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New Dimensions to Precision Medicine Through Omics

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ABSTRACT

We have subjective opinions with regard to our food, clothing and recreational activities. Similarly, do we have the scope to access medicines or drugs that are best compliant for us? Surprisingly, the answer is 'Yes', and thanks to the concept of precision medicine! Precision medicine is the ultimate paradigm of efficient clinical practice, as every individual would benefit from what is best for them. Drug development with individualized compatibility is achievable through the insight of omics. Omics comprehensively encompasses every field of biotechnology that structurally or functionally relates with the biological makeup of an organism, at various levels. It is interesting and equally promising to explore the new dimensions in precision medicine through various omics, such as genomics, transcriptomics, proteiomics, metabolomics, and epigenomics. Lipidomics, redoxomics and interactomics are the latest additions to the omics family.

Keywords: Omics; Genomics; Transcriptomics; Proteiomics; Metabolomics, Epigenomics; Lipidomics; Interactomics; Precision Medicine

Introduction

Precision medicine or personalized medicine refers to the radical approach of customizing medical treatments and prevention strategies in compliance with the genetic makeup, lifestyle factors and environmental influencers of every individual. This approach aims at providing precise methodologies for the prevention, diagnosis and treatment of health disorders. Drug efficacy is quantified through a biomarker. Every drug used as a preventive/treatment mode targets a biological pathway which manifests as a phenotypic trait with indispensable roots in various omics. At baseline lies the genomics, which studies an individual's entire DNA/Deoxyribonucleic acid sequence (genome) with relevance to instructions for protein synthesis. Genes within the nucleus of a cell instruct protein synthesis through a specific order of their base pair arrangement. This instruction expels the nucleus and translates in a cell's cytoplasm with the assistance of enzymes and messenger molecules (messenger ribonucleic acid/mRNA). The instructions carried by mRNA are read by ribosomes, and utilized for appropriately arranging the amino acids to form a specific protein. Protein is the verve of every cellular structure and function.

Thus genomics levels-up to transcriptomics where gene expression at the RNA level is studied. And the next ascend is 'metabolomics', which encompasses an understanding of metabolism, both at the cellular as well as organism level, through measurement of molecules (endogenous and exogenous) which serve as substrates or products of biochemical reactions. Apart from the genetic changes that are marked in a DNA sequence, a gene function or the activity of its encoded protein can be altered due to epigenetic mechanisms. Thus epigenomics or the study of epigenetic mechanisms like DNA acetylation, histone modification and non-coding RNA also finds its significance in precision medicine, especially in diseases like asthma where environmental pollutants exert interference to drug efficacy [1]. Precision medicine is achievable through an integration of multiple omics techniques in the biomedical field. And in the following sections, each field of omics is elaborated with relevant examples in drug efficacy.

Omics Integration in Precision Medicine – Illustrated with Practical Scenarios

Genomics

Precision pharmacotherapy strongly rests on genomics. Pharmacogenomics defines the usage of genomic technologies (like gene sequencing, gene expression analysis, and statistical genetics) in drug discovery with proposed clinical use and market value. It employs genomic approaches which are large scaled and systematic to quicken the discovery of 'drug response' markers. Such markers might notify an action at the drug target site, or it might be indicative of the drug metabolism, establishing a link with the disease pathway. Potentially, the implications of pharmacogenomics in clinical medicine and research would outline the selection of precise drugs and optimization of their dosage to suit an individual's 'drug-response' markers. The scope to categorize 'patient' population on the basis of genetics in drug response might upscale safety and efficacy in therapeutic areas such as diabetes, obesity, cardiology, neuropsychiatry, endocrinology and oncology. This can be attributed to a prior knowledge and preparedness to avert/manage the drug-induced, adverse outcomes [2].

