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Review

A review of the bioactivity of coffee, caffeine and key coffee constituents on inflammatory responses linked to depression

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ABSTRACT

Coffee is a widely consumed beverage containing numerous biologically active constituents predominantly belonging to the polyphenol and alkaloid classes. It has been established that coffee has a beneficial effect on numerous disease states including depression. A number of prospective and retrospective cohort studies have assessed the effects of coffee consumption on the relative risk of developing major depressive disorder in humans. These studies have identified an inverse relationship between the consumption of caffeinated coffee and the risk of developing depression. Caffeine, chlorogenic acid, ferulic acid and caffeic acid, all important constituents of coffee, have been shown to possess biological activities that highlight a possible mechanistic link to the pathology of depression. This review aims to assess the evidence from the biological evaluation of these constituents of coffee on markers of inflammation associated with depression in in vitro and in vivo models of inflammation, neuroinflammation and depression. The ability of bioactive coffee constituents to modulate the parameters of neuroinflammation has been shown with caffeine having strong antioxidant properties in vitro, chlorogenic acid and caffeic acid having strong anti-inflammatory and antioxidant properties in vitro and ferulic acid having activities in in vivo animal models of depression.

Contents

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1. Introduction

Coffee, one of the most consumed beverages worldwide (Lara, 2010), contains caffeine, the most widely used psychoactive substance (Nehlig, 1999) along with numerous other biologically active compounds. It has been reported that coffee has beneficial effects on a number of disease states including type 2 diabetes mellitus, Parkinson's disease, liver disease (Higdon & Frei, 2006), stroke risk (Ruiz-Crespo, Trejo-Gabriel-Galan, Cavia-Saiz, & Muñiz, 2012), Alzheimer's disease (Butt & Sultan, 2011), some cancers (reviewed in Nkondjock, 2009) and depression (Kawachi, Willett, Colditz, Stampfer, & Speizer, 1996; Lucas et al., 2011; Ruusunen et al., 2010). This suggests that this extremely popular beverage may manipulate health outcomes relating to many prevalent health conditions including depression.

Depression is a leading cause of morbidity with approximately 5% of the world's population diagnosed with a mood disorder, including depression, dysthymia and bipolar affective disorder (2007 National Survey of Mental Health and Wellbeing (SMHWB), 2007; Marcus, Taghi, van Ommeren, Chisolm, & Saxena, 2012). In addition to the high prevalence of this condition, literature shows that there is up to a 65% rate of failure with current antidepressant therapies (Crown et al., 2002; Fava, 2003; Fava et al., 2006; Paykel et al., 1995). Furthermore, the World Health Organisation (WHO) has identified the need for the development of effective strategies to shorten episodes and prevent recurrences of depression and as a result, has identified depression as a priority health area (Ustün, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004). The burden of disease associated with depression is multifactorial and includes aspects of mortality burden, disability burden, family burden and economic burden along with the classical burden of depression (Lépine & Briley, 2011).

1.1. Hypotheses of depression

Over the last 50 years there have been numerous proposed theories of depression starting with the well-accepted monoamine theory (Heninger, Delgado, & Charney, 1996) and progressing through to the neuroinflammatory hypotheses of depression (Maes, 2011; Maes et al., 2009; Myint & Kim, 2003). The evolution of the hypotheses of depression is mainly due to the shortcomings identified with the monoamine theory (Elhwuegi, 2004) in relation to the aetiology of depression. Given the complex, heterogeneous nature of depression (Vaidya & Duman, 2001) the neuroinflammatory hypotheses of depression better describe the aetiology and pathophysiology of depression.

Numerous hypotheses of neuroinflammatory depression have been proposed but all feature the essential features of neuroinflammation, altered tryptophan catabolism, through both the serotonergic and the kynurenine pathways and oxidative stress (Maes, 2011; Maes et al., 2009; Myint & Kim, 2003). Neuroinflammation is critical for the health of neurons in the CNS and for the maintenance of the integrity of the blood brain barrier (Petty & Lo, 2002). In the CNS the balance between the Th1 and Th2 immune responses is critical as when this balance is disturbed and favours the Th1 response, neuronal damage and death occur (Guillemin et al., 2001; Mándi & Vécsei, 2012; Najjar, Pearlman, Alper, Najjar, & Devinsky, 2013). Activated microglial cells regulate the production of the pro-inflammatory Th1 immune response (Xiao & Link, 1999), and the astroglial cells regulate the anti-inflammatory Th2 immune response (Xiao & Link, 1999). The role of each of these responses and the cells responsible are shown in Fig. 1 below.

