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Mini-review

The role of dietary acid load and mild metabolic acidosis in insulin resistance in humans

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ABSTRACT

Type 2 diabetes is increasingly being recognised as a global health crisis (World Health Organisation). Insulin resistance is closely associated with obesity and precedes the development of type 2 diabetes. However, there is now increasing evidence to suggest that diet itself may independently be associated with type 2 diabetes risk. A diet with a high acid load (or high potential renal net acid load, PRAL) can result in a decrease in pH towards the lower end of the normal physiological range, which may in turn lead to the development of insulin resistance. Conversely, reducing dietary acid load (the so called 'alkaline diet') may be protective and prevent the onset of type 2 diabetes. Here, we explore the influence of dietary acid load on the development of mild metabolic acidosis and induction of insulin resistance. Whilst large prospective cohort studies link high dietary acid load or low serum bicarbonate with the development of type 2 diabetes, the effect of a diet with a low acid (or high alkaline) load remains unclear. Further interventional studies are required to investigate the influence of dietary composition on the body's acid/base balance, insulin resistance and incidence of type 2 diabetes.

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1. Introduction

The role of nutrition in the induction of insulin resistance has received increasing attention since the recognition of the type 2 diabetes epidemic as a global health crisis by the World Health Organisation [1]. Diet plays a major role in the development of excess body weight, a key risk factor for type 2 diabetes [2]. However, there is also evidence to suggest that diet itself may independently be associated with type 2 diabetes risk [3]. The rapid rise in the prevalence of type 2 diabetes, in combination with earlier disease onset, has led to increased public health concern and a greater focus on the delineation of dietary strategies that may prevent or delay the onset of type 2 diabetes [2].

The analysis of dietary patterns has become increasingly popular in the study of diet–disease relationships. This concept considers the potential synergistic or antagonistic interaction between individual foods and nutrients within the overall diet [4,5]. Dietary patterns better reflect real-life behaviour by representing the effect of the whole diet, which may in turn reveal stronger associations with diet-related disease risk. Within the current literature, two major dietary patterns have been identified, the "Western diet" and the "Prudent diet" [6]. The Western diet is typically high in animal proteins such as those derived from red meat, processed meat and eggs, as well as processed foods including high-energy drinks, dessert foods, and French fries. The Prudent diet is comparatively rich in fruit, vegetables, legumes and whole grains [7], and is therefore rich in fibre, magnesium, potassium, folate, and vitamin B6. It is also relatively low in fat, particularly saturated fat, in contrast to the Western diet [6]. A third dietary pattern is the Mediterranean diet, which is similar to the Prudent diet, but consists of a higher dietary intake of plant based fats, and moderate consumption of alcohol (in particular red wine) [5]. It is postulated that the higher monounsaturated fat and antioxidant content of the Mediterranean diet may confer additional benefit in improving insulin

Abbreviations: GLP-1, glucagon-like peptide 1; PRAL, potential renal acid load; NEAP, net endogenous acid production; RNAE, renal net acid excretion; HOMA-IR, homeostasis model of assessment of insulin resistance.

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sensitivity, and preventing type 2 diabetes [8], cardiovascular disease and mortality [9].

1.1. Definition of diet-induced (or 'mild') metabolic acidosis

The influence of the Western diet on health and disease outcomes in terms of its effect on the body's acid/base balance, has gained increasing interest in recent years [2]. Under normal conditions, blood pH is maintained within a narrow physiological range. The pH of arterial blood is close to 7.40, with a normal range considered to be approximately 7.35–7.45. An arterial pH less than 7.35 is classified as acidaemia, whilst the underlying condition characterised by hydrogen ion retention or loss of bicarbonate or other bases, is referred to as acidosis [10]. The acidogenic Western diet is associated with an increase in the body's hydrogen ion load, and has been hypothesised to lead to chronic mild metabolic acidosis, if proceeded by the failure of compensatory processes that aim to restore the homeostatic acid/base balance [2,11]. Consumption of foods with a high acid load (e.g. animal protein) results in the net production of nonvolatile acids such as hydrogen chloride (HCl) and hydrogen sulphate (H_2SO_4). These acids are buffered through the excretion of carbon dioxide via the lungs, and the production of sodium salts from nonvolatile acids which are excreted by the kidneys, predominately in association with ammonium, as NH_4Cl and $(NH_4)_2SO_4$. The bicarbonate generated in this process is reabsorbed and returned to the plasma, to replace the bicarbonate used to buffer the nonvolatile acid [11]. If acid production in the body exceeds acid excretion from the lungs and kidneys, the plasma bicarbonate and pH decrease. Previous authors have labelled this downward shift in body pH to the lower end of the normal range as a result of dietary acid load 'latent acidosis' [12], 'diet induced acidosis' [13–15], 'low grade acidosis' [13,16], 'chronic metabolic acidosis' [2], 'subclinical acidosis' [13] or 'mild metabolic acidosis' [17–19]. For the purpose of this review, we describe this dietary associated shift in pH as 'mild metabolic acidosis'.

