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Nutritional intakes of patients at risk of pressure ulcers in the clinical setting

Running Title: Nutritional intakes of patients with reduced mobility

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Authorship

Shelley Roberts: Conception and design of the study; collection, assembly, analysis and interpretation of data; drafting and revision of manuscript; and approval of final version of manuscript.

Wendy Chaboyer and Ben Desbrow: Conception and design of the study; analysis and interpretation of data; drafting and revision of manuscript; and approval of the final version of the manuscript.

Michael Leveritt and Merrilyn Banks: Interpretation of data; drafting and revision of the manuscript; approval of the final version of the manuscript.

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Conflict of Interest Statement

The authors declare no conflict of interest.
ABSTRACT

Objective: Malnutrition is a risk factor for pressure ulcers. The aim of this study was to describe the energy and protein intakes of hospitalised patients at risk of pressure ulcers and identify predictors of eating inadequately.

Research Methods & Procedures: An observational study was conducted in four wards at two hospitals in Queensland, Australia. Adult patients with restricted mobility were observed for 24 hours, and information such as oral intake and observed nutritional practices was collected. A chart audit gathered other demographics, clinical, anthropometric and dietary information. T-tests or one-way analysis of variance tests were used to identify differences in total energy and protein intakes. Univariate and multivariate regression analysis was conducted to determine predictors of eating inadequately (i.e. intake of <75% of estimated energy and protein requirements).

Results: Mean energy and protein intakes of the 184 patients were 5917±2956kJ and 54±28g, respectively. Estimated energy and protein requirements were calculated for 93 patients. Only 45% (n=42) and 53% (n=49) met ≥75% of estimated energy and protein requirements, respectively. In multivariate analysis, patients on the renal ward were 4.1 and 4.6 times more likely to be eating inadequately for energy and protein, respectively (p<0.05). Patients who consumed any amount of oral nutrition support were 5.1 and 15.5 times more likely be eating adequately for energy and protein, respectively (p< 0.05).

Conclusions: Renal patients appear to be more likely to be eating inadequately, whilst any consumption of oral nutrition support seems to increase likelihood of eating adequately.

Keywords: Pressure ulcer; nutrition; oral intake, hospital.
Introduction

Malnutrition is a common and costly problem in the hospital setting, affecting as many as 20–50% of patients [1-2]. Its consequences are severe, including impaired immunity, delayed recovery and healing, loss of muscle mass and function and poorer quality of life [3]. Malnutrition increases hospital length of stay (LOS) and hospital costs amongst various groups of patients [4-6], and is also directly associated with the development and severity of pressure ulcers (PU) [7-8].

PU place a large burden on both the patient and the healthcare system. The prevalence of PU ranges from around 5–30% of all hospitalised patients [7, 9]. For the patient, PU result in numerous medical complications, including increased risks of infection and mortality, and lengthy healing times [3, 10]. Other problems arising from PU include pain, discomfort, decreased mobility and independence, wound exudates and odour and social isolation [11-12]. PU result in severe consequences to the health care system, including increased hospital costs and LOS [13-14].

Malnutrition has been associated with at least twice the odds ratio of having a PU [7]. Mechanisms by which malnutrition increases PU risk may be related to body composition, skin and tissue integrity, and mobility [3, 11, 15]. Low body weight may be associated with PU due to an increase in bony prominences and less fat tissue to distribute pressure [11]. Malnutrition may also result in impaired skin integrity and resistance to pressure due to decreased nutrient availability for tissue maintenance and repair [3]. Furthermore, malnutrition is associated with decreased mobility, which is an independent risk factor for PU [3, 15].
Oral or enteral nutritional supplementation in groups of older patients deemed at risk of PU may contribute to PU prevention [16]. Although most studies have failed to reach statistical significance individually, likely due to small sample sizes, a meta-analysis found that the provision of oral or enteral nutrition support resulted in a 26% lower incidence of PU in high risk patients compared to routine care [16].

