

Abstract

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Pyridoxal phosphate inhibits pituitary cell proliferation and hormone secretion.

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BACKGROUND: Pyridoxal phosphate (PLP), a bioactive form of pyridoxine, dose-dependently (10-1000 microm) inhibited cell proliferation in rat pituitary MMQ and GH3 cells and in mouse AtT-20 cells.

RESULTS: After 4 d, MMQ cell numbers were reduced by up to 81%, GH3 cell numbers were reduced by up to 64% ($P < 0.05$), and AtT-20 cell numbers were reduced by up to 90%. Cell proliferation rates recovered and dose-dependently reverted to control levels after PLP withdrawal. After 4 d, PLP (400 and 1000 microm) decreased [3H]thymidine incorporation by up to 71% ($P < 0.05$). PLP (400-1000 microm) reduced GH3 cell GH and prolactin secretion and AtT-20 cell ACTH secretion (adjusted for cell number) by approximately 70% after 2 d. The 100 microm PLP also inhibited prolactin secretion (65%, $P < 0.05$) in primary rat pituitary cells treated for 2 d. PLP decreased the percentage of AtT-20 and GH3 cells in S phase and increased those in G0-G1 phase. Furthermore, PLP induced AtT-20 and GH3 cell apoptosis (28 vs. 6, $P < 0.05$; 26 vs. 3, $P < 0.05$, respectively) and dose-dependently reduced content of the antiapoptosis gene Bcl-2.

CONCLUSION: These results indicate that pharmacological doses of PLP inhibit pituitary cell proliferation and hormone secretion, in part mediated through PLP-induced cell-cycle arrest and apoptosis. Pyridoxine may therefore be appropriate for testing as a relatively safe drug for adjuvant treatment of hormone-secreting pituitary adenomas.

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