Bone Health and Nutrient Intake 2 to 5 years after Biliopancreatic Diversion with Duodenal Switch

Master Theses in Clinical Nutrition

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Summary

This report describes findings from anthropometric, bone mineral density, biochemical and dietary assessments in relation to bone health during follow-up consultations of biliopancreatic bypass with duodenal switch (BPD-DS) patients followed at the Department of Preventive Cardiology, Ullevål University Hospital from November 2006 to May 2007.

The study population consisted of 65 BPD-DS patients, 55 women and 10 men, who had undergone surgery 2-5 years earlier. Average age was 40 years, and ranged from 21 to 59. Mean BMI was 51 kg/m^2 pre-surgery and 30 kg/m^2 post-surgery, and ranged from 38 - 84 pre-surgery to 20 – 47 kg/m^2 post-surgery. All patients in this study had achieved satisfactory weight loss at the time of follow-up assessments. The study population had a mean weight loss of 62 kg, and an average of 76 % of excess weight was lost.

Analyses showed a high incidence of increased bone turnover and fat soluble vitamin deficiency (vitamin A and D) after BPD-DS. No significant relationship between nutrient status and length of common channel was detected.

Average bone mineral density (BMD) was within normal range, but 25-32 % had decreased levels (less than -1.0 SD of reference population) and men had significantly lower BMD than women. Patients with low BMD tended to be older, have lower BMI pre-surgery and lost less weight after the surgery. Reductions in BMI had the strongest correlation to BMD.

Biochemical markers of bone turnover were increased in a large proportion with serum-PTH levels increased in 92 % and serum-I-CTP increased in 45 % of cases. We found that a greater reduction in BMI was associated with higher levels of the bone resorption marker I-CTP, which indicated that the high rate of weight loss post-surgery increases bone turnover. Our results showed moderate effect on BMD at 2-5

years post-surgery, but the significantly increased rates of bone turnover may affect BMD in the longer term.

We found no relationship between nutrient intake and nutrient status or BMD. Several factors may have affected these findings. Most importantly, we only obtained dietary registration from 45 % of the study population.

No relationship was detected between supplement compliance and nutrient status or BMD. This could indicate that absorption of supplement derived nutrients may be even poorer than initially anticipated, however considering that supplement compliance was self-reported the reliability of this data may be poor. Nutritional monitoring over a longer time period, and for several years post-surgery, may be necessary to detect any relationship between such parameters.

Our data is not sufficient to accept the H_0 hypothesis which states that nutrient intake after BPD-DS is not associated with bone health. Further studies are needed to elucidate this, and we continue to emphasize the importance of ensuring patient compliance to post-surgery nutritional recommendations and follow-up procedures.

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Abbreviations

BPD-DS	Biliopancreatic bypass with duodenal switch
RYGB	Roux-en-Y- gastric bypass
BMD	Bone mineral density
DXA	Dual energy X-ray absorptiometry
TBBD	Total body bone density
AP-spine	Lumbar spine
WHO	World Health Organization
BMI	Body mass index
DM2	Diabetes mellitus type II
EWL	Excess weight loss
PTH	Parathyroid hormone
I-CTP	C-terminal telopeptide of type I collagen
b-ALP	Bone-specific alkaline phosphatase
25(OH)D	25 hydroxyvitamin D
$1.25(OH)_2D_3$	1.25 dihydroxyvitamin D_3, calcitriol
UUS	Ullevål University Hospital

1. Background

1.1 Obesity

Body weight is a result of interactions between genetic, environmental and psychosocial factors, mediated trough the physiological factors; energy intake and energy expenditure. Adiposity occurs when energy intake exceeds energy expenditure over a prolonged period of time. An increase in energy intake or decrease in energy expenditure, which causes even a slight imbalance, may lead to obesity in the long-term. The causal relationship is complex and there are several factors which affect energy balance.[2] chapt 1.2

As illustrated in Table 1, the World Health Organization (WHO) recommends the use of the body mass index (BMI) as a tool to classify varying stages of obesity. BMI is calculated as weight (kg) divided by height squared (m^2) and is frequently used in diagnostic setting.

Table 1. WHO table of BMI classification

Classification of overweight in adults according to BMI[a]

Classification	BMI (kg/m^2)	Risk of comorbidities
Underweight	<18.5	Low (but risk of other clinical problems increased)
Normal range	18.5–24.9	Average
Overweight	⩾25.0	
Pre-obese	25.0–29.9	Increased
Obese class I	30.0–34.9	Moderate
Obese class II	35.0–39.9	Severe
Obese class III	⩾40.0	Very severe

[a] These BMI values are age-independent and the same for both sexes. However, BMI may not correspond to the same degree of fatness in different populations due, in part, to differences in body proportions. The table shows a simplistic relationship between BMI and the risk of comorbidity, which can be affected by a range of factors, including the nature and the risk of comorbidity, which can be affected by a range of factors, including the nature of the diet, ethnic group and activity level. The risks associated with increasing BMI are continuous and graded and begin at a BMI below 25. The interpretation of BMI gradings in relation to risk may differ for different populations. Both BMI and a measure of fat distribution (waist circumference or waist : hip ratio (WHR)) are important in calculating the risk of obesity comorbidities.

Source [3] chapt 2, page 8

BMI does not distinguish between weight from muscle and that from fat tissue, which may limit its applicability in determining health risk in some populations. Measuring waist circumference can be used as an indicator of abdominal, visceral obesity. Several studies have shown that the amount of visceral fat accumulation is a valid indicator of co- morbidities, independent of BMI. There is an increased risk of metabolic complications for men with a waist circumference above 102 cm, and women with a waist circumference above 88 cm .[4] chapt 1, page 2. Measurement of hip circumference enables the assessment of gluteofemoral muscle mass and bone structure. Waist-hip ratio (WHR) also takes into consideration the accumulation of fat on the hips relative to that accumulated in the abdomen. This is useful when considering potential health implications of obesity. A tendency to accumulate surplus fat on the hips rather than in the stomach may protect from the development of co-morbid conditions.[4] chapt 22, page 282.

Morbid obesity is defined as a BMI above 40 or a BMI above 35 accompanied by existing co-morbid conditions. [5] This patient group has been determined to be eligible for obesity surgery.[4] chapt 22, p 287.

1.1.1 Prevalence

Current data indicate a progressive rise in obesity worldwide. WHO reported an incidence of overweight and obesity among Norwegian men and women to be 31.4 % and 19.6 % respectively (data from 2002). In developed countries there is a higher prevalence of obesity in the lower socio-economic part of the population while the opposite is true for developing countries.[4] chapt 1, page 1-20

In Norway, the proportion of obese men and women has increased significantly between 1960 and 1999. In this period there was an increase in average body weight of 9.1 and 3.7 kg in men and women respectively.[6]

Further studies confirm that average weight is still increasing. Data from the Oslo region show that 10 % of 30 year olds had a BMI above 30, 14 % among 40-year olds

and 18 % among 60-year olds. The incidence of obesity was slightly higher for men than women, especially within the younger population. Among women the prevalence of obesity was lower in the higher education group, whilst this difference was less apparent for the male population. There was a higher incidence of obesity among immigrants from non-western cultures compared to ethnic Norwegian women.[7]

The most recent population statistics for the Nordic countries is from 2006 and indicate a 9 % incidence rate in Norway for persons with a BMI above 30. It should be noted that only the Norwegian data was obtained from self-reported height and weight measurements and is the lowest reported incidence rate among the Nordic countries.[8]

The obesity epidemic does not only impose an increased prevalence of obesity, but also increasing degrees of this condition. The most rapid increase is seen in the subgroup classed as morbidly obese.[3] chapt 2, page 20

1.1.2 Health implications of obesity

Obesity is increasingly being recognized as an important public health problem, and leads to high health care costs as well as economic productivity losses. The disease has detrimental effects, leading to premature mortality, morbidity, disability and reduced quality of life.[4] chapt 3, page 28. The Framingham Heart Study showed that the risk of mortality increased by 1% for each extra 0.45 kg of excess weight between the ages of 30 and 42 years, and by 2% between the ages of 50 and 62 years.[9] The severity and the duration of obesity is positively related to the risk of developing co-morbidities.[4, 10]

Obesity increases the risk of developing the following conditions:

- type 2 diabetes and insulin resistance
- metabolic syndrome
- cardiovascular diseases and coronary heart disease (including ischemic heart disease, angina pectoris and myocardial infarction)
- hypertension

- dyslipidaemia
- stroke
- various types of cancer (such as endometrial, cervical, ovarian, prostate, breast, colon, rectal, kidney, liver and gall bladder)
- end-stage kidney disease
- fatty liver disease
- osteoarthritis
- pulmonary embolism
- deep vein thrombosis
- reproductive disorders, complications in pregnancy and polycystic ovary syndrome
- hyperuricaemia and gout
- gastro esophageal reflux disease
- gallbladder disease and gallstones
- pancreatitis
- low back pain
- breathlessness and sleep apnoea
- psychological and psychosocial problems

1.1.3 Dietary intake and obesity

Dietary intake is an important determinant of body weight, but the links between diet and obesity are difficult to determine due to methodological challenges in mapping dietary intake. Obesity occurs when energy intake exceeds energy expenditure over a prolonged period of time. According to the WHO there is sufficient evidence to conclude that the three main categories in habitual dietary intake which may increase the risk of consuming excess energy are; a diet high in energy dense foods (foods high in fat and/ or sugar and low in fibre); intake of sugary drinks and large portion sizes. Frequent consumption of fast-food may increase the risk of weight gain, whilst breast feeding and the traditional Mediterranean diet may prevent obesity. [4] chapt 5, page 46-51

The progressive increase in availability of food supplies seen in the past 40 years is apparent. The proportion of energy derived from fat is higher than recommended in most European countries, whilst fruit, vegetables and dietary fiber intake is lower than recommended. This results in a tendency towards energy-dense diets in our part of the

world. The lower socio-economic subgroups have a higher occurrence of dietary risk factors for obesity.[4] chapt 7, page 64.

1.1.4 Treatment of obesity

The overall aim in the management of obesity is to improve health and quality of life. This requires appropriately trained health professionals, who can manage associated medical complications as well as aiding weight loss. It will also require sufficient consultation time and long-term follow-up to ensure maintenance of weight loss.

WHO recommended the following aims of the treatment of obesity: A 5-10 % weight loss or maintenance of weight where the achievement of weight loss is not possible, reduction in waist circumference and changes in body composition by reducing fat percentage. It is important to reduce co-morbid conditions or prevent the development and/ or progression of these. A 5 % weight reduction is associated with some improvement in health parameters, while a 10 % reduction is likely to achieve significant improvement in blood pressure, blood cholesterol and triglyceride levels as well as improving the control of blood glucose levels in patients with insulin resistance and diabetes.[4] chapt 22, page 282-284.

Methods of treatment

Conventional treatment for obesity involves lifestyle intervention, including dietary modification and/ or increasing physical activity with or without the aid of pharmaceutical agents. Pharmaceutical agents used in the treatment of obesity include Sibutramine and Orlistat. Sibutramine affects central regulation of food intake whilst Orlistat is a lipase inhibitor which reduces fat absorption.[4] chapt 22, page 282-287.

There is a positive association between level of physical activity and the degree of weight loss sustained. However, physical activity as an isolated intervention only achieves modest weight loss (2-3 kg) hence a dietary component is likely to be necessary. There is convincing evidence that physical activity alone, diet alone and both components combined are all effective in the treatment of adult obesity. Physical

activity play a major role in maintaining weight-loss. New evidence suggests that 60 min of physical activity per day, 5 days per week is necessary to maintain weight loss. It also suggests that frequent physical activity in every-day life is more important than structured exercising in the prevention of obesity. [4]chapt 22, page 282-290.

Dietary treatment

Dietary treatment for obesity should restrict energy intake in order to achieve an energy deficit. Several methods have been tested for this purpose. Low-fat diets (≤ 30 % of energy from fat) have shown to achieve spontaneous weight loss and are also recommended for healthy normal-weight individuals because it is beneficial to health in the long term. A fixed energy deficit diet is useful in the development of diet plans. It is based on patients' individual energy requirements and may for example aim to achieve an 500 kcal energy deficit per day. These dietary treatment plans may be helpful for patients who are making overall lifestyle changes with the intent of maintaining them in the long term. They also enable combined increase in physical activity and impose minimal adaptations on a persons life compared to other diets as they include readily available "normal food" and do not exclude any food groups. Evidence suggests that these methods increase patient compliance. Other dietary treatment methods include meal-replacement programmes especially designed portion based products to replace anything from one to all meals in a day. Such products are often used in very low or low energy diets (VLED and LED diets respectively). VLED diets have better short-term, but not long-term results. Low glycaemic index diets exclude foods which cause rapid postprandial glycaemia such as sugar, white bread and potatoes. The diet favours foods which are low in carbohydrates.

High protein and low carbohydrate diets are popular dietary interventions in the weight management industry. Such diets are often composed of a high protein- and fat energy proportion, and severely restricted carbohydrate intake. These diets are associated with rapid weight loss and increased satiety, but also poor compliance and short term maintenance of weight loss. Determining the efficacy of the different

dietary interventions requires further research.[4] chapt 5, page 46-51 and chapt 22, page 284-285.

Another element in the lifestyle intervention treatment for obesity focuses on long-term behavioural modification aimed at enabling patients to sustain changes in diet and physical activity patterns. This requires health professionals with good interpersonal skills. There is strong evidence that these methods combined with advice on diet, physical activity and health is the most effective conventional approach to maintaining weight loss.[4] chapt 22, page 286

The 1991 National Consensus Development Conference on Gastrointestinal Surgery for Severe Obesity recommended that most patients seeking treatment for morbid obesity should first attempt conventional treatment before they are considered for obesity surgery.[11] However currently such treatment programmes appear to show a persistent lack of success among those suffering with morbid obesity.[12]

1.2 Obesity surgery

Bariatric surgery is becoming increasingly popular as the treatment of choice for morbid obesity. A few years ago this type of surgery was performed at very few hospitals in Norway and they were not able to accommodate the increasing demand. As a result, several Norwegians traveled abroad to have the surgery. Today the capacity of Norwegian hospitals is increasing, but there is still a significant number who travel abroad to avoid long waiting lists. Patients who have not qualified for the operation in Norway may also travel abroad to have the procedure performed.

Surgical treatment is the only intervention proven to maintain long-term significant weight loss (over 10 years) in the morbidly obese. This weight reduction is associated with significant metabolic benefits, particularly in patients with type 2 diabetes.[3] chapt 22, page 287-290. Studies show good patient satisfaction and significantly

improved quality of life in this patient group, despite frequently reported side effects.[13]

1.2.1 Indications for obesity surgery

Obesity surgery is intended for high-risk morbidly obese patients. In order to qualify for obesity surgery patients must undergo medical, surgical, psychiatric and nutritional assessments by a multidisciplinary team. The American Association of Clinical Endocrinologists, the Obesity Society and American Society for Metabolic & Bariatric Surgery, Medical Guidelines for the Clinical Practice for the Perioperative Nutritional, Metabolic, and Non surgical Support of the Bariatric Surgery Patient 2008, [1] have recommended several criteria for patient eligibility for obesity surgery. There is international consensus that bariatric surgery can be considered in patients with a BMI above 40, or a BMI above 35 accompanied by co-morbid conditions. Patients should be committed to comply with post-operative treatment guidelines including taking recommended supplements and attending follow-up consultations. Exclusion criteria include serious mental illness and/ or drug/alcohol misuse, reversible endocrine or other disorders being an important causal factor of the obesity, inability to comprehend risks, expected outcomes, alternatives and lifestyle changes required after bariatric surgery. Surgical treatment requires a multidisciplinary team to provide long-term follow-up of each patient.[1, 3, 14]

1.2.2 Obesity surgery techniques

Bariatric surgery includes both restrictive and malabsorptive methods. Some procedures involve either a restrictive or a malabsorptive change, while some involve both components.

Gastric restriction causes weight loss by producing early satiety and limiting food intake. This method creates a small pouch (which is able to hold less than 20 ml). A modified version of this technique (the laparoscopic adjustable gastric band), uses an inflatable circumgastric band attached to a subcutaneous reservoir, which allows

access to a hypodermic syringe that injects or withdraws fluid, thereby tightening or enlarging the band. Gastric restriction operations require strict dietary compliance in order to achieve satisfactory weight loss because the narrow outlet does not inhibit intake of high energy liquids and soft foods. However, there is a lower risk of long-term nutritional deficiencies because they do not entail as dramatic alterations in the gastrointestinal system. Reported excess weight loss after 3-5 years is 40-60 %, but after the initial period there is a slow regain and some patients do not achieve any long term weight loss after these procedures.[1,4] chapt 22, page 287-288

The most frequently performed gastric bypass surgery is the roux-en-Y gastric bypass (RYGB). A small proximal pouch (10-30 ml) is created from the stomach and connected to the jejunum 50cm from the ligament of Treitz. Most of the stomach, duodenum and a small portion of the jejunum are bypassed. This procedure limits food intake and produces some nutrient malabsorption. The distal gastric bypass procedure bypasses a greater length of the small intestine and may induce significant macronutrient malabsorption. RYGB achieves greater weight loss than purely restrictive methods, but adverse effects such as dumping syndrome are frequent. There is a moderate risk of nutrient deficiencies, and this patient group require thorough dietary guidance and close follow-up.[1, 4] chapt 22, page 287-288.

Obesity surgery is usually successful in inducing substantial weight loss, primarily by inducing a necessary reduction in energy intake. The Swedish obesity study (SOS study) is a prospective intervention study evaluating the medical outcomes of obesity surgery over a long period of time. After 8 years they reported an average weight loss of 16.3 kg, compared to no loss in a conventionally treated group.[15]

Table 2: Common types of Bariatric surgery procedures.

Purely malabsorptive procedures	Restrictive > Malabsorptive
Jejunoileal bypass Jejunocolonic bypass	Roux-en-Y gastric banding (RYGB) standard, long-limb
Purely restrictive procedures	**Restrictive < Malabsorptive**
Gastric banding Laparoscopic adjustable gastric band (LAGB) Vertical banded gastroplasty Silastic ring gastroplasty	Biliopancreatic diversion (BPD) BPD with duodenal switch Very long limb RYGB

Table 2 illustrates the different types of bariatric surgery procedures, classified into groups according to degree of stomach restriction and malabsorptive component. The purely malabsorptive procedures, jejunoileal- and jejunocolonic bypass are no longer performed due to a high incidence of intolerable side effects.[1]

1.2.3 Biliopancreatic Diversion with Duodenal Switch

Biliopancreatic diversion (BPD)

The biliopancreatic diversion was developed by Scopinaro et al.[16] It includes a subtotal gastrectomy which creates a 200 to 500 mL gastric pouch. Biliopancreatic juices are diverted to the terminal ileum 50 cm from the ileocecal valve, which significantly reduces the absorption of nutrients. An anastomosis between the proximal limb of the jejunum and ileum is made 50 cm proximal to the ileocaecal valve. The mixing of nutrients with digestive juices is in this way limited to the 50 cm common channel resulting in significant limitations of digestion and absorption. BPD causes weight loss primarily through energy and fat malabsorption and is associated with several micronutrient deficiencies, in particular iron and fat soluble vitamins as well as metabolic bone disease.[10]

The biliopancreatic diversion with duodenal switch (BPD-DS)

The BPD was modified by Hess and Hess into the biliopancreatic diversion with duodenal switch (BPD-DS).[17] This procedure maintains the pylorus and the first part of the duodenum. It is preferred by many amongst the obesity surgery techniques, because it results in the most significant weight loss.[13, 18, 19] Its' malabsorptive component prevents weight gain, but can also lead to severe malnutrition, particularly in cases with poor follow-up and/ or compliance to post-operative dietary recommendations. Stomach volume is reduced leaving approximately 20-30% of the stomach. The duodenum is cut just below the stomach and the small intestine is divided approximately 250 cm from the passage between the small and large bowel (ileocoecal passage). The lower part of the bowel is anastomosed to the remaining part of the stomach, making the new food channel. The remaining part of the bowel (biliopancreatic channel), is connected to the lower part of the small intestine, 50 to 100 cm from the ileocoecal passage making up the common channel.[1, 20] BPD-DS combines malabsorption with a moderate restriction of dietary intake and improves protein absorption and results in fewer side effects compared to the BPD.[21] The BPD-DS achieves up to 78 % excess weight loss at 18 years, but nutritional deficiencies are still relatively common (5-40% long-term). Alterations in bowel movements are frequent, with 3-5 movements, commonly offensive, occurring each day. The level of malabsorption is related to length of common channel.[10]

Figure 1. Illustration of the BPD and BPD-DS procedures .The duodenal switch is a modification of biliopancreatic bypass.

Fig. 5: Biliopankreatisk bypass

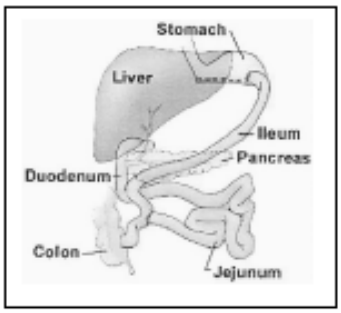


Fig. 6: Duodenal switch

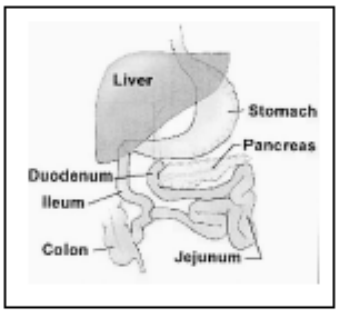
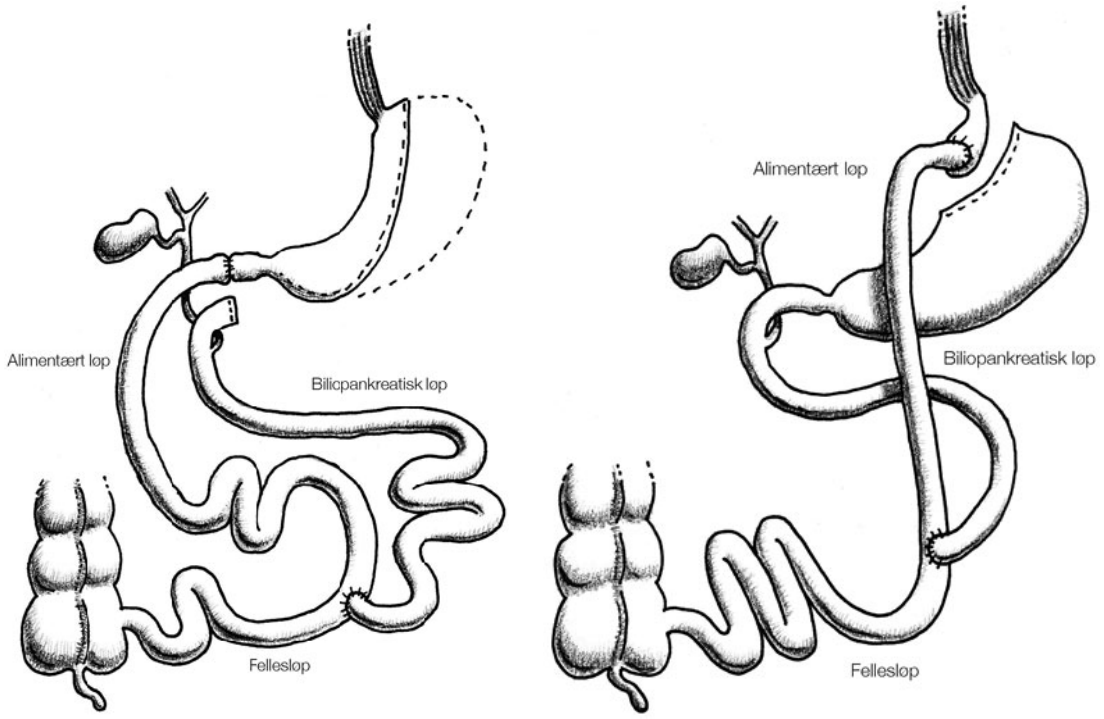


Figure 2. Illustration of the difference between BPD-DS and RYGB bariatric procedures.



Health benefits

Bariatric surgery is considered the most effective treatment for co-morbid conditions in the morbidly obese[22] BPD-DS has shown to result in rapid weight loss, with a mean percentage excess weight loss (% EWL) of 78 % sustained in the long-term.[10] Excess weight is the amount of weight above a normal BMI (BMI above 25 kg/m^2).

Reports show that weight loss, in particular visceral fat, leads to better management of insulin sensitivity and type 2 diabetes, obesity-related cardiomyopathy, cardiac function, lipid profile, respiratory function, disordered sleep, obesity-related infections, venous stasis, non-alcoholic liver disease, asthma, polycystic ovary syndrome, infertility and complications in pregnancy, joint problems (arthritis) and increases adiponectin levels. Subsequent reduction in abdominal pressure may also improve urinary incontinence, gastroesophageal reflux, hypertension, pseudotumor cerebri, venous stasis disease and hypoventilation. Less fat around the neck area

relieves obstruction too breathing and sleep apnoea. Most bariatric surgery patients also report improved psychosocial status and quality of life. [1]

Bypassing the proximal bowel also improves the physiological responses to gut hormones involved in glucose regulation and appetite control. [23]

Complications

Bariatric surgery is associated with several complications both in the early post-operative period and in the long-term. Due to the nature of these procedures many are related to metabolic disturbances and several can lead to the need for re-constructive surgery.

Table 3. Complications related to bariatric surgery

Immediate complications	**Early complications**	**Long- term complications**
Anastomoses rupture Problems with wounds • Infections, included fistulas • Rupture Heart- and lung problems - breathing - pneumonia - mycardial infarction - deep veinous thrombosis - lung embolism Death in connection with surgery (usually within 30 days)	Narrowing of anastomosis • Dysphagia • Emesis • Reflux	Malabsorption • Diarrhoea • Constipation • Nausea, emesis Dumping syndrome Conditions following malabsorption: vitamin deficiency iron deficiency calcium deficiency development of gall stones Mechanical complications: (a) ileus (b) malfunction of uncoupled part of intestine (c) incisinal hernia (d) gastrointestinal bleeding

Current knowledge regarding long-term nutritional complications following bariatric surgery is limited. This is especially true in relation to the BPD-DS technique. Nutrient deficiencies are common, and may become clinically significant if not recognized and treated. [24] Nutrient deficiencies previously identified in this patient group include protein, iron, vitamin B12, folate, calcium, fat soluble vitamins A, D, E and K, and other micronutrients. RYGB procedures commonly cause B_{12}, iron and folic acid deficiencies. The deficiencies appear to be more severe in malabsorptive procedures such as BPD-DS. Research is needed to evaluate the clinical significance

of these deficiencies and to determine guidelines for supplementation.[25] This section focuses on complications which are related to long-term metabolic complications in BPD-DS patients (as listed in table 2), relevant to bone health.