To our knowledge, no hospitals within Australia routinely prescribe oral nutrition support (ONS) to at-risk patients for the prevention of PU. Given this, understanding the oral intake of patients at risk of PU and factors determining oral intake in routine care is important if we are to ensure those at risk of PU are eating adequately. Whilst investigations of dietary intakes of hospitalised patients have been conducted [17-20], no studies have described nutritional intakes amongst a group of patients at risk of PU. Therefore, it is unknown whether the current knowledge about the intakes of hospital patients in general can be applied to patients at risk of PU. The aim of this study was to describe the nutritional intakes of hospitalised patients at risk of PU, and predictors of inadequate energy and protein intakes.
Materials and Methods

Study overview

A multisite observational study was undertaken, consisting of two components; 24 hour observations and chart audits. Ethical approval was gained through Queensland Health (reference number HREC/11/QTHS/111) and Griffith University (reference number NRS/40/11/HREC).

Setting

Data collection was conducted in four medical wards (renal, immunology, respiratory medicine and general medicine) at two public metropolitan hospitals in Southeast Queensland, Australia. A randomised data collection schedule was used to allocate seven days of data collection (i.e. Monday to Sunday) to each ward (28 days in total) over nine weeks.

Study participants

 Patients were included in the study if they met the following eligibility criteria: able to provide consent (aged over 18 years, cognitively intact); at risk of pressure ulcers due to restricted mobility (i.e. use of mobility aid such as walking stick, frame, wheelchair; or presence of mobility-restricting equipment such as intravenous (IV) lines, oxygen therapy, as determined from medical notes); and length of stay ≥3 days. Reduced mobility was chosen as a conservative inclusion criteria to identify patients at risk of PU, as it is a widely recognised risk factor and strong predictor of PU in the clinical setting [10, 21-22]. The use of a PU risk assessment tool, such as the Braden or Norton scale, was not used to identify at risk patients.
as they are shown to have insufficient predictive validity and poor reliability [23-26]. Patients
could not be recruited into the study more than once. Eligible patients were provided with a
participant information sheet, and informed consent was obtained from agreeable patients.

**Tool Development and Pilot Testing**

The conceptual framework that underpinned data collection was developed from a review of
the literature and clinical experience. A number of predictor variables were identified and
grouped into categories, including patient related (eg. self-feeding ability; co-morbidies; level
of mobility; and nutrition impacting symptoms such as chewing or swallowing problems,
nausea, vomiting or mouth ulcers), service related (eg. hospital diet; dietetic input; food and
supplement provision), and care delivery related (eg. feeding assistance; malnutrition risk
assessment completion) factors. A semi-structured observational tool and a chart audit tool
were developed using this framework to determine the data to be collected. The tools were
assessed by five clinicians and academics with expertise in this area of research. The tools
were piloted and modified prior to data collection. Four researchers (including one author)
were involved in data collection, and undertook training in the use of the data collection
forms. A pilot study of ten patients was conducted prior to data collection to test intra- and
inter-rater reliability of the data collectors. Both intra-rater and inter-rater reliability were
>95%.

**Data Collection**

*Patient observations*

Using a semi-structured observational tool, each patient was observed for 24 hours
(commencing at 0700 hours). Observations were performed by three data collectors across
three 8-hour shifts. Patients’ oral intake was recorded for the 24 hour duration of data collection by observing each patient’s plate at the end of each meal (breakfast, lunch and dinner). Researchers indicated the amount (none, ¼, ½, ¾, all) of each component of a standard sized meal consumed on the observational data form. This method of observed food intake has previously been shown to be a valid and reliable method of collecting dietary intake data [19, 27]. Patients’ menu slips were collected to determine the specific meals and food items they received at each meal. At mid-meals (morning tea, afternoon tea, supper), researchers observed patients’ food and fluid intake, including any supplements consumed.

Researchers observed a number of nutrition-related practices, such as patients’ ability to feed themselves; whether feeding assistance was received at meals and mid-meals, and if so, provided by whom; who completed the patients’ menu; and whether the patient was involved in their menu choice if they did not complete their own menu. Each patient also answered some brief questions regarding appetite, nutrition impacting symptoms (such as chewing and swallowing abilities, nausea, vomiting, mouth ulcers, etc.), weight history and PU history.

**Chart audit**

For each patient recruited into the study, an independent chart audit was completed (by a researcher who did not collect observational data on that patient). Information was collected from patients’ medical records and bedside charts, and included patient demographics; medical information; height, weight and body mass index (BMI) when available; serum albumin levels; hospital diet; fluid restrictions; nutrition support (oral, enteral or parenteral); evidence of food/fluid intake and weight monitoring; and dietitian referrals and reviews.

