313.9 INTERFERON INDUCES DIFFERENTIAL REDISTRIBUTION OF THE 210KDALTON NEUROFILAMENT SUBUNIT IN CULTURED DORSAL ROOT GANGLIA CELL BODIES FROM NORMAL AND TRISOMY 16 MICE. D. MacFabe*, J. Lew*, B. Scott and A. V. Plioplys
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The human 21st chromosome, as the mouse 16th chromosome, codes for the alpha and beta interferon receptors. Cultured fibroblasts from both the human and mouse trisomic conditions have an exaggerated response to exogenously applied interferon. To test whether neurons may display a similar sensitivity to interferon, dorsal root ganglia (DRG) were dissociated and grown in tissue culture. They were taken from gestational age E19 normal and phenotypic trisomy 16 mouse fetuses, and normal 3 month old swiss mice. The cultures were grown on a rat tail collagen matrix in CMRL-1415 medium supplemented with 10% FCS for 42 days prior to interferon treatment. Cultures from each fetus or adult were divided into an untreated group or treated with identical medium with 6700 units of alpha and beta mouse interferon per ml for 48 hours. After fixation with cold methanol and acetone, the cultures were reacted with monoclonal antibody (mab) N210 which recognizes the phosphorylated 210 Kdalton subunit of neurofilaments. The reaction product was revealed using direct PAP techniques with 4-chloro-1-naphthol as colorant. Statistical comparisons were done with the chi-square test.

In normal fetuses and adult mice there was a significant ($p < 0.005$) increase in the percentage of DRG cell bodies stained with mabN210. Trisomic DRG's displayed a similar response to interferon ($p < 0.05$). However, the percentage of staining of untreated trisomic DRG cell bodies was similar to the interferon treated normals.

These results suggest that neurofilament antigen expression may in part be regulated by interferon. Also, trisomic DRG cell bodies express greater mabN210-immunoreactivity than do normals. These findings may be related to the observation of precocious neurofilament antigen expression in Down's syndrome early in life (A. V. Plioplys, J. Neurol. Sci., in print).

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