Health care does not really focus on maintaining optimal health, but rather on curing diseases. A large repertoire of tools, technologies, and treatments has been developed for this purpose, making disease care an enterprise that may become too costly. Also, within health care, citizens become patients in the literal sense of the word: patiently undergoing treatments instead of playing an active part in their own health. This needs to change and in theory should be simple as a huge health profit can be achieved if each person would eat their proper diet during their lifespan: reductions in obesity (95%), type-2 diabetes (80%), cardiovascular diseases (40%), and cancers (50%) could be achieved. Biomarkers should serve two crucial goals: to report on health improvement or health maintenance instead of disease progression and to empower the individual to achieve this.

Human health is based on a complex network of interactions between pathways, mechanisms, processes, and organs. Many of these processes have to function in a continuously changing environment (e.g. diet, infections, stress, temperature, and exercise) and thus strive to maintain internal homeostasis by adapting to these changes. We call this phenotypic flexibility and realize that disease onset occurs when and where these adaptive processes fail. Importantly, diet plays both a positive and a negative role here. Many nutrients serve specifically to optimize these 'flexibility processes' (fig. 1). Yet also, prolonged caloric excess and an unbalanced diet ('hidden hunger') cause loss of flexibility and long-term adaptation processes with negative health consequences.

Due to a wide variety of reasons (e.g. genetic and epigenetic factors, exposure, diet, stress, and exercise), individuals differ in their ‘wiring’ of phenotypic flexibility, will react differently to acute and chronic stressors, and develop a personal trajectory of metabolic-inflammatory health and disease. Thus, personalized diagnosis of the phenotypic flexibility system needs to reveal the ‘weak spots’ in this flexibility network. For one person, this may be impaired triglyceride storage in adipose tissue resulting in a fatty liver, for another the impaired excretion of VLDL particles from the
liver due to a shortage in choline, resulting in a fatty liver. A third person may accumulate liver fat due to a shortage of carnitine, causing inadequate fatty acid oxidation, for example. Each of these processes needs to be diagnosed and require a specific food-based therapy.

Ideally, this type of phenotypic flexibility biomarker develops into two dimensions. Firstly, from a single process to the complete system of flexibility (‘systems flexibility biomarker’), and secondly, along the timeline of an individuals’ health trajectory, building the life story of phenotypic flexibility, a ‘biopassport’. This biopassport is the ideal starting point for the design of both (food-based) personal health optimization strategies and self-empowerment strategies.