**Data analysis**
Oral nutrient intake data was analysed by an accredited practicing dietitian familiar with the foodservice systems of the two sites. Data was analysed using Foodworks version 6.0 (Xyris Software, Brisbane). A database was created with foodservice information from both sites, including energy and protein contents of each meal component and food item. Each patient’s food intake for the 24 hour observation period was entered into the database, including any supplements, enteral or parenteral feeds, and foods sourced from outside the hospital. Outcome variables were total energy and protein intakes.

Patients’ disease-specific estimated energy requirements (EER) and estimated protein requirements (EPR) were calculated for those patients who had adequate anthropometric and medical data available for comparison with their food intakes. This was done using Australian and international best practice clinical guidelines for patients with specific disease states [28-30], and 100 – 125kJ/kg (25 – 30kcal/kg) and 0.8 – 1.0g/kg protein for individuals without disease states affecting metabolic requirements [30-31]. When there was a range recommended for requirements (eg. 100 – 125kJ/kg), the average of the two values was taken as the recommended requirement.

All data were entered into SPSS. Following data entry, 10% of the data was checked for accuracy. Demographic data was analysed using descriptive statistics, and participant characteristics were compared between sites. The influence of environmental and patient-related factors on total energy and protein intakes were assessed using independent samples T-tests or one-way analysis of variance tests.

Patients were divided into two groups for each set of analysis. For analysis of energy intakes, patients were divided into a) inadequate intake (i.e. consuming <75% EER) and b) adequate intake (i.e. consuming ≥75% of EER). These values were chosen as previous research has
shown that patients who consumed <75% EER lost weight during their admission, suggesting that this level of intake is inadequate for patients to maintain their body weight [18]. Whilst there is no clinical evidence to suggest a threshold for adequacy of protein intake, the amount of protein required to maintain muscle mass and other bodily functions is likely to be related to the amount of energy required to maintain body weight. For this reason, patients were also divided into consuming <75% and ≥75% of EPR for analysis of protein intakes. Univariate logistic regression analysis was conducted to determine potential predictors of eating inadequately, using the conceptual framework of theoretically important variables. Significant factors were then entered in a multiple logistic regression model. For all associations, significance was set at p≤0.05.
Results

A total of 241 patients were recruited and participated in the study, however complete data was available for only 184 patients. Patient characteristics for these 184 patients are summarised in Table 1. There were significant differences between sites for LOS and serum albumin. The most common diagnoses were infection (22.3%), respiratory disease (16.3%), and gastrointestinal disease/condition (6.5%). The most common co-morbidities were hypertension (48.9%), chronic obstructive pulmonary disease (35.5%) and type 2 diabetes mellitus (31.0%).

There were a number of cases where nutritional intake data was incomplete. This occurred due to time constraints of data collectors, patients consuming food off the ward, patients unable to be observed at some times due to medical or privacy concerns, and patients being unexpectedly discharged prior to conclusion of the 24-hour observational period. Figure 1 represents the flow of patients available for each type of data analysis. There were no significant differences in age, LOS, BMI or albumin between patients with complete and missing data.

The mean total energy intake was 5917±2956 kJ, and mean total protein intake was 54±28 g protein. There were significant differences in energy and protein intakes in a number of variables, as shown in Table 2. Variables for which there were no differences in energy and protein intakes included site, ward, diagnosis, LOS, history of past or present PU, mobility status, dietitian referral or review, documented prescription of ONS, hospital diet, malnutrition screening tool (MST) score, serum albumin, reported recent weight loss, or other nutrition impacting symptoms such as chewing or swallowing difficulties.
Patients’ subjective appetite rating (very poor, poor, fair, good, very good) was related to both energy and protein intake, with improved appetite being associated with higher energy (p<0.001, F=7.503) and protein (p<0.001, F=6.686) intakes.

Energy and protein requirements were estimated for 93 patients. Mean requirements were 8271±1665kJ and 69.4±16.0g protein. On average, patients met 75.4±39.3% (median 72.0%, IQR 48.0 – 93.8%) of their EER and 80.6±43.3% (median 76.3%, IQR 53.5 – 102.1%) of EPR. Only 45.2% (n=42) of patients met ≥75% of their EER, whilst 53.1% (n=52) met ≥75% of their EPR.

