Egypt. J. Chem. **Vol. 68,** No. 12 pp. 11 - 20 (2025)

Egyptian Journal of Chemistry

http://ejchem.journals.ekb.eg/

Personalized Medicine in Managing Chronic Disease: A Role for Genetic and Socioeconomic Factors

Ali Mohammed Ali Al Sheban [1*], Zahra Nasser Ali Aljubran[2], Norah Abdulmohsen Alsuwailem[2], Seham Moteeb Al-Motairi[3], Samia Hassan Alfifi[3], Amjad Aiaid Almutairi[3], Roseline Deline Davids[3], Shaden Alzahrani[3]

1 King Saud University, College of Pharmacy, Department of Pharmaceutical Chemistry, Saudi Arbaia

2 Eastern Region, Al Nada Health Center, Ministry of Health, Saudi Arabia

3 Nurse Specialist, King Abdulaziz Medical City, Saudi Arabia

Abstract

Chronic noncommunicable diseases (NCDs), including type 2 diabetes, cardiovascular diseases, and cancers, impose a gradually growing global burden and are associated with chronic, low-grade systemic inflammation. Systemic inflammation can arise from both genetic susceptibility and modifiable environmental and lifestyle exposures that interact and contribute to the development of chronic diseases. The exposome—a framework that encompasses every environmental exposure throughout a person's life—is a powerful model through which we can understand how diet, air pollution, physical inactivity, and psychological stress contribute to disease-specific pathology. Additionally, social determinants of health—including food insecurity, urban stress factors, and inequities in health—also drive inflammation. The gut microbiome acts as a liaison between external environmental influence and internal immune responses. Precision medicine also offers a revolutionary paradigm through the use of multi-omics (genomics, epigenomics, metabolomics) and exposomics to establish individual disease risk and guidance for interventions. Artificial intelligence (AI) and machine learning (ML) are essential for applying these large datasets to identify new biomarkers of disease, ultimately predicting disease risk and informing personalized treatment. Wearable technology can further support health monitoring in real time. Although there are challenges to consider related to privacy and bias from algorithms, personalized medicine supplemented by public health can make chronic disease interventions more effective and equitable. From a health systems perspective, this represents a shift toward a preventative and data-based system to improve health outcomes and lessen the probability of a chronic disease occurring worldwide.

Keywords: Personalised Medicine, Chronic Inflammation, Exposome, Multi-Ome, Socioeconomic Factors

1. Introduction

The remarkable increase in clinical science and mass vitality over the past two centuries has transformed human longevity, doubling life expectancy through breakthroughs such as improved sanitation, widespread inoculation campaigns, and progressive medical treatment (Oeppen & Vaupel, 2002). These advances have significantly reduced mortality due to infectious diseases and acute situations. Still, the current widening of life has brought about a recent mass fitness obstacle: the rising incidence of chronic noncommunicable diseases (NCDs), which promptly dominate planet mortality statistics (GBD 2019 Diseases and Injuries Collaborators, 2020). Increasingly common are conditions such as type 2 diabetes, fleshiness, cardiovascular disease (CVD), metabolically associated fatso liver disease (MAFLD), cancer, chronic lung and kidney disease, autoimmune conditions, and neurodegenerative conditions which together account for over 50% of all deaths worldwide due to their association with chronic inflammatory methods (Furman et al., 2019). This adjustment is characterised by a complex interaction of contemporary lifestyles, environmentally friendly exposure, and natural sensibilities, which together drive the global burden of chronic diseases.

The current situation, defined by calorie-dense food, sedentary behaviors, and exposure to a sustainable pollutant, strikingly contrasts with the ancestral situations for which homosapiens genetics evolved to adapt (Cordain et al., 2005). Our ancestors, who have evolved over the millennia to thrive in an environment with limited support and increased body demands, are not well suited to the modern perspective of abundant food processing, urban life, and chemical exposure. The estimated contribution of non-genetic components, including lifestyle options and ecological adaptations, to chronic diseases is estimated to be 80–90 % of the attributable liability for chronic diseases, far exceeding the merely heritable input (Rappaport & Smith, 2010). The International Health Responsibility (GBD) examines, looking at information from 1990 to 2016 through 195 countries, shows that modifiable vulnerabilities factors such as needy diet, physical inactivity, smoking, and alcohol

*Corresponding author e-mail: alialasiri@ksu.edu.sa.; (Mohammed Ali Al Sheban, Ali).
Receive Date: 19 February 2025, Revise Date: 09 June 2025, Accept Date: 1 June 2025
DOI: 10.21608/ejchem.2025.362027.11330

consumption are reliable for approximately 60 % of global mortality (GBD 2016 hazard variables Collaborators, Year 2 17). Such factors disrupt physiologic homeostasis, leading to chronic low-grade inflammation known as metaflammation, which is a cornerstone of the evolution and progression of several NCDs (Hotamisligil, 1997).

The exposome theory has emerged as a complete outline in order to better distinguish these non-genetic effects. The exposome encompasses the entire environmental exposure an individual encounters, including diet, wind strength, interpersonal stressors, and internal organic methods such as the microbiome project and oxidative stress (Wild, 2012 CE). Exposomics seeks to quantify the aforementioned exposure and its physiological consequences, providing perceptions on how ecological components interact with hereditary sensitivity in order to accelerate disease (Miller & Jones, 2014). At the same time, precision medicine uses hereditary, ecological, and lifestyle data to develop appropriate prevention and treatment strategies (Collins & Varmus, 2015). The integration of AI, including machine learning, deep learning, and synthetic neural networks (ANN), brings about a further increase in accuracy by allowing the examination of large datasets generated by multi-omics (e.g., Genomics, Epigenomics, Metabolism, and Wearable Detector Technologies (Topol, 2019). These tools make it possible to accurately predict the risk of a disease and to design a personalized treatment plan, a paradigm shift in the management of chronic diseases.

That evaluates targets to synthesize the support of lifestyle and living components for chronic inflammation, clarify the position of the exposome in the pathogenesis of the disease, and discover how machine learning-based precision medicine can modify the prevention and treatment of NCDs. The current study underlines the current strategies for dealing with the global chronic disease epidemic through an examination of the inflammatory mechanisms underlying chronic diseases, the sustainable and organic causes of inflammation, as well as the ability of advanced computerized methods.

1. Chronic Inflammation

Inflammation is an important organ system that contributes to the body's innate defense mechanism against pathogens, body injury, and harmful stimuli (Medzhitov, 2010). Acute inflammation is a tightly controlled, short-lived reaction designed to neutralize dangers, such as bacterial infections, other tissue damage, and facilitate repair. The release of proinflammatory mediators, e.g., cytokines, interleukin-1, tumor necrosis factor-alpha, and chemokines, which recruit immune cells to the site of injury or infection. Anti-inflammatory signals restore homeostasis, preventing tissue damage (Medzhitov, 2010). This method is essential for perseverance, allowing the body to fight illness and heal effectively.

