-Alpha: TGF- Alpha (Calnexin) Density (CD-4)
Calcium – threonine – magnesium (BRCA1)   p16
Calcium – serine – magnesium (BRCA2)   p18
Calcium – cysteine - magnesium (BRCA3)   p19

TNF-Beta: TGF-Beta (Calmodulin) Motility (CD-8)
Calcium – phenylalanine – magnesium (HRas)   p21
Calcium – tyrosine – magnesium (KRas)   p27
Calcium – tryptophan – magnesium (NRas)   p57

TNF-Gamma: TGF-Gamma [VEGF] (Calcineurin) Modulatory Enzyme: IFNγ (CD-25)
Iron – serine – Manganese
Iron – cysteine – Manganese
Iron – threonine – Manganese

These are examples of the "enzymes" that have evolved with various designations; e.g. AKT, mTOR, PTEN, NF-kB, and MYC.

https://medicalxpress.com/news/2019-12-therapeutic-colorectal-cancer.html

DECEMBER 27, 2019

Researchers identify new therapeutic target for colorectal cancer

by Rockefeller University Press

Researchers at the University of Toronto have identified a key protein that supports the growth of many colorectal cancers. The study, which will be published December 27 in the *Journal of Cell Biology*, reveals that a protein called Importin-11 transports the cancer-causing protein βcatenin into the nucleus of colon cancer cells, where it can drive cell proliferation. Inhibiting this transport step could block the growth of most colorectal cancers caused by elevated βcatenin levels.

Around 80% of colorectal cancers are associated with mutations in a gene called *APC* that result in elevated levels of the βcatenin protein. This increase in βcatenin is followed by the protein's accumulation in the cell nucleus, where it can activate numerous genes that drive cell proliferation and promote the growth and maintenance of colorectal tumors. But how βcatenin enters the cell nucleus after its levels rise is poorly understood. "Because the molecular mechanisms underlying βcatenin nuclear transport remain unclear, we set out to identify genes required for continuous βcatenin activity in colorectal cancer cells harboring *APC* mutations," says Stephane Angers, a professor in the Department of Pharmaceutical Sciences at the University of Toronto's Leslie Dan Faculty of Pharmacy.

Using CRISPR DNA editing technology, Angers and colleagues, including graduate student Monika Mis, developed a new technique that allowed them to screen the human genome for genes that support βcatenin's activity in colorectal cancer cells after its levels have been elevated by mutations in *APC*. One of the main genes they identified was *IPO11*, which encodes a protein called Importin-11 that is known to be involved in nuclear import.

Angers and colleagues found that Importin-11 binds to βcatenin and escorts it into the nucleus of colorectal cancer cells with mutations in *APC*. Removing Importin-11 from these cells prevented βcatenin from entering the nucleus and activating its target genes.

The researchers discovered that Importin-11 levels are often elevated in human colorectal cancers. Moreover, removing Importin-11 inhibited the growth of tumors formed by *APC*mutant cancer cells isolated from patients.

"We conclude that Importin-11 is required for the growth of colorectal cancer cells," Angers says. Learning more about how Importin-11 transports βcatenin into the nucleus may help researchers develop new therapies that block this process and reduce the growth of colorectal cancers caused by mutations in *APC*