Univariate logistic regression analysis of potential predictors of eating inadequately (i.e. consuming <75% EER or EPR) is shown in Table 3. Significant predictors were entered into a multiple logistic regression model, which determined independent predictors of eating inadequately, displayed in Table 4. Factors not associated with eating inadequately, including mobility; serum albumin; nutrition impacting symptoms such as chewing or swallowing difficulties; self-feeding ability; dietitian referral or review; or MST score were not entered into the multivariate model.

Patients on the renal ward were four times more likely to be eating inadequately in relation to energy and protein compared to all other wards. Patients who did not consume any ONS were five times more likely not to meet energy requirements, and over 15 times more likely not to meet protein requirements.
Discussion

This study directly observed the oral intakes of hospital patients in an attempt to understand factors associated with improving dietary intake in patients at risk of PU. Predictors of eating inadequately (i.e. intake of <75% EER and EPR) were being on the renal ward, and the absence of any intake of ONS.

Mean energy and protein intakes in this study are comparable to previous studies describing the intakes of general hospitalised patients [17, 19-20, 32]. Many patients at risk of PU appear to be eating inadequately in the hospital setting. In fact, only 42 of the 93 patients (45.2%) met \( \geq 75\% \) of their EER, whilst only 49 (52.7%) met \( \geq 75\% \) of their EPR. This phenomenon is reflected in studies of hospitalised patients in general, with a number of studies reporting inadequate energy and protein intakes to meet estimated requirements in patients [17, 19-20].

Clearly, the methods used to calculate estimated energy and protein requirements and the criteria used to define an adequate intake will affect the results. In the current study, an adequate intake was defined as an intake of \( \geq 75\% \) of EER and EPR. This is based on a Danish hospital study that described an intake of <75% of EER as being inadequate, as patients within this category experienced weight loss during the study period [18]. Whilst there have been various methods used to define energy intake in relation to requirements in previous research, this method was chosen as it correlates with weight change in a clinical population.

Whilst there were a number of factors associated with differences in total energy and protein intakes, only two variables remained independent predictors of eating inadequately after
multivariate logistic regression analysis. These were: 1) being admitted to the renal ward, and
2) having no intake of ONS.

Admission to the renal ward was an independent predictor for eating inadequately, where patients were over four times more likely to consume <75% of their EER and EPR. This may be due to a number of reasons. Firstly, disease-related factors are likely to play a role, as renal failure was a potential predictor of eating inadequately in univariate analyses. Chronic renal failure (CRF) and dialysis both increase EER, making it more difficult to reach an adequate intake [28, 33]. Furthermore, food intake may be decreased due to anorexia, nausea or vomiting related to uraemic toxicity [33-34]. Therapeutic diets (such as low salt, low potassium) may restrict patients’ food choices and consequently, their intake. As renal failure did not reach statistical significance in the multivariate model, whilst the renal ward did, there appears to be other (i.e. non-disease related) factors involved in whether these patients meet their requirements. These may be related to hospital foodservice or ward practices in renal wards, such as meal times, availability of staff for set up / assistance with feeding, timing of meals, or missed meals due to extended periods of time off the ward (for example in dialysis).

Inadequate nutritional intake is of particular concern to this patient group, as malnutrition is common in patients with chronic renal failure [33]. It could be hypothesised that the high prevalence of malnutrition and prolonged periods of immobility (i.e. during dialysis sessions) in renal patients may increase the likelihood of PU. One study found that renal insufficiency (measured by elevated creatinine) was an independent risk factor for PU after multivariate regression analysis in the intensive care unit (ICU) [35]. However, another study failed to find this association among hip fracture patients admitted to an orthopaedic ward [36]. Future research should further investigate whether renal patients in hospital wards (outside the ICU) are at higher risk of PU than other patient groups.
The consumption of any amount of oral nutrition support was associated with adjusted OR of 5.1 and 15.5 of meeting ≥75% of EER and EPR, respectively. Patients were included in this category regardless of the actual amount of nutrition support consumed on the observation day. Being prescribed ONS or receiving a dietitian consultation were not associated with eating adequately, which highlights the importance of ensuring the provision of nutrition care ultimately results in patients actually consuming ONS products, rather than assuming that their prescription is sufficient. This is in agreement with a previous study of 1291 patients in a Swiss hospital, which reported the consumption of ONS as a predictor for eating adequately [20]. This may be due to some component of patient acceptance of the supplements, and perhaps greater acceptance of hospital food. Some evidence suggests that enteral nutrition may stimulate appetite; however these studies refer to naso-gastric feeding, and in general they are poorly designed with small sample sizes [37]. The notion of primary anorexia (due to disease) and secondary anorexia (due to malnutrition), proposed by Allison, is a cycle which seemed to be interrupted with enteral nutrition [38]. To our knowledge, there is little evidence about the effect of ONS on appetite and food intake. While it is possible that there may be a relationship between appetite and consumption of ONS, there is no evidence to support this notion. Clearly ONS is effective in improving total energy and protein intakes and increasing likelihood of patients meeting their requirements, however the factors which determine its consumption must be understood in order to effectively utilise ONS in nutrition interventions.