Still, scientists discovered a distinct structure of inflammation associated with fleshiness, called metaflammation, which differed significantly from acute inflammation (Hotamisligil et al., 1993). Metaflammation may be a chronic, low-grade systemic inflammation, characterized by persistently increased levels of inflammatory mediators, identical to CRP, IL-6, and TNF-, as well as increased immune cell infiltration in peripheral tissues, especially adipose tissues (Gregor & Hotamisligil, 2011). Unlike acute inflammation, metaflammation does not occur spontaneously and does not impair the primary function of the tissue concerned. Alternatively, it creates a sustained inflammation setting that disrupts metabolic and physiological homeostasis, facilitating the pathogenesis of multiple chronic diseases, including diabetes, cardiovascular disease, and certain malignancies (Furman et al., 2019).

Continuous stimulation, such as excessive calorie consumption, sedentary lifestyle, psychological stress, and ecological exposure, may be driving the chronic environment of metaflammation. These components preserve a cycle of immune activation and tissue damage, essential for progressive health deterioration. For example, in the fleshiness, adipose tissue becomes a major supporter of proinflammatory cytokines which disrupt insulin signal and increase insulin resistance, characteristic of type 2 diabetes (Shoelson et al., 2007). In the same way, chronic inflammation of the arterial wall plays a role in the progression of atherosclerosis, a crucial factor in CVDs (Libby et al., 2011). The prominent effect of metaflammation stresses the role of the primary mechanism in the evolution and progression of NCDs and calls for targeted interventions to mitigate their consequences (Franceschi et al., 2018).

2. The Role of the Exposome in Inflammation

The exposome provides a comprehensive outline for understanding the environmental drivers of chronic inflammation, encompassing both external and internal exposure of humans throughout their lives (Wild, 2012). External exposure variables include lifestyle options, e.g., diet, material handling, smoke, body, and chemical exposure, e.g., sky pollution, pesticides, habitat characteristics, e.g., urban environment, climate crisis, and social determinants, e.g., Rappaport & Smith, 2010; Miller & Jones, 2014; Vineis et al., 2017). In the exposome, organic processes such as the functioning of the gut microbiome, oxidative stress, and metabolic wastes interact with external exposure to modulate the inflammatory response (Clemente et al., 2018).

External exposure elements significantly contribute to chronic inflammation. For instance, natural chemicals such as linuron (an herbicide used in agriculture) and methyl carbamate (a compound used in industry) have been demonstrated to increase neuroinflammation through increased astrocyte activation, possibly contributing to neurodegenerative diseases (Kim et al., 2020). Air pollution is a well-documented trigger of systemic inflammation, increasing the risk of cardiovascular and respiratory diseases through oxidative stress and immune activation (Brook et al., 2010). The urban environment, characterised by high pollution rates and limited greenery, is further exacerbated by sedentary behavior and stress (Wild, 2012). Social components such as chronic stress from minimal socioeconomic conditions or societal isolation trip the hypothalamic-pituitary-adrenal (HPA) axis, promoting hydrocortisone and proinflammatory cytokine tiers which aggravate inflammation (Slavich & Irwin, 2014).

The involvement of intrinsic exposome variables, especially the intestinal microbiome, plays a key role in modulating inflammation. Dysbiosis, an imbalance of microbial composition, may trigger systemic inflammation by disrupting the gut barrier, allowing microbial entities to take advantage of lipopolysaccharides (LPS) to enter the bloodstream (Cani et al., 2007). Oxidative stress, caused by an imbalance of reactive oxygen species (ROS) and antioxidant defenses, increases inflammation

through damage to cellular components and a trip of inflammatory nerve pathways (Pizzino et al., 2017). The interaction of external and internal exposure components reveals the complexity of chronic inflammation and calls for a complete technique to understand and reduce its cause.

3. Lifestyle Factors Driving Chronic Inflammation

Diet is a major determinant of chronic inflammation, with modern diets, particularly Western diets (WD), playing an important role in its onset and progression (Christ et al., 2019). The western diet, which qualifies as a high consumption of ultraprocessed foods, sugar, trans fats, saturated fats, and sodium, disrupts the metabolic and immune homeostasis, promoting inflammation. This diet disrupts the functioning of the intestine, allowing microbial substances such as lipopolysaccharides (LPS) to leak into the blood, a phenomenon known as metabolic endotoxemia (Cani et al., 2007). For example, a high-fructose diet common in sugary drinks and industrial foods alters the intestinal microbiome, primarily affecting the production of acetate, which disrupts hepatic lipogenesis and contributes to dyslipidemia and MAFLD. The microbial-metabolic axis concentrates on the intricate connection of diet, microbiome, and inflammation.

Nutrient deficiency, additional compound, and chronic inflammation. Insufficient consumption of essential micronutrients, such as vitamin D, magnesium, and zinc, disrupts immune control and antioxidant defense and exacerbates the inflammatory response (Gombart et al., 2020). For instance, vitamin D deficiency is associated with increased levels of proinflammatory cytokines and an increased risk of autoimmune diseases (Holick, 2nd ed., 1997). On the contrary, an anti-inflammatory diet rich in fruit, vegetables, whole grains, and omega-3 fatty acids (e.g., fish can reduce inflammation by reducing cytokine production and improving gut health (Sears, 2015). The Mediterranean diet, which emphasizes plant foods and healthy fats, has been shown to lower inflammation markers and reduce the risk of CVD and type 2 diabetes (Estruch et al., 2018).

The wide range of dietary effects extends beyond human health to planet's structures. The Lancet Commission is leading the global Syndemic debate on how current food development and consumer forms contribute to the ternary burden of fleshiness, undernutrition, and global warming (Swinburn et al., 2019). The industrial food system, which relies on monoculture crops and highly processed foods, degrades green resilience while promoting an unhealthy diet. Systemic changes such as promoting long-term farming, reducing dependence on processed foodstuffs, and increasing access to nutrient-dense foodstuffs, especially in neglected populations, are needed to deal with this problem.

Physical inactivity is a major contributor to chronic inflammation and NCDs, alongside planet statistics that 27.5 % of adults and over 80 % of adolescents fail to obtain recommended body weight degrees (Guthold et al., 2018). Sedentary behavior contributes to inflammation by increasing the production of proinflammatory cytokines, such as IL-6 and TNF-α, while reducing the anti-inflammatory mediator adiponectin (Henson et al., 2013). Longer sitting, which is common in the current work and leisure environment, worsens insulin resistance and metabolic dysfunction, a major precursor to type 2 diabetes and CVD (Hamilton et al. 2014).

Compared to moderate doses, regular body activity has potent anti-inflammatory effects. Immune function, visceral fat reduction, and low circulating levels of inflammatory markers are enhanced by activities such as alert walking, cycling, or resistance training. For instance, aerobic exercise has been shown to increase the production of anti-inflammatory cytokines, such as IL-10, which counteract metaflammation (Pedersen & Febbraio, 2012). The material task also contributes to the diversity of the gut microbiome and further reduction in inflammation (Monda et al., 2017). In order to reduce the inflammation burden and prevent chronic diseases, population vitality projects promoting active lifestyles, similar to city systematic planning promoting walking and cycling, are key.

