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Page 206*a*, Abstract 12001 should read as follows:

**12001** Comparison of Changes in Surface Morphology of PC12 cells Induced by NGF, EGF and Insulin. J.L. Connolly, S.A. Green* and L.A. Greene*, Department of Pathology, Beth Israel Hospital, Harvard Medical School and Department of Pharmacology, New York University School of Medicine.

Scanning and transmission electron microscopy were conducted on the rapid cell surface responses of PC12 cells to 3 peptide hormones/growth factors for which they have receptors, nerve growth factor (NGF), epidermal growth factor (EGF), and insulin. EGF causes surface changes indistinguishable from those that have been reported for NGF (J. Cell Biol. 82:820-827) and these EGF induced changes also occur after 4 hr. pretreatment with NGF. After addition of NGF (50ng/ml) or EGF (10ng/ml) there is rapidly initiated a sequential change in the cell surface. Microvilli which are a prominent feature on the untreated PC12 cells are gone within 1 min. of treatment. Ruffles develop over the dorsal surface within 0.5 min., become prominent by 3 min., and are almost gone within 15 min. Peripheral ruffles are observed within 3 min. and are also absent by 15 min. Large blebs are seen in 45 min., are present on 50% of cells by 2 hrs. and are gone by 4 hrs. At high magnification an increase in the number of 60-130 nm coated pits is seen after NGF or EGF treatment. This increase reaches a maximum of 3 fold from 0.5 to 3 min. and then gradually decreases. The pits may represent the internalization site for these growth factors. Insulin (10ng/ml) does not cause the marked structural changes described above and preliminary observations do not reveal an increase in pit formation. NGF induces neuronal differentiation in the PC12 cells while EGF does not. These changes therefore do not represent early neuronal morphogenesis and may reflect a generalized response to some but not all peptide hormones/growth factors on certain cells. (Supported by NIH grants AM 26920, NS-16036 and the March of Dimes Birth Defects Foundation.