Nutritional Implications of BPD-DS

Sustained weight loss after biliopancreatic diversion with duodenal switch is primarily due to decreased calorie absorption secondary to fat malabsorption. Initial weight loss is also due to moderate gastric restriction. Scopinaro et al found that only 28 % of ingested fat and 57 % of ingested protein is absorbed after the BPD procedure. How much is absorbed after the BPD-DS procedure is unclear, but gastric and pancreatic enzymes are mixed with bile only in the distal part of the ileum (the last 50-100 cm) also after this procedure. Fat malabsorption may lead to insufficient uptake of other essential nutrients such as essential fatty acids and the fat soluble vitamins A, D, E and K. This may have severe clinical consequences and alter calcium metabolism. Among the metabolic complications protein, vitamin and mineral deficiencies as well as liver damage have been reported.[10, 25-27]

Table 4. Overview of reported metabolic complications associated with bariatric surgery.

Metabolic complications of bariatric surgery[a]

Complication	Clinical features	Management
Acid-base disorder	Metabolic acidosis, ketosis Metabolic alkalosis	Bicarbonate orally or intravenously; adjust acetate content in PN Salt and volume loading (enteral or parenteral)
Bacterial overgrowth (primarily with BPD, BPD/DS)	Abdominal distention Pseudo-obstruction Nocturnal diarrhea Proctitis Acute arthralgia	Antibiotics (metronidazole) Probiotics
Electrolyte abnormalities (primarily with BPD, BPD/DS)	Low Ca, K, Mg, Na, P Arrhythmia, myopathy	Enteral or parenteral repletion
Fat-soluble vitamin deficiency	Vitamin A—night vision Vitamin D—osteomalacia Vitamin E—rash, neurologic Vitamin K—coagulopathy	Vitamin A, 5000-10,000 U/d Vitamin D, 400-50,000 U/d Vitamin E, 400 U/d Vitamin K, 1 mg/d ADEK, 2 tablets twice a day (http://www.scandipharm.com)
Folic acid deficiency	Hyperhomocysteinemia Anemia Fetal neural tube defects	Folic acid supplementation
Iron deficiency	Anemia	Ferrous fumarate, sulfate, or gluconate Up to 150-300 mg elemental iron daily Add vitamin C and folic acid
Osteoporosis	Fractures	DXA, calcium, vitamin D, and consider bisphosphonates
Oxalosis	Kidney stones	Low oxalate diet Potassium citrate Probiotics
Secondary hyperparathyroidism	Vitamin D deficiency Negative calcium balance Osteoporosis	DXA Serum intact PTH level 25-Hydroxyvitamin D levels Calcium and vitamin D supplements
Thiamine deficiency (vitamin B_1)	Wernicke-Korsakoff encephalopathy Peripheral neuropathy Beriberi	Thiamine intravenously followed by large-dose thiamine orally
Vitamin B_{12} deficiency	Anemia Neuropathy	Parenteral vitamin B_{12} Methylmalonic acid level

[a] BPD = biliopancreatic diversion; BPD/DS = biliopancreatic diversion with duodenal switch; DXA = dual-energy x-ray absorptiometry; PN = parenteral nutrition; PTH = parathyroid hormone.

Source: [1]

According to Slater et al, [10] there is a progressive increase in the incidence and severity of A, D and K deficiency with time after BPD-DS. His team measured serum vitamins A, D, E, and K, zinc, parathyroid hormone, corrected calcium, and alkaline phosphatase levels in a cohort of patients who had previously undergone biliopancreatic diversion with or without duodenal switch. They found a mean % EWL loss of 48, 68, 59 and 59 % at one, two, three and four year post-surgery respectively. Serum vitamin A levels were low in 52 % of cases at one year and 69 % after four years, but no patients suffered from night blindness or visual abnormality. Vitamin K levels went from being abnormal in 51 % in the first to 68 % by the fourth year with unquantifiable levels (less than 0.1 nmol/L) in 42 % of cases. No patient had symptoms of excessive bleeding. All patients had normal levels of vitamin E one

year post-surgery and 96 % still had normal levels after four years. Serum zinc was abnormally low in 51 % at one year and 50 % after four years. Incidence of abnormal serum vitamin D levels was 57 % after one year and increased to 63 % in the fourth year. They also found that hypocalcaemia increased from 15 % in the first year to 48 % four years post-operatively. Secondary hyperparathyroidism was seen in 31 % after one year and 69 % after four. Of the latter, 27 % had developed clinically significant hyperparathyroidism (serum parathyroid levels more than 50 % above the upper limit for normal). Some patients (6 %) also had raised alkaline phosphate levels. No patients suffered from bone pain.[10]

In other studies biochemical measurements have shown that low levels of serum albumin, creatinine, retinol, 25(OH)D and elevated levels of PTH are common. BPD-DS with common channels less than 100 cm have a high incidence of complications and nutritional deficiencies.[21, 28]

Despite high patient satisfaction, the implication of the BPD-DS procedure may result in profound medical complications including, among others, malnutrition. It is therefore important to provide thorough and optimal follow up treatment for this patient group including long-term nutritional monitoring.[10] Some may require chewable or liquid supplements to help adequate absorption. [28]

Implications of BPD-DS DS for bone disease

Calcium and vitamin D deficiency, and elevated levels of PTH are common in morbid obesity. Up to 84 % of morbidly obese patients have vitamin D deficiency.[28] Sequestration of vitamin D in adipose tissue and subsequent limited bioavailability may be an important cause of this. In addition lifestyle factors associated with obesity, such as an unbalanced diet and limited sun exposure could lead to nutrient deficiencies and disturbances in bone metabolism. Bariatric surgery may exacerbate these disturbances (and consequently changes in bone mass).[29] Carlin et al, 2006 highlighted the importance of identifying vitamin D deficiency pre-operatively and found an inverse correlation between 25(OH)D levels with BMI and PTH values. [30]

Osteomalacia, osteopenia, secondary hyperparathyroidism and increased bone turnover comprise the major metabolic bone disturbances which have been reported after bariatric surgery. A common scenario is that post-surgical weight loss is accompanied by an increase in s-PTH levels, a decrease in s-25(OH)D levels while corrected calcium levels tend to remain within normal limits.[31] [32] Sinha and Bockman, 2007 noted that the degree of change in bone metabolism is probably related to amount of weight loss and malabsorption.[14,33]

In bariatric surgery there is a multifactorial aetiology to bone disease. Bypass procedures interfere with the normal mixing of intestinal contents with digestive juices which can lead to increased levels of fat in the intestinal lumen which in turn may decrease vitamin D absorption, leading to further decreases in calcium absorption. Steatorrhea also has a negative impact on calcium balance. In this condition calcium forms insoluble soaps with fatty acids in the gut. [14] The preferential sites for calcium absorption are primarily the duodenum and proximal jejunum. These are detached in the BPD-DS procedure. [34] When calcium absorption is decreased the body will maintain normal calcium levels by up-regulating PTH levels, increasing calcitriol and calcium resorption from bone. [35] Energy balance is also of importance for bone health. Profoundly restricted dietary intake, rapid weight loss, and impaired absorption of nutrients, is likely to implicate a high risk of developing metabolic bone disease after BPD-DS.[30, 32, 35-37]

The malabsorptive component of BPD-DS may also impair absorption of several other nutrients important in bone health, such as iron, vitamin C and vitamin K. For example when the distal small bowel is bypassed, magnesium deficiency may occur as a result of reduced absorption and chelation with unabsorbed fatty acids.[38] In patients with shorter absorptive surfaces it becomes especially important to optimize nutrient intake. Guidelines on optimal supplementation for BPD-DS patients have not been established.[28]

BPD-DS patients are given a high protein diet in order to prevent protein malnutrition. It is therefore important to assess postoperative protein intake. Although

urinary calcium loss is unchanged by inclusion of protein and phosphorous rich foods such as meat, cereal, beans and dairy products, phosphorous increases the calcium content of the digestive secretions and therefore increases endogenous calcium losses in the faeces. Thus increasing consumption of protein rich foods results in net-calcium loss, and this could exacerbate the risks of a low calcium diet. On the other hand, inadequate protein intakes compromise bone health and may contribute to osteoporosis.[25]

Thus BPD-DS patients depend on micronutrient supplementation to meet their needs. It is important to monitor compliance and adequate absorption. Crowley et al found that their gastric bypass patients who did not take vitamin D supplements, only achieved approximately half of required vitamin D intake.[39, 40] In patients with gastrointestinal side effects or a history of calcium oxalate renal stones, calcium citrate may be better tolerated, alter urine acidity and often prevent further stone formation.[28]

Bariatric surgery and bone mineral density

Studies looking at malabsorptive bariatric surgery and BMD have found an incidence of 12.5 % with a T-score below -2.5, 39 % with a T-score between -1.0 and - 2.5. Severe hypovitaminosis D (25(OH)D < 8ng/mL) was seen in 34 % and 42 % had low hypovitaminosis D (s-25(OH)D = 8-20 ng/mL). The magnitude of weight loss correlated negatively with s-25(OH)D, calcium and phosphorus, and positively with s-ALP levels. S-25(OH)D and calcium concentrations correlated positively with BMD. PTH, s-1.25$(OH)_2D_3$ and ALP concentrations correlated negatively with BMD. These characteristics were a reflection of the presence of secondary hyperparathyroidism, an accelerated conversion of 25(OH)D to 1.25(OH)D by the elevated PTH levels, and increased osteoblastic activity. They concluded that hypovitaminosis D and subsequent bone loss is common in patients who have undergone bariatric surgery. And these patients require rigorous vitamin D supplementation.[41]

Marceau et al investigated bone changes after BPD. They detected increased bone turnover and mineralization. Cortical thickness decreased, trabecullar bone volume increased. 10 years post surgery bone density was unchanged at the hip, but decreased by four percent at the lumbar spine. Elevated calcitriol was found to be a predictor for future bone loss. Elevated osteocalcin was associated with overall bone loss, and lower albumin levels were associated with bone loss at the hip. Lower calcium level was associated with bone loss at the lumbar spine. They concluded that bone was relatively tolerant to the metabolic changes after BPD, provided it was accompanied by close follow-up and treatment of metabolic disturbances and avoidance of malnutrition. They also concluded that the beneficial effects of surgery far outweigh the risk of post-operative bone disease.[42]

1.3 Bone Disease

Appendix 1 presents important dietary components related to bone metabolism, assessment methods of bone health including biochemical measurements and dual energy X-ray absorptiometry, and a brief outline of the physiology of bone metabolism

The prevention and treatment of bone disease is done in two stages during life. The primary prevention stage, during the first decades of life, is focused on maximising peak bone mass. Secondary prevention takes place in later stages of life and focuses on the prevention of excessive bone loss and subsequent osteoporosis. Both prevention stages have similar intervention strategies such as ensuring adequate nutrient intake and weight bearing physical activity. As discussed in appendix one, several important nutrients contribute to maintaining healthy bone. Many of these are co-dependent, interacting with each other, as well as genetic and environmental factors.[43] Bone disease may be caused by nutritional deficiency and nutritional excess.

Osteopenia

Osteopenia is a term that describes low bone density. Even though osteopenia is not considered a disease, preventive measures should be taken since osteoporosis may develop if there is further losses in bone density.[44]

1.3.1 Secondary hyperparathyroidism (SHP)

Too much PTH production caused by increased activity of the parathyroid glands is known as hyperparathyroidism. When this occurs in response to low blood calcium, caused by another condition, the hyperparathyroidism condition is called secondary hyperparathyroidism. Secondary hyperparathyroidism is characterized by s-PTH above 8,5 pmol/L combined with 25(OH)D below 50 nmol/L and normal s-calcium levels (Ca^{2+} 2.15 – 2.55 mmol/L).[45]

A fall in serum-calcium results in secondary increase in PTH secretion. This condition is seen in malabsorptive states, osteomalacia, liver failure and chronic renal failure.

1.3.2 Osteomalacia and rickets

Osteomalacia results from vitamin D deficiency or a disturbance in its metabolism. The main features include bone pain and tenderness, deformity and proximal muscle weakness. The biochemical changes include low plasma calcium levels, a reduction in plasma phosphate and a reduction in urine calcium excretion. However these changes depend on the underlying cause. A low plasma 25(OH)D level indicates vitamin D deficiency. Very low levels indicate nutritional osteomalacia, but not all patients with low levels develop bone disease. Diagnosis of osteomalacia is confirmed by a transiliac bone biopsy. The under calcified sections have an excessive thickness of osteoid and an increased coverage of the mineralized surfaces with unmineralized osteoid.

Nutritional rickets may be a problem in Asian immigrants and in elderly people who are housebound. A vegetarian diet has also been linked to this disorder. A significant

number of elderly patients with femoral neck fracture have osteomalacia as well as osteoporosis.[46] chapt 71, page 329-333

In osteomalacia the three classes of markers for osteoblast activity no longer correlate. PICP is normal, ALP is grossly elevated and osteocalcin is decreased. This is because the expression of osteocalcin is regulated by vitamin D. The increase in ALP may suggest that in the absence of vitamin D, the osteoblasts are halted the developmental phase immediately preceding matrix mineralization, which is characterized by ALP expression, leading to a situation where more cells than usual are producing the enzyme.[47]

Table 5. Overview of the different nutrients involved in bone metabolism and their role in the development of bone disease.

	Nutritional component	Associated disease
deficiency	Vitamin D	Rickets/ osteomalacia
	Vitamin C	Scurvy
	Copper	Fractures (premature infants; PN*)
	Zinc	Delayed bone growth
	Vitamin K	? osteoporosis
excess	Vitamin A	Hyperostosis
		Ligamentous ossification
	Vitamin D	Idiopathic hypercalcaemia in infancy
	Aluminium	Aluminium osteodystrophy (dialysis and parenteral nutrition)
	Fluoride	Endemic
		Iatrogenic
	Cadmium	Fanconi syndrome
		Rickets/ osteomalacia
nutrition related	Calcium	Osteoporosis
nutrition dependent	Vitamin D	Rare forms of rickets
	Pyridoxine	Homocystinuria

* Parenteral nutrition (PN)
Source: [48]

1.3.3 Osteoporosis

Osteoporosis is a systemic skeletal disease in which the bone density (amount of bone per unit volume) is decreased while the composition is unchanged (porous bone tissue). This occurs because of an imbalance between bone formation and resorption.

Prevalence

The incidence of osteoporosis is increasing rapidly in the western world and is a major socio-economic problem. Every 3rd female and every 20th male will experience at least one osteoporotic fracture. The incidence is increasing exponentially with age in both males and females.[46] chapt 38, page 198-202. Hip fractures are accompanied by up to 20 % reduction in remaining life expectancy: 50 % of patients who could walk prior to a hip fracture, are unable to walk without aid afterwards, a third will be completely dependant on care.[43]

In Norway osteoporosis has been recognized as a major public health problem with one of the highest incidences of bone fractures in the world. There is a significant high incidence rate of femur, wrist and spine fractures and it is believed that osteoporotic fractures will reach epidemic proportions in the near future.[44]

Causes

The most important determinants of osteoporosis is peak bone mass and subsequent rate of bone loss, [49] but causes of osteoporosis are multifactorial, and a range of nutrients and foods may influence its development. Long-term deficiency or excess of one or more bone related nutrients, as well as changes in requirements following physiological and metabolic influences may contribute in the development of osteoporosis.[43] In addition to a well balanced diet ensuring adequate calcium and vitamin D intake it is important to be physically active and avoid underweight.[44] Unexplained osteoporosis is seen in younger males and females (idiopathic osteoporosis), and secondary to several diseases (for example induced by steroids, impaired gonad function, malabsorption, hypertyreosis, and immobilization).

Diagnosis

The diagnostic criteria for osteoporosis involve both a clinical and statistical element. Clinically, the loss of bone is associated with micro architectural deterioration of bone tissue with a consequent increase in bone fragility and susceptibility to fracture. Statistically, the WHO defines osteoporosis as a T-score for bone mineral content of

more than 2.5 SD below the mean for young adults accompanied by at least one fragility fracture (a fracture that occurred as a result of slight trauma). It can be caused by several different pathogenetic mechanisms and therefore the biochemical indicators of bone metabolism will behave differently under several conditions that can all lead to osteoporosis. Increased bone resorption is reflected in pathological concentrations of bone collagen breakdown products e.g. 1-CTP.[47]

Bone fractures

In osteoporosis bone mass and bone strength is reduced to such a level that fractures occur without or after very little trauma (spontaneous fractures). Due to the structure and high metabolic activity, trabechular bone is most vulnerable. Common osteoporotic fractures are vertebral, forearm and femoral neck. The latter is considered the most important because it occurs most frequently and leads to considerable morbidity. Many osteoporotic fractures are frequent, lead to immediate disability, early and late mortality and considerably morbidity. Prevention of this disease is of considerable clinical and economic importance.[46] chapt 38, page 198-202

Treatment

Increasing calcium intake does not have the same effect as estrogen, bisphosphonates and calcitonin therapy, and it is therefore not adequate as the only treatment for osteoporosis. However adequate calcium intake should be the basis of any other treatment.[43]

Study rationale

The tremendous health consequences of morbid obesity and accompanying costs are considered important arguments for the surgical treatment of morbid obesity. However if the treatment contributes to morbidity from bone disease, one may argue that surgery could be counterproductive. Although nutritional complications are relatively well documented in respect to bariatric surgery in general, studies focusing on BPD-DS and bone health are limited, particularly in respect to long-term complications.[50]

Several studies have shown changes in bone metabolism after bariatric surgery. Such changes have included vitamin D deficiency, secondary hyperparathyroidism, hypocalcaemia and increased bone turnover. It is believed that the severity of these consequences depend on the degree of weight loss and malabsorption. Of the bariatric procedures commonly performed today, BPD-DS causes the most severe weight loss and malabsorption. [14] However few studies focus on BPD-DS in particular and even fewer give insight into dietary habits which may affect bone health in this patient group. This study hopes to contribute information about medium term bone health (2-5 years) following BPD-DS typical in Norwegian patients.

Study Aims

The primary aim of this study was to examine bone mineral density, nutrient intake and biochemical markers relevant to bone health in 65 BPD-DS patients 2 - 4 years post surgery, and identify potential long-term risk factors which may lead to disturbances in bone metabolism.

Research questions

1) Compare bone health status in patients and reference populations by measuring bone mineral density (BMD), using dual energy X-ray absorptiomentry (DXA) and by measuring biochemical markers relevant to bone metabolism.

H_0 BPD-DS patients have BMD and biochemical markers within normal range compared to reference population.

H_1 BPD-DS patients have decreased BMD, and altered biochemical markers compared to reference population.

2) Compare anthropometric measurements to pre-surgical values, and correlate with BMD and biochemical markers relevant to bone health.

H_0 Weight reduction after BPD-DS is not associated with biochemical markers or BMD

H_1 The degree of weight reduction is positively related to biochemical markers and inversely related to bone mineral density.

3) Examine nutrient intake relevant to bone health, compare to current recommendations, BMD and biochemical markers of bone metabolism.

H_0 Nutrient intake after BPD-DS is not associated with indicators of bone health.

H_1 Adequate nutrient intake is associated with protection against impaired biochemical markers and losses in BMD after BPD-DS.

2. Methods

The entire research project, from which this study is taken, is a result collaboration of two master students in clinical nutrition with medical personel at UUS, Department of Preventive Cardiology. These results have been divided between the two students and previous results have been presented in an earlier master thesis.

2.1 Arrangements

The two master students in clinical nutrition organised the procedures described here for all eligible patients who had undergone BPD-DS surgery at least two years earlier and up to five years earlier. The patients were all registered at the Department of Preventive Cardiology, UUS. The assessments, described in this study are part of the standard follow up procedure for this patient group in order to determine individual needs for medical and dietetic treatment. Routine blood samples required for the usual follow-up of the patients were obtained. Upon patient consent these were used in the study.

The patients were invited to attend consultations and assessments all scheduled on the same day. To the extent possible, consultations were arranged early in the morning to make it convenient for the patients to combine with taking fasting blood tests at the UUS clinical chemistry and haematology laboratory. The patients were offered breakfast before and/ or in between consultations. Patients attended a consultation with the same physician (Serena Tonstad) and one of the two masters students in clinical nutrition.

2.2 Study assessment overview

Table 6: Study assessments

Study Assessment Overview	
Consultation with professor Serena Tonstad	Bowel problems (including stool frequency), height, weight, BMI, waist circumference
Surgery details from patient records	Surgery location, length of common channel, corrective surgery, pre-surgery anthropometric measurements
Dual energy x-ray absorptiometry (DXA)	Bone mineral density was measured using the: Lunar iDXA, version 11.20.068 (Human GE, Madison, Wisconsin) by a specialist in endocrinology, Dr. Johan Halse and his team at the Osteoporosis clinic, Pilestredet Park 7, Oslo Norway. [51]
Biochemical measurements (blood-test results for the following parameters)	s-calcium, ionised calcium, 1.25-dihydroxyvitamin D, 25-hydroxyvitamin D, Parathyroid hormone, bone specific ALP, osteocalcin, 1-CTP, phosphate, magnesium, albumin, s-retinol, cobalamin, folate
Subjective interview with master students in clinical nutrition	Interview assessing the following factors in relation to dietary intake: Supplement intake and compliance, Problems relating to food intake, Discomforts and disturbances in relation to digestion
Dietary registrations	Dietary intake was assessed using a validated 7 day pre-coded food diary previously used in the Ungkost project.[52] The diary was developed at the Department for Nutrition Research, University of Oslo.

2.3 Study population

2.3.1 Inclusion criteria

All patients who were registered for post-surgical follow-up after BPD-DS surgery at the Department of Preventive Cardiology, UUS, and had undergone surgery 2 years or more previously (up to 5 years), were invited to attend a routine follow-up consultation. Patients who attended their consultations were asked to allow routine assessment results to be used in this study.

2.3.2 Exclusion criteria

There were no exclusion criteria. All patients known to be alive were invited. Patients who did not turn up for consultations after repeated invitations by post and telephone could not be included

2.3.3 Ethical approval

Ethical approval was obtained from the Regional Committees for Medical Research Ethics, south-east Norway (REK), Postboks 1130, Blindern, 0318 Oslo. UUHs' department for patient protection in research provided guidance on the development of forms for patient information and written consent. No additional assessment was conducted solely for the purpose of research, as this would have required the use of a bio bank. Data was stored in the research server of UUS. Written consent was obtained from all study participants.

2.3.4 Anthropomentric and physical assessments

Dr. Serena Tonstad conducted routine assessments, which included medical history including gastrointestinal symptoms, anthropometric measurements and physical examination. The patients were weighed in the morning after a small meal (usually the breakfast provided following the blood tests). Patients were weighed bearing light indoor clothing without shoes. A Seca Alpha model 770 scale (Seca, Hamburg, Germany, www.secaonline.com) was used.

Information about pre-surgical weight and BMI was obtained from the patients' records and compared to the results from the follow-up assessments. A letter of individual recommendations was sent to each patient based on blood test and other results obtained within 8 weeks of the consultation.

2.4 Bone mineral density (BMD)

Dual energy X-ray absorptiometry (DXA)
Bone mineral density was assessed by using DXA measurements [53], to identify potential long term nutrient deficiencies and disturbances in bone metabolism.

DXA measurements were obtained by referral to an experienced laboratory at the Osteoporosis clinic, (Pilestredet Park 7, Oslo, Norway). Assessments were conducted

by an endocrinologist, Dr. Johan Halse and his specialist team using the: Lunar DXA, version 11.20.068 (Human GE, Madison, Wisconsin) [51].

Z-score Results

The DXA measurement resulted in three different z-scores. All three were evaluated including total body bone mineral density z-score (TBBD), lumbar spine L1-L4 z-score (AP spine) and dual femur bone density mean total z-score (dual femur). The z-score compared BMD results to a reference population of age and gender matched controls.

2.5 Biochemical measurements

Results from biochemical measurements, relevant to bone health, taken during routine follow-up consultations, were obtained from the patients' records. The following biochemical markers were considered in this study: Parathyroid hormone (PTH), $1.25(OH)_2D_3$, calcitonin, C-terminal telopeptide of type I collagen (1-CTP), bone specific- alkaline phosphatase (b-ALP). Other relevant biochemical measurements included serum levels of nutritional status including 25(OH)D, s-calcium albumin corrected (s-calcium alb corr), s-ionised calcium (s-ion calcium), s-phosphate, s-cobalamin, s-folate and s-albumin levels.

Fasting venous blood had been collected at the clinical biochemistry laboratory, UUS, and analysed at the clinical chemistry and haematology laboratory at UUS, except the values for vitamin A, which were analysed at the nutrition laboratory at Aker University Hospital and vitamin D, calcitonin, PTH, 1-CTP and b-ALP which were analysed at the hormone laboratory at Aker University Hospital.

Parathyroid Hormone (PTH)

PTH was measured in frozen serum with a non- competitive immunoluminometric assay (ILMA) method obtained by using Immulite 2000 (Diagnostic Products Corporation (DPC), Los Angeles, CA. USA). At Aker Hormone lab they test for

intact PTH (1-84). This analysis has a high diagnostic accuracy and is able to differentiate between hypercalcaemia caused by hyperparathyroidism and hypercalcaemia caused by other diseases.

1,25-OH_2 vitD and 25-OH vitD

The amounts of calcitriol and calcidiol in serum were both measured by using a competitive radioimmunoassay (RIA) method (Kit from DiaSorin, Stillwater, MN, USA).

Osteocalcin

Osteocalcin was measured in serum using Brahms' Luminoimmunanalysis (LIA), article number 58.1. Brahms Aktiengesellschaft, Neuendorfstr. 25, D-16761 Hennigsdorf, [54]

Bonespecific-alcaline phosphatase (b-ALP)

The enzymatic activity of bone specific alkaline phosphatase (b-ALP) was measured with Kit (Metra Biosystems Inc., Mountain View, Ca, USA).