Tobacco smoke is a well-established risk factor for chronic inflammatory conditions, contributing to diseases such as COPD, lung cancer, and CVD (Arnson et al., 2010). Nicotine and other tobacco-derived compounds stimulate neutrophil activation, allowing the proinflammatory molecule to generate reactive oxygen species (ROS) and matrix metalloproteinases, which damage and sustain inflammation (Talukder et al., 2011). Smoking also has an adverse effect on the immune system, with increased susceptibility to diseases and autoimmune disorders (Sopori, 2002).

Furthermore, excessive alcohol consumption also contributes to systemic inflammation by disrupting the intestine and liver. Continuing alcohol consumption compromises gut blockade uprightness, leading to increased LPS translocation and hepatic inflammation, which leads to alcoholic liver disease and MAFLD (Wang et al., 2014). Alcohol also stimulates oxidative stress and disrupts the inflammatory nervous system, increasing the risk of tumors, especially the liver, esophagus, and pancreas (Seitz & Stickel, 2007). The risk elements of smoking and excessive alcohol consumption can be significantly reduced by cessation activities supported by population wellness standards, such as the tobacco tax and the recommendations for the use of alcohol.

4. Stress and Socioeconomic Factors

Chronic stress, regardless of the cause (e.g., anxiety, depression), or socioeconomic factors (such as. Poverty and communal isolation may be powerful drivers of inflammation. Stress tripped the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system, leading to increased hydrocortisone and catecholamine levels, which stimulate proinflammatory cytokine development (Slavich & Irwin, 2014). For instance, chronic stress is associated with increased IL-6 and CRP levels, which contribute to environments that favour depression, cardiovascular disease, and metabolic syndrome (Rohleder, 2014). Socioeconomic disparity contributes to these effects by restricting access to robust food, safe space for corporeal projects, and healthcare support, thereby perpetuating a cycle of inflammation and disease (Marmot & Bell, 2019).

An interpersonal factor identical to reduced earnings, education, and employment opportunities creates an environment promoting unhealthy behavior and chronic stress. For instance, food insecurity among low-income citizens regularly leads to reliance on cheap, calorie-dense processed foods, which stimulate inflammation (Seligman et al., 2010). In the same way, the urban environment, together with restricted green space or even high crime rates, prevents the development of the body,

thereby increasing the threat of diseases (Sallis et al., 2016). In order to alleviate inflammation and enhance wellness effects, the solution to the aforementioned social determinants calls for integrated strategies of mass well-being, such as community nutrition programs, low-cost medical assistance entry, and regulations aimed at reducing the revenue imbalance.

5. Biological Factors Driving Chronic Inflammation

Heritable variability, together with variations such as SNPs, interpolation, omission, and systematic genome changes, influences the risk of chronic diseases. Genome-wide association studies (GWAS) have been crucial in recognizing hereditary sites associated with noncommunicable diseases (NCDs), including type 2 diabetes, cardiovascular disease (CVD), fleshiness, asthma, and multiple cancers (Visscher et al., 22017) For instance, a landmark GWAS carried out in the Nipponese community analyzed heritable statistics from a thousand individuals and detected 320 independent ancestral signals in 276 sites related to 27 diseases, including 25 fresh sites unique to this society (Ishigaki et al., 2020). These conclusions highlight the diversity of heritable sensitivity among groups, as certain venues may be precise in order to limit the generalizability of hereditary information in worldwide populations (Popejoy & Fullerton, 2016).

However, this development, inherited variables, which account exclusively for a tiny fraction of the overall threat to chronic diseases, frequently explain less than 10–20 % of the variation in society analysis (Manolio et al., 2009). This restricted explanatory control underlines the importance of gene-environment interactions in the pathogenesis of diseases. For example, SNPs in the FTO gene are highly correlated with obesity traits, but their effects are significantly influenced by lifestyle factors such as diet and body mass index (Loos & Yeo, 2014). Similarly, hereditary differences associated with type 2 diabetes, such as those in the TCF7L2 gene, increase susceptibility but demand a green gun trigger to admire a needy diet or sedentary behavior in order to be considered a disease (Florez et al., 2006). The above interactions highlight the complexities of chronic disease etiology, where biological sensitivity acts as a cornerstone but is magnified or diminished by environmental exposure. Progress on genomic systems, such as next-generation sequencing, continues to reveal new ancestral associations, revealing an approach to targeted therapy and preventive measures specific to the human hereditary profile (Visscher et al., 2017).

Epigenetics, the study of inheritable changes in gene expression that do not alter DNA sequence, provides a crucial link between green exposure and disease threats (Feil & Fraga, 2012). An epigenetic mechanism involving DNA methylation and histone modification, for instance, acetylation, methylation, and noncoding RNA, controls gene expression in response to external stimuli such as diet, stress, pollution, and socioeconomic elements (Baccarelli & Bollati, 2009). These changes are akin to a molecular memory allowing eco-friendly exposure to the domination gene project throughout a being's second life and, in a few instances, throughout coevals. Maternal malnutrition during pregnancy may cause epigenetic changes in the fetus, such as an altered DNA methylation form of gene regulatory lipid and carbohydrate metamorphosis, increased susceptibility to obesity, type 2 diabetes, and metabolic syndrome later in life (Lillycrop & Burdge, 2015). Such epigenetic changes in early life may prevail, determining long-term health outcomes through developmental planning.

The evidence of the interplay of genetics and habitat in the design of the epigenetic profile is thus overwhelming. The study of more than 700 pairs of monozygotic and dizygotic twins shows that during biological components initiating a baseline epigenetic governance, sustainable exposure significantly alters DNA methylation form, influences gene expression, and disease risk (Kaminsky et al., 2009) For instance, exposure to environmental toxins, such as gas pollutant alternatively heavy metallic elements, may cause hypermethylation of the tumor suppressor gene, thereby increasing cancer susceptibilities (Baccarelli & Bollati, 2009). Similarly, chronic stress alters histone acetylation in brain regions associated with mood control, facilitating the development of psychiatric disorders like depression (Tsankova et al., 2007). Epigenetic changes are active and possibly reversible, like dietary supplements and methyl donors (e.g., vitamins, choline) or other approaches to reduce stress, aiming at reducing the risk of disease (Choi & Friso, 2010). The growing field of epigenomics, bolstered by high-throughput sequencers, is persevering to find out how natural exposures shape the epigenetic landscape, providing insights into the new curative target of chronic diseases.

The intestinal microbiome consists of a million microorganisms that live in the GI tract and regulate inflammation and immune function, acting as a link between ecological exposure and physiologic responses (Belkaid & Hand, 2014). A balanced microbiome supports immune homeostasis by producing an anti-inflammatory metabolite identical to short-chain fatty acids (SCFAs) that include butyrate, which reinforces the integrity of the intestine and inhibits the production of proinflammatory cytokines (Rooks & Garrett, 2016). However, dysbiosis, an imbalance of microbial composition, disrupts this balance, promoting chronic inflammation and contributing to diseases such as metabolically associated fatty liver disease (MAFLD), type 2 diabetes, and inflammatory bowel disease (Tilg et al., 2020).

