F314 Characterization of Poliovirus isolated from patients with aseptic meningitis.

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Poliovirus, a memeber of picornaviridae, major causative agent of paralytic poliomyelitis was isolated from feces of patients with aseptic meningitis in 1997. The virus was identified by cell culture methods and typed with WHO/RIVM enterovirus typing serum pool. RNA was preparated from characterized virus and reverse transcription was done using a commercially available random hexamer. After cDNA synthesis, a part of VP1 regions were amplified by PCR with UG1, UC1 primer set. Amplified viral cDNA was then confirmed by 0.8% AGE and specific fragment of 481 bp was obtained. PCR products were examined for restriction fragment length polymorphism(RFLP) with Hae III and confirmed as Sabin type 3. To make sure that this virus was originated from vaccine strain, RCT(reproductive capacity at different temperature) test was performed. The virus didn't show any plaque in case of elevated temperature. Sequence analysis of VP1 region and 5′ noncoding regions of the virus has shown that there was over 99% homogeneity between reference virus and isolate. Our findings suggest that there may be a circulating polio viruses which were orginated from vaccine and polio virus can induce aseptic meningitis, even in case of vaccine derived polio viruses.

F315 Counterbalances on CuZnSOD and Cat G conjugated Gene expression in Escherichia coli Double Mutants to Oxidative Stress

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In Escherichia coli, the counterbalances between superoxide dismutase (SOD) and catalase are more important for overall sensitivity to oxidative stress such as parquat(PQ, 0.1mM), H_2O_2 (1mM), CuSO4 (0.1mM) and heat shock (37℃→42℃) than the level of either SOD or HP1 alone. To evaluate relative coordinate defense, E. coli SOD and/or hydroperoxidase double mutants were conducted by the transformation of plasmid oriented in wild type, SOD^+HP1^-, $SOD^-HP1^+$and SOD^-HP1^- mutant cells, and their physiological response to oxidative stress was measured. The toxicity observed increasing expression of SOD than wild type by $CuSO_4$ or heat shock treat-ment results in more sensitivity to oxidative strss. This might be a result of an increase in hydrogen peroxide.The same pattern was found in the H_2O_2 treatment. However, SOD was found to be more important than hydroperoxide in preventing oxygen-mediated stress except in the case of H_2O_2 treatment in the presense of SOD. Neverthless, both enzymes were necessary for an effective defense against oxygen mediated radicals. Moreover, although HP1 could reduce the H_2O_2 or hydroxyl radicals, it also reduced the SOD function against oxygen radicals. This system offers much greater analysis on triple mutant including glutathione peroxidase.