Vitamin A

Vitamin A was measured by high performance liquid chromatography (Kit from Bio-Rad Laboratories, California USA) on Perkin Elmer systems (Perkin Elmer, Massachusetts USA).

1-CTP

C-terminal telopeptide of type 1 collagen (1-CTP) was measured using the UniQ radium immunoassay kit from Orion Diagnostica, P.O.Box 83, FI-02101 Espoo, Finland. [55]

Ionised calcium

Ionised calcium was run on a machine from Roche called Cobas b 221 (This machine uses an ion- selective electrode).

S-calcium, s-phosphate, s-magnesium and s-albumin

The results for the tests listed below were obtained on the Cobas Integra 800 (Roche Diagnostics):

Table 7. Biochemical parameters analysed using the Cobas Integra 800 (Roche diagnostics)

Analysis methods on the Cobas Integra 800	
magnesium	Colorimetric method with Chlorophosphonazo III
phosphate	Endpoint method
albumin	Colorimetric endpoint method
total calcium	Schwarzenbach with o-cresolphtalein complexone

Cobalamin and folate

For the analysis of haemoglobin a flowccytometer- based instrument called Sysmex XE 2100 (The Sysmex Corporation, Kobe Japan) was used. Ferritin, vitamin B12 and folic acid in serum was analyzed on Advia Centaur from Siemens

2.6 Dietary assessments

2.6.1 Consultation procedures.

The patients were scheduled for a one-hour visit with one of the two students to assess dietary habits and receive practical advice on food and supplement intake.

These consultations included a subjective interview to assess dietary intake, and to make recommendations for adequate supplement, fluid and protein intake. To ensure standardised questions were asked, a structured interview guide was used in these consultations. (See appendix 2) The patients were also asked if dietary components had changed as a result of the DS surgery. Food intolerances in particular those regarding dairy products and high fat foods were assessed.

The structured interview also included detailed questions about supplement intake and compliance. The patients were first asked an open question about general supplement

intake; this was then cross-checked by asking specific questions about certain supplements recommended to BPD-DS patients. Information about brand, frequency and amount was obtained for each supplement. The interview also considered frequency of occasions when supplements were not taken as planned, and which supplements were missed out.

Patients were considered to have an adequate supplement intake if they complied with the minimum recommended intake of at least 4000 mg of calcium (4 tablets of calcigran forte), 750 µg vitamin D (one AfiD2 forte) and one vitamin-mineral supplement (Nycoplus multi or equivalent) per day. Other supplement intake was not considered in relation to bone mineral density in this report.

2.6.2 Dietary records

After consultations, dietary intake was assessed using a 7-day (3 + 4) pre-coded, portion-size based food diary, which had been developed at the Department of Nutrition Research, University of Oslo were it was used in a population based epidemiological study, Ungkost, looking at dietary habits among the younger generations in Norway.[52] (See appendix 3) This also included record of supplement intake. The diary had one booklet for each day which consisted of lists of food products and beverages. The lists were divided into bigger/smaller food groups (e.g. drinks, bread, vegetables, snacks, sweets). Every group had additional space to record intake of products which were not listed in the diary. For every food item/ beverage there were units listed (e.g. glass, slice, can, plate, and tub). The diary was filled in by recording amount of the relevant food item taken at a set time interval. The days were divided into five periods (6- 10 am, 10 am- 2 pm, 2- 6 pm, 6 - 10 pm and 10 pm- 6 am). Accompanying the diary, was a booklet illustrating different portion sizes, as a guide to help the patients identify their respective portion sizes and ensure correct quantity of food item was recorded as accurately as possible. Patients also received detailed guidelines (verbal and written) on how to fill in the pre-coded food diary

(Appendix 4). Upon completion, the diaries were sent back to the clinic in a prepaid envelope.

The food diaries were scanned, optically read and proof read using Cardiff Teleform 6.0 scan station. The result file was then imported into KBS, a nutrient calculation computer programme, to calculate energy and nutrient contents from the food diaries. KBS was also developed at the Department of Nutrition Research, University of Oslo.[52]

2.7 Statistical Analysis

All variables were examined using descriptive statistics including measures of central tendency, standard deviations and ranges.

The Student t- test was used to compare continuously distributed variables to reference values. A paired samples t-test was used to test significant differences in anthropometric measurements, comparing pre-to post-operative values. Bone mineral density was compared between patients whose common channel length was => 100 cm or <100 cm, and between those who had a common channel length => 70 cm and < 70 cm. An independent samples t-test was used to investigate the three BMD z-scores according to gender.

Because several variables were not normally distributed Spearman correlation analysis was used in the assessment of potentially related variables. The following variables were tested for correlation with the three mean z-score values for BMD and biochemical markers of bone metabolism; age, weight and BMI characteristics including changes in pre- and post surgical values, waist circumference at follow up, calcium intake from food and supplements, intake of vitamin D from food and supplements, intake of multivitamin supplements, stool frequency, s-PTH, s-25-OH vitD, s-1.25-OH vitD, s-osteocalcin, s-1CTP, s-bone specific-ALP, s-ionized calcium,

s-magnesium, s-phosphate, s-albumin and months since surgery. Significant correlations were presented.

Bone Mineral Density Compared to Supplement Intake.

Relationship between supplement intake and BMD was investigated for correlation and differences between groups according to compliance with supplements. The three z-scores for BMD were also compared to calcium, vitamin D and general supplement intake using an independent samples t-test. Adequate intake was classified according to guidelines for supplement intake in this patient group.

Multiple Regression of Significantly correlated variables

Multiple regression analysis was performed to identify the relative interrelationships between significantly correlated variables to the three BMD mean z-scores and serum levels of I-CTP.

A p-value of < 0.05 was considered statistically significant. The software program used was SPSS 15.0.

3. Results

3.1 Subjects

Among the patients registered for post-surgical follow-up, 77 had undergone surgery 2-5 years prior to consultation dates. One patient was diseased, and ten patients did not attend follow-up consultations. Among the 66 patients who did attend, all consented to having their follow-up assessment results used in this study. One patient was excluded due to incomplete and suspected inaccurate measurements in several study parameters. This patient had incomplete biochemical measurements and was hospitalised for correctional surgery (to lengthen his common channel) during the post-surgery follow-up assessments. In total, twelve patients were excluded from the study. Thus the study population consisted of 65 BPD-DS patients (84.4 %of the total known patients), including 55 women and 10 men in total.

3.1.1 Patient characteristics

Country of surgery

Table 8 presents country and city where patients had their BPD-DS surgeries, including the respective number of patients operated in each location.

Table 8. Surgery location

Country of surgery	**number of patients** (N=65)
Spain, Alicante	35
Norway, Førde	6
Belgium	9
Russia, Moscow	12
Germany	2
Sweden, Gothenburg	1

The majority had undergone surgery at Dr. Baltasars' clinic in Alicante, Spain.

Common Channel Length

Table 9 presents common channel length for the study population for whom the information was available.

Table 9. Length of common channel.

Length of common channel**,** cm n=62	
mean (SD)	71 (17)
median (p25, p75)	65 (60, 75)
min-max	50-150

Information about common channel length was available in 62 cases. In three cases the medical records did not provide this information and the patients themselves stated that they had not been informed. Common channel length varied according to location of surgery and pre-surgical BMI. The shortest common channel was reported from surgery performed in Russia with 50 cm, and the longest was 150 cm from surgery performed in Germany.

3.1.2 Results anthropometric measurements

Table 10 presents age at follow-up and pre- and post surgical anthropometric data.

Table 10. Age and anthropometric characteristics

Characteristics	**total** N=65	**women** n=55	**men** n=10
age, years			
mean (SD)	40 (8.6)	40 (7.6)	39 (13.4)
median (p25,p75)*	38 (34.0, 46.5)	38 (34.0, 44.0)	38 (28.3, 51.0)
min- max.	21-59	26- 58	21- 59
weight pre-surgery, kg			
mean (SD)	150.0 (27.1)	146.9 (26.2)	167,3 (27.0)
median (p25,p75)*	145.0 (130.5,163.0)	141.0 (129.0, 160)	156.5 (144.3, 193.5)
min- max.	108.0 – 235.0	108.0 – 235.0	140.0 – 215.0
weight at follow up, kg			
mean (SD)	87.9 (15.3)	86.0 (15.5)	98.2 (9.6)
median (p25,p75)*	86.6 (77.5, 98.6)	85.0 (76.0, 96.0)	95.2 (92.4, 105.4)
min- max.	57.0 – 141.0	57.0 – 141.0	85.0 - 118.0
weight reduction, kg			
mean (SD)	62,1 (56.8, 67.4)	60.8 (55.3, 66.3)	69.1 (49.9, 88.3)
median (p25, p75)*	56.7 (49.2, 75.5)	56.0 (49.0, 74.0)	64.0 (46.8, 98.7)
min-max	27.0, 135.0	27.0 - 135.0	33.3 – 103.6
excess weight loss, %			
mean (SD)	76 (72, 79)	76 (72, 80)	74 (65, 84)
median (p25, p25)*	76 (65, 85)	75.8 (61, 95)	77 (71, 118)
min – max	51 – 120	51- 120	52- 90
BMI pre-surgery, kg/m²			
mean (SD)	51.1 (8.2)	51.1 (8.4)	50.5 (7.5)
median (p25,p75)*	50.6 (44.8, 55.9)	50.6 (45.0, 55.7)	49.1 (43.9, 58.5)
min- max.	38.0- 84.3	38.0- 84.3	39.6- 60.4
BMI at follow up, kg/m²			
mean (SD)	29.9 (4.4)	29.9 (4.6)	29.7 (3.0)
median (p25, p75)*	29.5 (26.6, 32.7)	29.8 (26.5, 32.6)	29.2 (27.3, 33.0)
min- max.	19.5- 47.1	19.5- 47.1	25.1- 34.3
BMI reduction, kg/m^2			
mean (SD)	21.1 (7.1)	21.2 (7.1)	20.9 (7.9)
median (p25, p75)*	20.1 (16.6, 25.7)	20.3 (16.7, 25.3)	18.9 (14.1, 28.2)
min – max	7.8- 35.5	7.8- 48.4	9.4- 31.6
waist circumference, cm			
mean (SD)	96 (12)	95 (12)	103 (11)
median (p25, p75)*	95 (89, 106)	95 (88, 102)	101(93, 115)
min- max.	72- 119	72- 119	90- 118

*p25 and p75 represents the 25th and 75th percentile respectively

Women had the greatest variation in anthropometrics, representing both the highest and lowest values for both weight and BMI, but the majority weighed between 110 and 170 kg pre-surgery. Three patients had extreme values, with a weight above 215 kg and one patient had a relatively low weight of 108 kg pre-surgery.

All patients in this study had achieved satisfactory weight loss at the time of the follow up consultations. The study population had a mean weight loss of 62.1 kg and on average 75.6 % of excess weight was lost.

3.2 Results Bone Mineral Density (BMD)

DXA measurements were obtained in 57 cases, representing 88 % of the study population. Table 11 presents z-score results from DXA measurements of bone mineral density (SD of age matched controls) and compares men and women.

Table 11. Bone mineral density (BMD) z-scores

DXA z-score results	total group	women	Men	p-value
total body bone density	(n=57)	(n=48)	(n=9)	0.02
mean (SD)	-0.31 (1.30)	-0.14 (1.22)	-1.23 (1.44)	
median (p25,p75)*	-0.20 (-1.25, 0.80)	-0.10 (-1.08, 0.98)	-1.80 (-2.45, 0.15)	
min- max.	-3.3 – +2.9	-2.9 - +2.9	-3.3 - +0.8	
lumbar spine L1-L4	(n=57)	(n=48)	(n=9)	0.055
mean (SD)	- 0.74 (1.54)	-0.58 (1.48)	-1.64 (1.60)	
median (p25, p75)*	-0,80 (-2.00, 0.40)	-0.80 (-1.80, 0.50)	-1.40 (-3.15, -0.40)	
min- max.	-4.1-+2.9	-3.1-+2.9	-4.1-+0.8	
dual femur bone density	(n=56)	(n=48)	(n=8)	0.591
mean (SD)	-0.38 (1.09)	-0.35 (1.12)	-0.58 (0.92)	
median (p25,p75)*	-0.45 (-1.10, 0.40)	-0.40 (-1.08, 0.40)	-0.60 (-1.55, 0.43)	
min- max.	-2.6-+3.5	-2.6-+3.5	-1.8-+0.5	

*p25 and p75 represents the 25th and 75th percentile respectively

Mean total body bone density (TBBD) z-score for entire group indicated an average BMD in the low-normal range for the study population. The lowest TBBD z-score was measured in the male group, in which the mean z-score was much lower. Mean z-score for lumbar spine in the male group was also much lower than in the female group. This group also represented the lowest measured value of -4.1 SD. Independent samples t-test showed significant differences in TBBD z-score between genders (p-value 0.02). There were no significant differences between genders in mean z-scores for lumbar spine L1-L4 or dual femur mean total.

Table 10 shows number of patients with normal or abnormal BMD scores. A z-score between –1 and 1 was considered within normal range of reference population. A z-score below -1.0 SD indicated a BMD lower than average compared to their peers and within the lowest 1 - 14 % of age matched controls. A z-score below -2.5 SD indicated a BMD lower than 99 % of the reference population.

Table 12. Classification of BMD results

Total body bone density z-score	group (n=57)	Women (n=48)	men (n=8/9*)
=> -1.0	39 (68 %)	36 (75 %)	4 (54 %)
< -1.0	14 (25 %)	12 (25 %)	5 (56 %)
=< -2.5	4 (7 %)	2 (4.2 %)	2 (22 %)
lumbar spine bone density z-score			
=> -1.0	32 (56 %)	30 (62 %)	4 (45 %)
< -1.0	18 (32 %)	18 (38 %)	5 (56 %)
=< -2.5	7 (12 %)	4 (8 %)	3 (33 %)
dual femur bone density z-score			
=> -1.0	39 (69 %)	36 (75 %)	5 (63 %)
< -1.0	16 (29 %)	12 (25 %)	3 (38 %)
=< -2.5	1 (2 %)	1 (2 %)	0

*In the male group n=9 for total body bone density and AP spine, for dual femur n=8

Approximately 25-30 % had BMD results below normal. In the male group, the prevalence of low BMD was much higher at 56 %, but this difference was only significant in TBBD z-scores. Only 7-12 % had z-scores above 1.0 SD

3.3 Results biochemical measurements

Table 13 presents blood test results relevant to bone health for each gender. These were available for all patients.

Table 13. Biochemical measurements

Biochemical results			
Nutrient status markers relevant in the maintenance of bone health			
s-25(OH)D, nmol/L	All (n=65)	Women (n=55)	Men (n=10)
mean (SD)	62.2 (27.4)	61.7 (25.5)	65.0 (38.0)
median (p25,p75)*	59.0 (39.5, 81.5)	60.0 (41.0, 81.0)	52.5 (37.8, 93.8)
min- max.	17.0 – 135.0	17.0 – 114.0	18.0 – 135.0
s-calcium albumin corrected, mmol/L	(n=64)	(n=54)	(n=10)
mean (SD)	2.23 (0.17)	2.22 (0.17)	2.27 (0.12)
Median (p25, p75)*	2.25 (2.16, 2.33)	2.23 (2.17, 2.32)	2.32 (2.14, 2.36)
min – max	1.17 – 2.43	1.17 – 2.42	2.08 – 2.43
s-ion calcium active, mmol/L	(n=61)	(n=51)	(n=10)
mean (SD)	1.24 (0.04)	1.24 (0.04)	1.25 (0.06)
Median (p25, p75)*	1.24 (1.22, 1.26)	1.23 (1.21, 1.25)	1.26 (1.21, 1.29)
min – max	1.14 – 1.34	1.16 – 1.36	1.14 – 1.33
s-phosphate, mmol/L	(n=64)	(n=54)	(n=10)
mean (SD)	1.26 (0.17)	1.28 (0.15)	1.16 (0.25)
Median (p25, p75)*	1.28 (1.12, 1.37)	1.29 (1.17, 1.37)	1.15 (0.92, 1.31)
min – max	0.88 – 1.66	0.91 – 1.59	0.88 – 1.66
s-magnesium, mmol/L	(n=62)	(n=52)	(n=10)
mean (SD)	0.88 (0.05)	0.88 (0.05)	0.88 (0.07)
Median (p25, p75)*	0.89 (0.84, 0.91)	0.89 (0.84, 0.91)	0.88 (0.84, 0.95)
min – max	0.75 – 1.01	0.76 – 0.98	0.75 – 1.01
s-cobalamin, pmol/L	(n=63)	(n=53)	(n=10)
mean (SD)	233 (102)	238 (108)	209 (60)
Median (p25, p75)*	212 (168, 273)	213 (162, 276)	199 (172, 243)
min – max	101 – 684	101 – 684	116 – 328
s-folate, nmol/L	(n=64)	(n=54)	(n=10)
mean (SD)	18.4 (9.4)	19.68 (9.38)	11.67 (6.26)
median (p25, p75)*	16.1 (10.7, 24.5)	17.55 (12.63, 25.60)	10.45 (7.55, 13.80)
min – max	4.8 – 45.7	6.00 – 45.70	4.80 – 27.30
s-vitamin A, µmol/L	(n=62)	(n=53)	(n=9)
mean (SD)	1.3 (0.5)	1.3 (0.5)	1.4 (0.5)
median (p25, p75)*	1.3 (1.0, 1.8)	1.3 (1.0, 1.7)	1.4 (1.0, 1.9)
min – max	0.2 – 2.5	0.2 – 2.5	0.7 – 2.0
s-albumin, g/L	(n=64)	(n=54)	(n=10)
mean (SD)	42.2 (2.9)	41.7 (2.9)	44.7 (1.9)
median (p25, p75)*	42.0 (41.0, 44.0)	41.5 (40.0, 44.0)	44.5 (43.0, 46.0)
min – max	32.0 – 49.0	32.0 – 49.0	43.0 – 49.0
Markers of bone metabolism			
s-PTH, pmol/L	(n=64)	(n=54)	(n=10)
mean (SD)	13.7 (6.3)	14.2 (6.6)	11.0 (4.0)
median (p25,p75)*	12.6 (9.8, 16.3)	12.7 (10.3, 18.8)	11.2 (8.4, 13.4)
min – max	4.8 – 31.7	4.8 – 31.7	4.9 – 13.4
s-1.25(OH)$_2$vitD, nmol/L	(n=64)	(n=54)	(n=10)
mean (SD)	145.3 (47.4)	143.2 (48.6)	156.8 (40.2)
median (p25,p75)*	140.0 (115.3, 179.0)	131.0 (110.5, 175.3)	144.5 (130.5, 193.0)
min- max.	66.0 – 326.0	66.0 – 326.0	90.0 – 224.0
Bone resorption markers			
s-1CTP, µg/L	(n=54)	(n=44)	(n=10)
mean (SD)	5.5 (1.8)	5.5 (1.9)	5.3 (1.4)
median (p25, p75)*	5.3 (4.3, 6.8)	5.4 (4.3, 6.9)	4.9 (4.3, 6.1)
min – max	1.5 – 10.7	1.5 – 10.7	3.6 – 8.1

Bone formation markers			
s-bone specific ALP, E/L	(n=54)	(n=44)	(n=10)
mean (SD)	40.0 (15.3)	38.3 (15.2)	47.1 (14.6)
median (p25, p75)*	38.0 (28.8, 50.0)	36.0 (27.3, 47.5)	43.5 (38.5, 56.8)
min- max.	18.0 – 107.0	18.0 – 107.0	25.0 – 78.0
s-ALP, IU/L	(n=64)	(n=54)	(n=10)
mean (SD)	89,3 (29.6)	86.8 (29.1)	102.9 (30.0)
median (p25, p75)*	84.0 (68.5, 103.3)	78.0 (67.3, 101.0)	95.5 (84.5, 126.8)
min – max	49.0 – 207.0	49.0 – 207.0	50.0 – 154.0
s-osteocalcin, nmol/L	(n=54)	(n=44)	(n=10)
mean (SD)	1.9 (0.8)	1.8 (0.8)	2.1 (0.7)
median (p25,p75)*	1.8 (1.5, 2.2)	1.7 (1.5, 2.1)	2.2 (1.9, 2.8)
min- max.	0.5 – 5.4	0.5 – 5.4	0.7 – 2.9

*p25 and p75 represents the 25th and 75th percentile respectively

Table 14 shows the prevalence of abnormal results in relation to reference values.

Table 14. Prevalence of abnormal biochemical results

Biochemical results - abnormal values	no of cases (%)	Reference
Nutrient status markers		
s-25(OH)D (n=65, f= 55, m= 10)	14 (23 %)	*< 37 nmol/L*
s-calcium albumin corrected (n=64, f=54, m=10)	16 (25 %)	*>2.17 mmol/L*
s-ionized calcium active (n=61, f=51, m=10)	1 (2 %)	*<1.15 mmol/L*
s-phosphate (n= 64, f= 44, m= 10)	11 (17 %)	*> 1.4 mmol/L*
s-magnesium (n= 62, f= 52, m= 10)	0	*< 0.71 mmol/L*
s-ferritin (n=64, f=54, m=10)	10 (16%)	*men <25, women <10 µmol/L*
s- vitamin. B12 (n= 63, f= 53, m= 10)	7 (11 %)	<140 pmol/L
s-folate (n= 64, f= 54, m= 10)	1 (2 %)	< 6 pmol/L
s-vitamin A (n= 62, f= 53 m= 9)	40 (66 %)	*< 1,44 µmol/L*
s-albumin (n= 64, f= 54, m= 10)	9 (14 %)	*< 40 g/L*
s-total protein (n= 64, f= 54, m= 10)	7 (11 %)	*< 64 g/L*
Markers of bone turnover		
s-PTH (n= 64, f= 55 m= 9)	60 (92 %)	*> 5,7 pmol/L*
s-1,25(OH)2vitD (n= 64, f= 54, m= 10)	16 (28 %)	*> 169 pmol/L*
Markers of bone resorption		
s-1CTP (n= 53, f= 44, m= 9)	24 (45 %)	*men >5,0 ug/L women >5,6 µg/L*
Markers of bone formation		
s-b-ALP (n= 54, f= 44, m= 10)	37 (69 %)	*men > 41 E/L women > 31 E/L*
s-Osteocalcin (n= 54, f= 44, m= 10)	1 (2 %)	*< 3,4 nmol/L*

Table: Number of cases and frequency of abnormal biochemical markers, (above or below reference values stated for each).

Mean 25(OH)D values for entire group was normal, but there was a wide range in these values and some patients had very low levels. Fourteen patients (22 %) had low vitamin D, while none had values above the normal range. The female group had slightly lower values compared to the male group. Serum levels of the active vitamin

D metabolite $1.25(OH)_2D_3$ was within normal range in 72 % of the study population, the remaining 28 % had increased levels, indicating increased bone turnover.

Forty patients (66%) had serum vitamin A levels below the reference range. Mean albumin corrected s-calcium was in the normal range, but 25 % presented with sub-normal levels. There was no significant difference between genders.

There was a high prevalence (92 %) of increased PTH. The female group had a slightly higher mean value compared to the male group. Only five patients (8 %) had normal s-PTH levels. One patient had a I-CTP level below normal, while twenty-four (44 %) had increased levels.

The majority (69 %) had increased serum bone-specific alkaline phosphatase. The remaining 31% had normal levels. S-bone specific ALP was higher in the male than in the female group. Mean s-osteocalcin was mostly within normal range and only two patients had abnormal levels. One patient had a value below normal and one patient had significantly increased levels.

3.4 Results Nutrient Intake

3.4.1 Dietary interview regarding supplements

Table 15 presents results on supplement intake as reported during consultations.

Table 15. Results of self-reported supplement intake

Self-reported supplement intake	no of patients (N=65)	study population, %
calcium supplementation[1]		
recommended dose or more taken (at least 2000mg calcium per day)	31	48
some calcium taken[2]	53	81
no calcium supplementation	11	17
high-dose vitamin D supplementation[3]		
recommended dose taken (750µg D_2 per day)	43	66
more than recommended dose taken	6	9
some high-dose vitamin D supplementation taken[4]	52	80
no high-dose Vitamin D supplementation	13	20
multivitamin with mineral supplementation[5]		
recommended dose or more taken (at least 2 tablets per day)	36	55
at least one tablet per day taken	55	85
no multivitamin & mineral supplementation	10	15
recommended dose of cod liver oil with vitamin A and D taken[6]	4	6
half of recommended dose cod liver oil taken	10	15
other omega 3 supplement, unregistered brand	5	8
intake of omega 3 supplement with B vitamins[7]		
recommended dose taken (two capsules per day)[8]	12	19
at least one capsule per day[9]	22	34
more than recommended dose taken	1	2
no supplements taken	7	11
other supplements than recommended (unspecified)	37	57

[1]Nycomed calcigran forte, one tablet contains 500 mg calcium carbonate and 10 µg cholecalciferol (vitamin D_3), produced by Nycomed Pharma AS. Dosage recommended to our patients was 2 × 2 tablets per day or equivalent calcium supplementation.
[2]At least 500 mg of calcium (one calcigran forte tablet or equivalent) taken per day.
[3]High-dose vitamin D supplement, AFI-D_2 forte from Nycomed Pharma, containing: Ergocalciferol (vitamin D_2) 0,75 mg equivalent to vitamin D 30 000 NE per tablet. Our patients were recommended taking one capsules per day.
[4]One or more tablets of AFID2 forte taken at least once per week
[5]Nycomed Nycoplus multivitamin and mineral supplement or equivalent. (One tablet Nycoplus Multi contains: vitamin A 500µg, vitamin D 5µg, vitamin E 10mg, B_1 1.4mg, B_2 1.6mg, B_3 18mg, B_6 2mg, B_5 6mg, B12 1µg, folic acid 200µg. vitamin C 60mg, zinc 15mg, copper 2mg, iodine 150µg, manganese 2.5mg, chromium 50µg, selenium 50µg, iron 14mg, magnesium 100mg) Our patients were recommended taking 1 × 2 tablets per day.
[6]Møllers tran or Møllers Dobbel: Cod liver oil either 5ml liquid or 2 capsules containing 10µg vitamin D, 250µg vitamin A and 10mg vitamin E (two capsules also contain 300mg EPA, 300mg DHA, 5 ml liquid contains 600mg DHA and 400mg EPA). These are standard doses which were also recommended to our patients.
[7]Omega 3 supplement (Møllers godt for hjertet) with 450mg of omega 3 (200mg EPA, 200mg DHA), 100µg folate, 1mg vitamin B6, 1µg vitamin B12 and 5mg vitamin E per capsule.
[8]Recommended dose was 2 capsules of the omega 3 supplement with B vitamins (møllers godt for hjertet) per day
[9] One or more capsules of the omega 3 with B vitamins taken per day

Most patients reported using supplements. The majority took calcium, vitamin D and a combined multivitamin & mineral supplement to some extent, but only twenty six patients (40 %) reported taking at least the recommended dose of all three of these supplements. Less than half reported taking at least the recommended dose of calcium per day, but 77 % reported taking 1000 mg of calcium or more each day. Forty three patients (66 %) complied with the recommended daily high-dose vitamin D supplementation of 750µg, while 5 % took more than recommended (they took two tablets which provided 1500 µg per day). Thirteen patients (20%) took no high-dose vitamin D supplement. Table 14 also presents content of vitamin and minerals in the

recommended multivitamin & mineral supplement, Nycoplus Multi. Approximately half of the study population (55%) took the recommended dose (two tablets) of this supplement, while fifty five patients (85%) took at least one tablet per day. Few patients took the recommended dose of cod liver oil with vitamin A, D and E, but ten patients took half the recommended dose. A large proportion (57 %) took other unspecified supplements in addition to and/ or instead of recommended supplementation. Two patients reported taking calcium citrate in addition to or instead of Calcigran forte. This intake was included in their estimated calcium intake. Seven patients (10.8 %) reported taking no form of supplementation at all.