Dysbiosis, altering microbial diversity and disrupting the function of the intestine, is a major driver of this diet, high in processed foods, sugar, and fructose. For instance, a high-fructose diet promotes the development of proinflammatory bacteria, particularly in order to increase the development of lipopolysaccharides (LPS), which trigger systemic inflammation via metabolic endotoxemia (Zmora et al., 2019). This method is particularly important in MAFLD, where microbial metabolites such as ethyl alcohol and acetaldehyde increase liver inflammation and fat accumulation (Leung et al., 2016). Similarly, a diet rich in fiber, prebiotics, and probiotics may restore microbial balance, improve SCFA production, and reduce inflammation (Roberfroid et al., 2010). For example, a supplement containing Lactobacillus and Bifidobacterium strains has been shown to increase insulin sensitivity and decrease inflammation in patients with type 2 diabetes (Tonucci et al., 2017).

The function of the microbiome extends beyond metamorphosis to manage the immune system and neurological health via the gut-brain axis. Dysbiosis has been linked to neuroinflammation, contributing to conditions such as Alzheimer's disease and depression (Cryan et al., 2019). Furthermore, emerging research proposes that microbiome-targeted interventions, such as faecal microbiota transplant (FMT), maintain safety in inflammatory situations despite difficulties with standardization and long-term safety (Gupta & Khanna, 2017). The energetic response of the gut microbiome to lifestyle and environmental factors underlines its potential to be a curative target for reducing chronic inflammation.

6. Precision Medicine

Precision medicine is a revolutionary technique for medical assistance, focusing on customizing prevention, diagnosis, and treatment methods based on the second distinctive biological, ecological, and lifestyle profile of an individual (Collins & Varmus, 2015). Unlike the conventional one-size-fits-all approach, clarity medicine uses detailed molecular and sustainable facts to identify the disease mechanism and enhance intervention. Progress in multi-omics systems, which provide a complete picture of the human body's composition, and exposomics, which quantify natural influences (Ashley, 2016). Homo sapiens Functional Genomics Project (HFGP), which analyses immune responses in 500 well-educated adults, shows significant inter-individual variability in cytokine production influenced by a combination of hereditary polymorphism, microbiome composition, and ecological exposure prefer seasonal variations (Ter Horst et al., 22016) Such conclusions require a personalized approach to adapt them to the same degree of variability in health and disease.

Precision medicine has already shown promise in a number of fields, from oncology to cardiology. Genomic Profiling identifies actionable mutants that enable targeted therapy of lung cancer patients with tyrosine kinase inhibitors and EGFR mutants (Lynch et al., 2004). In chronic disease leadership, precision medicine recognizes individuals with higher liability for circumstances such as type 2 diabetes established in inherited and lifestyle components, allowing early intervention such as a personalized diet plan or pharmacotherapy (Torkamani et al., 2018) Corrective medicine only enhances therapy efficacy in spite of, but it also shifts its focus to prevention, thereby reducing the responsibility of chronic diseases.

7. Multi-Omics and Exposomics

A multi-omics approach, including genomics, epigenomics, transcriptomics, proteomics, metabolomics, and microbiomics, has created huge datasets capturing the complex interaction among molecular and sustainable components of the disease (Hasin et al., 2017). Genetics identifies SNPs related to a disease and provides insights into inherited sensitivity (Visscher et al., 2017). Epigenomics reveals how ecological exposure, similar to diet or pollutants, alters gene expression by DNA methylation or histone modification (Rakyan et al., 2011). Transcriptomics and proteomics clarify gene and protein conformation, while metabolomics profiles small molecules to discover metabolic disturbances associated with inflammation (Wishart, 2016). Microbiomes, focusing on the intestinal microbiome, identify a microbial fingerprint related to the disease state (Tilg et al., 2020). Together, the above-mentioned omics disciplines provide a complete picture of human lifestyle and enable precise disease descriptions.

Exposomics complements multi-omics by systematically quantifying external and internal environmental exposure. External exposure includes lifestyle elements, e.g., diet, smoke, chemical pollutants, e.g., pesticides, heavy metallic elements, and social factors, e.g., stress, socioeconomic condition, whereas inside exposure embraces the microbiome project, oxidative stress, and metabolic waste (Wild, 2012). Environment-wide association studies (EWAS) attempt to quantify the contribution of these exposures to the threat of a disease, comparable to the way GWAS pinpoints biological associations (Patel et al., 2010). Rajagopalan & Brook (2012) correlated exposure to the EWAS research with the exposure of PM2.5 to increased type 2 diabetes risk due to the inflammation of the nerves. Wearable detectors, such as smartwatches and biosensors, additionally enhance exposomics by providing real-time statistics on physiological parameters (e.g., compassion assessment, glucose levels, and exposure to natural substances (e.g., Breathing capacity, UV radiation) (Dunn et al., 2018). The integration of multi-omics and exposomics generates huge statistics and requires sophisticated computer hardware for the system and interpretation of the resulting data.

8. AI and Machine Learning in Precision Medicine

Intelligent automation (AI) in ANN and machine learning has transformed the analysis of large, complex data sets in Clarity Medicine (Rajkomar et al., 2019). AI methods excel at pattern detection, prediction of outcomes, and identification of relationships in data from various omics and exposomics sources, which are quicker and more precise than traditional statistical techniques. Machine learning algorithms train on labeled data to identify patient anchoring on disease liability or treatment response, while deep learning algorithms, which mimic nervous systems, use unstructured data with a predilection for clinical images or genomic sequences (Obermeyer et al., 2016). For example, an ML model combining inherited, lifestyle, and natural factors to forecast type 2 diabetes susceptibility with more than 80% accuracy allows early intervention (Segal et al., 2017). Likewise, DL models performed at dermatologist-level accuracy for diagnosing skin cancer from images, showcasing their diagnostic ability (Esteva et al., 2017)

Automated reasoning capability to handle enormous information is particularly sought after in precision medicine, in which many genetic, metabolite, and wearable sensors are commonly a component of a large dataset. Such methods as supervised learning, unsupervised learning, and support training enable AI to identify new biomarkers, stratify patients according to their liability, and personalize treatment strategies (Topol, 2019). ANN models analyze EHRs to predict cardiovascular events and integrate genetics, lifestyle, and clinical data (Dey et al., 2019). Machine intelligence enables real-time monitoring and personalized information from wearable monitors for both patients and physicians.

