Introduction: *Cannabis* biosynthesizes Δ^9-tetrahydrocannabinolic acid (THCA-A), which decarboxylates into Δ^9-tetrahydrocannabinol (THC). There is growing interest in the therapeutic use of THCA-A, but its clinical application may be hampered by instability. THCA-A lacks cannabimimetic effects; we hypothesize that it has little binding affinity at cannabinoid receptor 1 (CB_1). **Materials and Methods:** Purity of certified reference standards were tested with high performance liquid chromatography (HPLC). Binding affinity of THCA-A and THC at human (h) CB_1 and hCB_2 was measured in competition binding assays, using transfected HEK cells and [^{3}H]CP55,940. Efficacy at hCB_1 and hCB_2 was measured in a cyclic adenosine monophosphase (cAMP) assay, using a Bioluminescence Resonance Energy Transfer (BRET) biosensor. **Results:** The THCA-A reagent contained 2% THC. THCA-A displayed small but measurable binding at both hCB_1 and hCB_2, equating to approximate K_i values of 3.1μM and 12.5μM, respectively. THC showed 62-fold greater affinity at hCB_1 and 125-fold greater affinity at hCB_2. In efficacy tests, THCA-A (10μM) slightly inhibited forskolin-stimulated cAMP at hCB_1, suggestive of weak agonist activity, and no measurable efficacy at hCB_2. **Discussion:** The presence of THC in our THCA-A certified standard agrees with decarboxylation kinetics (literature reviewed herein), which indicate contamination with THC is nearly unavoidable. THCA-A binding at 10μM approximated THC binding at 200nM. We therefore suspect some of our THCA-A binding curve was artifact-from its inevitable decarboxylation into THC-and the binding affinity of THCA-A is even weaker than our estimated values. We conclude that THCA-A has little affinity or efficacy at CB_1 or CB_2.