Table 16 presents average intakes of calcium and high-dose vitamin D.

Table 16. Intake of calcium and vitamin D from supplements reported in structured interview (Calcigran forte or equivalent and AFI-D_2 forte).

Calcium and vitamin D intake from self-reported supplementation	**total N=65**	**women** n=55	**men** n=10
calcium, mg			
mean (SD)	1352 (850)	1396 (847)	1107 (866)
median (p25,p75)*	1500 (1000, 2000)	1800 (1000, 2000)	1000 (54, 2000)
min- max.	0 – 3000	0 – 3000	0 – 2000
vitamin D, μg			
mean (SD)	635 (424)	655 (410)	525 (506)
median (p25,p75)*	750 (375, 750	750 (750, 750)	(0, 750)
min- max.	0 – 1500	0 – 1500	0 – 1500

*p25 and p75 represents the 25th and 75th percentile respectively

The female group had slightly higher mean calcium and vitamin D intakes than the male group. Median vitamin D intake however, was similar in both groups.

3.4.2 Dietary Records

We obtained dietary records from only twenty-nine patients. Hence we present data on food and nutrient intake from less than half (45 %) of the patients. Table 17 presents macronutrient intakes from dietary records, excluding supplements.

Table 17. Average daily macronutrient intake from dietary registrations.

Macronutrient intake from food & beverages	**tot group** n=29	**women** n=25
energy, kJ		
mean (SD)	10914 (3705)	10675 (3597)
median (p25,p75)*	10408 (8485, 12709)	10408 (8485, 12346)
min- max.	4577 – 23629	
protein,, g		
mean (SD)	105 (31)	103 (32)
median (p25,p75)*	105 (84, 122)	101 (81, 122)
min- max.	30 – 168	30 – 168
protein, %		
mean (SD)	16.7 (3.3)	16.7 (3.3)
median (p25,p75)*	16.7 (14.4, 18.5)	16.7 (14.5, 18.1)
min- max.	108- 235	11.0 – 25.1
fat, g		
mean (SD)	117 (49)	116 (51)
median (p25,p75)*	109 (83, 157)	109 (83, 145)
min- max.	26 – 296	26 – 296
fat, %		
mean (SD)	38.9 (6.2)	39.0 (6.6)
Median (p25,p75)*	40.0 (36.4, 43.5)	40.0 (35.4, 43.9)
min- max.	20.9 – 48.5	20.9 – 48.5
SFA, %		
mean (SD)	16.3 (2.5)	16.3 (2.6)
Median (p25,p75)*	16.7 (14.5, 18.3)	16.7 (14.5, 18.3)
min- max.	9.2 – 20.6	9.2 – 20.6
MUFA, %		
mean (SD)	12.8 (2.3)	12.9 (2.4)
median (p25, p75)*	12.7 (11.6, 13.8)	13.4 (11.7, 14.1)
min- max.	6.6 – 17.6	6.6 – 17.6
PUFA, %		
mean (SD)	7.0 (2.2)	7.0 (2.3)
median (p25, p75)*	7.1 (5.3, 9.1)	7.1 (5.0, 9.2)
min- max.	3.3 – 11.2	3.3 – 11.2
CHO, g		
mean (SD)	264 (95)	275 (91)
median (p25, p75)*	247 (194, 271)	262 (209, 288)
min – max	142 – 550	168 – 586
CHO, %		
mean (SD)	43.1 (7.6)	43.2 (8.0)
median (p25, p75)*	42.6 (38.8, 46.6)	41.7 (38.8, 46.2)
min- max.	29.2 – 68.0	29.2 – 68.0
fibre, g		
mean (SD)	18.3 (6.0)	17.9 (5.8)
median (p25, p75)*	18.0 (14.4, 21.6)	17.8 (14.4, 21.6)
min – max	8.0 – 35.9	8.0 – 35.9
sugar, %		
mean (SD)	10.0 (7.5)	10.1 (7.8)
median (p25, p75)*	7.8 (5.8, 12.2)	7.9 (5.8, 12.2)
min- max.	1.8 – 38.1	1.8 – 38.1

*p25 and p75 represents the 25th and 75th percentile respectively

Results show great variations in energy intake. Mean energy intake was high. The large majority however had an average daily energy intake between 7000 and 14000 kJ per day. One patient had an energy intake which was over 6400 kJ higher than any

of the others at 23629 kJ per day. Energy contribution from fatty acids was particularly high with a saturated fat intake contributing to 16.3 % of energy intake. One quarter of CHO intake was provided from added sugar which contributed to 10 % of energy intake.

Table 18 presents data on micronutrient intake from 3+4 day dietary registrations, (excluding supplements).

Table 18. Average daily micronutrient intake.

Nutrient intake from food and beverages	**tot group** n=29	**women** n=25
vitamin D, µg		
mean (SD)	5.5 (4.3)	5.7 (4.6)
median (p25, p75)*	4.5 (3.0, 6.6)	4.5 (2.8, 7.2)
min – max	0.65 – 18.8	0.64 – 18.81
vitamin A, RAE		
mean (SD)	1243.4 (624.7)	1263.2 (661.3)
median (p25, p75)*	1048.0 (872.0, 1599.0)	1048.0 (888.5, 1645.0)
min – max	267.0 – 3714.0	267.0, 3714.0
calcium, mg		
mean (SD)	1213.4 (464.0)	1189.1 (476.7)
median (p25, p75)*	1162.0 (778.5, 1559.0)	1158.0 (742.0, 1559.0)
min – max	406.0 – 2191.0	406.0 – 2191.0
magnesium, mg		
mean (SD)	341.8 (92.8)	337.8 (94.5)
median (p25, p75)*	346.0 (280.5, 383.0)	345.0 (280.5, 383.0)
min – max	123.0 – 590.0	123.0 – 590.0

*p25 and p75 represents the 25th and 75th percentile respectively

Average micronutrient intake from food and beverages mostly met the Norwegian governments' (shdir) Recommended Dietary Intake (RDI), apart from vitamin D , which was slightly lower than these recommendations. Micronutrient intake also varied greatly between individuals.

Table 19 presents supplement contribution of average vitamin A, vitamin D, calcium and magnesium intake as recorded in 7 day dietary registration. Of the 29 diaries returned, none answered all the questions about supplement intake, hence (n) for each micronutrient is stated. Table includes both genders.

Table 19. Average daily supplement contribution to vitamin A, vitamin D, calcium and magnesium intake from dietary registrations.

Nutrient intake from recorded supplements	mean (SD)	median (p25, p75)	min-max
vitamin D, µg (n= 22)	673 (589)	719 (57, 860)	10-2189
calcium, mg (n = 21)	1537 (789)	1857 (1000, 2000)	0- 2857
magnesium, mg (n= 21)	149 (46)	157 (100, 200)	86- 206
vitamin A, RAE* (n= 22)	951 (569)	840 (643, 1144)	0- 3025

* 1 RAE = 1 µg retinol or 12 µg β-carotene

Supplements were the main source of vitamin D, vitamin A, calcium and magnesium.

Table 20 presents contribution of food groups to nutrients, excluding that from supplement intake, from dietary registrations.

Table 20. Average daily nutrient contribution from food and beverages.

Percent contribution of food groups to nutrients (n=29)												
	energy %	**protein %**	**fat %**	**SFA %**	**MUFA %**	**PUFA %**	**CHO %**	**sugar %**	**fibre %**	**vitamin A**	**vitamin D**	**Ca**	**Mg**
Bread	12	9	3	1	2	7	23	-	34	-	-	2	18
Other cereal products	6	7	4	5	3	4	8	1	9	4	-	8	6
Cakes	5	2	6	6	5	7	6	10	4	4	5	2	3
Potatoes	2	1	-	-	-	-	4	-	7	-	-	-	4
Vegetables	2	2	-	-	-	1	3	1	14	12	-	2	5
Fruit & berries	6	1	-	-	-	1	13	7	18	1	-	3	8
Meat, blood & offal	13	28	18	17	24	9	2	-	-	28	-	3	9
Fish & shellfish	3	8	3	1	3	4	1	1	-	1	32	1	4
Egg	2	4	3	2	4	2	-	-	-	4	9	1	1
Milk, cream products etc.	10	12	8	12	7	1	10	10	-	8	9	32	10
Cheese	7	13	11	16	10	2	-	-	-	11	1	30	4
Butter, margarine, oils	11	1	27	22	21	51	1	1	-	21	42	-	-
Sugar & sweets	9	3	7	8	8	4	13	35	1	1	-	4	8
Drinks	8	3	3	4	2	-	11	33	-	3	1	8	11
Nuts and seeds	2	2	3	1	5	6	1	-	3	-	-	-	4
Miscellaneous	3	2	3	3	4	2	4	1	10	1	-	2	4

Bread and bread products contributed most to energy intake, but meat and meat products were also important energy sources. Other foods frequently consumed included dietary fats such as oil, butter, margarine and mayonnaise based sauces, milk products, sweets and chocolate. Popular spreads were cheese, cottage cheese and meat spreads such as liver pate, ham and salami. Fruit and vegetable intake varied between individuals, but generally intake was low.

Dairy products including milk, cheese and cream based products contributed the most to calcium intake and were important sources of magnesium. Bread was, however, the main food source of magnesium. Important sources of vitamin A were meat and meat products, dietary fats, and, to some extent, cheese. Dietary fat, in particular margarine, was the main source of vitamin D followed closely by fish and fish products of the more fatty varieties.

3.5 Relationships between nutrients, biochemical measurements and BMD measurements

3.5.1 Nutrient intake, nutrient status and bone health

No significant relationship was found between nutrient intake from dietary recordings and bone mineral density or markers of bone turnover (data not shown). Calcium and vitamin D status markers were not associated with BMD or bone turnover (data not shown). Compliance to supplement intake did not correlate with any indicators of bone health (data not shown).

3.5.2 BMI reduction

Table 21 presents results from t-tests testing differences between patients with a BMI reduction of less than the median (=<20.1 kg/m^2) and those with a reduction above the median (>20.1 kg/m^2).

Table 21. Comparison of patients with BMI reduction above or below median.

BMI reduction	mean	BMI reduction 7.8 - 20.1 kg/m^2	BMI reduction 20.2-35.5 kg/m^2	p-value	mean difference
Gender (N=65, f=55, m=10)		f=26, m=6	f=29,m=4	0.533	-0.057
Age, years (N=65)	39.65	41.97	37.39	0.035	-4.473
BMI pre-surgery, kg/m^2 (N=65)	51.06	45.58	56.37	0.000	10.605
common channel, cm (n=62)	71.21	76.77	65.65	0.011	-10.740
Waist at follow up, cm (N=65)	95.95	95.88	96.03	0.859	-0.525
s-vit.B12, pmol/L (n=63)	233.24	206.55	259.09	0.042	52.243
I-CTP, ug,L (n=54)	5.48	5.04	5.86	0.063	0.902
TBBD, z-score (n=57)	-0.31	-0.796	0.094	0.017	0.814
AP-spine, z-score (n=57)	-0.74	-1.112	-0.435	0.153	0.585
Dual femur, z-score (n=56)	-0.38	-0.764	-0.071	0.026	0.633

Variables related to weight and weight change, were significantly related to bone mineral density, and BMI reduction was significantly correlated to BMD. Those who had lost less weight were older, they had a lower initial BMI and a longer common channel. They also tended to have lower I-CTP. In relation to BMD those who lost the most weight had higher BMD z-scores for total body and dual femur but not for AP spine.

3.5.3 Bone mineral density

Table 22 compares patients with a total body bone density (TBBD) z-score equal to or above and below -1.0 SD.

Table 22. Comparison of patients with normal or abnormal total body bone density z-scores.

TBBD z-score above or below -1.0 SD	mean	mean <-1.0 SD	mean =>-1.0 SD	p-value	mean difference	95% confidence interval	
Gender		f=12, m=5	f=36, m=4	0.068	-0.194	-0.403	0.015
Age, years	39.7 (N=65)	43.4 (n=17)	37.1 (n=40)	0.007	-6.337	-10.829	-1.845
BMI before surgery, kg/m^2	51.1 (N=65)	48.6 (n=17)	52.8 (n=40)	0.084	4.223	-0.587	9.034
Waist at follow up, cm	96 (N=65)	101 (n=17)	95 (n=40)	0.070	-6.393	-13.330	0.545
Common channel, cm	71 (n=62)	72 (n=16)	69 (n=38)	0.539	-2.977	-12.639	6.685
BMI reduction, kg/m^2	21.2 (N=65)	18.2 (n=17)	22.9 (n=40)	0.023	4.726	0.679	8.773
months since surgery	36.5 (N=65)	40.6 (n=17)	35.2 (n=40)	0.031	-5.438	-10.359	-0.518

Patients with a z-score below -1.0 SD were significantly older compared to those with a TBBD above -1.0 SD. They also tended to have a higher BMI pre-surgery however this was not statistically significant. Patients with a higher TBBD had a greater

reduction in BMI (mean reduction was 23 kg/m^2), than those with a low TBBD (mean reduction 19.6 kg/m^2). Again this difference was not statistically significant.

We looked carefully at patients with a TBBD z-score below -2.5 (n=5) they had significantly longer time past since surgery (p= 0.000). Average time since surgery for those with a TBBD z-score above -2.5 SD was 35.9 (8.7 SD) months while it was 45.8 (2.6 SD) months for those below. Waist circumference was significantly higher for those with a z-score below -2.5 SD group, with a mean of 107 cm compared to a mean waist circumeference of 95.6 cm for those with a z-score above -2.5. These differences were not significant between those with a z-score above or below -1.0 SD.

Table 23. Correlation analysis of total body bone density z-scores with patient characteristics

Total body bone density	**tot group** (n=57)		**women** (n=48)		**Men** (n=9)	
	coefficients	p-value	Coefficients	p-value	coefficients	p-value
age, years	- 0.255	0.056	- 0.176	0.232	- 0.634	0.066
months since surgery	- 0.294	0.026	- 0.205	0.161	- 0.184	0.635
BMI reduction, kg/m^2	0.341	0.009	0.291	0.045	0.611	0.081
waist circumference, cm	- 0.507	0.000	- 0.421	0.003	- 0.703	0.035

Variables related to weight and weight change, related most significantly to bone mineral density. BMI reduction and BMI at follow-up were significantly correlated to TBBD.

Table 24 presents patient characterstics correlated with lumbar spine bone density.

Table 24. Relationship between lumbar spine bone density and patients characterstics

Lumbar spine bone densityL1-L4 z-score	**tot group** (n=57)		**women** (n=48)		**men** (n=9)	
	coefficients	p-value	coefficients	p-value	coefficients	p-value
age, years	- 0.006	0.963	0.021	0.886	-0.577	0.104
BMI reduction, kg/m^2	0.191	0.154	0.120	0.417	0.350	0.356
waist circuference, cm	- 0.357	0.006	0.002	0.991	-0.650	0.058

Table 25 presents patient characteristics correlated with dual femur bone density z-scores.

Table 25. Relationship between dual femur z-scores and patient characteristics.

Dual femur bone density	**tot group** (n=56)		**women**, (n=48)		**men**, (n=8)	
	coefficients	p-value	coefficients	p-value	coefficients	p-value
age, years	- 0.233	0.084	-0.085	0.564	-0.577	0.104
BMI reduction, kg/m^2	0.418	0.001	0.377	0.008	0.350	0.356
waist circumference, cm	- 0.308	0.021	-0.249	0.088	-0.650	0.058

Table 25 presents patient characteristics correlated with serum I-CTP.

Table 25. Relationship between serum I-CTP and patient characteristics

Correlation analysis of C-terminal telopeptide of type I collagen	**tot group**		**women**		**men**	
	coefficients	p-value	coefficients	p-value	coefficients	p-value
Age, years (n=54, f=48, m=6)	- 0.389	0.004	0.102	0.492	-0.890	0.001
BMI reduction, kg/m^2 (n=54, f=48, m=6)	0.302	0.027	0.143	0.334	-0.049	0.894
s-calcium alb corr (n=53, f=47, m=6)	0.452	0.001	0.164	0.272	0.431	0.214
s-albumin (n=53, f=47, m=6)	- 0.353	0.010	0.120	0.423	0.360	0.307

3.5.4 Regression analysis of BMD z-scores and I-CTP

The most important dependant variables including BMD and I-CTP were studied further to understand which of the clinical characteristics were the most important and statistically significant correlates. With a small population of about 50 individuals only about 4-5 characteristics could be included in the regression analyses.

Table 27, 28 and 29 show multiple regressions of the independent variable TBBD and the dependent variables BMI reduction and BMI pre-surgery controlling for age and common channel.

Table 27. Regression analysis of total body bone density z-scores

Multiple regression of total body bone density (TBBD)				
n=54	coefficient	p-value	95% confidence interval	
TBBD, z-score	-0,264	0,825	-2,657	2,129
age, years	-0,027	0,252	-0,075	0,020
BMI reduction, kg/m^2	0,049	0,046	0,001	0,096
common channel, cm	0,000	0,972	-0,023	0,023

Table 28. Regression analysis of total body bone density z-scores

Multiple regression for total body bone density (TBBD)				
n=54	coefficient	p-value	95% confidence interval	
TBBD, z-score	-0.362	0.826	-3.653	2.929
age, years	-0.028	0.257	-0.077	0.021
BMI pre-surgery, kg/m^2	0.024	0.246	-0.017	0.066
common of channel, cm	-0.001	0.934	-0.025	0.023

Table 29. Regression analysis of total body bone density z-scores

Multiple regression of total body bone density (TBBD)				
n=54	coefficient	p-value	95% confidence interval	
TBBD, z-score	0,795	0,645	-2,653	4,243
Age, years	-0,030	0,215	-0,078	0,018
BMI reduction, kg/m^2	0,078	0,068	-0,006	0,163
common channel, cm	0,000	0,978	-0,023	0,023
BMI pre-surgery, kg/m^2	-0,031	0,394	-0,103	0,041

Total body bone density was positively correlated with BMI reduction controlled for age and common channel. This was not the case for BMI pre-surgery, but BMI reduction was not significantly correlated with TBBD when controlled for this variable.

Table 30, 31 and 32 show multiple regressions of the independent variable lumbar spine bone density and the dependent variables BMI reduction and BMI pre-surgery controlling for age and common channel.

Table 30. Regression analysis of lumbar spine z-scores

Multiple regression of lumbar spine L1-L4 (AP spine)				
n=54	coefficient	p-value	95% confidence interval	
AP spine, z-score	-2,137	0,151	-5,080	0,806
age, years	0,011	0,699	-0,047	0,070
BMI reduction, kg/m^2	0,041	0,166	-0,018	0,100
common channel, cm	0,002	0,895	-0,026	0,030

Table 31. Regression analysis of lumbar spine z-scores

Multiple regression for lumbar spine bone density (AP-spine)				
n=54	Coefficient	p-value	95% confidence interval	
AP-spine, z-score	-2.435	0.225	-6.417	1.547
age, years	0.011	0.701	-0.048	0.071
BMI pre-surgery, kg/m^2	0.024	0.345	-0.027	0.075
common channel, cm	0.001	0.949	-0.028	0.029

Table 32. Regression analysis of lumbar spine z-scores

Multiple regression of lumbar spine L1-L4 (AP spine)				
n=54	coefficient	p-value	95% confidence interval	
AP spine, z-score	-1,611	0,452	-5,878	2,657
agecons, years	0,010	0,736	-0,050	0,070
BMI reduction, kg/m^2	0,056	0,289	-0,049	0,161
common channel, cm	0,002	0,898	-0,027	0,030
BMI pre-surgery, kg/m^2	-0,015	0,731	-0,105	0,074

Lumbar spine bone density was not correlated with changes in BMI or BMI pre-surgery when controlling for age and common channel.

Table 33, 34 and 35 show multiple regressions of the independent variable dual femur bone density and the dependent variables BMI reduction and BMI pre-surgery controlling for age and common channel.

Table 33. Regression analysis of dual femur z-scores

Multible regression of dual femur bone density z-scores				
n=53	coefficient	p-value	95% confidence interval	
dual femur, z-score	-0,750	0,477	-2,853	1,353
age, years	-0,021	0,306	-0,062	0,020
BMI reduction, kg/m^2	0,055	0,011	0,013	0,096
common channel, cm	0,000	0,969	-0,019	0,020

Table 34. Regression analysis of dual femur z-scores

Multiple regression for Dual femur bone density				
n=53	Coefficient	p-value	95% confidence interval	
dual femur, z-score	-1.818	0.205	-4.659	1.024
age, years	-0.019	0.358	-0.061	0.022
BMI pre-surgery, kg/m^2	0.043	0.019	0.007	0.079
common channel, cm	0.000	0.982	-0.020	0.019

Table 35. Regression analysis of dual femur z-scores

Multible regression of dual femur bone density z-scores				
n=53	coefficient	p-value	95% confidence interval	
dual femur, z-score	-1,285	0,393	-4,283	1,712
age, years	-0,020	0,346	-0,061	0,022
BMI reduction, kg/m^2	0,040	0,275	-0,033	0,112
common channel, cm	0,000	0,965	-0,019	0,020
BMI pre-surgery, kg/m^2	0,016	0,614	-0,046	0,077

Dual femur bone density was positively correlated with reduction in BMI and BMI pre-surgery when controlled for age, and common channel length, but not when controlled for each other.

Table 36, 37 and 38 show multiple regressions of the independent variable I-CTP and the dependent variables BMI reduction and BMI pre-surgery controlling for age and common channel.

Table 36. Regression analysis of total serum I-CTP

Multiple regression of serum-I-CTP				
n=51	coefficients	p-value	95% confidence interval	
I-CTP, ug/L	7,628	0,001	3,435	11,821
age, years	-0,072	0,017	-0,131	-0,013
BMI reduction, kg/m^2	0,087	0,006	0,026	0,147
common channel, cm	-0,017	0,467	-0,062	0,029

Table 37. Regression analysis of total serum I-CTP

Mulitple regression for serum-I-CTP				
n=51	coefficient	p-value	95% confidence interval	
I-CTP, ug/L	7.355	0.005	2.288	12.423
age, years	-0.071	0.026	-0.134	-0.009
BMI pre-surgery, kg/m^2	0.051	0.071	-0.005	0.107
common channel, cm	-0.024	0.305	-0.072	0.023

Table 38. Regression analysis of total serum I-CTP

Multiple regression of serum-I-CTP				
n=51	coefficients	p-value	95% confidence interval	
I-CTP, ug/L	9,098	0,001	4,046	14,151
age, years	-0,077	0,012	-0,137	-0,018
BMI reduction, kg/m^2	0,139	0,021	0,022	0,256
common channel, cm	-0,011	0,633	-0,058	0,035
BMI before surgery, kg/m^2	-0,054	0,300	-0,157	0,049

Serum levels of I-CTP at > 2 years post surgery had a strong negative correlation with age. Those who had I-CTP levels above reverence range were younger with a mean age of 36 years compared to 42 years in the group with normal values. This relationship was significant when controlled for all the other variables. In addition I-CTP levels were positively correlated with BMI reduction, but not BMI pre-surgery.

4. Discussion

This study confirm previous findings that there is a high incidence of increased bone turnover and fat soluble vitamin deficiency (vitamin A and D) after BPD-DS.[1] No significant relationship between nutrient status and length of common channel was detected. Markers of bone metabolism were related to the degree of weight loss. Average bone mineral density was within normal range compared to the reference population, but 25-32 % had decreased levels and men had significantly lower BMD than women ($p=0.02$). Patients with low BMD tended to be older, have lower BMI pre-surgery and had a lower weight reduction. BMI reduction had the strongest correlation to BMD results. There was no relationship between nutrient intake and biochemical measurements or BMD.

4.1 Patient characterstics

The purpose of the study was to investigate patients 2 - 5 years after BPD-DS surgery to gain insight into their nutrient and bone health status after they had returned to a stable metabolic state. Participation rate was 84 %, which included all patients who showed up for follow-up consultations a part from one. Patients did not attend consultations for several reasons. Some had too far to travel and/ or psychological problems, some had been transferred to other follow-up programs, some stated that they did not wish to have follow-up treatment and others did not respond to repeated attempts of being contacted. One patient was excluded due to incomplete and suspected inaccurate measurements in several study parameters. He was also admitted for correctional surgery (to lengthen his common channel) during the post-surgery follow-up assessments. It is likely that this patient was in an unstable metabolic state, and that his test results do not resemble that of a more metabolically stabilized > 2 years post surgery patient. All these factors lead to the decision to exclude all his data.

Gender distribution

Our study population included 85 % women and 15 % men. The uneven distribution between genders may be representative to the general bariatric patient population. One university hospital in Norway which performs such procedures informed us that 60-70% of referred patients were women. [56] However findings related to differences between gender requires further investigation before any conclusion can be met.