In renal transplantation, the drug 'tacrolimus' is considered as the cornerstone of immunosuppression. Tacrolimus gets metabolized by a cytochrome enzyme (cytochrome P 450 3A or CYP3A subfamily) in the liver and small intestine. The CYP3A5 gene regulates this enzyme activity; and hence, a variation in intron 3 affects the gene expression, effecting tacrolimus trough blood levels through an altered enzyme activity. A study was conducted by Nair et al., 2015, to observe the influence of CYP3A5 polymorphism on tacrolimus-induced nephrotoxicity and acute rejection. Study participants included 25 adults who underwent renal transplantation, and were on tacrolimus. The tacrolimus dosage was 0.1 mg/kg/body weight, distributed across two divided doses. The 'tacrolimus' trough blood levels were evaluated on day 6, post-operative. Amongst the various genotypes of CYP3A5, the frequency of 'acute rejection' episodes was significantly higher in CYP3A5*1/*1 homozygotes (40%), compared to the other genotypes, namely CYP3A5*1/*3 (20%) and CYP3A5*3/*3 (13%). The carriers of the mutant allele CYP3A5*3 (A6986G) were comparatively more in number. A significant correlation was evident for tacrolimus drug level and CYP3A5 polymorphism. The CYP3A5 'expressors' exhibited frequent acute rejection episodes, physiologically demanding a higher dose of tacrolimus. While the 'non-expressors', had higher frequency of tacrolimus nephrotoxicity. Thus, CYP3A5 genetic assessment prior to a renal transplantation might aid in determining the optimal tacrolimus dosage to prevent drug toxicity and acute rejection episodes [3].

Transcriptomics

Transcriptome refers to the entire RNA (ribosomal nucleic acid) transcripts namely, messenger RNA/mRNA, transfer RNA/tRNA, ribosomal RNA/rRNA, and other non-coding RNAs. The RNA transcripts are produced by the genome of every cell, consequently transcriptomics refers to the study of gene expression at the RNA level. Transcriptomics comprises of genome wide information on gene structure and function pertaining to their molecular mechanisms in certain biological pathways. By comparing transcriptomes, we can identify the differential expression of genes in response to various drug treatments in a diverse cell population [4]. Inter-individual variability in drug response has manifold reasons; still gene expressions and its outcome, the transcriptome have a significant role. For instance, assessing the urinary levels of FOXP3 mRNA might serve as a non-invasive marker for predicting acute rejection outcome in renal transplant patients [5]. The absence of Foxp3, due to its unfavorable gene expression, might lead to an abnormality in Tregs' production. Amongst kidney transplant patients with reported acute rejection, an increased urinary level of Foxp3 mRNA expression was observed by Muthukumar et al. [5]. The drug, cyclosporine (CsA), belongs to the family of calcineurin inhibitors (CNI). The usage of cyclosporine is imperative for immunosuppression and the consequential prevention of allograft rejection after a kidney transplantation. Cyclosporine exerts immunosuppression by inhibiting the activation and propagation of T cells.

The regulatory T cells are a special subcategory of CD4+CD25+ T lymphocytes, which are also known as the 'regulatory T lymphocytes' or Tregs/Treg cells. Tregs are vital in suppressing immune responses, as well as in maintaining tolerance to self-antigens, including the grafts. The transcription factor, namely, Foxp3 or forkhead box P3, programs the development and function of Tregs. Since Tregs (CD4+ CD25+ Foxp3+) are targets of CsA, single nucleotide variations in the FOXP3 gene can effect CsA drug efficacy, demonstrating its role in transplantation tolerance. A single nucleotide polymorphism in FOXP3, rs3761549, has been significantly correlated with the renal allograft function. A supporting scientific evidence by Xu et al., 2017 involved 166 renal transplant patients with at least a 5 year follow-up. During the five year follow-up period after transplantation, the carriers of the T/TT genotype in rs3761549 of FOXP3 gene exhibited a quick decline in the estimated glomerular filtration rate (eGFR) compared to the carriers of C/CC genotype (24.0% vs. 6.3%, P=0.004) [6].

Metabolomics

Metabolomics encompasses an understanding of metabolism, both at the cellular as well as organism level, through measurement of molecules (both endogenous and exogenous) which are either substrates or products of a biochemical reaction. It is the study of metabolism in a comprehensive way, and thus finds its importance in health and disease. Metabolomics also proves its role in drug discovery as proposed drug targets are ascertained through drug metabolism [7].

Recent Advances in Metabolomics Unveil the Pros and Cons of Antimetabolite Chemotherapy: Metabolic enzymes which are a part of nucleotide metabolism or the folate cycle are the targets of antimetabolite compounds. These metabolic enzymes can have a direct role in nucleotide biosynthesis or they can indirectly influence met-

