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INSTANT TREATMENT BY EPIGENETIC BIOMARKER

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The wealth of latest data and knowledge relating to epigenetics obtained in recent years highlights a vibrant future for epigenetics research. It involves the integration of high-throughput sequencing technologies and the means to maintain and manipulate the large amount of data produced by sequencing epigenomes. As more epigenetic markers are associated with specific diseases and tools can be developed to diagnose patients and gauge the severity of disease. There is also great concern in therapeutic epigenetics many drugs, such as DNA methyltransferase inhibitors and histone deacetylase inhibitors are already used in cancer treatment. It is a very exciting time to study epigenetic, and this field of research has the potential to completely transform medicine and greatly improve human lives.

The goal of clinical biomarkers is to provide physician with relevant information about the detection of a disease as well as about patient and disease characteristics that influence treatment decision. Conceptually, epigenetics biomarkers consist of two complementary building blocks: An experimental assay that provides accurate measurement of epigenetic modification in a given patient sample, either at a single locus or at multiple genomic region, Several mechanism of epigenetic regulation have been discovered in recent years with DNA methylation, histone acetylation, microRNAs and other ncoRNA being among the most prominent. Their prevalence in current applications the reminder of this paper concentrates on DNA methylation biomarker although we note that epigenetics biomarker based on histone modifications and non-coding RNAs may increasingly complement DNA methylation biomarker in the future.

To be suitable for population screening, diagnosis biomarkers are usually based on readily available body fluids. Further more extensive efforts have been made to develop accurate biomarker based on blood. the blood test for detecting solid tumors is that tumor cells may shed epigenetically alter DNA into the blood stream or that blood cells may have undergone epigenetics changes representative of those present the tumor, for example in response to specific environmental influence. Both arguments have limitations the amount of heterogeneity of normal blood and it is doubtful whether blood is suitable proxy for epigenetic alteration elsewhere. Complementary to early diagnosis of developing tumors a second class of epigenetic biomarkers aim to support clinical decision making once a tumor has been identified.

The epigenetic markers associated with the production of pluripotent embryonic stem cells is also of high interest for its relevance in reprogramming differentiated cells to make induced pluripotent stem cells [2]. DNA Methylation patterns often association with clinical parameters such as cancer stage, survival time and chemotherapy resistances, which give rise to new opportunity for treatment decisions as well as survival prognosis, thus enabling more modified cancer therapy. DNA Methylation biomarkers have been slow to generate measurable impact on clinical



cancer therapy. Beyond a number of conceptual reasons discussed elsewhere the gap between discovery and clinical adaption is aggravated by inefficiencies of the biomarker development process itself, foremost talented biomarkers being missed or discarded, while poor candidates fail at late stage of the validation process. We are confident that systematic, bioinformatics driven strategies can increase the efficiency and reduce the cost of biomarker development projects thus helping to fulfill the clinical promise of epigenetic biomarkers in cancer and beyond. Published research on epigenetic testing becomes an increasingly attractive option for researchers working on other common disease. In fact several theoretical frameworks have already been proposed for integrating and combining the power of genetic and epigenetic association studies. Specifically, it appears that epigenetic alteration in complex disease other than cancer are orders of magnitude weaker and rarer than those observed in tumors.

References

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2. Mikkelsen, TS et al. Nature 2008, 454, 49-55.