Control group

No other group had undergone similar metabolic change and therefore we did not include a control group in our study. It could have been relevant to compare data from a morbid obese population which had not undergone surgery to determine whether BPD-DS is more detrimental to bone health than remaining morbidly obese. Although studies show increased incidence of vitamin D deficiency and increased serum PTH levels, data on BMD in the morbidly obese population before bariatric surgery is limited.[57, 58]

4.1.1 Results anthropometric characteristics

All patients had achieved satisfactory weight loss, after > 2 years post- surgery, with at least 50 % of excess weight loss maintained at follow-up. The study population had a mean loss of 62 kg and 76 % of excess weight. Hence the average patient was no longer classified as obese 2-5 years post-surgery. According to the WHO classification, these results meant that our population sample significantly reduced obesity associated health risk. Some patients achieved sufficient weight loss to achieve a normal weight (BMI 18.5-25), while some patients sustained a BMI in the morbidly obese category. One patient had a BMI above 45 at follow-up consultations, however with an initial BMI above 70, the weight loss and subsequent health benefits were invaluable.

Height measurements

Results from pre-surgery height measurements were used to determine changes in BMI. Although the changes in height were not significant (p=0.703), some patients were several centimetres shorter at follow-up, which would have underestimated their weight loss had the new height measurements been used in BMI calculations.

Mean and median values

We presented mean and median values because there was an uneven distribution of several study parameters. The large majority of this patient group weighed between 110 and 170 kg pre surgery, but one weighed 215 kg, while one patient weighed only 108 kg. The low value did not have the same effect on the distribution, as her weight did not range as far from the majority of the patient group as the extreme high value.

4.2 Bone mineral density (BMD)

DXA measurements were obtained from 88 % of the study population. Average bone mineral density was within normal range for the study population with a mean TBBD z-score of -0.31, but 25-32 % had decreased levels (less than -1.0 SD of reference population). (Only 7-12 % had z-scores above 1.0 SD). Mean values for the whole group indicated normal BMD for all three z-scores. However there was a wide range and only TBBD 68 % had normal values. The male group had much lower total body and AP spine bone density than the women, with a mean BMD below normal at -1.23 and -1.64 SD for the two values respectively. These results indicated that BPD-DS has some detrimental effect on bone mineral density, although it is uncertain how much is attributable to BPD-DS and how much is a result of disturbances seen in morbid obesity. Further bone mineral density studies later than 5 years post-surgery may be necessary to determine whether BPD-DS decreases bone mineral density.

Case

The lowest TBBD was measured in the male group. This man had a z-score of -3.3 and a T-score of -2.7 SD which could qualify him, according to WHO diagnostic criteria, as having severe osteoporosis at the age of 59 years. Mean difference in z-score between gender was not significant in any of the parameters measured, most likely due to the male group being much smaller than the female group (n=9 vs n=49 respectively).

4.2.1 Z-score vs t-score

BMD z-score was used as opposed to t-score results because the purpose was to investigate BPD-DS effect on bone mineral density and it was considered more relevant to compare results with individuals of equal age and gender. Guidelines also state that z-score should be used when evaluating DXA measurements in pre-menopausal patients, although fewer patients are diagnosed with low bone mineral density (osteopenia) when z-scores are used. [59] It would have been of great value if patients had measured BMD before surgery. This would have made it possible to gain some insight into how much bone mineral loss is attributable to BPD-DS as opposed to their previous condition as morbidly obese.

Menopause

Menopause was not taken into account in this study and may have been relevant because most patients were in their forties and fifties (when menopause often presents). The perimenapausal stage (the first five to eight years after menopause initiates) is associated with particular increase in bone turnover and bone loss, a result of the large impact estrogen withdrawal has on bone. [43] This could have been an important confunding factor and should have been considered.

4.3 Biochemical measurements

4.3.1 Nutrient status

The majority of our patients had normal or low serum values of all nutrients despite high doses of supplements. Corrected calcium and 25(OH)D levels were decreased in 25 and 22 % respectively. The highest incidence of nutrient deficiency was vitamin A, which was low in 66 %. Vitamin A supplementation was recommended through a multivitamin and mineral supplement as well as from cod liver oil with vitamin A, D and E. Considering the high incidence of vitamin A deficiency and low compliance in taking the latter supplement, this may indicate that alternative vitamin A supplementation is necessary. However we found no correlation between supplement compliance and any of the markers of nutrient status which may indicate that the differences in level of malabsorption, is the most important determining factor for nutrient status. If this is true, a possible solution to the high incidence of vitamin D deficiency could be to recommend increased sun exposure to ensure that vitamin D needs are met.

4.3.2 Markers of bone metabolism

Biochemical markers of bone turnover were significantly increased in a large proportion of the study population. Increased incidence of secondary hyperparathyroidism and vitamin D deficiency have also been established in the morbidly obese popualtion.[12] The relative contribution of morbid obesity and BPD-DS surgery to these disturbances has yet to be determined.

Balsa et al stated that approximately 70% of patients who undergo biliopancreatic diversion develop secondary hyperparathyroidism in the long term. A particularly high incidence of increased serum-PTH levels in our study confirmed that which has been shown in previous studies. [25, 35] A total of 92 % had increased serum-PTH. Other markers of increased bone turnover, such as I-CTP was raised in 45 %, and 28 % had

increased active vitamin D levels (calcitriol). Bone specific-ALP, a marker of bone formation, was also raised in 69 %, but osteocalcin was normal in all but one patient. The high incidence of increased bone turnover and resorption markers was an indication of abnormal rates of bone resorption. These results confirm previous findings that there is a high incidence of increased bone turnover after BPD-DS. [28, 60]

Considering that BMD results appeared to be less affected by the apparent increased bone turnover than expected, further study is necessary to determine whether it will affect BMD in the longer term. There is a lot of uncertainty around the long term effects of increased PTH and bone turnover after bariatric surgery. Studies persistently show a high incidence of secondary hyperparathyroidism, but long term data has not yet shown expected effects on BMD. The morbid obese population have decreased vitamin D and increased PTH levels, a result of increased uptake and clearance of 25(OH)D by adipose tissue. [29] One question is whether the increased food intake that happens over time after BPD-DS combined with less adiposity and subsequent increased bioavailability of vitamin D, will moderate PTH levels later on and prevent long term effects on BMD.[60]

4.4 Dietary intake

4.4.1 Dietary records

Great difficulty was experienced in having the 7 day pre-coded food diaries returned. A prospective pre-coded food diary which was quite easy, but required a little time each day to complete, was chosen in order to obtain as accurate information about nutrient intake as possible. This was considered necessary to detect any relationship between dietary habits with bone health parameters, but seven days of filling out this diary proved to be too demanding for the majority of our patients. This was despite thorough explanation, both written and verbal, about the food diary and providing patients with an addressed, pre-paid envelope in which to return the diary. As a result,

dietary records were obtained from 45 % of the study population. No patient expressed hesitation when they were asked to complete this assessment.

Considering that there was a wide variety in patient compliance, data on less than half of the study population may, for example, not include those who were less compliant to dietary recommendations, which could have resulted in a misrepresentation of dietary habits.

Alternative methods of dietary analysis

In retrospect, we could have obtained dietary information from more cases if we had used a less demanding dietary analysis tool. A food frequency questionnaire filled out on the day of consultation, perhaps together with a student clinical nutritionist, might have obtained the best response. However patients were asked to fill out a number of forms on the consultation day and therefore this may not have been the case. Another alternative would have been to conduct several 24-hour recall interviews over the telephone. These methods however would have relied on memory, and estimation of nutrient intake would probably not have been as accurate. Short food questionnaires are not able to provide data on individual nutrients, and are better use in estimating food choice such as dairy products and vegetables etc. [61] For the purpose of detailed dietary analysis in the future, the same pre-coded food diary could be used, but the amount of days expected to record intake could be limited to four days.

Limitations in assessing nutrient intake.

Other studies have shown that there are many weaknesses in available methods for dietary analysis. Filling out a dietary record may in it self affect intake, as patients tend to become more aware of what they eat when they have to write it down. Underreporting is common in all methods of dietary analysis, especially in the obese population. [62] Diet records suffer from errors in estimating portion size, from variability in food composition and from inadequacies of existing food composition tables. In addition, a snap shot of one week can not represent all intake. [61]

In this study patients recorded dietary intake after consultations with a student clinical nutritionist where they had received thorough dietary assessment followed by appropriate guidelines and recommendations for future intake. This may have significantly affected the recordings and must also be considered an important weakness in the dietary assessments. In contrast, results on mean intake of calcium and vitamin D from dietary registrations of supplement intake showed small differences when compared to those from the subjective interview. This may indicate a greater accuracy than anticipated within these results.

Nutrient deficiency and compliance to dietary recommendations.

There was a high incidence of nutrient deficiency and non-compliance to diet and post-surgery supplement recommendations in our patient group, but this varied greatly between individuals. Negative experiences in the initial post-operative period may influence compliance to supplement intake in the long term. General nutrient status and adverse reactions to foods may also determine a patients' willingness to comply. It may be beneficial to start supplementation in the pre-operative period. This could prepare patients for the use of supplements under normal conditions without the bowel discomforts and nausea often experienced in the early post-operative period. This could also correct any pre-surgery nutrient deficiencies and may help determine if the patient is in fact willing to comply with supplement recommendations post-surgery. Williams et al recommended starting a multivitamin with minerals and a calcium with vitamin D supplement from the first preoperative visit.[28]

Energy and macronutrient intake

Results show great variations in energy intake and one patient had an average energy intake which was over 6400 kJ higher than any of the others (23629 kJ per day). This particular case should have been investigated further. The extreme recorded energy intake may have been wrong due to a misunderstanding during dietary recording, but it may also have been accurate and a result of severe malabsorption, but this was never clarified.

Energy contribution from fatty acids was particularly high with a saturated fat intake contributing to 16.3 % of energy intake. Fat intake was well above the governmental recommendations and may have been an important contributing factor to the extreme frequency of stéatorrhea frequently experienced in this patient group. Some reported up to 20 toilet visits per day.

Important dietary recommendations in BPD-DS patients include ensuring a high intake of good quality protein. Our data illustrate an average protein intake of 16.7%, which is adequate for the general population, but is probably insufficient after BPD-DS because of the need to compensate for malabsorption. Carbohydrate intake was lower than that advised for the general population, but expected for our patient group to allow for more protein rich foods. It should be noted however, that one quarter of CHO intake was provided from added sugar, and even though this contributed no more than recommended limits for the general population it may indicate a high intake of CHO sources low in micronutrients. This combined with a high saturated fat intake may give cause for concern about the quality of the diet in this patient group, in terms of providing adequate amounts of essential nutrients.

Malabsorption

Fat malabsorption may lead to insufficient uptake of essential fatty acids and fat soluble vitamin deficiencies. This may have severe clinical consequences and can alter calcium metabolism. Previous studies have shown a progressive increase in the incidence and severity of hypovitaminemia A, D and K with time after BPD-DS. [15, 63] This may affect calcium metabolism and increase the incidence of secondary hyperparathyroidism.[10] This should also be considered in relation to the uncertain effectiveness of absorption from supplements.

Micronutrient intake

The dietary analysis software KBS, did not include functions for analysing folate, B_{12} and vitamin K, so dietary intake of these nutrients could not be estimated. Average intake of the other micronutrients mostly met the Norwegian governments (Sosial- og

helsedirektoratet) Recommended Dietary Intake (RDI)[64], apart from vitamin D intake which was only 6 µg per day on average. Considering the large malabsorption characterised in this patient group however, the net absorbed micronutrients from food intake is likely to be insufficient. Micronutrient intake also varied greatly between individuals, which again illustrates the profound variation in the quality of our patients' diets.

Food contribution to nutrient intake

Several patients had become intolerant to dairy products after the BPD-DS procedure and this has also been shown elsewhere [14]. Dairy products were, however, were still the most important source of calcium, and patients often reported using these products despite symptoms of lactose intolerance. Fruit and vegetables appeared to contribute less to Mg and vitamin A (in the form of β-carotene) than could be expected, illustrating a low intake of these food groups.

Important sources of vitamin A, such as meat and meat products and dietary fats, were frequently consumed. According to biochemical measurements this did not ensure adequate supply for a large proportion of our patients.

The Framingham heart study found that high intakes of fruit, vegetables, magnesium and potassium had a greater BMD in hip and forearm.[65] The protective effects of fruit and vegetables on bone health, mediated through vitamin C and other antioxidants, their content of vitamin K, magnesium, potassium, zinc and fibre as well as their alkaline ash may also be important reasons to ensure adequate intakes in BPD-DS patients. [43] A high intake of this food group will also be beneficial for the maintenance of long-term weight loss.

Supplement intake

Several studies have emphasized the risk of metabolic bone disease in bariatric surgery patients if adequate calcium and vitamin D supplementation is not given. [34, 36, 66]

Most patients took some kind of supplement. Reasons for not taking supplements included difficulties in swallowing the tablets, feeling of nausea or general stomach upsets associated with intake. Many struggled with taking the large chewable calcium supplements. In addition, iron supplements were often associated with cramps and disturbed bowel habits. Some patients took B_{12} injections regularly, but most had normal serum values despite only obtaining this vitamin from dietary sources and the multivitamin- and mineral supplement.

It was difficult to obtain accurate information about the content of multivitamins and iron tablets due to great variations in brand choice, from known and unknown companies. Dietary registrations often did not disclose type of brand and / or number of tablets tablets taken per day. Some stated that they used some supplements from abroad but could not recall brand name or content. Nycomed Pharma, Nycoplus Multi was assumed brand were no other information was given.

Studies evaluating the effectiveness of aggressive high-dose vitamin D supplementation in preventing secondary hyperparathyroidism in this patient group are lacking. Good compliance in high-dose vitamin D supplementation resulted in 77% having vitamin D intake above the safe upper limit in our patient group. Despite this fact, no patient presented with high values and 22 % still had low serum levels, and other studies have found a higher incidence of vitamin D deficiencies.[67]

4.5 Associated links

The BPD-DS is a modification of BPD aimed at limiting consequences of malabsorption. But several biochemical measurements of nutrient status and bone metabolism were abnormal in this study.

I-CTP and changes in weight

C-terminal telopeptide of type I collagen (I-CTP) is known to correlate well with bone resorption rates in metabolic bone disease. [46] This biochemical marker was chosen to

investigate further for this reason. Biochemical markers may be better indicators of effects on bone metabolism in the shorter term as changes are seen more rapidly than in BMD. I-CTP levels were positively correlated with BMI reduction, but not BMI pre-surgery. This indicated an important relationship between the high rate of weight loss, charactersitic in BPD-DS, and effects on BMD.

Bone mineral density compared to weight loss

Weight reduction in the obese is associated with loss of bone. The amount of bone loss relative to the degree of weight loss seems to vary among populations and there is a strong positive correlation between rate of weight loss and effect on bone density. [28] This association is important in BPD-DS because both the rate and degree of weight loss is high. In this study, the strongest correlation to bone mineral density was level of BMI reduction. One study suggested that the weight loss induced in BPD-DS is associated with a significant loss of bone mass even at sites that are not affected by weight overload. [68]

Study results indicated that those who lost the most weight post surgery had the highest bone density. Considering that there was a significant positive correlation between BMI pre-surgery and reduction in BMI, this may indicate that those with the highest pre-surgery BMI had the highest bone density, but this difference was not significant and is contrary to previous studies which have found that morbid obesity is detrimental to bone density. [63] The inverse relationship between waist circumference and TBBD may have been confounded by gender, as men tend to have a greater waist circumference, and they had significantly lower bone density compared to women. However there was also a significant relationship between waist circumference and TBBD in the female group.

Bone mineral density compared to supplement intake.

We found no significant difference in any of the bone mineral density z-scores between those who had an adequate intake of supplements and those who did not. BMD was compared to calcium, vitamin D and general supplement intake using an

independent samples t-test. Adequate intake was classified according to guidelines for supplement intake in this patient group. Patients were considered to have an adequate supplement intake if they complied with the minimum recommended intake of at least 4000 mg of calcium (4 tablets of calcigran forte), 750 µg vitamin D (one AfiD2 forte) and one vitamin-mineral supplement (Nycoplus multi or equivalent) per day. Other supplement intake was not considered in relation to bone mineral density in this report.

Nutrient intake compared to BMD and biochemical markers of bone metabolism. We found no relationship between nutrient intake and BMD or biochemical markers of bone metabolism. Several factors may have affected these results. Most importantly, we only obtained dietary registration from 45 % of the study population and, as discussed, tools used in estimating nutrient intake have several limitations. However, we also did not detect any relationship between supplement compliance and nutrient status (which was available for all participants) to BMD. This could indicate that absorption of supplement derived nutrients may be even poorer than initially anticipated, however considering that supplement compliance was self-reported the reliability of this data may be poor.

5. Conclusion

Our results confirm previous findings that there is a high incidence of increased bone turnover and fat soluble vitamin deficiency after BPD-DS. We did not find any significant relationship between nutrient status and length of common channel, but markers of bone metabolism were related to degree of weight loss.

Average bone mineral density was within normal range for the study population, but there was a high incidence of low BMD (25-30 %) and men had significantly lower BMD than women. These results indicated that BPD-DS has some detrimental effect on bone mineral density, although it is uncertain how much is attributable to BPD-DS and how much a consequence of disturbances as a result of morbid obesity.

Biochemical markers of bone turnover were significantly increased in a large proportion of the study population.This indicated that there may be marked alterations in bone metabolism after BPD-DS. The relative contribution of morbid obesity and BPD-DS surgery to these disturbances has yet to be determined.

Associated links
All patients had achieved satisfactory weight loss after > 2 years post- surgery. Those with low BMD tended to be older, have lower BMI pre-surgery and had lost less weight after the surgery. Greater reduction in BMI was associated with higher level of I-CTP. This marker of bone resorption indicated that the high rate of weight loss post-surgery increases bone turnover. Although our results showed moderate effect on BMD at 2-5 years post-surgery, the significant high rates of bone turnover may affect BMD in the longer term. Further studies are needed to clarify this relationship.

We found no relationship between nutrient intake and BMD or biochemical markers of bone metabolism. We also did not detect any relationship between supplement compliance to BMD. Due to the nature of nutritional effects on bone health, nutritional monitoring over a longer time period, and for several years post-surgery,

may be necessary to detect any relationship. Our data is not sufficient to accept the H_0 hypothesis that nutrient intake after BPD-DS does not affect bone health. Previous studies have shown a clear relationship between nutrient status and bone metabolism and we continue to emphasize the importance of ensuring patient compliance to post-surgery nutritional recommendations and follow-up procedures.

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6. Appendix

Appendix 1

Further background information: Important dietary components related to bone metabolism, biochemical markers of bone metabolism, dual energy X-ray absorptiometry and physiology of bone metabolism

Appendix 2

Structured interview with student clinical nutritionist design

Appendix 3

Food diary

Appendix 4

Information sheet for food diaries

Appendix 5

Patient information and consent form

Appendix 6

Ullevål university hospital registration form for research projects

Appendix 1

6.1 Bone health

6.1.1 Physiology of bone metabolism – a brief overview

Bone tissue consists of a mineral component and an organic component, and has a mechanical as well as a biochemical function. Calcium, phosphate and magnesium are the most important minerals in bone tissue. An adult skeleton consist of calcium bound to phosphate in the complex crystalline material hydroxyapatite which is laid down on an organic matrix primarily made up of collagen. The skeleton contains more than half of the body's collagen, mainly in the form of type 1 fibrillar collagens. These extra-cellular proteins have a three-dimensional overlap structure called quarter stagger array, which provides gaps or hole zones where mineralization occurs. Bone metabolism maintains adequate levels of calcium for physiological processes in the body. The organic matrix also contains non-collagen proteins classified into sialoproteins (containing γ-carboxyglutamic acid (Gla proteins)), phosphoproteins (osteonectin) and bone proteoglycans.[48, 46] chapt 3, page 17-26

Bone formation and resorption

The turnover of bone is controlled by the activities of osteoblasts, osteoclasts, and osteocytes influenced by mechanical, nutritional and hormonal factors. Only a fraction of bone cells are active at a time. Remodelling ensures bone tissue vitality and elasticity. The synthesis, deposition and mineralization of the organic matrix involves expression of a number of genes by the osteoblasts. Production of type I collagen takes place during the proliferation of osteoblast precursor cells. After cessation of cell proliferation the expression of alkaline phosphatase starts. Osteocalcin is a calcium-binding protein expressed during matrix mineralization.[47]

Osteoblasts

Osteoblasts are differentiated from osteoprogenitor cells located in the periosteum and the bone marrow. Osteoblasts control bone turnover and are responsible for bone formation. They respond to endocrine, nutritional and mechanical signals released by osteoclasts and osteocytes. Communication of osteoclasts and osteoblasts is achieved through cytokine signaling. The signaling pathways are regulated by several hormones such as parathyroid hormone (PTH), vitamin D, growth hormone, steroids and calcitonin. Osteoblasts have receptors for PTH, $1.25(OH)_2D_3$, estrogen, and prostaglandin E_2. Remodellation and metabolic activity is far greater in trabecullar bone than in corticalis, and has a larger surface area.[48]

Osteoclasts

After activation and stripping of bone collagen, osteoclasts will start bone resorption. Osteoclasts derive from the fusion of cells of the monocyte-macrophage cell line. They are large and characterized by multiple nuclei, a homogenous, foamy cytoplasm and high concentration of vesicles and vacuoles. Osteoclasts dissolve bone mineral and release lysosymal enzymes, such as tatrate resistant acid phosphatase and proteins which create an acidic environment which break down the organic matrix.[47]

When the resorption process is finished, the bone formation cells (osteoblasts) start depositing osteoid composed mainly of type I collagen. Osteoid is mineralized into osteoid matrix under the influence of vitamin D and osteocalcin. Provided there is pre-existing bone surface after resorption, it will always be followed by a formation phase. As implied this will not be possible if the resorption process has resulted in a breakthrough (perforation) of the trabechular bone.[69]

Determinants of bone mineral density (BMD)

Bone mineral density is dependent on peak bone mass and subsequent bone loss.[44] Bone mass is steadily increasing in early stages of life until it reaches its' peak between 20 and 30 years of age. Total body bone mass remains relatively constant through reproductive years, then age-related bone loss occurs, which varies between

individuals, but occurs most rapidly during the first three years after menopause in women (perimenapausal stage). Average adult bone loss is 1% per year. Age related decreases in calcium absorption and increases in urinary calcium excretion contribute to this loss. Some women will lose up to 6 % per year in the perimenapausal stage. Because women generally have a lower peak bone mass, the development of osteoporosis is seen most frequently in women. The explanation for bone loss during aging includes declining calcium intakes, declining physical activity, decreased levels of gonadal hormones, decreased levels of circulating calcitriol, and intestinal resistance to this hormone.[43, 28]

Genetic predeterminants of peak bone mass account for 60 – 80 %. Environmental determinants of BMD include nutritional intake, smoking, excessive alcohol intake and starvation (anorexia) which may decrease bone mass. Peak bone mass is approximately 25 % less in females compared to males, but there are great individual variations. Adequate diet and weight bearing exercise is important to ensure potential peak bone mass is achieved. Immobilization, tobacco and alcohol increases bone mineral loss, as does early onset menopause which is related to a fall in oestrogen levels.[46, 70]

Dietary determinants of bone mineral density

Nutrition is one of the important modifiable risk factors in the development and maintenance of bone mass and the prevention and treatment of osteoporosis. Calcium and phosphorous compose about 80-90 % of bone mineral content. Protein is part of the organic matrix collagen structure where mineralization takes place and several other vitamins and minerals also play vital roles in bone metabolism.[43]

Energy and protein

Body weight is positively correlated with BMD. This may be due to greater weight bearing stress on the skeleton which stimulates bone formation. Weight loss is associated with bone loss. A 10 % weight loss typically results in 1 – 2 % bone loss.[58, 43] Severe weight loss and malnutrition is therefore considered important risk factors

for osteoporosis. This may be due to subsequent low protein intake, low general micronutrient intake including those necessary for healthy bone metabolism, muscle weakness which increases the risk of falling, and less padding to protect the skeleton during falls.[43]

Protein intake is associated with urinary excretion of calcium. The higher the protein intake the more calcium is lost. This is mainly due to the acid load of the sulphate produced in the metabolism of sulphur containing amino acids. A high protein diet may therefore be detrimental to bone health. However protein rich foods typically also contain phosphorous, which has a hypocalciuric effect and may inhibit the hypercalciuric effect of the protein. The link between high protein intake, calciuria and risk of osteoporosis is prevented when adequate calcium intake is ensured. There is also evidence that a protein deficient diet may reduce bone density due to subsequent reduced calcium absorption and IGF-1 levels.[43] Studies have found higher levels of bone resorption markers in diets with a lower protein intake compared to diets with a higher protein intake.[71]

Acid- Alkaline Ash

The skeleton may provide ion exchange as a buffer system to neutralize acid or alkaline disturbances after food intake. Some foods may produce and acid ash, for example meat, while fruit and vegetables have an alkaline ash and dairy products have a neutral ash. One hypothesis is that metabolic acidosis following a meal may cause increased bone resorption in order to release calcium ions and neutralize low pH. High intakes of meat could for example cause depletion in calcium stores and increase the risk of bone disease.[43]

Caffeine

Caffeine is believed to impair intestinal calcium absorption, but studies are inconclusive as to its effect on bone health. Its' negative effect is only relevant when calcium intakes are low.[43]

Calcium

The adult human body contains approximately 1000-1250 grams of calcium (depending on body size, gender and race) and 99 % is bound up in skeletal tissue. Free calcium is present in extra-cellular fluid, in all cells of the body and in the circulation as an easily exchangeable pool of calcium electrolytes.[39]

Calcium has both a structural role in the body as an important component of bone mass, and a mechanical role. The regulation of calcium metabolism is primarily concerned with protecting its' mechanical role which is vital in muscular, neurological and endocrine systems within the body. Free calcium plays a key role in signal transduction within and between cells, in neuromuscular transmission, in glandular excretion and in enzymatic reactions. Ionized calcium is the most common signal transduction element in cells, because of its ability to reversibly bind to proteins. These processes are dependent on a constant supply of calcium ions which is provided by the circulation.[39]

Calcium balance is maintained by several mechanisms including gut absorption, bone resorption and renal reabsorption.[28] This exchange is controlled by calciotrophic hormones, such as PTH, $1.25(OH)_2vitD_3$ (calcitriol) and calcitonin (but also locally acting hormones including cytokines).[48]

Absorption

Vitamin D status, intestinal transit time and mucosal mass affect calcium absorption. Dietary calcium is combined with calcium from the digestive juices in the small intestine. Active calcium uptake occurs in the duodenum and, when there is increased physiologic need, also in the ileum and to a small extent the jejunum and colon. Passive calcium absorption appears to occur across the lining the whole length of the intestine.[46] chapt 21, page 104-107. The active calcium absorption process is dependent on vitamin D. The difference between ingested calcium and that excreted in the faeces is termed net absorption. Net absorption fraction increases with decreased intake and with increased physiological needs. This adaptation is dependent on sufficient vitamin D being present and may become less efficient with increasing

age. In normal healthy individuals, 20-60 % of dietary calcium is absorbed.[39, 46, 72] Calcium absorption is inefficient in compensating for a fall in calcium intake. Absorption from the colon accounts for about 5 % of total absorption in normal individuals but may be important in patients with small bowel resections and where colonic bacteria break down dietary complexes. Decreased stomach acid reduces the solubility of insoluble calcium salts and reduces absorption unless fed with a meal.