Previous studies have found a number of predictors of poor oral intake, including higher BMI, modified diets, absence of ONS, poor appetite, requiring feeding assistance, LOS of <8 days or >90 days, and diagnoses of infection, cancer or delirium [17, 19-20]. The logistic regression model in the current study was based on a small sample, due to the exclusion of a
number of patients with missing data. Therefore, the model may be somewhat unstable, and these alternate potential predictors of oral intake should not be disregarded.

Whilst not statistically significant in the multivariate model, univariate logistic regression analysis identified a number of other predictors of oral intake in the current study. These factors should not be discounted, as with a larger sample size they may have reached statistical significance. There was a trend for patients who were categorised as underweight to be more likely to eat adequately for EER compared to all other weight categories (p=0.054). Underweight patients may be more likely to meet estimated requirements because a) requirements are based on body weight, therefore a lower body weight results in lower requirements which are easier to meet; and b) may be more likely to be seen by a dietitian and prescribed supplements (however in this study, a dietitian review or prescribed supplements were not associated with improved oral intakes). Surprisingly, overweight patients were more likely to eat adequately for EER (p=0.040) in univariate analysis. This is contrary to previous findings, which reported higher BMI was associated with inadequate intakes [17, 20]. Overweight patients would have higher requirements and may not be identified as at risk during nutritional screening, which may explain these previous findings. The conflicting results in the current study may be due to small sample size. A modified or restricted diet (compared to general or HPHE diet) trended towards being a predictor for eating inadequately for EER (p=0.056) and was significant for EPR (p = 0.049) in the univariate analyses. This may be due to modified diets restricting patients’ choices, and/or general and HPHE diets providing more energy and protein. Other studies have also reported modified diets as predictors of eating inadequately [17, 20].
Future nutritional interventions for the prevention of PU should focus on ONS as a method of improving the oral intakes of hospitalised patients, as this study and previous findings provide evidence for ONS as a predictor of eating adequately. Other factors previously shown to influence patients’ intakes should also be considered, as well as potential high risk groups such as renal patients.

**Conclusion**

Many hospitalised patients at risk of PU have insufficient oral intakes to meet their requirements. Predictors of eating inadequately were being on the renal ward and having no intake of ONS. Nutritional interventions targeting PU prevention should focus on ONS and consider other factors that may influence oral intake.
References


Figure and Table Legends

Figure 1 – Flow chart of patients’ data analysis

Table 1 – Patient characteristics

Table 2 – Factors related to total energy and protein intakes in patients with reduced mobility

Table 3 – Univariate logistic regression analysis of potential predictors of eating inadequately

Table 4 – Multivariate logistic regression analysis of predictors of eating inadequately
Table 1 – Patient characteristics

<table>
<thead>
<tr>
<th>Patient variable</th>
<th>Total (Mean±SD)</th>
<th>Site A (Mean±SD)</th>
<th>Site B (Mean±SD)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>66.7±16.8</td>
<td>69.9±15.1</td>
<td>65.2±17.5</td>
<td>0.076</td>
</tr>
<tr>
<td>Length of stay (days)</td>
<td>8.8±11.4</td>
<td>12.6±16.9</td>
<td>7.0±6.7</td>
<td>0.015</td>
</tr>
<tr>
<td></td>
<td>5.0 (3.0 – 9.0)(^a)</td>
<td>7.0 (4.0 – 13.5)(^a)</td>
<td>4.5 (3.0 – 8.0)(^a)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td>27.0±9.7</td>
<td>28.1±9.6</td>
<td>26.7±9.8</td>
<td>0.652</td>
</tr>
<tr>
<td>Serum albumin (g/L)</td>
<td>31.9±6.3</td>
<td>28.9±5.7</td>
<td>33.3±6.0</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