1.2. Previous reviews

The association of the acid/base balance with metabolic disease, including type 2 diabetes and cardiovascular disease has been the topic of previous reviews. Salas-Salvadó and colleagues [5] summarised the effect of dietary components on the risk of developing type 2 diabetes, and concluded that following either a Prudent or Mediterranean dietary pattern was the best strategy to reduce diabetes risk. The benefit of a Mediterranean diet in preventing type 2 diabetes in those with high cardiovascular risk was evidenced in the PREDIMED study [20]. Berkmeyer and colleagues [2] explored the relationship between the Western diet, acid/base balance and obesity. They concluded that a Western (or acidogenic) diet in the absence of respiratory compensation is associated with a rise in the hydrogen ion load in the body, and this effect could be attenuated by consuming fruit and vegetables, or by taking an alkaline supplement. Adeva and Souto [14] discussed how a Western diet is linked with the development of metabolic acidosis despite renal physiological alterations such as increasing renal net acid excretion (RNAE). They hypothesised that metabolic acidosis induces insulin resistance in skeletal muscle to permit protein degradation and to generate ammonium required to promote hydrogen ion excretion, resulting in an increased risk of type 2 diabetes and hypertension. They additionally suggest that metabolic acidosis may induce glucocorticoid production, and the resulting rise in plasma cortisol could in turn contribute to insulin resistance and proteolysis [21,22]. Souto and colleagues [23] further hypothesised the risk of developing renal impairment (heralded by microalbuminuria) due to metabolic acidosis induced insulin resistance, which in turn was considered to be associated with increased risk of cardiovascular disease and mortality [24]. However, a significant development in evidence since the publication of these articles has been recently provided by large prospective studies examining the link between the dietary acid load and diabetes risk [19,25].

In the present review we will explore the role of dietary acid load and mild metabolic acidosis in insulin resistance and type 2 diabetes. We will present the evidence regarding the influence of dietary acid load on the development of mild metabolic acidosis. Next, we will explore the evidence regarding the role that metabolic acidosis may play in insulin resistance and type 2 diabetes and finally, discuss evidence gathered in large cohort studies suggesting that dietary acid load predicts insulin resistance and type 2 diabetes.

2. The influence of dietary acid load on the development of mild metabolic acidosis

2.1. Quantifying the acidogenic potential of foods

The acidogenic potential of foods can be calculated using potential renal acid load (PRAL) [19,26] and net endogenous acid production (NEAP) [19]. PRAL is based on the nutrient ionic balance and intestinal absorption rates of protein, phosphorous, potassium, magnesium and calcium as well as the production of sulphate from metabolised protein [19,27]. PRAL may be calculated by the following equation [19]:

$$PRAL\left(\frac{mEq}{day}\right) = 0.49 \times Protein\left(\frac{g}{day}\right) + 0.037 \times Phosphorous\left(\frac{mg}{day}\right) - 0.021 \times Potassium\left(\frac{mg}{day}\right) - 0.026 \times Magnesium\left(\frac{mg}{day}\right) - 0.013 \times Calcium\left(\frac{mg}{day}\right)$$

Table 1
Examples of the dietary acid load score potential renal acid load (PRAL) of common foods and beverages.

Food	*PRAL (mEq/100 g)
Cheese varieties	4.3–34.2
Meat/meat products	6.7–13.2
Bread/grain products	1.7–12.5
Whole egg	8.2
Milk chocolate	2.4
Beer	−0.1–0.9
Whole milk	0.7
Coca-Cola	0.4
Red wine	−2.4
Fruit	−21–5.5
Vegetables	−14–−0.4

The higher the PRAL the higher the acid load, and vice versa [19].

$$*PRAL\left(\frac{mEq}{day}\right) = 0.49 \times Protein\left(\frac{g}{day}\right) + 0.037 \times Phosphorous\left(\frac{mg}{day}\right) - 0.021 \times Potassium\left(\frac{mg}{day}\right) - 0.026 \times Magnesium\left(\frac{mg}{day}\right) - 0.013 \times Calcium\left(\frac{mg}{day}\right)$$

Adapted from Remer et al. [26].

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