Integrating AI in managing chronic diseases, employing high-tech computational devices to analyze intricate datasets, and personalizing data-driven treatments. AI, encompassing Machine Acquiring Knowledge (ML), Deep Learning (DL), and Simulated Neural Networks (ANN), offers a groundbreaking goal in four major arenas: risk prediction, personalized medicine, biomarker discovery, and mass fitness intervention. Multi-omics, exposomics, and wearables' data in real time enhance the prediction, diagnosis, and treatment of noncommunicable disorders like type 2 diabetes, cardiovascular disease, cancer, and neurodegenerative disease. AI enables medical device makers to go beyond the conventional one-size-fits-all solution and deliver accurate and capable interventions that address the distinct demands of individuals and societies through collaboration with huge volumes of ancestral, natural, and lifestyle information (Dey et al., 2019).

An ML model integrates statistics like genome profiles, green exposure, and lifestyle factors to predict disease risk accurately. It identifies those at high risk of developing a disease even before symptoms arise, allowing early intervention to

postpone or prevent disease occurrence. Machine learning algorithms integrate genomic data with types of diet to predict fleshiness so that clinicians can prescribe individualized lifestyle changes like personalized nutrition plans or exercise (Choi et al., 2020). For type 2 diabetes, the ML model exhibits a consistent ancestral divergence like the TCF7L2 gene with more than 80% precision based on lifestyle statistics to aid load monitoring and glucose measurement (Segal et al., 2017). Real-time information from wearable technology, like smartwatches tracking body functions and center determination, gives an energy risk assessment that adapts to the changing well-being of a person (Dunn et al., 2018). AI empowered medical treatment systems to choose support effectively, thus cutting the long-term burden of chronic diseases by finding the right people in time.

AI enhances treatment procedures by analyzing patient data to suggest likely effective treatments, reducing the trial-and-error approach prevalent in conventional medicine. In oncology, artificial intelligence-powered platforms admire IBM Watson for Oncology analyze genomic and clinical information to identify a suitable mutant and propose a target therapy identical to a tyrosine kinase inhibitor in lung cancer patients with an EGFR mutation (Johnson et al., 2018). The current strategy aims at reducing medication delays and improving outcomes by matching patients to treatment based on their molecular profile. In diabetes management, AI algorithms utilize real-time data from CGMs and lifestyle factors like diet and physical activity to personalize insulin injection, enhance glycemic control, and minimize hypoglycemic events (Doupis et al., 2020). Closed-loop insulin delivery systems, also known as artificial pancreas, leverage ML for making real-time insulin dosing adjustments, milestones of great importance for type 1 as well as type 2 diabetics (Bekiari et al., 2018). Individualized care enhances the efficacy of care and facilitates attachment by matching interventions to the needs of people, improving life satisfaction.

Deep knowledge processes of acquisition are especially able to discover novel biomarkers with intricate, high-dimensional information from multi-omics analysis for earlier and more precise diagnosis. The DL model identifies a proteomic signature in cerebrospinal fluid that predicts Alzheimer's development and allows for early treatment, perhaps hastening cognitive deterioration (Litjens et al., 2017). In oncology, machine intelligence enhanced non-invasive examination through the detection of tumor DNA (ctDNA) in blood with high specificity and sensitivity (Cohen et al., 2018). A DL model of ctDNA enabled early detection of colorectal and lung cancer, providing a less invasive option compared to the conventional biopsy (Cristiano et al., 2019). Biomarkers yield invaluable insights into disease processes, allowing physicians to risk-stratify patients and create personalized treatment plans. Data-driven biomarker discovery also speeds new therapeutics, including cancer immunotherapy and anti-inflammatory drugs for autoimmune disorders (Vamathevan et al., 2019). The capacity of DL to handle unstructured data, like clinical images or a gene sequence, qualifies it as a pillar of proper medicine.

Machine learning enhances population planning, optimizes supply allocation, and recognizes cohorts for prevention. Predictive algorithms also detect high tobacco disease-burden districts to target cessation programs (Beam & Kohane, 2018). For example, ML on EHRs and socioeconmic data triggered interventions in high CVD districts, financing study, screening, and lifestyle programs (Herrin et al., 2016).

Automated reasoning has also been critically involved in epidemic surveillance, as shown during the COVID-19 pandemic, in which forecasting models monitor transmission and lightning lockdown schemes (Lalmuanawma et al., 2020). With the integration of data from various sources, including environment sensors, community determinants, and medical aid use, AI helps population health officials to prioritize interventions, like inoculation, political campaigns, and nutrition projects, in underserved areas. Population-level intention precision medicine refines by tackling chronic disease drivers, promoting equitable wellness in multiethnic populations (Khoury et al., 2016).

9. Challenges and Future Directions

Despite its promises, machine intelligence in precision medicine poses useful challenges. Information confidentiality remains a major concern given that multi-omics and exposomics datasets contain delicate, unique data, which increases the risk of breach or misuse (Price & Cohen, 2019). Algorithmic discrimination may also be a problem since a model trained on non-representative datasets is likely to produce skewed predictions, disproportionately painful oppressed collectives (Obermeyer et al., 2019). A highly used medical assistance algorithm underestimated liability in African American patients due to biased training information, emphasizing the need for different datasets (Obermeyer et al., 2019). Furthermore, integration into the clinical workflow is hindered by the need to train medical practitioners on how to interpret the final product of AI and integrate it into the verdict (Sendak et al., 2020).

Such obstacles are likely to be the focus of future advances in AI. A Federated study, which enables model training via decentralised datasets lacking shared responsive facts, provides a solution to the loneliness panics (Konen et al., 2016). Explainable AI, which provides a clear explanation of model predictions, will increase clinician confidence and acceptability (Holzinger et al., 2018). Moreover, active, personalized vitality monitoring, including a method for proactive disease prevention, will be enabled by integrating machine intelligence with real-time data from wearable detectors and EHRs. As these systems mature, AI will increasingly play a key role in precision medicine, transforming chronic disease management.

10. Conclusion

The global epidemic of non-communicable diseases, including type 2 diabetes, cardiovascular disease, and cancer, is fuelled by chronic inflammation triggered by a complex interaction between lifestyle and living components. The Exposome Foundation sheds light on the ways eco-friendly exposures, ranging from diet and pollution to societal stressors, interact with biological sensitivity and epigenetic changes in order to regulate the pathogenesis of diseases. The gut microbiome is even more amplified, acting as a key mediator of inflammation and immune function. Precision medicine, empowered by multi-omics, exposomics, and machine intelligence, provides a ground-breaking strategy for chronic disease management through personalized prevention and treatment to profile. In order to support personalized health monitoring, machine learning and deep learning methods excel in investigating large datasets, predicting disease severity, determining biomarkers, and improving therapy, while wearable detectors provide real-time information to support personalized health monitoring. However, in order to fully exploit AI's potential, privacy of data, differential partiality, and clinical integration must be overcome.

Public wellness approaches take a complementary position by addressing interpersonal factors such as food insecurity and the sedentary environment through systemic interventions for sustainable food systems and urban planning. Health care systems can address the root cause of chronic inflammation by integrating corrective medicine with community initiatives, thereby reducing the global responsibility for diseases. As AI and omics continue to advance, their integration into standard health care structures in clinics and communities will redefine chronic disease prevention and treatment, paving the way for healthier, longer lives. In order to ensure that clarity medicine rewards a wide range of populations around the world, the next phase of exploration should focus on the development of simple, accessible, and transparent machine learning-based treatments.

