CLINICAL OBSERVATIONS OF 10 CASES OF ENCEPHALOPATHY/EPILEPSY/CEREBRAL PALSY USING STANDARDIZED NATURAL PRODUCT CANNABIS

10 cases where caregivers requested cannabis therapy for treating refractory encephalopathies/epilepsy/cerebral palsy were observed from November 2014 while ingesting standardized quantities of cannabidiol as a natural product cannabis preparation. Positive effects were observed in 9 of the 10 patients, with reduced seizure frequency and spasm, with improved motor skills and mood. These results were achieved by administering between 1 and 6 mg/kg/day of natural product cannabidiol with no adverse side effects noted.

METHODS Caregivers requesting cannabis therapy for young persons with refractory epilepsy were provided standardized* all/CBD: THC: 2.5 mg/g (0.25%); CBD 34 mg/g (3.4%); CBN 0.6 mg/g (0.06%) natural product cannabis capsules and instructed to administer at dosages from 1 to 6 mg/kg/day, while continuing their routine medications. Various drugs such as Dilantin, Phenyltoin and Topamax were co-administered throughout the study. Patients were seen for routine check-ups and diagnostics, where caregivers were questioned as to observed effects and testimonials.

RESULTS The first patient, an 8-year-old male with epileptic seizures resulting from encephalopathy began in November 2014 and was immediately without seizures from the first dose of 1 mg/kg/day...that had not returned. In addition there was improvement in cognition and mood. Of the others that began later in 2014/2015, 7 of the ten now have no seizures (100% reduction in all cases), two had 50% and 30% reduction in seizures, respectively and one there was no response and stopped. All of the patients in the study had underlying encephalopathies (one diagnosed as Lizen) and 3 were with comorbid cerebral palsies. In the 9 of 10 cases that responded favorably all showed better behavior, less absences, improved motor skills and less aggression. There were no adverse side effects in any cases. The antiepileptic mechanisms of CBD are not known, but may include effects on the equilibrative nucleoside transporter; the orphan G-protein-coupled receptor GPR55; the transient receptor potential of vanilloid type-1 channel; the 5-HT1a receptor; and the α3 and α1 glycine receptors, all being sites of CBD activity.

CONCLUSIONS Admittedly this is mere observation of 10 patients in a non-controlled study, but it is the dramatic reduction in seizure frequency that calls direct attention to the phenomenon of a simple cannabis formulation. Definitely warranting further controlled studies of standardized cannabidiol preparations that do not appear to present adverse side effects when used in conjunction with other anti-seizure drugs.