\(^a\) Median (interquartile range)
Table 2 – Factors related to total energy and protein intakes in patients at risk of PU

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value (n) %</th>
<th>Total energy intake (kJ) mean±SD</th>
<th>p value</th>
<th>Total protein intake (g) mean±SD</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female (72) 39%</td>
<td></td>
<td>5363±2831</td>
<td>0.041</td>
<td>51±27</td>
<td>0.221</td>
</tr>
<tr>
<td>Male (112) 61%</td>
<td></td>
<td>6273±2992</td>
<td></td>
<td>56±28</td>
<td></td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18 – 50 (26) 14%</td>
<td></td>
<td>7118±3592</td>
<td>0.011a</td>
<td>65±35</td>
<td>0.012a</td>
</tr>
<tr>
<td>51 – 70 (76) 41%</td>
<td></td>
<td>6280±2887</td>
<td>0.06b</td>
<td>56±25</td>
<td></td>
</tr>
<tr>
<td>≥ 71 (82) 45%</td>
<td></td>
<td>5200±2633</td>
<td></td>
<td>48±26</td>
<td></td>
</tr>
<tr>
<td><strong>Eats independently</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>No (20) 11%</td>
<td></td>
<td>4330±3569</td>
<td>0.012</td>
<td>40±35</td>
<td>0.075</td>
</tr>
<tr>
<td>Yes (162) 89%</td>
<td></td>
<td>6069±2809</td>
<td></td>
<td>55±26</td>
<td></td>
</tr>
<tr>
<td><strong>Completess own menu</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>No (44) 25%</td>
<td></td>
<td>4611±3267</td>
<td>0.002</td>
<td>42±31</td>
<td>0.005</td>
</tr>
<tr>
<td>Yes (133) 75%</td>
<td></td>
<td>6342±2687</td>
<td></td>
<td>57±25</td>
<td></td>
</tr>
<tr>
<td><strong>Nausea past 24 hours</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (118) 77%</td>
<td></td>
<td>6225±2943</td>
<td>0.023</td>
<td>57±27</td>
<td>0.006</td>
</tr>
<tr>
<td>Yes (36) 23%</td>
<td></td>
<td>4922±3088</td>
<td></td>
<td>43±28</td>
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<tr>
<td><strong>Vomiting past 24 hours</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (143) 93%</td>
<td></td>
<td>6067±3015</td>
<td>0.008</td>
<td>55±27</td>
<td>0.002</td>
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<td>Yes (10) 7%</td>
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<td>3455±1916</td>
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<td>28±17</td>
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<tr>
<td><strong>Poor appetite past 24 hours</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (131) 85%</td>
<td></td>
<td>6169±2992</td>
<td>0.014</td>
<td>56±27</td>
<td>0.014</td>
</tr>
<tr>
<td>Yes (23) 15%</td>
<td></td>
<td>4500±2828</td>
<td></td>
<td>41±30</td>
<td></td>
</tr>
<tr>
<td><strong>Anti-emetics prescribed</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (163) 89%</td>
<td></td>
<td>6036±2900</td>
<td>0.135</td>
<td>55±27</td>
<td>0.034</td>
</tr>
<tr>
<td>Yes (20) 11%</td>
<td></td>
<td>4985±3375</td>
<td></td>
<td>42±29</td>
<td></td>
</tr>
<tr>
<td><strong>Weekly weight</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (52) 71%</td>
<td></td>
<td>4965±2883</td>
<td>0.009</td>
<td>47±28</td>
<td>0.038</td>
</tr>
<tr>
<td>Yes (21) 29%</td>
<td></td>
<td>7112±3566</td>
<td></td>
<td>63±33</td>
<td></td>
</tr>
<tr>
<td>BMI category</td>
<td>BMI</td>
<td>Mean ± SD</td>
<td>P-value</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>-------------------</td>
<td>------</td>
<td>------------</td>
<td>----------</td>
<td>-----------</td>
<td></td>
</tr>
<tr>
<td>Underweight (8) 14%</td>
<td>9383±3893</td>
<td>0.022c</td>
<td>82±32</td>
<td>0.065c</td>
<td></td>
</tr>
<tr>
<td>Healthy weight (21) 36%</td>
<td>6170±2615</td>
<td></td>
<td>56±29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight (15) 25%</td>
<td>7855±2433</td>
<td></td>
<td>74±20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese (15) 25%</td>
<td>7034±1815</td>
<td></td>
<td>60±18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any intake of ONS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (111) 72%</td>
<td>5749±2856</td>
<td>0.012</td>
<td>52±27</td>
<td>0.007</td>
<td></td>
</tr>
<tr>
<td>Yes (43) 28%</td>
<td>6587±3373</td>
<td></td>
<td>70±27</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\( ^a \) 18 – 50 vs ≥71