References

1. Arnson, Y., Shoenfeld, Y., & Amital, H. (2010). Effects of tobacco smoke on immunity, inflammation and autoimmunity. *Journal of Autoimmunity, 34*(3), J258–J265. https://doi.org/10.1016/j.jaut.2009.12.003
2. Ashley, E. A. (2016). Towards precision medicine. *Nature Reviews Genetics, 17*(9), 507–522. https://doi.org/10.1038/nrg.2016.86
3. Baccarelli, A., & Bollati, V. (2009). Epigenetics and environmental chemicals. *Current Opinion in Pediatrics, 21*(2), 243–251. https://doi.org/10.1097/MOP.0b013e32832925cc
4. Bayés-Genís, A., Voors, A. A., Zannad, F., et al. (2017). Transitioning from usual care to biomarker-based personalized medicine in heart failure. *European Heart Journal, 38*(24), 1843–1845. https://doi.org/10.1093/eurheartj/ehx297
5. Beam, A. L., & Kohane, I. S. (2018). Big data and machine learning in health care. *JAMA, 319*(13), 1317–1318. https://doi.org/10.1001/jama.2017.18391
6. Bekiari, E., Kitsios, K., Thabit, H., et al. (2018). Artificial pancreas treatment for outpatients with type 1 diabetes: Systematic review and meta-analysis. *BMJ, 361*, k1310. https://doi.org/10.1136/bmj.k1310
7. Belkaid, Y., & Hand, T. W. (2014). Role of the microbiota in immunity and inflammation. *Cell, 157*(1), 121–141. https://doi.org/10.1016/j.cell.2014.03.011
8. Bloom, D. E., Cafiero, E. T., Jané-Llopis, E., et al. (2011). *The global economic burden of noncommunicable diseases*. World Economic Forum. https://www.weforum.org/publications/the-global-economic-burden-of-non-communicable-diseases/
9. Brook, R. D., Rajagopalan, S., Pope, C. A., et al. (2010). Particulate matter air pollution and cardiovascular disease. *Circulation, 121*(21), 2331–2378. https://doi.org/10.1161/CIR.0b013e3181dbece1
10. Cani, P. D., Amar, J., Iglesias, M. A., et al. (2007). Metabolic endotoxemia initiates obesity and insulin resistance. *Diabetes, 56*(7), 1761–1772. https://doi.org/10.2337/db06-1491
11. Choi, E., Bahadori, M. T., Song, L., et al. (2020). Machine learning for healthcare: Clinical applications and challenges. *IEEE Reviews in Biomedical Engineering, 13*, 116–129. https://doi.org/10.1109/RBME.2019.2950981
12. Choi, S. W., & Friso, S. (2010). Epigenetics: A new bridge between nutrition and health. *Advances in Nutrition, 1*(1), 8–16. https://doi.org/10.3945/an.110.1004
13. Christ, A., Lauterbach, M., & Latz, E. (2019). Western diet and the immune system: An inflammatory connection. *Immunity, 51*(5), 794–811. https://doi.org/10.1016/j.immuni.2019.09.020
14. Clemente, J. C., Manasson, J., & Scher, J. U. (2018). The role of the gut microbiome in systemic inflammatory disease. *BMJ, 360*, j5145. https://doi.org/10.1136/bmj.j5145
15. Cohen, J. D., Li, L., Wang, Y., et al. (2018). Detection and localization of surgically resectable cancers with a multi-analyte blood test. *Science, 359*(6378), 926–930. https://doi.org/10.1126/science.aar3247
16. Collins, F. S., & Varmus, H. (2015). A new initiative on precision medicine. *New England Journal of Medicine, 372*(9), 793–795. https://doi.org/10.1056/NEJMp1500523
17. Cordain, L., Eaton, S. B., Sebastian, A., et al. (2005). Origins and evolution of the Western diet: Health implications for the 21st century. *American Journal of Clinical Nutrition, 81*(2), 341–354. https://doi.org/10.1093/ajcn/81.2.341
18. Cristiano, S., Leal, A., Phallen, J., et al. (2019). Genome-wide cell-free DNA fragmentation in patients with cancer. *Nature, 570*(7761), 385–389. https://doi.org/10.1038/s41586-019-1272-6
19. Cryan, J. F., O'Riordan, K. J., Cowan, C. S. M., et al. (2019). The microbiota-gut-brain axis. *Physiological Reviews, 99*(4), 1877–2013. https://doi.org/10.1152/physrev.00018.2018
20. Dey, D., Slomka, P. J., Leeson, P., et al. (2019). Artificial intelligence in cardiovascular imaging. *Journal of the American College of Cardiology, 73*(11), 1317–1335. https://doi.org/10.1016/j.jacc.2018.12.054
21. Doupis, J., Festas, G., Tsilivakos, V., et al. (2020). Smartphone-based technology in diabetes management. *Diabetes Therapy, 11*(3), 607–619. https://doi.org/10.1007/s13300-020-00768-4
22. Dunn, J., Runge, R., & Snyder, M. (2018). Wearables and the medical revolution. *Personalized Medicine, 15*(5), 429–448. https://doi.org/10.2217/pme-2018-0044
23. Esteva, A., Kuprel, B., Novoa, R. A., et al. (2017). Dermatologist-level classification of skin cancer with deep neural networks. *Nature, 542*(7639), 115–118. https://doi.org/10.1038/nature21056
24. Feil, R., & Fraga, M. F. (2012). Epigenetics and the environment: Emerging patterns and implications. *Nature Reviews Genetics, 13*(2), 97–109. https://doi.org/10.1038/nrg3142
25. Florez, J. C., Jablonski, K. A., Bayley, N., et al. (2006). TCF7L2 polymorphisms and progression to diabetes in the Diabetes Prevention Program. *New England Journal of Medicine, 355*(3), 241–250. https://doi.org/10.1056/NEJMoa062418

