Fallacy of HPV p16 and p18 Virus and Cancer

History of HPV

"In 1972, the association of the human papillomaviruses with skin cancer in epidermodysplasia verruciformis was proposed by Stefania Jabłońska in Poland. In 1978, Jabłońska and Gerard Orth at the Pasteur Institute discovered HPV-5 in skin cancer. In 1976 Harald zur Hausen published the hypothesis that human papilloma virus plays an important role in the cause of cervical cancer. In 1983 and 1984 zur Hausen and his collaborators identified HPV16 and HPV18 in cervical cancer." [1]

As the algorithm for quantum biology (QB) was being developed, the roles of p16 and p18 as viruses that could cause cancers became skeptical following discovery of nanobacteria when atomic force microscopy was used to assay cellular activity in the late 1990s. [2]

Over the following years, the discovery of "nanobacteria" was dispelled as being the ability to visualize ionic polarity (Van der Vaals forces) relative to hydroxyapatite. In essence, the inability to understand observations without a frame of reference resulted in the formulation of theories that could not be supported by the principles of physical science.

In our opinion, lacking the technology to provide a basis to identify early findings for observations at the cellular level for HPV 16 and 18 resulted in hypotheses that they were active viruses in a manner similar to nanobacteria.

When HPV16 and HPV18 are linked to p16 and p18, [3, 4, 5]

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[1] https://en.wikipedia.org/wiki/Human_papillomavirus_infection
[2] https://en.wikipedia.org/wiki/Nanobacterium
[3] https://www.ncbi.nlm.nih.gov/pubmed/15608419
[4] http://www.nature.com/bjc/journal/v110/n6/full/bjc201442a.html
[5] https://www.ncbi.nlm.nih.gov/pubmed/23482761

it becomes obvious that the observations were based on epigenetic signaling that had not been visualized previously and were assumed to be viral. Since p16 and p18 are linked to cervical cancers, the oncology research community erroneous assumed they were viral organisms; an error that has created chaos by leading research down an incorrect path and away from epigenetic activity.

As quantum biology was emerging, the following tool was developed to enable computational biologists to have a frame of reference for the levels of cellular interaction; i.e. density v motility.

Cell Alignment: For Explanation and Discussion

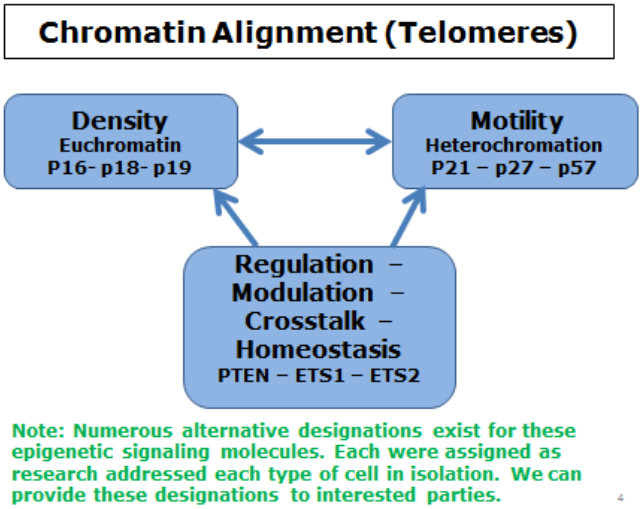
TNF-Alpha: TGF- Alpha (Calnexin) Density (CD-4)
Calcium – threonine – magnesium (BRCA1) **p16**
Calcium – serine – magnesium (BRCA2) **p18**
Calcium – cysteine - magnesium (BRCA3) **p19**

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.

The following illustration depicts the roles of p16 and p18.



Summary

With instances of head and neck as well as anal cancers increasing, in our opinion, the time has come to rethink the causes of cancer, focus on prevention, discontinue the use of HPV vaccines as revenue sources that are virus-based strategies and focus on alignment of molecules as a course to prevent cancers, plaques clots and other “clumping” that inclides but is not limited to kinases