abolic processes which are coupled to the flux into a nucleotide pool, and one carbon metabolism. Thus, metabolic enzymes with their direct effects on biological mechanisms, may encode a path for identifying biomarkers to assess cytotoxic response which could eventually improve the precision of a drug. Such an endeavor will bring precision medicine to the forefront overcoming the past claims of 'lack in specificity'. In neoplastic disease treatment, the antimetabolite chemotherapy is considered as a successful therapeutic strategy. Owing to the risk of high toxicity which might arise from its varied efficacies, there is a compelling biomedical need to identify the precise situation where it may definitely be effective. Quoting the beneficial side, anti-metabolite agents succeed in targeting one carbon metabolism thus being labelled as 'frontline' chemotherapy agents for numerous cancers. A common antimetabolite, the 5-fluorouracil/5-FU, disrupts the pyrimidine homeostasis, thus inducing specific alterations to nucleotide metabolism. The cellular response to 5-FU through metabolomics analysis revealed intracellular uracil accumulation. In deoxyuridine levels, an increased flux into the extracellular space was observed culminating in an induction of overflow metabolism. This nucleotide overflow might serve as a biomarker for ascertaining a positive response to the 5-FU antimetabolite chemotherapy [8].

An Intersection of Metabolomics and Genomics Defines New Frontiers in Chemotherapy: The serine-glycine-one carbon/SGOC metabolic network comprises of folate and methionine cycles. This network combines the nutritional status (glucose, amino acids and vitamins), with distinct outputs like biosynthesis (of nucleotides, proteins, and lipids), redox status maintenance, and substrate supply in methylation reactions. Genes of this metabolic pathway have crucial roles in tumorigenesis, and thus this metabolic network is the target for a wide spectrum of chemotherapies. Let's understand with a few examples… The Phosphoglycerate Dehydrogenase (PHGDH) is a protein coding gene, and its related pathways are serine and glycine biosynthesis and one-carbon metabolism. It qualifies as a cancer-driving gene as its variant product diverts glucose metabolism into one carbon metabolism. Serine and glycine uptake correlate with cell proliferation, and this ascertained through a metabolic analysis of uptake and excretion rates measured in cancer cells. Thus, the gene SHMT2 which encodes a mitochondrial serine hydroxymethyltransferase, provides context-dependent susceptible liabilities for tumor maintenance [8].

What is Semi-Targeted Metabolomics? Why is it Gaining Momentum Currently?:

So far, we have heard about targeted experiments in metabolomics which profoundly perceive a specific hypothesis through absolute quantitation of molecules or measuring the rates/fluxes of converting of one molecule to another. Targeted metabolomic analysis mandates substantial pre-existing knowledge with its success relying on the strength of the tested hypothesis. On the other hand, when we have a list of defined metabolites with which we intend to explore a hypothesis, then our experiment becomes semi-targeted. When it comes to new discoveries, the approach of 'semi-targeted metabolomics' is valuable and convenient; as molecules, in significant numbers, can be identified and quantified, unambiguously. Such an affluent approach can generate data to characterize the properties of a biological pathway, which otherwise should be obtained from multiple biochemical assays, each analyzed separately. Metabolomics obtains data through the testing of existing hypotheses from transcriptiomics or proteomics. While semi-targeted metabolomics can provide insights when there is hypothesis lack or a rejection of original hypothesis. Semi-targeted metabolomics' analysis has led to a deeper understanding of various physiological as well as pathophysiological concepts such as diabetes, metabolic syndrome, cardiovascular diseases, and cancer. An increased level of branched-chain amino acids or BCAAs in plasma associates with a higher risk for metabolic disorders like diabetes and cardiovascular diseases, emphasizing the vitality of mitochondrial amino acid oxidation in the etiology of such diseases [9].

Lipidomics

Cellular lipids have significant role in biological mechanisms or pathways. They are fundamental constituents of cellular membranes and lipid particles such as lipoproteins. The concentration of cellular lipids ranges from amol to nmol/mg protein, and their molecular species are tens to hundreds of thousands. The complete set of lipids in the cell of an organism is called a lipidome. Lipidomics encompasses the study of a lipidome, its structure, functions as well as interactions with other cellular components [10].

Lipidomics in Cardiovascular Medicine: Lipidomics, a subset of metabolomics, is a rapidly emerging technology of biomedical research. Lipidomics detects deviations in a metabolic and/or signaling pathway at various stages of a disease. It also finds its role in elucidating the mechanism of drug action or as readouts in Mendelian randomization approaches. At every stage of a disease, proteins and other metabolites (mainly lipids) mark their fundamental role as biomarkers. Proteomics covers the characterization and quantification of a wide range of proteins which are involved in gene expression. While lipidomics occupies the opposite end of the 'omics' spectrum, with its essential role in mediating signal transduction. Statins are the cornerstone for dyslipidemia management, and they are widely prescribed for reducing the atherogenic LDL (low density lipoprotein) cholesterol in the blood. Despite the benefits of LDL-C lowering, statin-induced myopathy (rhabdomyolysis) is a major obstacle for long-term statin compliance. A high dosage of simvastatin (80 mg/day) affects several metabolic and signaling pathways in a skeletal muscle. A combined metabolomic/lipidomic approach might facilitate the usage of serum biomarkers for precisely representing the 'early' statin toxicity. For instance, periodic assessment of cholesterol abnormalities and creatinine kinase levels, combined with detection of mitochondrial DNA levels and enzyme activity of respiratory chain in skeletal muscle might predict statin toxicity, early on. These biomarkers will aid in