26. Franceschi, C., Garagnani, P., Parini, P., et al. (2018). Inflammaging: A new immune–metabolic viewpoint for age-related diseases. *Nature Reviews Endocrinology, 14*(10), 576–590. https://doi.org/10.1038/s41574-018-0059-4
27. Furman, D., Campisi, J., Verdin, E., et al. (2019). Chronic inflammation in the etiology of disease across the life span. *Nature Medicine, 25*(12), 1822–1832. https://doi.org/10.1038/s41591-019-0675-0
28. GBD 2016 Risk Factors Collaborators. (2017). Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016. *The Lancet, 390*(10100), 1345–1422. https://doi.org/10.1016/S0140-6736(17)32366-8
29. GBD 2019 Diseases and Injuries Collaborators. (2020). Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019. *The Lancet, 396*(10258), 1204–1222. https://doi.org/10.1016/S0140-6736(20)30925-9
30. Gombart, A. F., Pierre, A., & Maggini, S. (2020). A review of micronutrients and the immune system. *Nutrients, 12*(1), 166. https://doi.org/10.3390/nu12010166
31. Gregor, M. F., & Hotamisligil, G. S. (2011). Inflammatory mechanisms in obesity. *Annual Review of Immunology, 29*, 415–445. https://doi.org/10.1146/annurev-immunol-031210-101322
32. Gupta, S., & Khanna, S. (2017). Fecal microbiota transplantation: The evolving role in treating *Clostridium difficile* and beyond. *Current Infectious Disease Reports, 19*(11), 39. https://doi.org/10.1007/s11908-017-0600-4
33. Guthold, R., Stevens, G. A., Riley, L. M., & Bull, F. C. (2018). Worldwide trends in insufficient physical activity from 2001 to 2016. *The Lancet Global Health, 6*(10), e1077–e1086. https://doi.org/10.1016/S2214-109X(18)30357-7
34. Hasin, Y., Seldin, M., & Lusis, A. (2017). Multi-omics approaches to disease. *Genome Biology, 18*(1), 83. https://doi.org/10.1186/s13059-017-1215-1
35. Henson, J., Yates, T., Edwardson, C. L., et al. (2013). Sedentary time and markers of chronic low-grade inflammation in a high-risk population. *PLoS ONE, 8*(10), e78350. https://doi.org/10.1371/journal.pone.0078350
36. Herrin, J., St. Andre, J., Kenward, K., et al. (2016). Community factors and hospital readmission rates. *Health Services Research, 51*(1), 20–39. https://doi.org/10.1111/1475-6773.12317
37. Holzinger, A., Biro, M., & Dehmer, M. (2017). Explainable AI: Methods and applications. *Wiley Interdisciplinary Reviews: Data Mining and Knowledge Discovery, 7*(5), e1210. https://doi.org/10.1002/widm.1210
38. Horton, R., Beaglehole, R., Bonita, R., et al. (2017). From public to planetary health: A manifesto. *The Lancet, 383*(9920), 847. https://doi.org/10.1016/S0140-6736(14)60409-8
39. Hotamisligil, G. S. (2017). Inflammation, metaflammation and immunometabolic disorders. *Nature, 542*(7640), 177–185. https://doi.org/10.1038/nature21363
40. Hotamisligil, G. S., Shargill, N. S., & Spiegelman, B. M. (1993). Adipose expression of tumor necrosis factor-alpha: Direct role in obesity-linked insulin resistance. *Science, 259*(5091), 87–91. https://doi.org/10.1126/science.7678183
41. Ishigaki, K., Akiyama, M., Kanai, M., et al. (2020). Large-scale genome-wide association study in a Japanese population. *Nature Communications, 11*(1), 3879. https://doi.org/10.1038/s41467-020-17745-8
42. Johnson, K. B., Wei, W. Q., Weeraratne, D., et al. (2018). Precision medicine, AI, and the future of personalized health care. *Clinical and Translational Science, 11*(1), 1–4. https://doi.org/10.1111/cts.12526
43. Kaminsky, Z. A., Tang, T., Wang, S. C., et al. (2009). DNA methylation profiles in monozygotic and dizygotic twins. *Nature Genetics, 41*(2), 240–245. https://doi.org/10.1038/ng.286
44. Khoury, M. J., Iademarco, M. F., & Riley, W. T. (2016). Precision public health for the era of precision medicine. *American Journal of Preventive Medicine, 50*(3), 398–401. https://doi.org/10.1016/j.amepre.2015.08.031
45. Kim, J., Kim, Y., & Yang, M. (2020). Environmental chemicals and neuroinflammation. *Environmental Research, 187*, 109676. https://doi.org/10.1016/j.envres.2020.109676
46. Konečný, J., McMahan, H. B., Yu, F. X., et al. (2016). Federated learning: Strategies for improving communication efficiency. *arXiv preprint arXiv:1610.05492*. https://arxiv.org/abs/1610.05492
47. Lalmuanawma, S., Hussain, J., & Chhakchhuak, L. (2020). Applications of machine learning and artificial intelligence for COVID-19 (SARS-CoV-2) pandemic: A review. *Chaos, Solitons & Fractals, 139*, 110059. https://doi.org/10.1016/j.chaos.2020.110059
48. Leung, C., Rivera, L., Furness, J. B., & Angus, P. W. (2016). The role of the gut microbiota in NAFLD. *Nature Reviews Gastroenterology & Hepatology, 13*(7), 412–425. https://doi.org/10.1038/nrgastro.2016.85
49. Lillycrop, K. A., & Burdge, G. C. (2015). Epigenetic mechanisms linking early nutrition to long-term health. *Best Practice & Research Clinical Endocrinology & Metabolism, 29*(5), 667–676. https://doi.org/10.1016/j.beem.2015.06.001
50. Litjens, G., Kooi, T., Bejnordi, B. E., et al. (2017). A survey on deep learning in medical image analysis. *Medical Image Analysis, 42*, 60–88. https://doi.org/10.1016/j.media.2017.07.005
51. Loos, R. J. F., & Yeo, G. S. H. (2014). The bigger picture of FTO: The first GWAS-identified obesity gene. *Nature Reviews Endocrinology, 10*(1), 51–61. https://doi.org/10.1038/nrendo.2013.227
52. Lynch, T. J., Bell, D. W., Sordella, R., et al. (2004). Activating mutations in the epidermal growth factor receptor underlying responsiveness of non-small-cell lung cancer to gefitinib. *New England Journal of Medicine, 350*(21), 2129–2139. https://doi.org/10.1056/NEJMoa040938
53. Manolio, T. A., Collins, F. S., Cox, N. J., et al. (2009). Finding the missing heritability of complex diseases. *Nature, 461*(7265), 747–753. https://doi.org/10.1038/nature08494
54. Marmot, M., & Bell, R. (2019). Social determinants and non-communicable diseases: Time for integrated action. *BMJ, 364*, l251. https://doi.org/10.1136/bmj.l251

