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Anxiolytic properties of green tea polyphenol (-)-epigallocatechin gallate (EGCG).Vignes M¹, Maurice T, Lanté F, Nedjar M, Thethi K, Guiramand J, Récasens M.

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Abstract

Naturally occurring polyphenols are potent antioxidants. Some of these compounds are also ligands for the GABA(A) receptor benzodiazepine site. This feature endows them with sedative properties. Here, the anxiolytic activity of the green tea polyphenol (-)-epigallocatechin gallate (EGCG) was investigated after acute administration in mice, using behavioral tests (elevated plus-maze and passive avoidance tests) and by electrophysiology on cultured hippocampal neurons. Patch-clamp experiments revealed that EGCG (1-10 μM) had no effect on GABA currents. However, EGCG reversed GABA(A) receptor negative modulator methyl beta-carboline-3-carboxylate (beta-CCM) inhibition on GABA currents in a concentration dependent manner. This was also observed at the level of synaptic GABA(A) receptors by recording spontaneous inhibitory synaptic transmission. In addition, EGCG consistently inhibited spontaneous excitatory synaptic transmission. Behavioral tests indicated that EGCG exerted both anxiolytic and amnesic effects just like the benzodiazepine drug, chlordiazepoxide. Indeed, EGCG in a dose-dependent manner both increased the time spent in open arms of the plus-maze and decreased the step-down latency in the passive avoidance test. GABA(A) negative modulator beta-CCM antagonized EGCG-induced amnesia. Finally, state-dependent learning was observable after chlordiazepoxide and EGCG administration using a modified passive avoidance procedure. Optimal retention was observed only when animals were trained and tested in the same state (veh-veh or drug-drug) and significant retrieval alteration was observed in different states (veh-drug or drug-veh). Moreover, EGCG and chlordiazepoxide fully generalized in substitution studies, indicating that they induced indistinguishable chemical states for the brain. Therefore, our data support that EGCG can induce anxiolytic activity which could result from an interaction with GABA(A) receptors.

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