treatment decisions such as considering the likelihood of tolerating the dosage reduction, switching between various statin types or using alternative means to LDL-C in the at-risk group [11,12].

Epigenomics

The epigenome comprises of attachments (like chemical compounds and proteins) to the genome which modify its function by 'marking' it. These marks on the genome influence gene function which consequently affect the production of its encoded protein, without altering the DNA sequence. The epigenome or the marks on a genome could be a resultant of epigenetic processes such as, hypo/hyper methylation of DNA, histone modification and expression of non-coding RNA.

Potential Asthma Therapies Targeting Histone Modification: Histones are proteins around which a DNA is wrapped. Histones are vital for normal cell function, as they maintain DNA in an intact form. In a DNA, they can either enhance transcription by enabling accessibility to transcription factors (euchromatin/open state), or they can stop transcription tightly coiling the DNA (heterochromatin/closed state). The post-transcriptional modifications in the N-terminal tail of a histone protein controls its strength of binding with DNA. This usually pertains to addition of an acetyl or a methyl group to the amino acids like lysine and arginine. The histone acetyltransferases or HATs add an acetyl group to the lysine residue of the histone, which can turn on/off certain genes. The activity of HATs was seen to be high in biopsy samples from asthmatics (both children and adults). Site-specific histone acetylation is an important drug target in asthma. For instance, the acetylation on histone 3 lysine 9 (H3K9) is shown to increase the transcription of specific genes such as the pro-inflammatory transcription factor NF-κB (nuclear factor kappa B). Another example is H3K9 methyltransferases or H3K9me3 which is associated with airway remodeling and repression of inflammatory genes. In asthma, airway remodeling is an anticipated feature, mostly in response to an allergen, wherein the airway walls are thickened and there is mucus hypersecretion by human airway smooth muscle cells/HASM. Amongst asthmatics, there is a decrease in H3K9me3 repressive complex at the promoter region of the VEGF/vascular endothelial growth factor gene in the HASm cells. Drugs that are potent Inhibitors of both H3K9 methyltransferases are recently being developed. An H3K9me3 demethylase (JMJD2D) which removes H3K9me3 repression complexes, can favorably activate transcription of VEGF gene [13,14].

B A noncoding RNAs or ncRNA refers to the functional RNAs which aren't transcribed. The small noncoding RNAs namely siRNA and microRNAs/miRNAs have a mentionable role in respiratory diseases. In asthma, the ncRNAs are differentially expressed in a cell-specific manner, thus etching a role for single cell omics. The evaluation of ncRNA's role in a disease like asthma encompasses the inspection of differential expression in different patient subsets, and also a comparison with their healthy counterparts. This especially targets a single ncRNA which changes expression significantly, over time, to inhibit or overexpress its action. Treatments aiming at reducing reduce eosinophilia through suppression of cytokine signaling (SOCS3) can effectively target such and siRNA [13,14].

Drug-Induced Epigenetic Effects on Genome: Asthma is amongst the top concerning biomedical conditions with its contribution of one in each 250 deaths, globally. There is a heterogeneity of nearly 10% in treatment response with severe asthmatic symptoms remaining unresolved through current therapies, in this sub-group. Currently, the first-line bronchodialator, albuterol (a short-acting β2-agonist) is widely used. On a population-based comparison, the bronchodilator drug response/BDR of albuterol is less in African Americans and Puerto Ricans than the Mexican Americans. However, owing to a cost curtail, there is a likelihood to overuse albuterol ignoring its low BDR. As a result, there could be molecular effects on airway epithelium due to an overuse. This epigenome effect associates the hereditary component, genome expression and environmental influence on asthma. The commonly used nasal samples are non-invasive proxies of lung condition, as nasal airway transcriptome reflects the asthma status correlating with bronchial airway cells [15].

