Nature Reviews Endocrinology **7**, 249 (2011); published online 29 March 2011; doi:10.1038/nrendo.2011.48

PITUITARY TUMORS

MGMT EXPRESSION PATTERNS EXPLORED

In aggressive pituitary adenomas and carcinomas, hypermethylation of the *O*-6-methylguanine-DNA methyltransferase (MGMT) gene promoter does not correlate with MGMT immunoexpression in all patients, according to a multinational research team.

The researchers explain that aggressive pituitary adenomas are clinically difficult to manage. Pituitary carcinomas are particularly challenging, as they generally progress to craniospinal or systemic metastases.

The treatment options for patients with these tumors are limited. The chemotherapeutic agent temozolomide is emerging as a possible treatment for patients who have not responded to other therapies. Low levels of MGMT are associated with a good response to temozolomide, as MGMT disrupts the therapeutic action of temozolomide. The authors suggest that measuring MGMT gene promoter methylation should be an indication of protein expression levels and thus response to temozolomide.

The researchers examined the methylation status and expression of MGMT in 12 silent subtype 3 pituitary adenomas and 10 primary pituitary carcinomas. Methylation was observed in two of the adenomas, MGMT immunostaining was negative or low in 11 of the adenomas and high in one. Of the carcinomas, three were methylated, six were unmethylated and one gave no result. MGMT immunostaining was low in five carcinomas and high in five carcinomas.

The lack of correlation between methylation and immunostaining suggests that the regulation of MGMT is complex and requires further study. We feel strongly that determination of MGMT immunoreactivity is of clinical value and that assessment of MGMT immunoreactivity as well as MGMT promoter methylation status should be pursued, conclude the researchers.

Claire Greenhill

Original article Salehi, F. *et al.* MGMT promoter methylation and immunoexpression in aggressive pituitary adenomas and carcinomas. *J. Neurooncol.* doi:10.1007/s11060-011-0532-6