The first discriminatory step illustrating the PPPM-oriented survey is estimating of the correlation strength between genetic polymorphism and risks of the disease, and subsequent construction of the groups at risks. Those goals can be solved by using of BioChip methodology (each disease has individual fingerprints). As a result, a patient becomes a data carrier, i.e., he/she knows about possible risks of a disease, and the physician can reasonably select of preventive protocol, proceeding from the assays made. Individuals, selected at the first stage, undergo the second phase of the survey, which uses a panel of phenotypic biopredictors.

Two examples to illustrate the topic: T1D and MS models.

T1D is an chronic autoimmune disease resulting in a destruction of pancreatic beta-cells capable alone of producing insulin, by two autoreactive tools, i.e., cytotoxic T lymphocytes and anti-islet autoAbs. For T1D, about half of the total risk is genetic and about half of that genetic risk is in the HLA region on the chromosome 6 to be used for gene-based predictive testing! Other genes are associated with T1D as well, but their contribution to risks is small.

Subclinical stages are also determined by identification of highly-specific proteomic-related biopredictors, i.e., different anti-islet autoAbs whose presence would determine risks for initiating subclinical abnormalities and then result in clinical manifestations of T1D.

MS is an autoimmune disorder of the central nervous system resulting in a destruction of neuro-myelin compartment including demyelination, axon loss and development of disability. Most of the studies confirmed the supreme role of the variations within HLA genes, particularly HLA-II DR region as MS gene-related risk factors!

The crucial step in the MS evolution is a primary myelin damage which is mediated by cytotoxic anti-myelin autoAbs! A portion of those are autoAbs against myelin-basic protein/MBP endowing with MBP-targeted proteolytic activity (so-called, Ab-proteases). Screening for those could become the next step to secure subclinical diagnosis of MS and to predict the clinical course.

The activity of Ab-proteases established markedly differs: (i) between MS patients and healthy controls; (ii) among different disease courses; and would correlate with EDSS scales of demyelination and thus the disability of the patients.

The sequence-specificity of Ab-proteases would illustrate a common but specific pattern revealing six sites of preferential proteolysis. Some of those sites are located within the immunodominant and encephalitogenic regions of MBP that are responsible for the induction of severe and aggressive forms of MS or for getting the disease run in a similar way. The other sites are less immunogenic to be responsible for either the induction moderate courses of MS or for maintaining clinically moderate courses.

The Ab-proteases were initially registered at the subclinical stages of MS, presumably, presumably demonstrating their defined sequence specificity with low immunogenicity.

Meanwhile, apart from MS patients, about 24% of the direct relatives were also seropositive for Ab-proteases but to reveal much lower activity as compared with the patients. Half of those seropositive relatives being monitored for 2 years have been demonstrating stable growth of the activity of Ab-proteases which reached their mid-level indices at a time point when had coincided with the initial occurrence of the primary clinical and MRI manifestations.

A unique value of Ab-proteases for monitoring MS at different stages of the disease whilst providing a fantastic tool for predicting demyelination is becoming evident and thus a reality. Moreover, Ab-proteases can be programmed and re-programmed to directly affect the remodeling of tissues (for instance, myelin). Such functionally valuable tools may thus be designed for the newer drug catalysts to act as preventive therapeutic tools!

Just comments and reference to malignant neoplasms: the initiation and progression of tumors depend on the stepwise acquisition of specific functions by cancer cells at both the primary and metastatic sites.

Tumor initiation (at subclinical stages) is provided by oncogenic mutations and inactivation of tumor-suppressor genes to be identified by genomic tailoring approach!

The initiation of metastasis would have to be predicted by a combination of genomic and proteomic approaches! And newer algorithms to suit the need of making the subclinical diagnosis of tumor are already in progress!

It would be extremely useful to integrate data harvesting from different databanks for applications such as prediction and personalization of further treatment. Medical practitioners will be able to thus provide more tailored prevention and treatment programs for their patients resulting in improved patient outcomes, reduced adverse events, and more cost effective...