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Cannabis has a long history of anecdotal medicinal use and limited licensed medicinal use. Until recently, alleged clinical effects from anecdotal reports and the use of licensed cannabinoid medicines are most likely mediated by tetrahydrocannabinol by virtue of: 1) this cannabinoid being present in the most significant quantities in these preparations; and b) the proportion:potency relationship between tetrahydrocannabinol and other plant cannabinoids derived from cannabis. However, there has recently been considerable interest in the therapeutic potential for the plant cannabinoid, cannabidiol (CBD), in neurological disorders but the current evidence suggests that CBD does not directly interact with the endocannabinoid system except in vitro at supraphysiological concentrations. Thus, as further evidence for CBD's beneficial effects in neurological disease emerges, there remains an urgent need to establish the molecular targets through which it exerts its therapeutic effects. Here, we conducted a systematic search of the extant literature for original articles describing the molecular pharmacology of CBD. We critically appraised the results for the validity of the molecular targets proposed. Thereafter, we considered whether the molecular targets of CBD identified hold therapeutic potential in relevant neurological diseases. The molecular targets identified include numerous classical ion channels, receptors, transporters, and enzymes. Some CBD effects at these targets in in vitro assays only manifest at high concentrations, which may be difficult to achieve in vivo, particularly given CBD's relatively poor bioavailability. Moreover, several targets were asserted through experimental designs that demonstrate only correlation with a given target rather than a causal proof. When the molecular targets of CBD that were physiologically plausible were considered for their potential for exploitation in neurological therapeutics, the results were variable. In some cases, the targets identified had little or no established link to the diseases considered. In others, molecular targets of CBD were entirely consistent with those already actively exploited in relevant, clinically used, neurological treatments. Finally, CBD was found to act upon a number of targets that are linked to neurological therapeutics but that its actions were not consistent with modulation of such targets that would derive a therapeutically beneficial outcome. Overall, we find that while >65 discrete molecular targets have been reported in the literature for CBD, a relatively limited number represent plausible targets for the drug's action in neurological disorders when judged by the criteria we set. We conclude that CBD is very unlikely to exert effects in neurological diseases through modulation of the endocannabinoid system. Moreover, a number of other molecular targets of CBD reported in the literature are unlikely to be of relevance owing to effects only being observed at supraphysiological concentrations. Of interest and after excluding unlikely and implausible targets, the remaining molecular targets of CBD with plausible evidence for involvement in therapeutic effects in neurological disorders (e.g., voltage-dependent anion channel 1, G protein-coupled receptor 55, CaV3.x, etc.) are associated with either the regulation of, or responses to changes in, intracellular calcium levels. While no causal proof yet exists for CBD's effects at these targets, they represent the most probable for such investigations and should be prioritized in further studies of CBD's therapeutic mechanism of action.