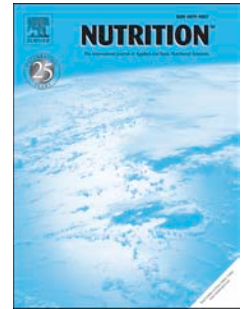


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Nutritional intakes of patients at risk of pressure ulcers in the clinical setting

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28 **Authorship**

29 Shelley Roberts: Conception and design of the study; collection, assembly, analysis and
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31 manuscript.

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35 Michael Leveritt and Merrilyn Banks: Interpretation of data; drafting and revision of the
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40 **Conflict of Interest Statement**

41 The authors declare no conflict of interest.

42 ABSTRACT

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

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

54 *Results:* Mean energy and protein intakes of the 184 patients were 5917±2956kJ and 54±28g,
55 respectively. Estimated energy and protein requirements were calculated for 93 patients. Only
56 45% (n=42) and 53% (n=49) met ≥75% of estimated energy and protein requirements,
57 respectively. In multivariate analysis, patients on the renal ward were 4.1 and 4.6 times more
58 likely to be eating inadequately for energy and protein, respectively (p<0.05). Patients who
59 consumed any amount of oral nutrition support were 5.1 and 15.5 times more likely be eating
60 adequately for energy and protein, respectively (p< 0.05).

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

63 *Keywords:* Pressure ulcer; nutrition; oral intake, hospital.

64 **Introduction**

65 Malnutrition is a common and costly problem in the hospital setting, affecting as many as 20
66 – 50% of patients [1-2]. Its consequences are severe, including impaired immunity, delayed
67 recovery and healing, loss of muscle mass and function and poorer quality of life [3].
68 Malnutrition increases hospital length of stay (LOS) and hospital costs amongst various
69 groups of patients [4-6], and is also **directly associated with the development and severity**
70 **of pressure ulcers (PU)** [7-8].

71 PU place a large burden on both the patient and the healthcare system. The prevalence of PU
72 ranges from around 5 – 30% of all hospitalised patients [7, 9]. For the patient, PU result in
73 numerous medical complications, including increased risks of infection and mortality, and
74 lengthy healing times [3, 10]. Other problems arising from PU include pain, discomfort,
75 decreased mobility and independence, wound exudates and odour and social isolation [11-
76 12]. PU result in severe consequences to the health care system, including increased hospital
77 costs and LOS [13-14].

78 Malnutrition has been associated with at least twice the odds ratio of having a PU [7].
79 Mechanisms by which malnutrition increases PU risk may be related to body composition,
80 skin and tissue integrity, and mobility [3, 11, 15]. Low body weight may be associated with
81 PU due to an increase in bony prominences and less fat tissue to distribute pressure [11].
82 Malnutrition may also result in impaired skin integrity and resistance to pressure due to
83 decreased nutrient availability for tissue maintenance and repair [3]. Furthermore,
84 malnutrition is associated with decreased mobility, which is an independent risk factor for PU
85 [3, 15].

86 Oral or enteral nutritional supplementation in groups of older patients deemed at risk of PU
87 may contribute to PU prevention [16]. Although most studies have failed to reach statistical
88 significance individually, likely due to small sample sizes, a meta-analysis found that the
89 provision of oral or enteral nutrition support resulted in a 26% lower incidence of PU in high
90 risk patients compared to routine care [16].

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

100 **Materials and Methods**

101 Study overview

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

106 Setting

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

112 Study participants

113 Patients were included in the study if they met the following eligibility criteria: able to
114 provide consent (aged over 18 years, cognitively intact); at risk of pressure ulcers due to
115 restricted mobility (i.e. use of mobility aid such as walking stick, frame, wheelchair; or
116 presence of mobility-restricting equipment such as intravenous (IV) lines, oxygen therapy, as
117 determined from medical notes); and length of stay ≥ 3 days. Reduced mobility was chosen as
118 a conservative inclusion criteria to identify patients at risk of PU, as it is a widely recognised
119 risk factor and strong predictor of PU in the clinical setting [10, 21-22]. The use of a PU risk
120 assessment tool, such as the Braden or Norton scale, was not used to identify at risk patients

121 as they are shown to have insufficient predictive validity and poor reliability [23-26]. Patients
122 could not be recruited into the study more than once. Eligible patients were provided with a
123 participant information sheet, and informed consent was obtained from agreeable patients.

