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Electromagnetic (EMG) signals of nerve growth factor (NGF) may induce differentiation of rat pheochromatocytoma cells PC 12

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Aims: In this study we investigated whether EMG signals may induce differentiation of PC12 cells.
Methods: The NGF EMG signals were recorded by a device emitting EMG waves and recording the resonance to the waves emitted by the substance tested. Emission of NGF signals was performed by *Multi Channel Dynamic Exciter 100 VI* utilizing an EMF generator of RW, at various intensities. The PC12 cells were cultured for 10 days and then in a Faraday cage: 1.Experimental sample (ExS) cells were exposed to static EMF signals of NGF, for 12 hours a day for 3 consecutive days, 2. Control sample (CS) cells were exposed with the device switched off, for the same period of time. Comparison samples (COS) of cells were cultured in presence of human NGF at various concentrations (1to20 μg/ml).
Results: A band of frequencies between 10 kHz to 200 kHz, was recorded for NGF. **CS cells** showed a slight increase in proliferation rate with no morphological changes at the end of the experiment. **COS cells** showed a dose dependent increase of its proliferation rate and differentiation in nervous cells.**ExS** cells showed no significant increase of proliferation rate after the exposure to the signals of NGF. After the end of 3rd exposure a high percentage (>50 %) of PC12 cells presented morphological features of nervous cells and a formation of neuronal networks. Repeated cultures of the EMF exposed cells revealed that they conserve their differentiation features for long, while, if no NGF is added in the culture media of the comparison samples, cells abolish their differentiation within 3 days.
Conclusions: NGF as other substances[1] emit signals of EMG nature that, if transmitted to target cells (PC12), may cause their differentiation to nervous cells, similar and more permanent to that the substance itself can do.

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Somatic mutations and activation-induced cytidine deaminase (AID) expression in a rheumatoid factor producing lymphoblastoid cell line

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Aims: The question whether Epstein-Bar virus (EBV) transformed lymphoblastoid cell lines (LCLs) exhibit somatic mutations in their Ig variable region genes (IgV) during in vitro growth was studied.
Methods: The sequences of the rearranged V_H of an adult-LCL which secretes a monoclonal IgM rheumatoid factor (RF-line) and of the V genes of cord blood LCLs were determined.
Results: EBV infection of adult and cord blood lymphocytes induces a rapid induction of AID, a mutator responsible for somatic hypermutation (SHM) in the IgV. SHM were not found in the rearranged V of cord blood LCLs. By contrast, the rearranged V_H gene of the RF-line, exhibited a low level of somatic mutations in culture. The mutations were preferentially targeted to the WRCH/DGYW hot spot motifs and biased for GC nucleotides, indicating that they were due to AID mediated SHM. Two point mutations in the CDR1&2 of the V_H of "non-antigen binding" RF clones, correlated with loss of antigen binding activity.
Conclusions: Induction AID expression and SHM in the rearranged V_H of adult-LCL, may explain the occasional loss of antigen binding activity occurring in freshly established antibody secreting LCLs. In addition, our results support the possibility that AID may act as an oncogene, since the tumorigenic outcome of EBV infection in B-cells, may be partly mediated by the induction of the mutatory activity of AID.