\( ^b \) 51 – 70 vs ≥71

\( ^c \) Underweight vs healthy weight

BMI  Body mass index

ONS  Oral nutrition support
Table 3 – Univariate logistic regression analysis of potential predictors of eating inadequately

<table>
<thead>
<tr>
<th>Variable</th>
<th>Intake &lt;75% EER</th>
<th>Intake &lt;75% EPR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crude odds ratio (95% CI)</td>
<td>p value</td>
</tr>
<tr>
<td>Age ≥70 (age &lt;70)</td>
<td>1.3 (0.6 – 3.0)</td>
<td>0.534</td>
</tr>
<tr>
<td>Underweight – BMI &lt;18.5 (other BMI categories)</td>
<td>0.1 (&lt;0.1 – 1.0)</td>
<td>0.054</td>
</tr>
<tr>
<td>Overweight – BMI 25.0 – 29.9 (other BMI categories)</td>
<td>0.3 (&lt;0.1 – 0.9)</td>
<td>0.040</td>
</tr>
<tr>
<td>Renal ward (other wards)</td>
<td>4.0 (1.2 – 13.1)</td>
<td>0.024</td>
</tr>
<tr>
<td>Renal failure (No renal failure)</td>
<td>3.0 (1.1 – 8.0)</td>
<td>0.031</td>
</tr>
<tr>
<td>Restricted diet (general/HPHE diet)</td>
<td>2.4 (1.0 – 5.6)</td>
<td>0.056</td>
</tr>
<tr>
<td>No intake of ONS (any intake ONS)</td>
<td>5.0 (1.3 – 19.6)</td>
<td>0.021</td>
</tr>
<tr>
<td>Poor appetite ratings (good appetite ratings)</td>
<td>1.7 (0.6 – 4.8)</td>
<td>0.335</td>
</tr>
</tbody>
</table>

*a Renal failure as a primary diagnosis or co-morbidity

b Poor appetite ratings included very poor, poor; good appetite ratings included fair, good, very good

HPHE High Protein High Energy
ONS Oral Nutrition Support
Table 4 – Multivariate logistic regression analysis of predictors of eating inadequately

<table>
<thead>
<tr>
<th>Variable (reference value)</th>
<th>Intake &lt;75% EER&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Intake &lt;75% EPR&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adjusted odds ratio (95% CI)</td>
<td>p value</td>
</tr>
<tr>
<td>Renal ward (other wards)</td>
<td>4.1 (1.2 – 14.0) 0.027</td>
<td></td>
</tr>
<tr>
<td>No intake of ONS (any intake ONS)</td>
<td>5.1 (1.2 – 21.2) 0.023</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Regression based on 93 cases. Cox and Snell $R^2 = 0.122$; Nagelkerke $R^2 = 0.163$; Homer and Lemeshow Test: $x^2 = 0.005$, df = 2, p = 0.998.  
<sup>b</sup> Regression based on 93 cases. Cox and Snell $R^2 = 0.175$; Nagelkerke $R^2 = 0.234$; Homer and Lemeshow Test: $x^2 = 1.841$, df = 2, p = 0.398.
Figure 1 – Flow chart of patient inclusion

Patients recruited to the study
n=241

- Patients with incomplete nutritional intake data
  n=57 (23.7%) excluded from analysis

- Patients with complete nutritional intake data
  n=184 (76.3%)

- Patients with missing anthropometric data
  excluded from analysis
  n=91 (49.5%)

- Patients with sufficient anthropometric data to calculate EER and EPR
  n=93 (50.5%)

Data analysis of predictors of eating inadequately

Data analysis of total energy and protein intake