124 Tool Development and Pilot Testing

125 The conceptual framework that underpinned data collection was developed from a review of
126 the literature and clinical experience. A number of predictor variables were identified and
127 grouped into categories, including patient related (eg. self-feeding ability; co-morbidities; level
128 of mobility; and nutrition impacting symptoms such as chewing or swallowing problems,
129 nausea, vomiting or mouth ulcers), service related (eg. hospital diet; dietetic input; food and
130 supplement provision), and care delivery related (eg. feeding assistance; malnutrition risk
131 assessment completion) factors. A semi-structured observational tool and a chart audit tool
132 were developed using this framework to determine the data to be collected. The tools were
133 assessed by five clinicians and academics with expertise in this area of research. The tools
134 were piloted and modified prior to data collection. Four researchers (including one author)
135 were involved in data collection, and undertook training in the use of the data collection
136 forms. A pilot study of ten patients was conducted prior to data collection to test intra- and
137 inter-rater reliability of the data collectors. Both intra-rater and inter-rater reliability were
138 >95%.

139 Data Collection

140 *Patient observations*

141 Using a semi-structured observational tool, each patient was observed for 24 hours
142 (commencing at 0700 hours). Observations were performed by three data collectors across

143 three 8-hour shifts. Patients' oral intake was recorded for the 24 hour duration of data
144 collection by observing each patient's plate at the end of each meal (breakfast, lunch and
145 dinner). Researchers indicated the amount (none, $\frac{1}{4}$, $\frac{1}{2}$, $\frac{3}{4}$, all) of each component of a
146 standard sized meal consumed on the observational data form. This method of observed food
147 intake has previously been shown to be a valid and reliable method of collecting dietary
148 intake data [19, 27]. Patients' menu slips were collected to determine the specific meals and
149 food items they received at each meal. At mid-meals (morning tea, afternoon tea, supper),
150 researchers observed patients' food and fluid intake, including any supplements consumed.

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

157 *Chart audit*

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

164 Data analysis

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

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

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

185 Patients were divided into two groups for each set of analysis. For analysis of energy intakes,
186 patients were divided into a) inadequate intake (i.e. consuming <75% EER) and b) adequate
187 intake (i.e. consuming \geq 75% of EER). These values were chosen as previous research has

188 shown that patients who consumed <75% EER lost weight during their admission, suggesting
189 that this level of intake is inadequate for patients to maintain their body weight [18]. Whilst
190 there is no clinical evidence to suggest a threshold for adequacy of protein intake, the amount
191 of protein required to maintain muscle mass and other bodily functions is likely to be related
192 to the amount of energy required to maintain body weight. For this reason, patients were also
193 divided into consuming <75% and \geq 75% of EPR for analysis of protein intakes. Univariate
194 logistic regression analysis was conducted to determine potential predictors of eating
195 inadequately, using the conceptual framework of theoretically important variables.
196 Significant factors were then entered in a multiple logistic regression model. For all
197 associations, significance was set at $p \leq 0.05$.

198 Results

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

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

213 The mean total energy intake was 5917 ± 2956 kJ, and mean total protein intake was 54 ± 28 g
214 protein. There were significant differences in energy and protein intakes in a number of
215 variables, as shown in Table 2. Variables for which there were no differences in energy and
216 protein intakes included site, ward, diagnosis, LOS, history of past or present PU, mobility
217 status, dietitian referral or review, documented prescription of ONS, hospital diet,
218 malnutrition screening tool (MST) score, serum albumin, reported recent weight loss, or other
219 nutrition impacting symptoms such as chewing or swallowing difficulties.

220 Patients' subjective appetite rating (very poor, poor, fair, good, very good) was related to
221 both energy and protein intake, with improved appetite being associated with higher energy
222 ($p < 0.001$, $F = 7.503$) and protein ($p < 0.001$, $F = 6.686$) intakes.

223 Energy and protein requirements were estimated for 93 patients. Mean requirements were
224 8271 ± 1665 kJ and 69.4 ± 16.0 g protein. On average, patients met $75.4 \pm 39.3\%$ (median 72.0%,
225 IQR 48.0 – 93.8%) of their EER and $80.6 \pm 43.3\%$ (median 76.3%, IQR 53.5 – 102.1%) of
226 EPR. Only 45.2% ($n = 42$) of patients met $\geq 75\%$ of their EER, whilst 53.1% ($n = 52$) met $\geq 75\%$
227 of their EPR.

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

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

239 Discussion

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

244 Mean energy and protein intakes in this study are comparable to previous studies describing
245 the intakes of general hospitalised patients [17, 19-20, 32]. Many patients at risk of PU
246 appear to be eating inadequately in the hospital setting. In fact, only 42 of the 93 patients
247 (45.2%) met $\geq 75\%$ of their EER, whilst only 49 (52.7%) met $\geq 75\%$ of their EPR. This
248 phenomenon is reflected in studies of hospitalised patients in general, with a number of
249 studies reporting inadequate energy and protein intakes to meet estimated requirements in
250 patients [17, 19-20].