Another important component of the neuroinflammatory hypotheses of depression is altered tryptophan catabolism. Neuroinflammation strongly influences the catabolism of tryptophan primarily through the enzyme indoleamine 2,3-dioxygenase (IDO), the rate-limiting step in the kynurenine pathway as seen in Fig. 3 below (Chopra, Kumar, & Kuhad, 2011; Leonard & Maes, 2012; Myint & Kim, 2003). IDO is up-regulated in activated microglial cells during neuroinflammation, resulting in increased production and accumulation of the neuroactive kynurenine metabolites (Alberati-Giani & Cesura, 1998; Costantino, 2009). Figs. 2 and 3 below show the effects of inflammatory mediators and compounds on the enzymes and metabolites of the kynurenine pathway. Quinolinic acid, 3-hydroxykynurenine, and 3-hydroxyanthranilic acid are known kynurenine metabolites with neurotoxic properties. Quinolinic acid is an excitotoxin through its NMDA receptor agonism and secondary free radical production (Plangar, Majlath, & Vecsei, 2012; Stone, Stoy, & Darlington, 2012). 3-Hydroxykynurenine and 3-hydroxyanthranilic acid free radical generators elicit their cytotoxicity through this mechanism (Oxenkrug, 2010; Stone et al., 2012).

The third proposed component of the neuroinflammatory hypotheses of depression is oxidative stress. A number of changes to free radical production and alteration to antioxidant defences are seen in depression (Black, Bot, Scheffer, Cuijpers, & Penninx, 2014). Amongst these include lipid peroxidation (Gałecki, Szemraj, Bienkiewicz, Florkowski, & Galecka, 2009; Sarandol et al., 2007) and peroxides, xanthine oxidase (Herken, Gurel, Selek, Armutcu, Ozen, Bulut, et al., 2007a,b; Maes et al., 2010) and 8-iso-prostaglandin F2 (Dimopoulos, Piperi, Psarra, Lea, & Kalofoutis, 2008). The effects of the various individual constituents of coffee will be evaluated for their mechanistic links to the various parameters outlined above.

The purpose of this review is to evaluate the evidence for coffee and bioactive constituents of coffee in the prevention and treatment of depression. To date, there have been no specific reviews highlighting

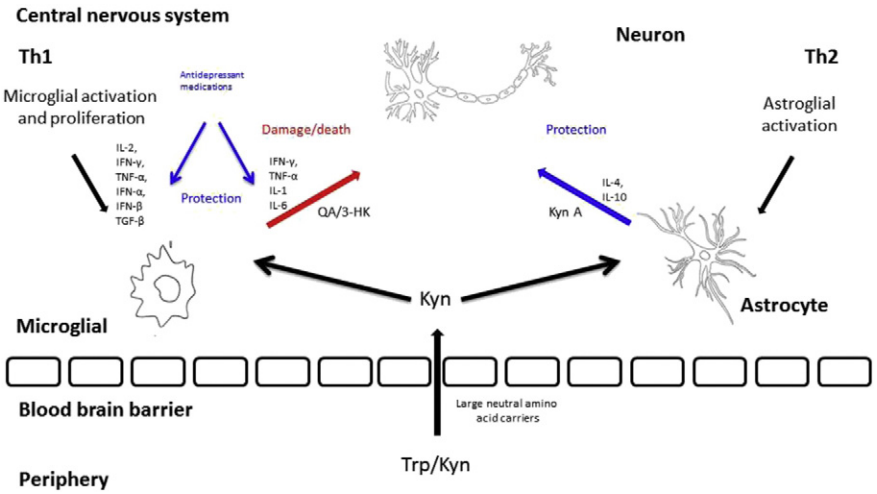

Fig. 1. The role of tryptophan catabolism in neuroinflammation.