Cancer Causes and Solutions

Applying the MCFIP designed iteration of bioinformatics to DNA Repair and Gene Entanglement allows the primary causes of the spectrum of cancers to be verified and used for discussion purposes.

- Theories for the consequences of infant phenylketonuria (PKU) and the adult form have been misconstrued.
  Modeling of interactions and imbalances of cellular mechanisms using the MCFIP tools identified the aggregation of amino acids and the inability to disassemble them as being responsible for the formation of kinases.
  When subjected to the modeling process, it became apparent that, with near certainty, PKU was the result of mutation of autophagy and the inability to convert phenylalanine to tyrosine and tyrosine to tryptophan.
  In-depth review of interactions between these three aromatic amino acids identified their aggregation as being tyrosine kinase; the most aggressive form of cancers.
  It is a fact that adult PKU exists and it is known as hyperphenylalaninemia.
  Correlating infant PKU with hyperphenylalaninemia can be accomplished through our discussions with experts in bioinformatics.
  The following link addresses vitamins B12 and B6 as enzymes that can resolve adult PKU.
  https://www.ncbi.nlm.nih.gov/pubmed/16601867
- Studies have identified more than 500 kinases.[1] Applying the DNA Repair modeling methodologies has identified a large number of kinases that are primary factors for the causes of cancer. Examples of additional kinases selected for discussion include the branched-chain amino acids (leucine – isoleucine – valine) - BCKD kinase, threonine – serine – cysteine (Cysteine-targeted Irreversible Protein kinase [a.k.a. Thrombocytopenia or Cysteinome]) and Abl-1 (c-abl), Abl-2 (v-abl) and BCR-Abl. This latter kinase is noteworthy because, as outlined in the following link, it is a primary factor for the spectrum of leukemia http://www.mcfip.net/upload/Leukemia%20Biomarkers%20-%20o.pdf

  Summary Kinase Activity
  Research identifies the ability to use enzymes to mitigate kinase formation; i.e. the PKU process. Given the fact that autophagy can be established as a cellular mechanism regulated by granzymes E - F - G, we have opted to focus the role of these enzymes as a means of treating or curing kinase driven cancers.

- Oncology research has established excessive cell division as a primary cause for the proliferation of cancers. Calcitonin has been identified as the biomarker for the process of division. However, this epigenetic signaling molecule has not previously been reduced to its elemental constituents (elements in conjunction with amino acids) and the cause of over-expression has not been elucidated.

[1] **http://www.sciencedaily.com/releases/2012/04/120412121359.htm**

MCFIP can provide explicit and verifiable explanations to bioinformatic professionals of TBD DNA Repair strategic partners for these modeling processes.

- Epigenetic modeling can be verified as being comprised of signaling molecules that exist in trefoils (3s). Mutation of BRCA1 and 2 is known as a primary cause of breast and cervical cancers, To date, however, the third member of the BRCA family has been hidden in plain sight and its over-expression is the near certain cause of disrupting the balance/equilibrium/homeostasis between BRCA1 and 2 with cancers being an outcome. MCFIP can provide explicit and verifiable explanations to bioinformatic professionals of the elemental constituents of BRCA1 - 3; these tools will allow for prevention of BRCA3 over-expression and the ability to prevent/treat/cure these cancers.

To avoid complexity and confusion due to terminology, we have opted to set aside explanations for how the aggregation of amino acids can create plaques that are responsible for the spectrum of neurodegenerative diseases. With that being said, as part of a strategic relationship, the ability of bioinformatics professionals to have access to our modeling methods and data will allow them to provide independent verification for the following:

- ✓ How neuroprotectins can prevent plaque formation
- ✓ How enzymes (i.e. granzymes E - F and G) offer the ability to treat/cure dementias and other neurodegenerative diseases that are the result of plagues in the synapse of cells