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

259 Whilst there were a number of factors associated with differences in total energy and protein
260 intakes, only two variables remained independent predictors of eating inadequately after

261 multivariate logistic regression analysis. These were: 1) being admitted to the renal ward, and
262 2) having no intake of ONS.

263 Admission to the renal ward was an independent predictor for eating inadequately, where
264 patients were over four times more likely to consume <75% of their EER and EPR. This may
265 be due to a number of reasons. Firstly, disease-related factors are likely to play a role, as renal
266 failure was a potential predictor of eating inadequately in univariate analyses. Chronic renal
267 failure (CRF) and dialysis both increase EER, making it more difficult to reach an adequate
268 intake [28, 33]. Furthermore, food intake may be decreased due to anorexia, nausea or
269 vomiting related to uraemic toxicity [33-34]. Therapeutic diets (such as low salt, low
270 potassium) may restrict patients' food choices and consequently, their intake. As renal failure
271 did not reach statistical significance in the multivariate model, whilst the renal ward did, there
272 appears to be other (i.e. non-disease related) factors involved in whether these patients meet
273 their requirements. These may be related to hospital foodservice or ward practices in renal
274 wards, such as meal times, availability of staff for set up / assistance with feeding, timing of
275 meals, or missed meals due to extended periods of time off the ward (for example in dialysis).
276 Inadequate nutritional intake is of particular concern to this patient group, as malnutrition is
277 common in patients with chronic renal failure [33]. It could be hypothesised that the high
278 prevalence of malnutrition and prolonged periods of immobility (i.e. during dialysis sessions)
279 in renal patients may increase the likelihood of PU. One study found that renal insufficiency
280 (measured by elevated creatinine) was an independent risk factor for PU after multivariate
281 regression analysis in the intensive care unit (ICU) [35]. However, another study failed to
282 find this association among hip fracture patients admitted to an orthopaedic ward [36]. Future
283 research should further investigate whether renal patients in hospital wards (outside the ICU)
284 are at higher risk of PU than other patient groups.

285 The consumption of any amount of oral nutrition support was associated with adjusted OR of
286 5.1 and 15.5 of meeting $\geq 75\%$ of EER and EPR, respectively. Patients were included in this
287 category regardless of the actual amount of nutrition support consumed on the observation
288 day. Being prescribed ONS or receiving a dietitian consultation were not associated with
289 eating adequately, which highlights the importance of ensuring the provision of nutrition care
290 ultimately results in patients actually consuming ONS products, rather than assuming that
291 their prescription is sufficient. This is in agreement with a previous study of 1291 patients in
292 a Swiss hospital, which reported the consumption of ONS as a predictor for eating adequately
293 [20]. This may be due to some component of patient acceptance of the supplements, and
294 perhaps greater acceptance of hospital food. Some evidence suggests that enteral nutrition
295 may stimulate appetite; however these studies refer to naso-gastric feeding, and in general
296 they are poorly designed with small sample sizes [37]. The notion of primary anorexia (due to
297 disease) and secondary anorexia (due to malnutrition), proposed by Allison, is a cycle which
298 seemed to be interrupted with enteral nutrition [38]. To our knowledge, there is little evidence
299 about the effect of ONS on appetite and food intake. While it is possible that there may be a
300 relationship between appetite and consumption of ONS, there is no evidence to support this
301 notion. Clearly ONS is effective in improving total energy and protein intakes and increasing
302 likelihood of patients meeting their requirements, however the factors which determine its
303 consumption must be understood in order to effectively utilise ONS in nutrition interventions.

304 Previous studies have found a number of predictors of poor oral intake, including higher
305 BMI, modified diets, absence of ONS, poor appetite, requiring feeding assistance, LOS of < 8
306 days or > 90 days, and diagnoses of infection, cancer or delirium [17, 19-20]. The logistic
307 regression model in the current study was based on a small sample, due to the exclusion of a

308 number of patients with missing data. Therefore, the model may be somewhat unstable, and
309 these alternate potential predictors of oral intake should not be disregarded.