The calcium from most supplements is absorbed as well as that from milk. Absorption of one very soluble salt, calcium-citrate, may be better than that of other salts because citrate appears to enhance paracellular calcium transport. Absorption of calcium supplements improves when they are taken with food, perhaps by slowing gastric emptying and extending time in which the calcium containing chyme is in contact with the absorptive surface.[46] chapt 21, page 104-107.

Calcium absorption may be inhibited by the presence of phytic acid, oxalic acid or phosphates. This interaction may become significant when calcium intake is low and/ or when there is a high intake of fibre rich foods.[39] There is some concern related to the intake of high phosphate containing drinks (such as coffee and dark coloured soft drinks) Elevated PTH levels, if sustained, could lead to bone resorption, but this effect has been reported in both diets with elevated phosphorous and low calcium levels. Low calcium levels alone may account for observed elevation in PTH.

Assessing calcium status

Calcium metabolism is in equilibrium with plasma calcium concentrations of about 2.1-2.60 mmol/L. Serum calcium level is regulated by calciotrophic hormones, and varies very little (1-2%). Total serum-calcium is a reliable analysis under normal conditions, but should also be corrected for serum-albumin. Serum calcium levels are rarely low as a result of low dietary intakes. Low serum Ca^{2+} may imply an abnormality of parathyroid function. Plasma calcium concentration rises in response to: increased calcium absorption; increased tubular reabsorption; and bone resorption. When this happens, the renal excretory threshold changes and extra calcium is excreted in the urine. Measuring urinary calcium in a 24-hour sample can be useful in

assessing adequate calcium intake. Abnormal low urine content where there is normal renal function suggests inadequate intake and/ or absorption.

If dietary supply is insufficient, calcium is released from bone tissue to maintain normal circulating levels. Hence normal s-calcium levels do not guarantee that there is adequate supply of calcium.[28] Plasma calcium concentrations may be abnormal for a number of reasons. Hypercalcaemia may be seen in diseases such as: malignant disease; primary hyperparathyroidism; granulomas; vitamin D overdosage; milk alkali syndrome; immobilization; thyrotoxicosis; hypercalcaemia of infancy; familial hypocalciuric hypercalcaemia. Hypocalcaemia may be seen in vitamin D deficiency (and disturbances in its metabolism); hypoparathyroidism and pseudohypoparathyroidism. Adequate calcium intake protects against osteoporosis and has been associated with reducing the risk of hypertension, colon cancer, lead poisoning and kidney stones in patients with short bowel syndrome.[46]

Recommended dietary calcium intake for healthy Norwegian adults is 800-900 mg per day.[39] Calcium requirements is the amount of dietary calcium required to replace losses in the urine, faeces and sweat plus the calcium needed for bone accretion during periods of skeletal growth. The recommended dietary intake for calcium is based on skeletal needs. The calcium intake required by older adults to achieve mean maximal retention or minimal loss was determined to be 1200 mg/day. This is also sufficient to protect against risk of hypertension. Bone metabolism responds to calcium intake up to a certain threshold when calcium needs are met, after this additional calcium supply will not improve bone mass. Ilich and Kersttner estimated that variations in calcium intake during early stages of life may account for 5 to 10 % of the difference in peak bone mass. This impact could contribute to more than 50 % of hip fracture rates later in life.[43] Supplementation is recommended when adequate intake is not met.[44]

Calcium intake up to 2.5 grams per day is considered safe. Higher intake is difficult to achieve through diet alone, but may be obtained through supplementation and can lead to hypercalcaemia if there is also a high intake of vitamin D.[39]

Phosphorous

Phosphorous is widespread throughout the body which contains 800-1200 g, 85 % is contained in the hydroxyapatite complex of bone. Hydroxyapatite contains calcium and phosphorous in the ratio of 1:2. In soft tissue phosphorous is mainly bound to organic compounds. Phosphorous plays a role in biochemical reactions involving organic phosphates such as adenosine tri-phosphate, and it is an integral part of phospholipids, phosphoproteins and phosphosugars.[39, 48] Phosphate is absorbed by several mechanisms mostly as inorganic phosphate achieved through hydrolysation of dietary organic compounds. Absorption of heavily soluble compounds such as phytate is believed to be low.[39]

Plasma phosphorous concentrations are relatively constant at 0.8-1.4 mmol/L and its balance is regulated by the amount reabsorbed in the kidneys. Vitamin D status affects the absorption of phosphate. Increased availability of calcitriol increases its absorption and decreased calcitriol has the opposite effect.[39] PTH reduces the renal reabsorption, calcitonin also increases phosphate excretion by the kidney.

Excessive phosphate consumption may be detrimental to bone. A rise in phosphate intake leads to a rise in serum concentrations which leads to reduced ionized calcium and increased PTH content in blood. A high phosphate – low calcium diet may cause hypocalcaemia or secondary hyperparathyroidism. Of particular concern is the phosphate content of carbonated beverages, which causes a high acid load from the phosphoric acid used as acidulant, and its frequent consumption in our population especially the younger generations. The tendency of these drinks to replace milk in the diet may further exacerbate their effect on bone health.[43]

Phosphorous deficiency (which may occur with prolonged use of aluminium-containing antacids) has widespread effects and there are important bone diseases related to phosphorous metabolism. Phosphate deficiency may cause osteomalacia, myopathy, growth failure and defects in leukocyte function. There is a difference between total body phosphorous deficiency, retention of phosphorous and its

redistribution between bone and soft tissue. Symptoms of phosphate deficiency include muscle weakness, lack of appetite, nausea and decalcification of bone.[39]

Recommendations

The Norwegian recommendations for phosphate intake is 600 mg per day for adults. [64] Phosphorous is present in all food groups and is often related to protein content.

Magnesium

Magnesium content in the body is approximately 25 grams. The skeleton contains 60 % and the remainder is distributed in soft tissue.[43] Magnesium is an important co-factor in the PTH-receptor complex. Plasma magnesium is kept constant by a rapid adaptation of the kidneys. Total plasma magnesium is 1.7-2.3 mg per 100 ml. Magnesium plays an important role in calcium and bone metabolism. It affects bone remodelling and strength and appears to have a positive correlation with hip bone mineral density.[28]

Recommended daily intake of magnesium is 350 and 280 mg for women and men respectively. [73] Net intestinal magnesium absorption appears to be linearly related to intake (like phosphorous), but at very low intakes a negative magnesium balance can occur. It is increased by both vitamin D and PTH. The tissues will only lose significant levels of magnesium if the intake is very low over a prolonged period of time or if intestinal losses are high. Hypomagnesaemia occurs in association with hypocalcaemia but may also be found on its own. The most common cause of magnesium deficiency is probably malabsorption, especially where this results from small bowel resection. Other causes include: dietary deficiency from protein energy malnutrition and prolonged parenteral nutrition; excessive renal loss, diabetes, in which there is both an increase in the renal loss and increased transport of magnesium (as well as potassium) into the soft tissues when insulin is given. Magnesium deficiency must be severe to impair calcium absorption. Chronic hypomagnesaemia impairs PTH secretion, resulting in altered calcium metabolism, hypocalcaemia and vitamin D abnormalities, This further decreases jejunal magnesium absorption.[38, 43]

Biochemical indicators of magnesium deficiency may be characterised by a tendency of higher serum-calcium and lower serum-phosphate. Magnesium deficiency decreases bone strength and volume, impairs bone development and leads to the uncoupling of bone formation and resorption. [43]

There is no relationship between magnesium and calcium absorption under normal magnesium levels.[74] Studies on magnesium supplementation after bariatric surgery are lacking.[28]

Fluoride

Fluoride is present in very small amounts in food and water. It is easily absorbed by passive diffusion in the intestines and is incorporated into teeth and bone.[43] Incorporation of fluoride into bone increases the size of the apatite crystals and hence decreases their solubility. In addition fluoride seems to stimulate osteoblast activity, increase bone formation and has shown a general increase of spinal bone mass of 5 to 10 %.[43]

Iron

Iron may act as a cofactor for enzymes involved in collagen synthesis. Ilich and Kerstetter examined the relationship between bone mass and ferritin in a four year clinical trial in adolescent girls. They found a positive association between ferritin levels and BMD, but concluded that further studies are needed to establish this connection. Iron may also act as a toxin to bone cells and contribute to bone disease if iron metabolism is impaired or there is iron overload. It is not clear whether the detrimental effect on bone is a direct result of iron compounds or if it is a result of iron overload-induced hypovitaminosis C or both.[43]

Iron absorption is inhibited by other minerals and trace elements including calcium. This becomes particularly relevant when high doses of calcium supplementation is administered due to impaired nutrient absorption.[43]

Both Norkost and Ungkost showed that iron intake was lower than recommended among fertile women in the Norwegian population.[52]

Zinc

The body contains approximately one to two grams of which 90 % is found in muscle, bone, skin and hair. Zinc plays an important role in the metabolism of connective tissue, where it acts as a co-factor for enzymes such as alkaline phosphatase and collagenase, which enable bone mineralization and development of collagen structures in bone tissue.[43]

Zinc deficiency affects DNA synthesis and protein metabolism, this may impair bone formation, growth and epiphyseal formation. Zinc deficiency is associated with osteoporosis.[43] Phytic acid, dietary fiber, calcium and low protein intake may inhibit absorption.[43] Recommended dietary intake of iron is 9 mg for men, and 15 mg for women until menopause, after which requirements are similar to that of men.[64]

Copper

The body contains approximately 75 to 100 mg of copper. Copper deficiency is uncommon because it is readily available in most foods. Recommended intake of copper for adults is 0.9 mg per day.[64] Copper plays a role in collagen maturation, and thus could influence bone composition and structure. [43]

Sodium

Dietary sodium increases urinary calcium excretion. Because urinary calcium excretion account for 50 % of the variability in calcium retention, dietary sodium has a potentially big influence on bone loss. In adult women, each extra gram of sodium per day may produce an additional rate of bone loss of 1 % per year if the calcium loss in the urine comes from the skeleton. Animal studies have shown that salt causes bone loss, but there are few studies on its' effect on bone mass in humans. [43] The association between sodium and calcium loss is of particular relevance when considering the current trend in the Norwegian population, in which there are decreasing calcium intakes while sodium intake remains persistently high.[75]

Vitamin D

Calciferol or vitamin D, is photosynthesised in the skin of vertebrates by ultraviolet B radiation (UVB). The two most important forms are ergocalciferol (vitamin D_2) and cholecalciferol (vitamin D_3). Vitamin D requires two obligate hydroxylations to become biologically active as the hormone $1.25(OH)_2D_3$ (calcitriol). [76] The activation process requires a cytochrome enzyme system similar to the P450 system in the liver and 25(OH)D-1α-hydroxylase in the kidney. The production of the active metabolite in the kidney is carefully regulated mainly by PTH in response to serum calcium and phosphorous levels.[76] PTH and calcitriol act synergistically to enhance renal tubular reabsorption of calcium and to mobilize calcium stores from bone.

Calcitriol maintains normal serum concentrations of calcium and phosphorous by ensuring efficient absorption of these minerals from the small intestine.[76] In the intestine, calcitriol stimulates the synthesis of calbindin (calcium binding protein).[43] Non-calciotrophic effects of calcitriol include effect on cell differentiation and the immune system. Calcitriol stimulates osteoblast activity, and among other things increases the synthesis of osteocalcin, which effects bone mineralization. It also has a direct effect on the mineralization of newly formed osteoid-tissue. Vitamin D deficiency hence leads to insufficient mineralization of osteoid-tissue (rakitt, osteomalacia). Calcitriol also stimulates differentiation of resorptive cells (osteoclasts).

Sources

Latitude, time of day and season has profound effects on UVB exposure, and dietary intake of vitamin D is clinically important because UVB exposion is often insufficient in northern latitudes. In the far northern countries such as Norway, there is very little vitamin D synthesis in the skin for up to six months of the year.[76]

Recommended dietary intake of vitamin D for the adult population is 7.5 µg per day, and 10 µg per day for persons older than 61 years. Both cholecalciferol and ergocalciferol are fat-soluble vitamins, which are absorbed in the entire length of the small bowel. Bile acids are necessary for absorption.

Vitamin D deficiency

Any disturbance of vitamin D production in the skin, absorption from the intestine or the conversion into its active form will lead to a deficient state. Disturbances in the calcitriol receptor may also cause deficiency, metabolic bone disease, and other biochemical abnormalities. [76] Vitamin D deficiency is characterized by inadequate mineralization or increased demineralization of the skeleton. In children this results in rickets, which is characterised by widening at the end of the long bones, rachitic rosary and deformations in the skeleton. In adults, prolonged vitamin D deficiency and secondary hyperparathyroidism can lead to osteopenia, osteoporosis and eventually osteomalacia.[66]

Vitamin D deficiency decreases serum concentrations of ionized calcium, which in turn leads to increased PTH levels stimulating calcium release from bone, reduced renal losses and increased excretion of phosphorous. This causes normal levels of serum calcium and low or low-normal serum phosphorous. Thus vitamin D deficiency is characterized either by normal or low-normal serum calcium with a low-normal or low serum phosphorous and increased PTH levels. Serum alkaline phosphatase (ALP) is usually also elevated.

The elderly are at risk of developing vitamin D deficiency. Decreased exposure to sunlight, decreased activation of vitamin D precursors in skin, reduced ability to hydroxylate vitamin D into the active form in the liver and kidneys, reduced end-organ response to calcitriol, reduced dietary intake and absorption of the vitamin may all be contributing factors. In addition, anticonvulsant and/ or steroid drug therapy impairs vitamin D status. [43] [76] Patients with impaired malabsorptive conditions may also be at risk of vitamin D deficiency. This is often seen in patients suffering from severe liver failure, Crohn's disease, Whipple's disease and sprue. The inability to secrete adequate amounts of bile or conditions that damage the small intestine are characteristics of these disorders.[76]

Serum concentrations 25(OH)D is a good indicator of vitamin D status as it reflects both that obtained from sunlight and that of dietary sources and should therefore be

used as the primary indicator of vitamin D status. Its half life is approximately ten days to three weeks.[76] Because of the short serum-half life of calcitriol (4-6 hours) and careful regulation of its production it is not a good indicator of vitamin D status.[76]

Vitamin D concentration above 30ng/mL is considered optimal for good bone health, and "normal" reference range may include individuals with significant vitamin D insufficiency. Serum levels should be above 25-30 ng/mL in order to prevent consequences of vitamin D deficiency.[77]

Vitamin K

Some evidence suggests that vitamin K may protect against age-related bone loss, mediated through the vitamin K-dependent γ-carboxylation of some proteins in bone (such as osteocalcin).[65] Vitamin K deficiency results in undercarboxylated osteocalcin which is associated with decreased BMD and higher incidence of hip fractures.[43]

Vitamin K is obtained from dietary sources and from the synthesis of intestinal bacteria. American authorities recommend a dietary intake of 55-70 μg of vitamin K per day. There are currently no recommendations for vitamin K intake from the Norwegian health authorities. Deficiency in healthy individuals is rare. At risk patients are newborn, those with conditions leading to fat malabsorption and prolonged use of antibiotics.[43]

The Framingham heart study found a relationship between the incidence of hip fractures and dietary intake of vitamin K in elderly men and women assessed by food frequency questionnaire. They found increased incidence of fracture in those who reported low dietary intakes, but no effect on BMD.[78]

Vitamin C

Vitamin C is necessary for collagen cross linking and the collagenous structure of bone is weakened as a result of scurvy. In addition vitamin C along with other antioxidant may protect bone tissue from oxidative damage. This could be of

particular relevance in smokers who are at increased risk of developing bone disease.[43]

Vitamin A

All functions of vitamin A are thought to be mediated through the regulation of gene expression in several different types of cells. Vitamin A is required for normal foetal development and for several other functions such as sight, cell differentiation and reproduction, haematopoiesis and differentiation of epithelial tissue.

Vitamin A plays a role in bone remodelling. Both osteoblasts and osteoclasts have nuclear receptors for retinoic acid. Both deficiency and excess of vitamin A is believed to be detrimental to bone. Too high levels of vitamin A may increase bone resorption while vitamin A deficiency impairs osteoblast proliferation which causes excessive deposition of periosteal bone. It is therefore important to maintain adequate but safe levels of vitamin A intake to protect bone.[43]

Cobalamine, B_{12}

B_{12} deficiency is associated with increased fracture risk and may be an important modifiable risk factor for osteoporosis.[79] Malabsorption is often a result of altered gut function in the gastric pouch or sleeve, but can also occur when 60 to 100 cm of the terminal ileum is bypassed. B_{12} supplementation is often recommended to all bariatric surgery patients because deficiency is common. BPD-DS patients are likely to be less prone to deficiency compared to RYGB patients because a larger portion of the stomach is maintained which produces intrinsic factor necessary for its' absorption. The distal part of the ileum also remains intact. Mild malabsorption of the vitamin may be corrected by oral supplementation of 350 μg, but many require lifelong subcutaneous injections.[28, 38]

6.1.2 Assessing bone health

Biochemical markers of bone metabolism

Collagen comprises about 90 % of the organic matrix. Thus how well the biochemical markers reflect the synthesis and degradation of type I collagen determines their representativeness of bone metabolism. The breakdown of bone causes increased renal excretion of its by-products including hydroxyproline, pyridinoline, deoxypyridinoline and N-telopeptide.

In patients with significantly altered gastrointestinal function it is important to consider markers which reflect regulatory situations and those which reflect disturbances in bone metabolism. Serum levels of calcium, PTH, phosphate, magnesium, 25(OH)D and $1.25(OH)_2D_3$ reflect the regulatory situation, while serum levels of bone markers such as bone-specific alkaline phosphatase, alkaline phosphatase, osteocalcin. I-CTP will reflect metabolic disturbances. Fasting urine levels of calcium/ creatinine ratio, DPYD and NTX will also reflect the latter. The concentration of ionized calcium in blood is strictly controlled, and therefore calcium can not be used as a general indicator of bone resorption.[47]

Urin-pyridiolin

Collagen consists of two alfa-1-chains and one alfa-2-chain attached in a triple helix. These structures are bound together by peptide bonds which will be released during bone resorption, and can be measured in urine as urin-pyridinolin.[55]

Alkaline phosphatase (ALP)

Alkaline phosphatase is an indicator of osteoblast activity. It is an enzyme associated with the plasma membrane of the osteoblast. ALP may be involved in extra cellular breakdown of pyrophosphate (which inhibits calcium phosphate deposition). It may be measured in serum using colorimetric methods. Its activity generally correlates with bone formation rate, for example in high or low turnover metabolic bone disease and during growth.[47]

Type I collagen

Type I collagen is synthesised as a larger protein (type I procollagen), it contains an extra domain on each end of the molecule which prevent premature association of the collagen into fibrils before it has been secreted out into the extra cellular space. The extension domains are then released and can be quantified in blood where they are known as propeptides of type I collagen, often abbreviated as PINP for the amino terminal and PICP for the carboxy terminal. If bone matrix formation and mineralization are synchronized, as they normally are, there is correlation between serum carboxy terminal propeptide of type I procollagen (PICP) and mineralization rate. In osteomalacia these processes are uncoupled and there is no correlation between the two. This reflects the formation of unmineralized osteoid.[47]

Osteocalcin (bone GLA-Protein (BGP))

Osteocalcin is a gla protein which is incorporated into bone matrix during bone formation. Osteocalcin is produced by the osteoblasts during the matrix mineralization phase. It's expression is strongly controlled by calcitriol and is not produced in the absence of this hormone. Gla residues are assumed to bind calcium and to be directly derived from osteoblasts. Approximately 30 % of newly formed osteocalcin remains in the circulation and is hence used as a marker for bone formation. However during bone resorption osteocalcin from bone is also released into the circulation, blood may contain osteocalcin from both formation and resorption. It may therefore be more appropriate to regard this protein as a marker of bone turnover rate rather than a formation marker.[43]

The biochemical indicators of bone resorption can be divided into two categories: Enzymes derived from the osteoclast and breakdown products of the organic matrix. The first category, biochemical markers of osteoclast function include tatrate-resistant acid phosphatase (TRAP). The other category, biochemical markers of bone matrix resorption, is a mixture of peptides and free amino acids from the breakdown of collagen fibres. (Risteli and Risteli 385-93)

C-terminal telopeptide of type I collagen (I-CTP)

I-CTP is the carboxy-terminal telopeptide region of type I collagen released during the degradation of mature type I collagen. This peptide is found in blood, where it seems to be derived from resorption and degradation of loose connective tissues. Increased serum concentrations of I-CTP are hence seen in conditions associated with increased lysis of bone, such as multiple myeloma, osteolytic metastases, rheumatoid arthritis and e.g. immobilization. In metabolic bone disease I-CTP concentration in serum correlates well with bone resorption rates. This is also true for osteomalacia and hyperthyroidism. [46]

Calcitonin (CT)

Calcitonin is a peptide hormone with binding sites in the kidney, bone and central nervous system. It is released from the thyroid gland in respond to the rise in serum calcium concentration and in response to gut hormone signalling. CT slows or halts osteoclastic resorption, thus stopping calcium release from bone. When calcium absorption stops CT concentrations fall. In skeletal tissue CT inhibits differentiation of osteoclasts and their resorptive properties.[80]

Parathyroid hormone (PTH)

PTH is a peptide with 84 amino acids. It is normally produced in the parathyroid glands which are located behind glandula thyreoidea in the neck. Calcium sensing receptors control the secretion and synthesis of PTH according to plasma calcium concentrations. The parathyroid glands are stimulated by a decline in calcium concentrations and their size and activity may increase by hyperplasia secondary to prolonged hypocalcaemia. An increase in serum-phosphate will also increase its production.

PTH helps maintain blood calcium levels by regulating bone turnover. A fall in plasma calcium concentrations stimulates the release of PTH from the parathyroid gland. PTH increases renal phosphate clearance, increases renal tubular reabsorption

of calcium and activates bone resorption. PTH promotes synthesis of calcitriol which then stimulates intestinal calcium absorption and regulates the synthesis of PTH by negative feedback. [76] This negative feedback loop ensures rapid response to correct hypocalcaemia, but in situations of a chronically deficient diet it has serious consequences in bone tissue.[48]

PTH effects bone tissue through stimulation of the osteoblasts. This leads to the release of several growth-factors, which in turn lead to an activation of osteoclasts. The net effect is increased bone turnover.

Modern methods of analysis use "two sites" antibody technique (IRMA) targeted at both the N- and C-terminal. This method has high sensitivity and specificity, and has made it easier to identify disturbances in calcium metabolism.

Table 39. Overview of established biochemical markers of bone metabolism and their origin.

Biochemical markers of bone metabolism			
	Cellular markers in serum (enzymes released by bone cells)	*Matrix markers in serum (agents from bone matrix)*	*Matrix markers in urine*
Formation markers	Alkaline phosphatase (ALP) Bone-specific alkaline phosphatase (b-ALP) Osteocalcin	PICP (Propeptide of type I procollagen) PINP (propeptide of type I procollagen)	
Resorption markers	Tatrate-resistant acid phosphatase (TRAP)	1-CTP Pyridinoline & deoxypyridinoline cross-links?	Hydroxyproline Pyridinoline & deoxypyridinoline cross-links INTP

Source:[47]

The pro-peptide parts of type I procollagen reflect the deposition of new type I collagen, while the non-collagenous indicators of osteoblast activity are alkaline phosphatase and osteocalcin. In a steady state situation, there should be reasonable correlations between all these markers, but they represent different phases of the osteoblast.

Other hormones in bone metabolism

Cytokines are locally acting hormones which facilitate changes in calcium metabolism. Immune cell products such as lymphotoxin, tumour necrosis factor (TNF) and the interleukins promote bone resorption. Several osteotrophic cytokines

may have a role in bone remodelling. Among the factors capable of stimulating bone resorption are tumour necrosis factor α and β, transforming growth factors, platelet derived growth factor, and arachidonic acid metabolites such as prostaglandins and leukotrienes. [48]

6.1.3 Measuring bone mineral density

Bone mineral density (BMD) is a measured calculation of the true bone mass (g/cm^2). In populations BMD correlates with bone strength, its ability to bear weight and tolerate impact, however this may vary between individuals. BMD measurements are used to predict the risk of bone fracture.

The World Health Organization has defined specific diagnostic categories for assessing bone health. However the reference population used in interpreting BMD is based on Caucasian data and there is significant variation in BMD between ethnic groups. For example, persons of African descent tend to have a greater BMD compared to those of Caucasian and Asian descent. This should be taken into account when interpreting BMD results [49]

Dual energy x-ray absorptiometry (DXA) is recognised as the most accurate method of measuring bone mineral density. This method uses the fact that a (foton) beam is weakened when it passes through bone tissue. To correct for surrounding soft tissue two foton beams with different energy levels are used. Common assessment areas are underarm, columna lumbalis and columna femoris. Bone mineral content (BMC) is the most important single risk factor in relation to osteoporosis. BMC changes according to rate of bone metabolism, because the remodelling space changes. If bone metabolism increases a fall in BMC can be seen. There is a clear relationship between BMC and fracture risk on population level, but as BMC only to a limited degree is related to biomechanical strength, BMC measurement in individuals can not predict future risk for developing osteoporosis accurately.[69] Results are defined in T-score and Z-score. On population level there is a two to three fold increase in the incidence

of spinal fractures in patients with low BMD. A BMD in the osteoporosis range, implies approximately 5 times increased fracture risk

T-score is the standard deviation from the mean BMD of a reference population (young healthy adults in their peak bone density years). Table 40 shows the WHO use of the T-score to define diagnostic criteria of metabolic bone disease.

