

## Is the prooxidant effect of polyphenols harmful?

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Diets rich in polyphenols are epidemiologically associated with lower risk of developing some age-related diseases in humans. Apparent disease-protective effect of polyphenols is often attributed to their powerful antioxidant activities, as established *in vitro*. However, polyphenols can also exert pro-oxidant activities under certain experimental conditions. Neither pro-oxidant nor anti-oxidant activities have yet been clearly established to occur *in vivo* in humans, nor are they likely given the limited levels of polyphenols that are achievable *in vivo* after consumption of foods and beverages rich in them [1]. Polyphenols oxidize readily in beverages [2-4]. They can also oxidize in cell culture media and in the oral cavity. Often, these pro-oxidant effects involve interactions of polyphenols with transition metal ions. Flavonoids can chelate metal ions, often decreasing the pro-oxidant activity of metal ions [5,6]. On the other hand for many individuals, minerals can be difficult for the body to absorb and use efficiently. Chelated minerals can include any ligand that helps the body to absorb the mineral during digestion. The benefits of proper trace mineral intake include support of muscle tissue and heart rhythm, immune system function, proper bone and collagen formation, mental wellbeing and healthy metabolism [7-9]. However, there are some studies that can make the link between this oxidant/pro-oxidant effective, but unfortunately they do not seem to have attracted the interest of many researchers. Though polyphenols may modulate human neutrophil functions, there are some documents showing that neutrophils may modify polyphenolic compounds biochemically [10]. It is hypothesized that inflammatory cell-specific metabolism of polyphenolics can modify the properties of these compounds at the local site of inflammation [11,12]. Polyphenols are capable of both scavenging and generating radicals and may exert their beneficial effects by a combination of both mechanisms [13], the more effective antioxidants the polyphenols were, the more are they cytotoxic and antiproliferative. This could be due to a dual antioxidant/pro-oxidant effect of polyphenols or better to their capacity to either scavenge or generate radicals depending on the environment. However, the reaction between flavonols and HOCl may be more complicated than a simple oxidant-antioxidant interaction, and that phenolic compounds can react with HOCl to form stable chlorinated components, with each product potentially having a unique reactivity [14,15]. The flavonoids and iso-flavonoids have been shown to react with peroxy radicals, superoxide, hydroxyl radicals, and ONOO<sup>•</sup>. The antioxidant behavior of the flavonoids and isoflavonoids is related to the structure of the compound [16-18]. The aromatic nature of polyphenols makes them potential targets of HOCl and ONOO<sup>•</sup>. These reactions may create novel pharmacophores at the site of inflammation [12]. Under certain pathological conditions *in vivo* (e.g., inflammation), flavonols may be converted to chlorinated derivatives, which exhibit an enhanced antioxidant potential and thereby play a role in cardio protection. Quercetin chlorinated derivatives exhibited significantly greater antioxidant capacity than the unmodified quercetin. The chlorination of quercetin enhances the inhibition of LDL oxidation, increases the Total Radical Antioxidant Potential and plays a role in cardioprotection [19].

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