An epigenome-wide association study/EGWAS used paired nasal epithelial samples from a population of 97 children (Puerto Rican) to identify almost 22 CpGs genome-wide associated sites for repeated use of albuterol ($p < 9 \times 10–8$). The predominant epigenetic effect of albuterol was hypomethylation on CpGs as captured using EPIC array across the genome (probability of hypomethylation being 76% with a p value of $3.3 \times 10–5$). From nasal epithelia, hypomethylation was validated on the CpGs including, cg23032799 (CREB3L1), cg10290200 (FLNC), cg05673431 (KSR1) and cg00483640 (MYLK4-LINC01600). Of particular interest, the hypomethylation at CpGs cg05673431 (KSR1) and cg10290200 (FLNC) increased the gene expression in their location site (with a false discovery rate/FDR of less than 0.05) [15]. Numerous stimuli like particles of air pollution, allergens, and viruses might trigger the epithelial cells which are frontline defenders against environmental factors. Subsequent to these triggers, there is an activation of airway epithelial cells and smooth muscle cells. This activation results in secretion of pro-inflammatory mediators (like cytokines) and growth factors (like vascular endothelial growth factor/VEGF). Thus epigenetics accounts, considerably, for the heritability of asthma. Hence it is important to target epigenetic mechanisms through omics' tools. In asthma susceptibility SNPs (ascertained through GWAS and candidate gene studies), the DNA methylation/acetylation modifiers (in response to an allergen) can be targeted to allow specificity of drugs. More importantly, as there is a cell-specific mechanism for epigenomes, it is necessary to relate this with 'single cell' omics.

Single Cell Omics

The heterogeneity of healthy and diseased tissues at the genetic, functional, or compositional level presents major challenges in the discovery and development of drugs. The setbacks concerning

the complexities of heterogeneous tissues has influenced the development of tools for single cell multi-omics such as genomics, transcriptomics, and multiplex proteomic analysis. Numerous technologies to comprehensively analyze the molecular level of a single cell are emerging. For instance, single cell tools with the capacity to assay a huge quantum (>40) of elements including secreted proteins, cell surface markers, and components of phosphoprotein signaling pathways are existing. Additionally, analyzing single cell's genome at a focused/high coverage, its transcriptome at sparse coverage or the entire transcriptome with moderate coverage and high cell statistics, is now possible. Single cell analysis tools are categorized based on the measured analytes of various omics individually or in a combination. Microfluidic methods correlate the molecular study with analysis of small cell populations (say 2 to 3 numbers), alongside enabling the assessment of definite cell functions like motility. Microfluidic designs may permit cell analysis within a highly controlled and customized environment. They also favor non-destructive cell analysis; wherein an interesting finding, like the 'B' cell's ability to produce a specific antibody can be harvested for a broader use. Recently, a couple of tissue staining methods, namely, in situ RNA profiling via sequential hybridization, and proteomic analysis via ion beam profiling have enabled single cell analysis within fixed, intact tissues. Remarkably, the level of multiplexing in these methods has significantly exceeded the traditional immunohistochemical staining methods.

In cancer immunotherapy, single cell analytic tools claim significant guidance across several stages of biological mechanisms. The 'T' cells are the primary tumor killer cells, irrespective of the immunotherapy being based on either one or a combination of the following-dendritic cell vaccines, adoptive cell transfer or checkpoint inhibitors. For any given patient, across his course of therapy regimen, the crucial biomarkers include, kinetic persistence and functional behaviors of specific anti-tumor T cell phenotypes. In cell-based therapies, it is important to consider 'T' cell differentiation while designing clinical protocols. In the recent past, the neoantigens or the patient-specific mutant epitopes were stated to be essential in understanding and controlling the immunotherapy's anti-tumor specificity. Further on, the T cell receptor/TCR α or β -chain sequence is a strongly associated factor for recognizing the expressed neoantigen with a high avidity [16]. The recent era witnesses the advent of an 'omics' approach, eminently implicating in precision medicine for disease prevention and treatment. The exploration of various omics (namely genomics, transcriptomics, proteiomics, metabolomics, epigenomics, lipidomics, redoxomics and their interaction or interactomics) has set-foot in precision medicine with its high-throughput technologies substantiating the insights on biological pathways as drug targets. Personalized medicine or precision medicine refers to the timely accessibility of the right drug at the right dosage for an individual, paving way for tailor-made and effective treatment regimens. Multi-omics approaches can increase the efficiency of drug-development platforms. Nevertheless, we will have to ensure appropriate handling of large data sets through parallel development of computational approaches and systems bioinformatics.

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