Table 40. DXA T-score classification as a diagnostic tool for osteoporosis.

T-score	
T-score =/> -1	Normal bone density
T-score between -1 and -2.5	Low bone mineral density (osteopenia)
T score =/< -2.5	Osteoporosis if a fragility fracture has occurred (established) or severe osteoporosis is present.

Source: [49]

These criteria only apply to DXA of the posterior-anterior spine, femoral neck, and the proximal radius (33%) radius in post-menopausal women and men over the age of 50 years.

The z-score is the patients BMD expressed in SD from the mean of a reference population (a healthy reference population of the same gender and age) and should be used instead of the t-score for pre-menopausal women and men under the age of 50.

If BMD is solely a result of normal aging process, the z-score will be close to zero. If the z-score is less than -1.5, secondary causes of bone disease should be considered. All patients with a z-score below -2.0 should be evaluated for necessary treatment therapy.[28]

Williams et al recommended that all bariatric surgery patients have pre-operative DXA measurements as part of their evaluation and that measurements are conducted at 12 month intervals post-operatively, particularly in the first few years when metabolic change is most dramatic.[28]

Newer DXA machines tolerate weights up to 204 kg as opposed to the older ones which had a limit of 125 kg. For those who cannot be accommodated on a dxa table, dxa of the forearm may be used to assess bone density and fracture risk.[49]

Appendix 2

Structured interview with student clinical nutritionist

- Konsultasjon – Huskeliste

1. BES- skjema utleveres mellom konsultasjonene, slik at de kan fylle den ut mens de venter.

2. Subjektivt intervju – eget ark
3. DS konsultasjon – eget ark

5. Pedometer

vis og forklar

bestemme hvilke 3 dager de skal gå med den.

6. Matdagbok

Instruksjoner for matdagbok: Bruk mal for instruksjoner + sette opp plan for hvilke dager den skal fylles ut.

7. IPAQ

Insturksjoner for utfylling av spørreskjema om fysisk aktivitet

Denne kan de ta med hjem å fylle ut etter at de er ferdig med kostdagbok og pedometer.

8. Blodprøverekvisisjoner

3 skjemaer må sendes med

9. DEXA- rekvisisjon

Henvisning sendes med.

10. Samtykkeskjema – invitasjon til å være med i studien

- 2. Subjektivt intervju

- Har matlysten/ appetitten din endret seg sammenlignet med før operasjonen?
- Har matlysten/ appetitten din endret seg det siste året?
- Opplever du begrensninger ved måltid med tanke på mengder/ porsjonsstørrelser du kan ta i ett måltid?
- Reagerer du på fettrik mat?
  - Hvis ja, hvordan reagerer du?
- Har du opplevd smaksforandringer?
- Opplever du søtsug?

- Hvis ja, hvordan takler du dette?

- Er det noe du ikke tåler lenger?
  - I så fall hva? Og hvordan reagerer du?

- Er du plaget av:
  - Diare/ forstoppelse?
  - Kvalme/ oppkast?
  - Tygge/ svelgeproblemer?

- Er det sosiale situasjoner som påvirkes?

3. DS konsultasjon

- Kartlegging av måltidsytme/ mønster
  - Hovedmåltider
  - Mellommåltider
  - Inntak mellom måltidene
  - Væske både til og mellom måltidene.

- Tilskudd
  - Hvilke tabletter tar du, hvor ofte, og hvor mange av hver?

Tilskudd	▪ **Hvor ofte**	**Antall per gang**	**Tot antall per dag**
Multivitamineraler (med zink og magnesium?) eks. Nycoplus Multi Spesifiser:			
Calcigran forte tyggetablett Hvis annet kalsium tilskudd, spesifiser:			
AFI D 2 Forte			
Ferromax (65mg) (kvinner i fertil alder)			
Hemofer			
Omega 3 m/ folat			
B12 injeksjon			
Zink (mangel/ hårtap)			
K-vitamin			
Idoform (v/ diare)			
Imodium (v/ diare)			
Bio-dophilus, naturlige tarmbakterier			

*kalsium og jern bør ikke tas samtidig (konkurerer om opptak)

- Hender det at du ikke tar enkelte tilskudd?
  - I så fall, hvor ofte?
  - Hvilke?
  - Hvorfor?
- Opplever du vanskeligheter med tilskuddene?
  - Er de vanskelig å huske på?
  - Er de vanskelige å svelge?

- Proteininntak
  - Forenklet kostanamnese med fokus på proteinholdige matvarer
    - Husk: pålegg, middag, meieriprodukter.
    - Evt gi enkle råd og svar på spørsmål

- Væskeinntak

  Hva og hvor mye drikker de?
  Anbefal minst 2 liter per dag, helst mellom måltidene.

Appendix 3

Food diary

Dagbok

Fyll inn:

Kjønn

Alder ÅR

Ukedag DAG

Dato . .

Var denne dagen en vanlig dag? Skriv ja eller nei i rutene.

Hvis det var en uvanlig dag, forklar hvorfor denne dagen var uvanlig:

Hvor finner jeg matvarene i dagboken?

HUSK:

Absolutt alt du spiser/drikker skal skrives opp

Absolutt ikke kryss i dagboken

Absolutt bare bokstaver i de orange rutene

Absolutt bare tall i de sorte rutene

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Drikke

For størrelsen på glasset du drikker av, se bildeserie 1.
Fyll inn bokstaven i den orange ruten.

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Vann	glass					
Helmelk, søt/sur	glass					
Lettmelk, søt/sur	glass					
Ekstra lett lettmelk	glass					
Skummet melk	glass					
Drikkeyoghurt	glass					
Sjokolademelk av helmelk (eks. O'boy, Nesquick)	glass					
Sjokolademelk av lettmelk (eks. O'boy, Nesquick)	glass					
Sjokolademelk av skummet melk/ ekstra lett lettmelk (eks. O'boy, Nesquick)	glass					
Litago sjokolademelk	glass					
Litago sjokolademelk	1/2 liter					
Kakao av helmelk	kopp					
Kakao av lettmelk	kopp					
Kakao av skummet melk/ ekstra lett lettmelk	kopp					
Appelsinjuice	glass					
Eplejuice/eplemost	glass					
Nektar (eks. eple, tropisk frukt, annen frukt)	glass					
Brus med sukker	glass					
Brus med sukker	1/2 liter					
Brus, kunstig søtet	glass					
Brus, kunstig søtet	1/2 liter					

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Drikke forts.

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Saft med sukker (eks. appelsin, solbær)	glass					
Saft, kunstig søtet (eks. Fun light)	glass					
Usøtet mineralvann (eks. Farris)	glass					
Te, vanlig (eks. Earl Grey, solbær, mango)	kopp					
Fruktte (eks. nype, kamille)	kopp					
Instant urtete med sukker	kopp					
Kaffe	kopp					
Sukketter/Natrena/ Canderel	stk					
Sukker til te/kaffe	teskje					
Melk til te/kaffe	spiseskje					

Annet
beskriv best mulig hva, hvor mye og når:

Brød m.m.

Skriv antall skiver i sort rute. For tykkelse på brødskiven se bildeserie 2 og fyll inn bokstaven i orange rute.

1 skive = 1/2 rundstykke

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Loff/fint rundstykke	skiver					
Mellomgrovt brød, grovt rundstykke	skiver					
Grovbrød	skiver					
Baguette/Ciabatta	stk					
Knekkebrød lyst, skonrok, kavring	stk					
Knekkebrød, mørkt	stk					
Lompe	stk					
Pølsebrød, hamburgerbrød	stk					
Pitabrød	stk					
Flatbrød (eks. Mors flatbrød, Ideal)	stk					

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Smør eller margarin på brød.

1 skive= 1/2 rundstykke= 1 knekkebrød
=2 vaffelhjerter= 2 kjeks= 1/2 ciabatta

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Meierismør	til antall skiver					
Myk margarin (eks. Soya soft)	til antall skiver					
Lett margarin (eks. Soft light)	til antall skiver					
Hard margarin (eks. Per, Melange)	til antall skiver					

Annet
beskriv best mulig hva, hvor mye og når:

Hvor mye smurte du på brødet?

Se bildeserie 3 og skriv bokstaven for det bildet som ligger nærmest opp til den smør-/margarinmengden du brukte på brødet. Hvis du hadde forskjellig mengde smør/margarin på de brødskivene du spiste innenfor det angitte tidsrommet, kan du anslå et gjennomsnitt for skivene.

	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Bildeserie 3					

Pålegg

Du skal oppgi mengde pålegg i forhold til brødskiver. Har du spist to typer pålegg på samme brødskive, fører du opp begge (eks. 1 hvitost helfet og 1 skinke). Hvis du bare har spist pålegg og ikke brød, anslå til hvor mange skiver du kunne brukt dette pålegget.

1 skive= 1/2 rundstykke= 1 knekkebrød
=2 vaffelhjerter= 2 kjeks= 1/2 ciabatta

Kjøttpålegg	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Servelat, vanlig	til antall skiver					
Skinke, spekeskinke, lett servelat	til antall skiver					
Salami, spekepølse, fårepølse	til antall skiver					
Leverpostei, vanlig	til antall skiver					
Leverpostei, mager	til antall skiver					
Kalkun-/ kyllingpålegg	til antall skiver					

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Ost

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Hvitost helfet 27% fett (eks. Jarlsberg, Norvegia)	til antall skiver					
Hvitost halvfet 16% fett (eks. Norvegia lettere)	til antall skiver					
Brunost helfet (eks. Geitost G35, Fløtemysost)	til antall skiver					
Brunost halvfet, prim	til antall skiver					
Smøreost, vanlig (eks. Baconost, Snøfrisk)	til antall skiver					
Smøreost, mager (eks. mager skinkeost, mager prim)	til antall skiver					
Dessertoster (eks. Brie, Gräddost, Gourmetfrukt)	til antall skiver					

Fiskepålegg

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Kaviar	til antall skiver					
Røkt laks/ørret	til antall skiver					
Makrell i tomat, røkt makrell	til antall skiver					
Sardiner, sursild, ansjos	til antall skiver					

Syltetøy/søtpålegg

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Syltetøy vanlig, gelé, marmelade	til antall skiver					
Syltetøy lett, frysetøy	til antall skiver					
Honning	til antall skiver					
Peanøttsmør	til antall skiver					
Nugatti	til antall skiver					
Sjokade	til antall skiver					
Hapå/Litagopålegg	til antall skiver					

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Annet pålegg

1 skive= 1/2 rundstykke= 1 knekkebrød
=2 vaffelhjerter= 2 kjeks= 1/2 ciabatta

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Egg, kokt/stekt	til antall skiver					
Majonessalat (eks. italiensk salat, rekesalat)	til antall skiver					
Majonessalat, lett (eks. italiensk salat, lett)	til antall skiver					
Tomat som pålegg	til antall skiver					
Banan som pålegg	til antall skiver					

Annet
beskriv best mulig hva, hvor mye og når:

Pynt på brødskiver

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Majones/remulade, vanlig	til antall skiver					
Majones/remulade, lett	til antall skiver					
Agurk/salatblad/ tomat	til antall skiver					

Annet
beskriv best mulig hva, hvor mye og når:

Yoghurt

	Antall	kl. 6-10	kl.10-14	kl.14-18	kl.18-22	kl.22-6
Yoghurt naturell	beger (175 ml)					
Yoghurt med frukt	beger (175 ml)					
Lettyoghurt	beger (150 ml)					
Litagoyoghurt	beger (125 ml)					
Litagoyoghurt m/müsli	beger inkl. müsli					
Go'morgen yoghurt m/müsli	beger inkl. müsli					

Annet
beskriv best mulig hva, hvor mye og når:

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Frokostgryn og grøt

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Havregrøt	bildeserie 5					
Havregryn	bildeserie 4					
Firkorn	bildeserie 4					
Søtet müsli (eks. Crusli, Solfrokost)	bildeserie 4					
Müsli, usøtet (eks. Go'Dag, Frukt müsli)	bildeserie 4					
Cornflakes	bildeserie 4					
Honnikorn/ Frosties/Chocofrokost	bildeserie 4					
Puffet ris/ havrenøtter/hvetenøtter	bildeserie 4					

Annet
beskriv best mulig
hva, hvor mye og når:

Melk/sukker/syltetøy brukt sammen med frokostgryn og grøt

3 teskjeer=1 spiseskje

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Helmelk, søt/sur	dl					
Lettmelk, søt/sur	dl					
Skummet melk, søt/sur Ekstra lett lettmelk	dl					
Syltetøy vanlig, gelé, marmelade	teskjeer					
Syltetøy lett, frysetøy	teskjeer					
Sukker	teskjeer					

Annet
beskriv best mulig
hva, hvor mye og når:

For drikkeyoghurt som tilbehør til frokostgryn og grøt se side 2
For yoghurt som tilbehør til frokostgryn og grøt se side 6

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Kjøtt og kjøttretter

Pølse	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Grillpølse/wienerpølse, vanlig	stk					
Grillpølse/wienerpølse, lett	stk					
Middagspølse/ kjøttpølse/medisterpølse	kjøttpølse (15cm)					
Middagspølse/ kjøttpølse, lett	kjøttpølse (15cm)					

Kjøttretter/pasta/pizza

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Kjøttkaker/karbonadekaker	stk					
Medisterkaker	stk					
Elg-/reinkarbonader	stk					
Hamburger med brød (eks. vanlig, McDonalds mfl.)	stk					
Tacoskjell med kjøttdeig og salat	fylte skjell					
Kebab/Pitabrød med kjøtt og salat	fylte pita					
Kjøttdeigsaus/tomatsaus med kjøttdeig	bildeserie 11					
Pasta med tomatsaus uten kjøtt	bildeserie 6					
Pasta med hvit saus	bildeserie 6					
Lasagne	stk (10x8cm)					
Pizza, trekantstykker	bildeserie 12					
Pizza, firkantstykker	bildeserie 13					
Ekstra revet ost	spiseskjeer					

For lompe, pølsebrød og hamburgerbrød se side 3
For ketchup og sennep se side 13
For saus se side 13
For kokt pasta (uten saus) se side 12

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Kjøtt forts.

Rent kjøtt	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Biff (okse, lam, svin)	stykker					
Koteletter (svin, lam, okse)	koteletter					
Stek (svin, lam, okse)	skiver					
Kokt skinke	skiver					
Elg-/reinstek	skiver					
Grillet kylling	1/4 kylling					
Kyllingfilet	filéer					
Bacon	skiver					

Gryteretter	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Risotto/risretter	bildeserie 11					
Fårikål	bildeserie 11					
Lapskaus	bildeserie 11					
Elg-/reingryterett	bildeserie 11					
Kjøttgryte (kjøtt og grønnsak er i samme gryte)	bildeserie 11					
Leverretter	bildeserie 11					

Annet
beskriv best mulig hva, hvor mye og når:

For saus se side 13

Fisk og fiskeretter

Fiskefarse

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Fiskeboller	stk					
Fiskekaker/fiskepudding	stk/skiver					

Ren fisk

Torsk/sei/uer, kokt	stykke					
Torsk/sei/uer, stekt	bildeserie 14					
Laks/ørret/kveite, kokt	stykke					
Laks/ørret/kveite, stekt	bildeserie 14					
Makrell, kokt	stykke					
Makrell, stekt	bildeserie 14					
Flyndre/steinbit, kokt	stykke					
Flyndre/steinbit, stekt	bildeserie 14					

Tillagede fiskeretter og fiskepinner

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Fiskepinner	stk					
Panert fisk	stk					
Fiskegryte/fiskesuppe	tallerken					
Fiskegrateng	stk (10x8cm)					

Reker og fiskeinnmat

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Reker (uten skall)	bildeserie 9					
Torskerogn	skiver					
Fiskelever	spiseskjeer					

Annet
beskriv best mulig hva, hvor mye og når:

Andre retter

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Risengrynsgrøt	bildeserie 5					
Pannekaker	stk					
Suppe (eks. blomkål, tomat)	tallerken (3dl)					
Ertesuppe/betasuppe	tallerken (3dl)					
Kjøttsuppe (eks. Trøndersodd)	tallerken (3dl)					
Omelett	antall egg i stykket					
Ostepai	stykke (10x8cm)					
Eggerøre	til antall skiver					

Vegetarrett
beskriv hva (oppskrift), hvor mye og når:

Annet
beskriv best mulig hva, hvor mye og når:

Blandet salat m/ost/kjøtt/skalldyr

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Blandet salat (med ost, kjøtt eller skalldyr)	bildeserie 10					
Blandet salat (med pasta og ost, kjøtt eller skalldyr)	bildeserie 10					

Annet
beskriv best mulig hva, hvor mye og når:

For dressing se side 13

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Potet/ris/pasta

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Potet, kokt	stk					
Potet, bakt	stk					
Potetmos	bildeserie 7					
Stekt potet	bildeserie 8					
Pommes frites	bildeserie 8					
Potetsalat med majones/rømmedressing	spiseskjeer					
Potetsalat med oljedressing	spiseskjeer					
Ris, kokt (eks. parboiled, naturris)	bildeserie 6					
Ris, kokt (eks. jasmin, basmati, hurtigris)	bildeserie 6					
Pasta, kokt (eks. spaghetti, makaroni, tagliatelle)	bildeserie 6					
Nudler (eks Mr.Lee)	pose					

Annet
beskriv best mulig hva, hvor mye og når:

Grønnsaker

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Gulrot, rå	stk					
Gulrot, kokt	stk					
Kålrot, rå/kokt	skive					
Brokkoli, blomkål, rå/kokt	bildeserie 9					
Hodekål, rå/kokt	skalk					
Råkost (blandet av flere grønnsaker)	bildeserie 9					
Grønnsaksblanding, kokt	bildeserie 9					
Blandet salat (eks. kinakål, mais, tomat og agurk)	bildeserie 10					

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Grønnsaker forts.

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Tomat	skiver					
Sopp, fersk	stk					
Paprika	ringer					
Mais	spiseskjeer					
Løk, stekt	spiseskjeer					

Annet
beskriv best mulig hva, hvor mye og når:

Saus/dressing

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Hvit saus	spiseskjeer					
Hvit ostesaus	spiseskjeer					
Brun saus	spiseskjeer					
Sjysaus	spiseskjeer					
Smeltet smør/ margarin	spiseskjeer					
Tomatsaus (uten kjøtt)	spiseskjeer					
Ketchup	spiseskjeer					
Sennep	spiseskjeer					
Bernaise saus ol.	spiseskjeer					
Dressing, vanlig (eks. Thousand Island)	spiseskjeer					
Dressing, lett (eks. Thousand Island light)	spiseskjeer					
Seterrømme 35% fett	spiseskjeer					
Lettrømme 20% fett	spiseskjeer					
Majones/remulade, vanlig	spiseskjeer					
Majones/remulade, lett	spiseskjeer					

Annet
Beskriv best mulig hva, hvor mye og når:

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Is/dessert

Is

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl.22-6
Is (eks. vanilje, krokan)	bildeserie 15					
Yoghurtis (eks. Dream, Living Lite)	bildeserie 15					
Ispinne (eks. Gullpinne, Pinup)	stk					
Kremmerhus (eks. Kroneis, Kronevaffel)	stk					
Saftispinne (eks. Lollipop)	stk					

Gelé, pudding, fromasj

Gelé (eks. sitron, jordbær)	bildeserie 15					
Pudding (eks. sjokoladepudding)	bildeserie 15					
Riskrem, multekrem, fromasj	bildeserie 5					

Hermetisk frukt, fruktgrøt

Fruktcoctail	bildeserie 5					
Ananas (ring), pære/fersken (halv)	stk					
Fruktgrøt, kompott	bildeserie 5					

Annet
beskriv best mulig hva, hvor mye og når:

Dessertsauser/krem

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Fløte	spiseskjeer					
Krem, pisket	spiseskjeer					
Sjokoladesaus	spiseskjeer					
Karamellsaus	spiseskjeer					
Vaniljesaus	spiseskjeer					
Rød saus	spiseskjeer					

Annet
beskriv best mulig hva, hvor mye og når:

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Kaker, gjærbakst

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Boller	stk					
Julekake, kringle	skive/stykke					
Skolebrød, skillingsbolle	stk					
Wienerbrød	stk					
Vafler	hjerter					
Eplekake, pai med frukt/bær	stykker					
Formkake, muffins	skive/stk					
Sjokoladekake	stykker					
Marsipankake, bløtkake	stykker					
Fyrstekake, nøttekake	stykker					
Smultring	stk					

Kjeks	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Kjeks (eks. Mariekjeks, Gjende)	stk					
Fylte kjeks (eks. Ballerina, Monaco, Pepita)	stk					
Havrekjeks (eks. Bixit, Sibas)	stk					
Smørbrødkjeks (eks. Kornmo, GoldenCrisp)	stk					
Smørbrødkjeks (eks. Kaptein, Start)	stk					
Salte kjeks (eks. Ritz, Salinas)	stk					
Kjeks med sjokolade (eks. Maryland cookies, Bixit med sjokoladetrekk)	stk					

Annet
beskriv best mulig hva, hvor mye og når:

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Frukt/bær	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Eple	stk					
Banan	stk					
Pære	stk					
Appelsin	stk					
Mandarin/klementin	stk					
Druer	stk					
Fersken/nektarin	stk					
Jordbær (friske/frosne)	stk					
Rosiner	neve					
Kiwi	stk					

Annet
beskriv best mulig hva, hvor mye og når:

Snacks	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Potetgull, vanlig (1 neve= 8 flak)	neve					
Potetgull, vanlig	pose (250g)					
Potetgull, lett/ potetskruer (1 neve= 8 flak)	neve					
Potetgull, lett/ potetskruer	pose (250g)					
Ostepop (1 neve= 8 ostebuer)	neve					
Maischips (1 neve= 8 flak)	neve					
Peanøtter	pose (100g)					
Popcorn	neve					
Popcorn	pose (100g)					
Dip (eks. rømme m/dipmix)	spiseskjeer					

Annet
beskriv best mulig hva, hvor mye og når:

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Godterier

Sjokolade/konfekt	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Melkesjokolade (Melkesjokolade, Firkløver, Helnøtt)	plate (100g)					
Mørk kokesjokolade	staver/4 ruter					
Marsipan med sjokolade (eks. Gullbrød, marsipangris)	som Gullbrød (65g)					
Sjokoladebiter (eks. Twist, konfekt)	biter					
Kinderegg	stk					
Snickers, Japp	stk (60g)					
Kjekssjokolade (eks. Kvikklunsj, Twix)	som Kvikklunsj (46g)					
Troika	stk					
New Energy	stk					

Annet
beskriv best mulig hva, hvor mye og når:

Smågodt	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Lakris (eks. "salte sild", lakrisbåter)	stk					
Gelé godt (eks. seigmenn, vingummi, "colaflasker")	stk					
Skumgodt (eks. "viskelær", "sopp", marshmellows)	stk					
Syrlige drops (eks. "bringebær", salte og sure bomber)	stk					
Karamell (eks. Fudge, Smørbukk, Fox)	stk					
Godteripose (Godt & blandet, Søppeldynga, Partymix)	pose (150g)					
Kjærlighet på pinne	stk					

Annet
beskriv best mulig hva, hvor mye og når:

Drops/pastiller

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Drops/pastiller med sukker (eks. kamferdrops, Halslinser, Doc)	stk					
Pastiller, kunstig søtet (eks. Dent)	stk					
Tyggegummi med sukker	stk					
Tyggegummi, kunstig søtet (eks. Extra, V6)	stk					

Annet
beskriv best mulig
hva, hvor mye og når:

Tran/kosttilskudd

1 barneskje= 5 ml

	Antall	kl. 6-10	kl. 10-14	kl. 14-18	kl. 18-22	kl. 22-6
Tran	barneskje					
Trankapsler	stk					
Sanasol	barneskje					
Biovit	barneskje					
Multivitamin (eks. Vitaplex, Vitamineral)	stk					
Fluortabletter	stk					
Jerntabletter (9mg)	stk					
C-vitaminer (eks. Ester C)	stk					

Annet
beskriv best mulig
hva, hvor mye og når:

Appendix 4

Information about food diary

Informasjon om Matdagboken

Dette er matdagboken som fylles ut i løpet av de neste 2 ukene. Den første uken registrerer du alt i __ dager etter hverandre, og den andre uken registrerer du alt i__ dager etter hverandre. Til sammen registrerer du alt du spiser i 7 dager. I den første konvolutten ligger skjemaene du skal fylle ut i den første uken, og i den andre konvolutten ligger skjemaene du skal fylle ut i den andre uken. Til sammen er det 7 skjemaer som skal fylles ut – en for hver dag.

Det er viktig at du er nøye med registreringen, og fyller inn alt du spiser. Det inkluderer mat og drikke til hovedmåltidene, mellommåltidene og det du spiser og drikker utenom disse.

Ingen restriksjoner
Det er også viktig at du ikke forandrer på kosten din på grunn av matdagboken. Du har ingen restriksjoner utenom det du har fra før.

Les nøye
I spørreskjemaene er det grundige forklaringer til hver seksjon. Leser du nøye gjennom alt som står i hver av seksjonene, vil du unngå misforståelser. Mye av det du lurer på, vil du også finne svar på her.

De oransje rutene
I matdagboken vil du se at det flere steder er en oransje rute ved siden av rutene for antall matvare. I disse rutene fyller du inn bokstaven for porsjonsstørrelse som passer til det du har spist. For å bestemme porsjonsstørrelse bruker du det oransje hefte, der finner du bilder av forskjellige porsjonsstørelser, og en bokstav som hører til hver størrelse. Dette gjøres hver gang det er en oransje rute ved siden av en matvare du har spist og fylt inn.

