Antiproliferative and antioxidant properties of anthocyanin-rich extract from açai

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\textbf{A B S T R A C T}

An anthocyanin-rich extract, generated from açai (AEA), was investigated for its antioxidant properties and antiproliferative activity against C-6 rat brain glioma cells and MDA-468 human breast cancer cells. AEA has an ORAC value of 2589 \textmu moles trolox equivalents (TE)/g dried powder and a DPPH radical-scavenging activity of 1208 \textmu moles TE/g, suggesting that AEA is an exceptional source of natural antioxidants. In addition, AEA remarkably suppresses proliferation of C-6 rat brain glioma cells, but has no effect on the growth of MDA-468 human breast cancer cells. Further experiments demonstrated that the AEA treatment dose-dependently inhibited the growth of C-6 rat glioma cells with an IC\textsubscript{50} of 121 \textmu g/ml. The DNA ladder fragmentation results indicated that AEA induced apoptosis of C-6 rat brain glioma cells. To compare açai with other anthocyanin-rich extracts, a number of berry extracts, including blueberry, strawberry, raspberry, blackberry and wolfberry, were assessed for potential antiproliferative activity against C-6 rat brain glioma cells. However, none of them showed suppressing effect. The results suggest that the active antiproliferative constituents in AEA are unlikely to be anthocyanins normally found in common berries.

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1. Introduction

A growing body of epidemiological and clinical evidence suggests a beneficial role of foods rich in antioxidants in reducing incidences and mortality of certain cancers (Bandera, Kushi, Moore, Gifkins, & McCullough, 2007; Parsons et al., 2008). Fruits, especially berries, have been shown to contain high levels of antioxidant compounds, such as polyphenols, phenolic acids, flavonoids, and carotenoids (Wang & Lin, 2000). These antioxidants are thought to prevent chronic complications in part through their interactions with reactive oxygen species (ROS) and ability to scavenge free radicals (Seifried, Anderson, Fisher, & Milner, 2007). Mammalian cells are constantly exposed to ROS as a result of normal metabolic processes occurring during aerobic respiration (Wei & Lee, 2002). For instance, superoxide is a type of ROS that is generated within the mitochondria and can result in the induction of additional ROS, such as hydrogen peroxide and hydroxyl radicals (Grivennikova & Vinogradov, 2006). As a result, increased levels of these ROS create an environment referred to as oxidative stress, which has the potential to lead to DNA damage and subsequently promote the mutations that initiate tumour progression (Wiseman & Halliwell, 1996). Human tumour cell lines in vitro have been shown to produce ROS at a greater rate than non-transformed cell lines (Sza-}

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