55. Medzhitov, R. (2010). Inflammation 2010: New adventures of an old flame. *Cell, 140*(6), 771–776. https://doi.org/10.1016/j.cell.2010.03.006
56. Miller, G. W., & Jones, D. P. (2014). The nature of nurture: Refining the definition of the exposome. *Toxicological Sciences, 137*(1), 1–2. https://doi.org/10.1093/toxsci/kft251
57. Monda, V., Villano, I., Messina, G., et al. (2017). Exercise modifies the gut microbiota with positive health effects. *Oxidative Medicine and Cellular Longevity, 2017*, 3831972. https://doi.org/10.1155/2017/3831972
58. Obermeyer, Z., Powers, B., Vogeli, C., & Mullainathan, S. (2019). Dissecting racial bias in an algorithm used to manage the health of populations. *Science, 366*(6464), 447–453. https://doi.org/10.1126/science.aax2342
59. Oeppen, J., & Vaupel, J. W. (2002). Broken limits to life expectancy. *Science, 296*(5570), 1029–1031. https://doi.org/10.1126/science.1069675
60. Patel, C. J., Bhattacharya, J., & Butte, A. J. (2010). An environment-wide association study (EWAS) on type 2 diabetes mellitus. *PLoS ONE, 5*(5), e10746. https://doi.org/10.1371/journal.pone.0010746
61. Popejoy, A. B., & Fullerton, S. M. (2016). Genomics is failing on diversity. *Nature, 538*(7624), 161–164. https://doi.org/10.1038/538161a
62. Price, W. N., & Cohen, I. G. (2019). Privacy in the age of medical big data. *Nature Medicine, 25*(1), 37–43. https://doi.org/10.1038/s41591-018-0272-7
63. Rajagopalan, S., & Brook, R. D. (2012). Air pollution and type 2 diabetes: Mechanistic insights. *Diabetes, 61*(12), 3037–3045. https://doi.org/10.2337/db12-0190
64. Rajkomar, A., Dean, J., & Kohane, I. (2019). Machine learning in medicine. *New England Journal of Medicine, 380*(14), 1347–1358. https://doi.org/10.1056/NEJMra1814259
65. Rakyan, V. K., Down, T. A., Balding, D. J., & Beck, S. (2011). Epigenome-wide association studies for common human diseases. *Nature Reviews Genetics, 12*(8), 529–541. https://doi.org/10.1038/nrg3000
66. Roberfroid, M., Gibson, G. R., Hoyles, L., et al. (2010). Prebiotic effects: Metabolic and health benefits. *British Journal of Nutrition, 104*(S2), S1–S63. https://doi.org/10.1017/S0007114510003363
67. Rooks, M. G., & Garrett, W. S. (2016). Gut microbiota, metabolites and host immunity. *Nature Reviews Immunology, 16*(6), 341–352. https://doi.org/10.1038/nri.2016.42
68. Sallis, J. F., Bull, F., Burdett, R., et al. (2016). Use of science to guide city planning policy and practice: How to achieve healthy and sustainable future cities. *The Lancet, 388*(10062), 2936–2947. https://doi.org/10.1016/S0140-6736(16)30068-X
69. Sears, B. (2015). Anti-inflammatory diets. *Journal of the American College of Nutrition, 34*(S1), 14–21. https://doi.org/10.1080/07315724.2015.1031355
70. Segal, E., Thieberger, R., & Zeevi, D. (2017). Personalized nutrition by prediction of glycemic responses. *Cell, 163*(5), 1079–1094. https://doi.org/10.1016/j.cell.2015.11.001
71. Seligman, H. K., Laraia, B. A., & Kushel, M. B. (2010). Food insecurity is associated with chronic disease among low-income NHANES participants. *Journal of Nutrition, 140*(2), 304–310. https://doi.org/10.3945/jn.109.112573
72. Sendak, M. P., D'Arcy, J., Kashyap, S., et al. (2020). A path for translation of machine learning products into healthcare delivery. *EMJ Innovations, 4*(1), 19–27. https://doi.org/10.33590/emjinnov/19-00172
73. Shoelson, S. E., Lee, J., & Goldfine, A. B. (2007). Inflammation and insulin resistance. *Journal of Clinical Investigation, 116*(7), 1793–1801. https://doi.org/10.1172/JCI29069
74. Slavich, G. M., & Irwin, M. R. (2014). From stress to inflammation and major depressive disorder: A social signal transduction theory of depression. *Psychological Bulletin, 140*(3), 774–815. https://doi.org/10.1037/a0035302
75. Sudlow, C., Gallacher, J., Allen, N., et al. (2015). UK Biobank: An open access resource for identifying the causes of a wide range of complex diseases. *PLoS Medicine, 12*(3), e1001779. https://doi.org/10.1371/journal.pmed.1001779
76. Swinburn, B. A., Kraak, V. I., Allender, S., et al. (2019). The global syndemic of obesity, undernutrition, and climate change: The Lancet Commission report. *The Lancet, 393*(10173), 791–846. https://doi.org/10.1016/S0140-6736(18)32822-8
77. Ter Horst, R., Jaeger, M., Smeekens, S. P., et al. (2016). Host and environmental factors influencing individual human cytokine responses. *Cell, 167*(4), 1111–1124. https://doi.org/10.1016/j.cell.2016.10.018
78. Tilg, H., Zmora, N., Adolph, T. E., & Elinav, E. (2020). The intestinal microbiota fuelling metabolic inflammation. *Nature Reviews Immunology, 20*(1), 40–54. https://doi.org/10.1038/s41577-019-0198-4
79. Tonucci, L. B., Olbrich dos Santos, K. M., de Oliveira, L. L., et al. (2017). Clinical application of probiotics in type 2 diabetes mellitus: A randomized, double-blind, placebo-controlled study. *Clinical Nutrition, 36*(1), 85–92. https://doi.org/10.1016/j.clnu.2015.11.011
80. Topol, E. J. (2019). High-performance medicine: The convergence of human and artificial intelligence. *Nature Medicine, 25*(1), 44–56. https://doi.org/10.1038/s41591-018-0300-7
81. Torkamani, A., Andersen, K. G., Steinhubl, S. R., & Topol, E. J. (2017). High-definition medicine. *Cell, 170*(5), 828–843. https://doi.org/10.1016/j.cell.2017.08.007
82. Torkamani, A., Wineinger, N. E., & Topol, E. J. (2018). The personal and clinical utility of polygenic risk scores. *Nature Reviews Genetics, 19*(9), 581–590. https://doi.org/10.1038/s41576-018-0018-x
83. Tsankova, N., Renthal, W., Kumar, A., & Nestler, E. J. (2007). Epigenetic regulation in psychiatric disorders. *Nature Reviews Neuroscience, 8*(5), 355–367. https://doi.org/10.1038/nrn2132
84. Vamathevan, J., Clark, D., Czodrowski, P., et al. (2019). Applications of machine learning in drug discovery and development. *Nature Reviews Drug Discovery, 18*(6), 463–477. https://doi.org/10.1038/s41573-019-0024-5

85. Visscher, P. M., Wray, N. R., Zhang, Q., et al. (2017). 10 years of GWAS discovery: Biology, function, and translation. *American Journal of Human Genetics, 101*(1), 5–22. https://doi.org/10.1016/j.ajhg.2017.06.005
86. Wild, C. P. (2012). The exposome: From concept to utility. *International Journal of Epidemiology, 41*(1), 24–32. https://doi.org/10.1093/ije/dyr236
87. Wishart, D. S. (2016). Emerging applications of metabolomics in drug discovery and precision medicine. *Nature Reviews Drug Discovery, 15*(7), 473–484. https://doi.org/10.1038/nrd.2016.32
88. Zmora, N., Suez, J., & Elinav, E. (2019). You are what you eat: Diet, health and the gut microbiota. *Nature Reviews Gastroenterology & Hepatology, 16*(1), 35–56. https://doi.org/10.1038/s41575-018-0061-2