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Editorial Open Access

Obesity, Intestinal Inflammation, and Antioxidant Bioavailability

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Editorial

Obesity has become a pandemic threatening public health and the resources used to combat it. Obesity is a complex syndrome that compromises various systems in the body and is heavily influenced by diet and physical activity. Recent evidence in animal models has highlighted changes in the gut microflora that may promote energy harvest, thereby contributing to the caloric imbalance that is present in obesity [1-4]. Other animal studies have shown that microbial metabolites may regulate food intake differently in obese and lean animals [5-7]. Taken together these suggest that an adaptation may occur in the obese gut depending on the nutrients available. While this line of research is new and emerging, the consequences of an altered gut environment have remained unstudied. For example, there are only two studies that have examined the bioavailability of compounds in obese vs. lean individuals [8,9] and this limited research supports a reduction in bioavailability of compounds or nutrients in obesity. While it is appreciated that nutrient bioavailability is a key factor in determining their biological impact, there is surprisingly little information on how obesity impacts the gastrointestinal tract, and whether obesity may alter the absorption of bioactive food compounds and thereby their systemic bioavailability.

Presently, there are several lines of research being conducted that show changes seen in the obese gut which may influence bioavailability of nutrients or compounds. A first line has examined the changes in gut bacteria (microflora/microbiota) to high-fat feeding. It has been reported that high-fat feedings, which often accompanies obesity, increases gut permeability of bacterial lipopolysaccharides (LPS) to plasma. Follow up studies have supported a role for glucagon-like peptide 2 (GLP-2) effects on gut barrier function, especially after prebiotic feedings [10]. Specifically, disruptions in tight-junction proteins like occludin and ZO-1 may be directly involved in regulating this LPS-induced inflammatory response. The authors postulate that this could potentially trigger the low-grade inflammation seen in obesity. This in turn leads to a cascade of events that modulate energy homeostasis and metabolism. In addition, as mentioned above, there is a recent exciting body of literature suggesting that alterations in gut microbiota can have dramatic effects on whole body energy homeostasis, and that the microbiota in obese individuals may be different than in lean counterparts [1-4].

A second line of literature has examined the link between diet-induced obesity (DIO) and intestinal inflammation. Three studies to date have been conducted looking at DIO and intestinal inflammation, which may be a critical to understand how bioavailability in obesity may be altered. These studies have suggested that inflammatory markers such as TNF-α and NF-κB are increased in the ileum within 2-6 weeks of high-fat feedings and that this correlates with degree of weight gain and increased fat mass [11]. Additional studies have shown that high-fat diets induce myeloperoxidase activity, an inflammatory protein associated with neutrophils and macrophages, in DIO-prone rats [12]. Finally de Wit et al. [13] found that DIO animals' ileums had induction of macrophage migration inhibitory factor, which was shown to enrich inflammation and interferon-γ-induced gene subsets in the ileum. These factors not only indicate inflammation presence but are associated with both obesity and insulin resistance.

A third line of evidence comes from researchers who have combined the first two lines mentioned above to support that changes in microbiota are necessary for the development of intestinal inflammation. Data from Ding et al. [11] showed that germ free mice fed a high fat diet do not exhibit an up regulation of TNF-α, while conventional mice do. This strongly suggests that there is some interaction which must occur between the diet and the microbiota to induce inflammation in the intestine. Furthermore, de La Serre et al. [12] showed that high-fat diets induced toll-like receptor 4 (TLR4) in obesity prone rats but not obesity resistant ones. TLR4 is a primary receptor mediating LPS, which provides additional support that this pro-inflammatory marker is critical for intestinal inflammation seen with high-fat feedings. Finally, de La Serre et al. [12] also showed that small intestine alkaline phosphatase, a LPS detoxifying enzyme, is reduced in high-fat fed mice.

Taken together these lines of research suggest that changes in the microbiota and intestine may impact the bioavailability of important dietary components in the obese state. This concept is critical because, for example, one can eat a certain amount of antioxidant-rich fruits and vegetables, but their bioavailability will determine the actual dose and actual metabolic form the body receives and can then utilize. Antioxidants have potent biological effects ranging from reduction in cardiovascular disease and cancer risk, reversal of age-related neurodegenerative declines, and improved glucoregulation [14]. Recently, anthocyanins and flavonols have increased in popularity due to the research on grapes and their derived products, such as wine and grape juice [15]. Their presence in foods and beverages is common and considered a normal dietary component. Anthocyanins have been reported to have antioxidant, anticarcinogenic, vasoprotective and anti-inflammatory properties [16]. However, the decrease in consumption of anthocyanins (12.5 mg/day), paired with other changes in the diet including increase saturated fat intake may reflect the alarmingly high rate of chronic diseases [17].

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Anthocyanins have been examined and data suggests that they are absorbed and excreted as intact glucosides and that antioxidant activity of the plasma increases after consumption [18]. Studies have shown that anthocyanins are metabolized by gut microflora via glycosylation and ring fission to produce phenolic acids and aldehydes accounting for the majority of anthocyanin metabolism *in vivo* [19]. However, with the evidence to support an altered gut microflora in obesity [2,5,6,20], it is possible that this alteration may further change the way anthocyanins are processed, potentially limiting their bioavailability. This appears to be supported by evidence that the microfloral profile can determine gut metabolites from anthocyanin breakdown as well as influencing the activity of phase I and II enzymes ultimately determining which metabolites will enter the bloodstream and be available systemically [21,22].

Currently there is little to no information linking the gut inflammation and antioxidant bioavailability changes with obesity, an area that our lab is currently investigating. It has been suggested that in order to determine which compounds in anthocyanins are most bioactive, their effects in disease models must be known, specifically with regards to absorption and metabolism [17]. Moreover, phenolic acid metabolites are mainly formed from gut microflora metabolism and it is suggested that this may be responsible for the disease-reducing properties associated with anthocyanin consumption [17]. A model for obesity and the gut with regards to bioavailability of bioactive compounds has not been established. This model can be used to understand metabolism and absorption in the obese state and use that to determine antioxidant efficacy in obesity and inflammation reduction.

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