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Nutritional Benchmarking: A Top Priority

Massimiliano Sansone*, Andrea Sansone, Elena Gangitano, Mariagrazia Romano, Silvia Seraceno and Francesco Romanelli

Department of Experimental Medicine, Sapienza - University of Rome, Italy

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***Corresponding author:** Massimiliano Sansone, Department of Experimental Medicine, Section of Medical Pathophysiology, Food Science and Endocrinology, Sapienza - University of Rome, Rome, Italy

Abbreviations: CHO: Carbohydrate Ratio: CR: Caloric Restriction: IGF: Insulin-Growth Factor: AMPK: Activated Protein Kinase: PPAR γ: Peroxisome Proliferator-Activated Receptor-γ: MTOR: Mammalian Target of Rapamycin: PGC: Proliferator-Activated Receptor Coactivator: KD: Ketogenic Diet

Introduction

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In the world of nutritional science, more and more studies have been performing for the last years with interesting results; nevertheless, at the moment outcomes of these studies do not provide consistent benchmarks that may use to build a nutritional plan or to lead further researches. Contrasting evidence represents an important drawback in this field creating a confusing background; indeed, these conflictual aspects concern the majority of topics in nutritional science. When attention is focused on total daily caloric intake, protein daily intake and daily carbohydrate (CHO)/fat ratio, it is possible to observe a great discordance of evidence. As regards total daily caloric intake, recent evidence shows that a daily reduction of 20-30% of caloric intake may significantly increase life span; in particular, this increased longevity seems to be confirmed in animal models whereas in humans long-term studies are not present at the moment to confirm this hypothesis [1-3]. Considering that actual dietary guidelines suggest approximately 2000 kcal/die diet for a man of ~70 kg, a reduction of 20-30% of daily caloric intake would be represented by a nutritional plan of ~1400 kcal/d for a man of ~70 kg [4-6].

Several mechanisms have been proposed to explain this evidence, and the reduction of mitogen stimuli caused at least partly by decreased hormonal levels, such as insulin, insulin-growth factor 1 (IGF-1), and testosterone appears to play a pivotal role [7-8]. This decrease in hormonal production affects also negatively the activity of mitogen pathways, such as mammalian target of rapamycin (mTOR) signalling pathway activated by insulin and FOXO a key checkpoint gene in the insulin-IGF-1 signaling pathway upregulated by CR[9]. Furthermore, caloric restriction (CR) seems to mimic physical exercise activating biological pathways which are stimulated by physical exercise, such as AMP-activated protein kinase (AMPK) signalling pathways, and to increase sirtuin levels [10]. In particular, the ability of sirtuins to influence metabolism and potentially life span is believed to revolve around the ability of sirtuin family members to function as protein deacetylases. Evidence suggests that mitochondrial biogenesis is regulated at least in part by proliferator-activated receptor coactivator-1α (PGC-1α), a transcriptional coactivator of peroxisome proliferator-activated receptor-γ (PPARγ) as well as other transcription factors [11]. It was therefore of considerable interest when it was shown that PGC-1α was in fact a deacetylation target of Sirt1 and that mechanism regulated PGC-1α activity [12].

On the other side, in sport nutrition evidence suggests that a high daily caloric intake has ergogenic properties increasing skeletal muscle glycogen replenishment and anabolic response to training induced stimuli in endurance activities [13-14]. In this framework a total daily intake of ~4000-6000 kcal is not uncommon taking into account that 5-6 hours of endurance training may cost ~3500-4200 kcal not including rest metabolism rate which may account for 1600-1800 kcal/die to sustain resting metabolic processes in a 70 kg man [15]. Indeed, an intake of ~7-12 g/kg/CHO/d is frequently reported among endurance athletes, accompanied by an intake of ~1.7-2 g/protein/d to sustain anabolic processes with the rest of daily caloric intake coming from fat intake [16-18]. Indeed, both endurance and resistance athletes try to maximize anabolic pathways and skeletal muscle synthesis by ingesting a high daily amount of protein and, in some cases, administering illegal performance enhancing drugs, such as insulin, IGF-1, growth hormone and testosterone [19]. These ways are clearly in contrast with what has been proposed by CR diet, and it seems that targets of maximizing physical performance and increasing life span could not be pursued at the same time. A similar conflict may be observed even when daily protein intake is considered; opposite evidence appears to suggest significant metabolic improvements brought about by both high and low protein diet [20-21].

Nevertheless, a similar situation can be observed when daily fat intake is concerned; indeed, in the last years a great number of studies support the properties of ketogenic diet (KD) to improve

metabolic parameters and body composition [22-27]. KD is generally characterized by a total carbohydrate intake of less than 50 g/d and a moderate protein intake of approximately 1.5 g/kg/d to induce ketogenesis. Indeed, despite no standard definition exists to define the ideal range of carbohydrate intake to achieve ketosis because of individual variability, a carbohydrate daily intake of 0.5 g/kg/d may represent an accurate assessment [28-29]. Daily protein intake should range from 1.76 to 2.2 g/kg lean mass/d or 1.2 – 1.7 g/kg body weight/d, and the remaining calories should come from fat, covering 70-80% of daily energy intake [30-31]. This low intake of CHO is clearly in contrast with daily intake recommended by nutritional guidelines; generally the ratio of macronutrients proposed by KD is opposite compared to the ratio suggested by the majority of nutritional guidelines [32-34]. Notwithstanding, KD appears to be a promising diet even though further evidence is required to sustain metabolic benefits of KD. Finally, this brief manuscript highlights the presence of contrasting and confusing evidence in different aspects of nutritional science; further studies are warranted to find significant benchmarks which may lead clinicians and scientists in their activities.

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