**Abstract**

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**HYPOPRARATHYROIDISM IN NORWAY – EPIDEMIOLOGY AND QUALITY OF LIFE**

Hypoparathyroidism (HP) is caused by a group of heterogeneous diseases in which hypocalcemia and hyperphosphatemia occur as a result of deficient or insufficient parathyroid hormone (PTH) secretion or target-organ receptor dysfunction. The most common cause is surgical damage to the parathyroid glands. Isolated nonsurgical HP can be of either autoimmune or genetic origin, but in many cases the cause remains unknown, referred as idiopathic HP. The epidemiology of HP is not known, except for some subgroups of HP. The main objectives and results from our studies so far can be summarized in the following points:

* *Prevalence and causes of HP in Norway*

We searched electronic hospitals record for HP-related diagnosis in the majority of the Norwegian hospitals. We found a prevalence of HP of 10 per 100 000, and the majority (63%) had HP due to surgical damage of the parathyroid glands. All identified patients were invited to participate in a more detailed survey and for registration in a national registry, which so far comprise 54% of the HP population. Idiopathic HP patients in the registry were scrutinized to identify the cause of HP by assay of autoantibodies and genetic tests, which led to identification of a genetic cause in 9 %.

* *Quality of life in HP*

Through a questionnaire based study we found that health related quality of life is reduced and patients with HP have more anxiety and depression than the background population.

* *Natural course and novel mutations in patients with Hypomagnesemia with secondary hypocalcemia*

Through the national survey we identified a patient with HP due to congenital deficient magnesium uptake in intestine which secondary causes a clinical picture of HP, called hypomagnesemia with secondary hypocalcemia. Another four patients were further identified. Five novel mutations in the causative gene were identified and the natural course of the disease was mapped.

* *The impact of PTH on corticosteroid secretion*

The relationship between PTH and secretion of hormones from the hypothalamic-pituitary-adrenal (HPA) axis is an unexplored area. Patients with high levels of PTH due to primary hyperparathyroidismhave an increased stimulated cortisol response, and PTH stimulates secretion from adrenocortical cells *in vitro*. We hypothesize that patients without PTH have diminished or blunted adrenal corticosteroid secretion and will test this hypothesis in HP-patients versus healthy subjects and patients with increased PTH-secretion (primary hyperparathyroidism). This pilot project is in planning phase.