Pålegg
I seksjonen for pålegg registreres mengden i forhold til antall skiver. Hvis du bruker mer pålegg en det som er vanlig på en skive, må du ta dette i betraktning. Registrer mengden i forhold til det du tar. Hvis du for eksempel bruker 2 skiver kokt skinke på en brødskive, isteden for 1 skive kokt skinke, må du skrive at du har spist kokt skinke til 2 brødskiver, selv om du bare har spist en brødskive. Antall brødskiver/ knekkebrød du har spist, registrerer du i en annen seksjon. Hvis du bruker ett tykt lag med leverpostei, som du tror vanligvis kan brukes til 2 skiver, må du skrive at du har spist leverpostei til 2 skiver. Hvis du har spist 2 skiver, som du hadde dobbelt opp med pålegg på, registrerer du dermed 4 porsjoner pålegg, altså pålegg til 4 skiver. Det samme gjelder for alt annet pålegg.

Kosttilskudd
Registrer alle kosttilskuddene du tar hver dag. Det er laget plass til å registrere disse på siste side i skjemaet, men du vil sannsynligvis finne at flere av kosttilskuddene du tar ikke er listet her. I så tilfelle kan du bruke baksiden av skjemaet til å skrive ned disse tilskuddene. Det er viktig at du registrerer hver eneste tablett, hver eneste dag. Hvis du ikke skriver de opp, går vi utifra at du ikke har tatt tilskudd.

Spørsmål
Hvis du har problemer med å fylle ut matdagbøkene, bruk av skritteller, spøreskjemaet for fysisk aktivitet, eller du har andre spørsmål, er det bare å ta kontakt med Julia eller Nicole:

Kontaktinformasjon, Julia
Telefon: 41046876
Mail: julia.mckenna@studmed.uio.no

Kontaktinformasjon, Nicole
Telefon: 97789991
Mail: n.s.warmbrodt@studmed.uio.no

Appendix 5

Patient information and consent form.

Informasjon og forespørsel om deltakelse i forskningsprosjekt

Næringsstatus, næringsinntak og fysisk aktivitet hos pasienter operert med biliopankreatisk bypass med duodenal switch for fedme

Hensikten med denne studien:
Hensikten med denne studien er å kartlegge kosthold, fysisk aktivitet, ernæringsstatus, benstatus, potensielle kostrelaterte problemer og fedmerelaterte komorbiditeter hos duodenal switch pasienter 2 år eller mer etter operasjon.
Studien utføres for å øke kunnskapen rundt følgene av denne typen operasjoner, og dermed gi et bedre behandlingstilbud for denne pasientgruppen.

Hvorfor blir du forespurt
Du er blitt spurt om å delta fordi du har gjennomgått biliopankreatisk bypass med duodenal switch for fedme, det er 2 år eller lengre siden operasjonsdato, og du blir fulgt opp ved avdeling for preventiv kardiologi, UUS.

Frivillighet:
Det er frivillig å delta i studien. Dersom du velger å ikke delta, trenger du ikke oppgi grunn. Du kan når som helst trekke deg uten at dette vil påvirke den behandlingen du får ved Ullevål universitetssykehus.

Hva innebærer dette for deg:
Mye av det du må gjøre i forbindelse med prosjektet inngår allerede i ditt behandlingsopplegg her ved Ullevål universitetssykehus. Blodprøver, dexa måling (mål på bentetthet) og ett spørreskjema er standard prosedyre, men du vil i tillegg bli bedt om å fylle ut en kostdagbok og et spørreskjema om fysisk aktivitet og gå med en skritteller i 3 dager.

Når du er ferdig med utfylling av kostdagbok og skrittellerskjema, må du returnere disse til oss.

Vi ber om din tillatelse til at disse opplysningene fra din journal, brukes i forskningsprosjektet: alder, kjønn, vekt, resultater fra blodprøvetaking og DEXA- målinger.

Slik ivaretas dine prøver og personopplysninger:
Alle opplysningene vil bli behandlet konfidensielt. I prosjektet har du et prosjektnummer som knytter deg som person til prosjektinformasjon gjennom en egen liste. Kun prosjektansvarlige med taushetsplikt har adgang til adresselisten. Alle opplysningene som inngår i prosjektet vil bli oppbevart i et eget datasystem ved sykehuset i tråd med interne retningslinjer for datasikkerhet.

Hvor lenge skal materiale og opplysninger lagres?
Når prosjektet avsluttes i november 2008 vil gjenværende materiale og informasjon bli destruert/slettet etter gjeldende retningslinjer. Du har rett til å se de personlige dataene som er samlet inn om deg og få dem rettet hvis noen er feil. Dersom du ikke lenger vil være med i studien kan du forlange analyseresultatene slettet, og da kan du også forlange at prøvene som er tatt av deg i forbindelse med forskningsprosjektet skal ødelegges.

Annet

Når du deltar i denne studien vil du være forsikret mot uforutsette skader forårsaket av studien etter gjeldende bestemmelser i Norge (pasientskadeerstatningsordningen). Undersøkelser i studien vil ikke medføre kostnader for deg. Vi dekker ikke tapt arbeidsfortjeneste. Forskningsetiske komite i Helseregion Øst har vurdert utprøvingen og funnet at den tilfredstiller de krav som forskriftene stiller.

Prosjektansvarlig/Mer informasjon

Hvis du har spørsmål om studien, kan du kontakte overlege Serena Tonstad. Hun vil være beredt til å svare på dine spørsmål på telefon: 22119979.

SAMTYKKE- PROSJEKTDELTAKELSE

Deltakelse i studien er basert på ditt frivillige, informerte samtykke. Dersom du ønsker informasjon utover det som framkommer i dette informasjonsskrivet og den muntlige informasjon du har mottatt, har du full anledning til å be om dette. Dersom du etter å ha fått den informasjonen du synes er nødvendig, sier ja til å delta i studien, må du signere samtykkeerklæringen:

Jeg bekrefter at jeg har mottatt den informasjonen (skriftlig og muntlig) om prosjektet som jeg synes er nødvendig og sier ja til å delta i studien:

Forsøkspersonens etternavn og fornavn:……..

Dato: / 2006 …… (signatur)

Muntlig bekreftelse: Jeg bekrefter at jeg på en korrekt måte har informert

…….. om hensikten og hvordan studien skal gjennomføres:

Dato: / 2006 ……. (signatur)

Appendix 6

Ullevål university hospital registration form for research projects

UUS' meldeskjema for forsknings-, kvalitetsstudier og annen aktivitet som medfører behandling av personopplysninger som er melde- eller konsesjonspliktig i henhold til helseregisterloven og personopplysningsloven med forskrifter

Utfylt skjema sendes elektronisk til sykehusets personvernombud: heidi.thorstensen@ulleval.no
Spørsmål ifm utfylling av skjemaet kan sendes til: heidi.thorstensen@ulleval.no

1 INFORMASJON OM SØKEREN

A. BEHANDLINGSANSVARLIG VIRKSOMHET

Ullevål universitetssykehus HF			Organisasjonsnummer 983971784
Postadresse: Kirkeveien 166	Postnr.: 0407	Sted: Oslo	Land: Norge
Telefonnummer: 22118080	Telefaksnummer: 22119950	E-postadresse/hjemmeside: www.uus.no	

B. DIVISJON/AVDELING VED UUS HVOR PROSJEKTET GJENNOMFØRES

Avdeling for preventiv kardiologi

C. DAGLIG ANSVAR FOR OPPFYLLELSE AV DEN BEHANDLINGSANSVARLIGES PLIKTER ER ADMINISTRERENDE DIREKTØR

D. PROSJEKTETS KONTAKTPERSON (må være ansatt ved UUS)

Navn og stilling ved UUS

Professor Serena Tonstad, overlege ved anvdeling for preventiv kardiologi

Telefonnummer	E-postadresse
22117939	Serena.Tonstad@uus.no

E. MULTISENTERSTUDIE[1]

Er prosjektet en multisenterstudie? ☒ Nei ☐ Ja Dersom ja, angi øvrige virksomheter som deltar.

Skal noen av disse også ha kopi av elektronisk database/informasjon som etableres i prosjektet? ☐ Nei ☐ Ja

F. LEGEMIDDELFIRMA ELLER ANNEN VIRKSOMHET HAR KONSESJON FOR PROSJEKTET

Er prosjektet organisert fra et legemiddelfirma eller annen virksomhet som allerede har konsesjon?

☒ Nei

☐ Ja Dersom ja, angi virksomhetens navn. (Kopi av konsesjonen skal sendes UUS' personvernombud, og prosjektet skal meldes personvernombudet som meldepliktig prosjekt, dvs skjemaet fylles ut med unntak av punktene 8.4, 8.5, 9 og 10.)

Skal den eksterne også ha kodelisten/navnelisten over deltakere inkludert fra UUS? ☐ Nei ☐ Ja

Dekker denne meldingen utlevering av opplysninger fra UUS til firma/virksomhet som har konsesjon? ☐ Nei ☐ Ja

[1] For å merke av i boksene, dobbeltklikkes det på venstre museknapp med markøren på boksen som skal avkrysses. I dialogboksen som kommer frem, velges "aktivert". Ved å gjenta prosessen og klikke på "deaktivert" fjernes krysset

2 PROSJEKTETS NAVN/TITTEL

Næringsstatus, næringsinntak og fysisk aktivitet hos pasienter operert med biliopankreatisk bypass med duodenal switch for fedme.

3 BESKRIV FORMÅLET MED BEHANDLINGEN/PROSJEKTET[2]

Formålet med studien er å kartlegge kosthold, fysisk aktivitet, ernæringsstatus, benstatus, potensielle kostrelaterte problemer og fedmerelaterte komorbiditeter hos duodenal switch pasienter, to år eller mer etter operasjon. Med dette håper vi å øke kunnskapen rundt disse faktorene som kan bidra til å forbedre oppfølgingstilbudet til denne pasientgruppen.

4 BEHOV FOR KONSESJON ELLER MELDING

4.1 Meldepliktige prosjekter – Siden UUS har internt personvernombud, vil de fleste studier være meldepliktige

Ett av de tre hovedpunktene må være oppfylt for at studien skal være meldepliktig:

- Prosjektet er omfattet av personopplysningsforskriften § 7-27. (Punkt a under må være oppfylt, samt enten b eller c)
  a) Tilrådd av personvernombud, og REK for prosjekter med medisinsk eller helsefaglig forskning.
  b) Ikke stort omfang, men lang varighet og identifiserbart, eller
  c) store datasett og tilfredsstillende avidentifisert eller pseudonymisert.

- Prosjektet/behandlingen har hjemmel i lov utføres i regi av organ i stat eller kommune (eks. kvalitetssikring etter helsepersonellovens § 26) – se personopplysningsloven § 33, fjerde ledd.

- Prosjektet er regulert i forskrift som spesielt angir at det er unntatt fra konsesjonsplikt eller underlag meldeplikt (f.eks. de sentrale helseregisterforskriftene)

[2] Behovet for konsesjon/melding er knyttet opp til hvilket formål man har med behandlingen av personopplysningene. UUS' journalsystemet er i sin helhet meldt, og har lovhjemlet formål. Når informasjon i journalsystemet skal benyttes til andre formål, kommer behovet for konsesjon, alternativt ny melding, opp, og man må angi formålet med den nye bruken/behandlingen av personopplysningene. Formulering av formålet er derfor viktig. Tilsvarende gjelder for annen innsamling og behandling av pasient-/personopplysninger. Formålet må samsvare med det som beskrives i samtykket fra hver enkelt person som deltar i studien.

4.1.1 Avklaringspunkter[3]:

☐ Nei ☒ Ja Respondenten eller verge samtykker i alle deler av undersøkelsen (formål og varighet på oppbevaring av personopplysningene)

☒ Nei ☐ Ja Det benyttes kobling mot andre personregistre, eks fødselsregister, kreftregister, dødsårsaksregister eller tilsvarende

☒ Nei ☐ Ja Studien inkluderer et stort omfang av personer og/eller data – dvs mer enn 5000 og/eller opplysninger av svært inngripende karakter

Angi totalt inkluderte 77

Angi hvilke type opplysninger som vurderes til å være av svært inngripende karakter

Oppgi i feltet under tidspunkt for når studien avsluttes og personopplysningene slettes eller anonymiseres (kodelisten slettes). Dersom det er vanskelig å angi tidspunkt, skriv i feltet under en tekstforklaring sammen med antatt tidspunkt.

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NB! Husk på å inkludere tid for innsamling, bearbeiding og publisering ved angivelse av tidspunkt.

Det innsamlede materiale skal ved angitt tidspunkt slettes eller anonymiseres (kun et av alternativene anonymisering eller sletting skal avkrysses)

☐ Nei ☐ Ja anonymiseres, forklar hvordan

☐ Nei ☒ Ja slettes ved prosjektavslutning

☐ Nei ☐ Ja Personopplysninger, inkludert avidentifiserte/kodede, lagres kun på papir.

ELLER

4.1.2 Intern kvalitetsoppfølging

☐ Nei ☒ Ja Oppfyller Helsepersonelloven § 26

Opplysningene må være slettet eller anonymisert før eventuell publisering av resultater.
Krever ikke samtykke, ref punkt 8, Personopplysningsloven § 33 4. ledd gir unntak for konsesjon men krever melding.
Pasienter som har reservert seg mot slik bruk av opplysningene, skal respekteres.

ELLER

4.1.3 Annet som hjemler melding, angi årsak/hjemmel

Dersom meldepliktig studie, besvares ikke punktene 9 og 10.

4.2 Konsesjonspliktig prosjekt
Dette punktet benyttes av sykehusets personvernombud dersom studien er konsesjonspliktig

☐ Ja - Prosjektet tilfredsstiller ikke forutsetninger for melding (se punktene under 4.1) og må ha konsesjon

5 PROSJEKTPERIODE

Ny melding, angi dato	Oppdatert melding (forutsetter samme formål som forrige melding), angi dato
SVAR:	SVAR:

Angi dato for prosjektavslutning. NB! Må inkludere innsamling, analyse/vurdering, artikkelskriving/konklusjon og eventuelt tid for oppbevaring etter ferdigstilling. Dette gir dato for sletting/anonymiseringer av personopplysningene.

SVAR: November 2008

[3] For å merke av i boksene, dobbeltklikkes det på venstre museknapp med markøren på boksen som skal avkrysses. I dialogboksen som kommer frem, velges "aktivert". Ved å gjenta prosessen og klikke på "deaktivert" fjernes krysset

Eventuelle tilleggskommentarer:

6 HUMANT, BIOLOGISK MATERIALE

Medfører prosjektet bruk av humant, biologisk materiale, som tas kun for denne studien, dvs ikke i diagnostisk eller behandlingsmessig hensikt?

☒ Nei

☐ Ja - Forskningsprosjekt vil da alltid måtte etablere biobank

(Kvalitetssikringsprosjekt, ref HPL § 26 vil benytte diagnostisk/behandlingsmessig biobank og trenger ikke melde forskningsbiobank.)

7 DETALJER OM PROSJEKTETS INFORMASJONSBEHANDLING

7.1 Behandlingen skal omfatte typer personopplysninger:

7.1.1 Ikke-sensitive personopplysninger

Identifikasjonsopplysninger

☒ Navn, adresse, fødselsdato
☐ Fødselsnummer (11 siffer)
☐ Fingeravtrykk, iris
☐ Annet

Atferdsopplysninger

☒ Loggføring av adferd, herunder tidspunkt og sted for handlinger
☒ Preferanser (ønsker, behov o.l.)
☐ Annet

Opplysninger om tredjepersoner

☐ Navn, adresse, fødselsdato
☐ Fødselsnummer (11 siffer)
☐ Annet

Fjernsynsovervåkning og lydopptak (sett eventuelt. flere kryss)

☐ Fjernsynsovervåkning
☐ Billedopptak
☐ Lydopptak

7.1.2 Sensitive personopplysninger, jf. pol. § 2 nr. 8

Behandlingen omfatter opplysninger om

☐ rasemessig eller etnisk bakgrunn, eller politisk, filosofisk eller religiøs oppfatning
☐ at en person har vært mistenkt, siktet, tiltalt eller dømt for en straffbar handling
☒ helseforhold
☐ seksuelle forhold
☐ medlemskap i fagforeninger.

Ved konsesjonssøknad, presiser nærmere: (eventuelt i eget vedlegg)

Behandling av sensitive personopplysninger:

☒ skjer helt eller delvis med elektroniske hjelpemidler
☐ som kun inngår eller skal inngå i et manuelt personregister

7.2 Behandlingen omfatter opplysninger om (beskriv også eventuell kontrollgruppe)

☐ Ansatte i egen virksomhet ☐ Elever/studenter/barnehagebarn ☒ Pasienter ☐ Tilfeldig utvalgte

☐ Adgangskontrollerte ☐ Medlemmer ☐ Kunder/klienter/brukere ☐ Seleksjonsutvalg

☐ Annet: (skriv inn i feltet nedenfor)

Angi størrelsesorden på utvalget 77

Inkluderer utvalget personer med begrenset samtykkekompetanse, eks mindreårige, demente, annet, ☒ Nei ☐ Ja Dersom ja, forklar

7.3 Hvordan samles personopplysningene inn?

☒ Manuelt ☐ Annet: (skriv inn i feltet nedenfor)

☒ Elektronisk (bilde og tekst)

☐ Videoopptak

☐ Lydopptak

7.4 Personopplysningene innhentes fra

☒ Fra den registrerte selv

☒ Annet: (skriv inn i feltet nedenfor)

Fra pasientjournal

7.5 Hvordan skal opplysningene brukes?

☒ Registreres

☐ Lagres

☒ Sammenstilles, med hva

(eksterne registre eller interne konsesjonsbelagte registre): Enkelte parametre skal sammenlignes med verdier fra tidligere i pasientens behandlingsperiode.

☐ Utleveres, til hvem:

☐ Annet:
(skriv inn i feltet)

7.6 Lagring og behandling av opplysninger – utstyret som benyttes, skal alltid eies av UUS dersom ikke annet er oppgitt

☒ Elektronisk –
På server i UUSs nettverk
Angi navn på server,
Dersom UUS – forskningsserver benyttes, er risikohåndtering og sikkerhet beskrevet i UUS standard vedlegg 2 og 3, – henvis til dem.

UUS – forskningsserver

Se UUS standard vedlegg 2 og 3

☐ Elektronisk –
På frittstående PC eiet av UUS (dvs ingen tilknytning til andre PC-er eller nettverk, interne eller eksterne)
Forklar hvordan PC er sikret

☐ Papir –
Forklar hvordan dette sikres mot uvedkommende

☐ Video, tape eller annet opptak
Beskriv hva og hvordan dette er sikret mot uvedkommende og om personen kan identifiseres

☐ Annet, angis i feltet

7.7 Hvordan gjenfinnes opplysningene? Bruk av direkte identifisering som personnummer og navn skal søkes å unngå, se sykehusets instruks i den kliniske håndboken

Opplysningene lagres med navn, personnummer eller annet som entydig angir det enkelte individ ☒ Nei ☐ Ja

Opplysningene lagres avidentifisert ☐ Nei ☒ Ja

Hvordan er krysslister/kodelister beskyttet/lagret (skriv inn i feltet under)

Kodelistene vil lagres i ett excel ark på en passordbeskyttet UUS PC, som er låst inne på ett kontor ved avdeling for preventiv kardiologi.

7.8 Blir personopplysningene gjort tilgjengelige/utlevert til andre

☒ Nei

☐ Ja, oppgi mottakeres navn og adresse, samt hvilken rolle mottaker har i prosjektet. (Videre utfylling i punkt 7.8 skal bare gjøres dersom dette er besvart med JA.)

Kryss av dersom opplysningene overføres til utlandet? ☐ Ja

Hva blir overført?

Informasjon med navn, personnummer eller annet som entydig angir det enkelte individ? ☐ Nei ☐ Ja

Avidentifisert informasjon? ☐ Nei ☐ Ja (Forklar i feltet under hvordan kryssreferanseliste beskyttes dersom dette ikke er likt som i pkt 7.7)

Anonymisert informasjon? ☐ Nei ☐ Ja

Annet? (Forklares i feltet under, eksempelvis hvordan opptak håndteres.)

Hvordan oversendes informasjonen?

Overført til diskett eller tilsvarende og sendt i vanlig postgang? ☐ Nei ☐ Ja

Sendt over eksterne linjer? ☐ Nei ☐ Ja

Hvordan er det sikret mot uautorisert avlytting? (Forklares i feltet under)

Annet? (Forklares i feltet under)

Er det inngått avtale med mottaker, slik at personvernet er sikret ved oversendt informasjon? ☐ Nei ☐ Ja

7.9 Annen årsak til overføring i eksternt datanett

Overføres opplysningene i eksternt datanett ut over det som er spurt om i 7.8? ☒ Nei ☐ Ja Dersom ja, forklar

8 RETTSLIG GRUNNLAG FOR BEHANDLING AV PERSONOPPLYSNINGENE

Som hovedregel skal informert samtykke innhentes. Dersom personens helse eller annen tilstand umuliggjør dette, kan det likevel søkes konsesjon. Det må da utdypes

- hvordan ulempen for den enkelte og personvernet alternativt håndteres
- hvilke samfunnsmessig interesse gjennomføring av prosjektet innebærer

8.1 Behandling av personopplysninger i henhold til personopplysningsloven § 8

(Gjelder ikke-sensitive personopplysninger)

- ☒ med den registrertes samtykke
- ☐ ved at det er fastsatt i lov at det er adgang til slik behandling
- ☐ for å oppfylle en avtale med den registrerte, eller utføre gjøremål etter den registrertes ønske før en slik avtale skal inngås
- ☐ for at den behandlingsansvarlige skal kunne oppfylle en rettslig forpliktelse
- ☐ for å utføre en oppgave av allmenn interesse
- ☐ for å utøve offentlig myndighet
- ☐ for at den behandlingsansvarlige eller tredjepersoner som opplysningene utleveres til kan vareta en berettiget interesse, og hensynet til den registrertes personvern ikke overstiger denne interessen

8.2 Behandling av personopplysninger i henhold til personopplysningsloven § 9

(Gjelder sensitive personopplysninger)

- ☒ med den registrertes samtykke
- ☐ når det er fastsatt i lov at det er adgang til slik behandling
- ☐ når behandlingen er nødvendig for å beskytte en persons vitale interesser, og den registrerte ikke er i stand til å samtykke
- ☐ når det utelukkende behandles opplysninger som den registrerte selv frivillig har gjort alminnelig kjent
- ☐ når behandlingen er nødvendig for å fastsette, gjøre gjeldende eller forsvare et rettskrav
- ☐ når behandlingen er nødvendig for at den behandlingsansvarlige kan gjennomføre sine arbeidsrettslige plikter eller rettigheter
- ☒ når behandlingen er nødvendig for forebyggende sykdomsbehandling, medisinsk diagnose, sykepleie eller pasientbehandling eller for forvaltning av helsetjenester, og opplysningene behandles av helsepersonell med taushetsplikt
- ☐ når behandlingen er nødvendig for historiske, statisktiske eller vitenskapelige formål, og samfunnets interesse i at behandlingen finner sted klart overstiger ulempene den kan medføre for den enkelte
- ☐ i henhold til personopplysningsloven § 9 annet ledd

8.3 Hvordan skal informasjonsplikten i POL §§ 19 og 20 oppfylles? Se sykehusets instruks for krav til innhold

Når opplysninger samles inn fra den registrerte	☐ Muntlig	☐ Skriftlig	Legg ved kopi av dokumentasjonen
Når opplysninger samles inn fra andre enn den registrerte	☐ Muntlig	☐ Skriftlig	

8.4 Annet grunnlag for behandlingen?

Forklar, for eksempel HPL § 26, se punkt 4.1.2:

8.5 Andre tillatelser

Fremleggingsplikt for REK Nei ☐ Ja ☒

Dispensasjon fra taushetsplikten Nei ☒ Ja ☐ Hvis ja, legg ved/ettersend dispensasjonen

Andre tillatelser, forklar og legg ved/ettersend

9 OPPFYLLELSE AV ENKELTE BESTEMMELSER I PERSONOPPLYSNINGSLOVEN

9.1 Hvordan skal det sikres at opplysningene er tilstrekkelige og relevante i forhold til formålet med behandlingen? Legg ved en beskrivelse av rutinen.

Opplysningene som hentes inn er del av behandlingen for duodenal switch opererte ved avdeling for preventiv kardiologi, UUS. Se vedlegg: Konsultasjon - huskeliste

9.2 Hvordan skal det sikres at opplysningene er korrekte og oppdaterte i forhold til formålet med behandlingen. Legg ved en beskrivelse av rutinen.

Opplysningene som hentes inn er del av behandlingen for duodenal switch opererte ved avdeling for preventiv kardiologi, UUS. Se vedlegg: Konsultasjon - huskeliste

9.3 Hvordan skal det sikres at opplysningene slettes når formålet er oppfylt? Legg ved en beskrivelse av sletterutinen.

9.4 Hvor lenge må opplysningene være identifiserbare, angis med måned og år: 2 år, 2 måneder

Til tiden for prosjektet må det inkluderes tiden det tar å gjennomføre innsamling av opplysninger, bearbeiding av opplysningene, konkludering, utforming av eventuelle artikler og eventuelle krav om oppbevaring etter publisering.

9.5 Er det meningen at opplysningene skal oppbevares etter at formålet er oppfylt, dvs etter tid angitt i punkt 9.4?

Nei ☒ Ja ☐ Hvis ja; angi grunnlag:

☐ i medhold av arkivloven

☐ annen lovgivning, hvilken lovhjemmel:

☐ historiske etter statistiske formål

☐ vitenskaplige formål

Dersom opplysningene behandles for historiske, statistiske eller vitenskapelige formål, legg ved en beskrivelse av de samfunnsinteresser som vil oppveie personvernulempene.

Presiser nærmere: (eventuelt i eget vedlegg)

10 INFORMASJONSSIKKERHET

UUSs sikkerhetsmål, sikkerhetsstrategi og sikkerhetsorganisering se VEDLEGG 1 (vedlegges konsesjonssøknad)

UUS risikovurdering og etablert forskningsserver, se VEDLEGG 2 og 3 (vedlegges konsesjonssøknad)

UUS har internkontrollsystem

Utføres behandlingen i henhold til annet regelverk som regulerer sikkerheten.

Nei ☒

Ja ☐ Hvis ja; hvilket regelverk.

11 Underskrift

Sted og dato	Underskrift
Oslo, 21 sep 06	Serena Tonstad