310 Whilst not statistically significant in the multivariate model, univariate logistic regression
311 analysis identified a number of other predictors of oral intake in the current study. These
312 factors should not be discounted, as with a larger sample size they may have reached
313 statistical significance. There was a trend for patients who were categorised as underweight to
314 be more likely to eat adequately for EER compared to all other weight categories ($p=0.054$).
315 Underweight patients may be more likely to meet estimated requirements because a)
316 requirements are based on body weight, therefore a lower body weight results in lower
317 requirements which are easier to meet; and b) may be more likely to be seen by a dietitian
318 and prescribed supplements (however in this study, a dietitian review or prescribed
319 supplements were not associated with improved oral intakes). Surprisingly, overweight
320 patients were more likely to eat adequately for EER ($p=0.040$) in univariate analysis. This is
321 contrary to previous findings, which reported higher BMI was associated with inadequate
322 intakes [17, 20]. Overweight patients would have higher requirements and may not be
323 identified as at risk during nutritional screening, which may explain these previous findings.
324 The conflicting results in the current study may be due to small sample size. A modified or
325 restricted diet (compared to general or HPHE diet) trended towards being a predictor for
326 eating inadequately for EER ($p=0.056$) and was significant for EPR ($p = 0.049$) in the
327 univariate analyses. This may be due to modified diets restricting patients' choices, and/or
328 general and HPHE diets providing more energy and protein. Other studies have also reported
329 modified diets as predictors of eating inadequately [17, 20].

330 Future nutritional interventions for the prevention of PU should focus on ONS as a method of
331 improving the oral intakes of hospitalised patients, as this study and previous findings
332 provide evidence for ONS as a predictor of eating adequately. Other factors previously shown
333 to influence patients' intakes should also be considered, as well as potential high risk groups
334 such as renal patients.

335 **Conclusion**

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

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440 **Figure and Table Legends**

441 Figure 1 – Flow chart of patients' data analysis

442 Table 1 – Patient characteristics

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

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

445 Table 4 – Multivariate logistic regression analysis of predictors of eating inadequately

Table 1 – Patient characteristics

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

^a Median (interquartile range)

Table 2 – Factors related to total energy and protein intakes in patients at risk of PU

Variable	Value (n) %	Total energy intake (kJ) mean±SD	p value	Total protein intake (g) mean±SD	p value
Gender	Female (72) 39%	5363±2831	0.041	51±27	0.221
	Male (112) 61%	6273±2992		56±28	
Age	18 – 50 (26) 14%	7118±3592	0.011 ^a	65±35	0.012 ^a
	51 – 70 (76) 41%	6280±2887	0.06 ^b	56±25	
	≥ 71 (82) 45%	5200±2633		48±26	
Eats independently	No (20) 11%	4330±3569	0.012	40±35	0.075
	Yes (162) 89%	6069±2809		55±26	
Completes own menu	No (44) 25%	4611±3267	0.002	42±31	0.005
	Yes (133) 75%	6342±2687		57±25	
Nausea past 24 hours	No (118) 77%	6225±2943	0.023	57±27	0.006
	Yes (36) 23%	4922±3088		43±28	
Vomiting past 24 hours	No (143) 93%	6067±3015	0.008	55±27	0.002
	Yes (10) 7%	3455±1916		28±17	
Poor appetite past 24 hours	No (131) 85%	6169±2992	0.014	56±27	0.014
	Yes (23) 15%	4500±2828		41±30	
Anti-emetics prescribed	No (163) 89%	6036±2900	0.135	55±27	0.034
	Yes (20) 11%	4985±3375		42±29	
Weekly weight	No (52) 71%	4965±2883	0.009	47±28	0.038
	Yes (21) 29%	7112±3566		63±33	

BMI category	Underweight (8) 14%	9383±3893	0.022 ^c	82±32	0.065 ^c
	Healthy weight (21) 36%	6170±2615		56±29	
	Overweight (15) 25%	7855±2433		74±20	
	Obese (15) 25%	7034±1815		60±18	
Any intake of ONS	No (111) 72%	5749±2856	0.012	52±27	0.007
	Yes (43) 28%	6587±3373		70±27	

^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

Variable (reference value)	Intake <75% EER		Intake <75% EPR	
	Crude odds ratio (95% CI)	p value	Crude odds ratio (95% CI)	p value
Age ≥ 70 (age <70)	1.3 (0.6 – 3.0)	0.534	1.3 (0.6 – 2.9)	0.559
Underweight – BMI <18.5 (other BMI categories)	0.1 (<0.1 – 1.0)	0.054	0.2 (<0.1 – 1.4)	0.104
Overweight – BMI 25.0 – 29.9 (other BMI categories)	0.3 (<0.1 – 0.9)	0.040	0.4 (0.1 – 1.3)	0.137
Renal ward (other wards)	4.0 (1.2 – 13.1)	0.024	4.1 (1.3 – 12.6)	0.014
Renal failure ^a (No renal failure)	3.0 (1.1 – 8.0)	0.031	2.2 (0.9 – 5.6)	0.090
Restricted diet (general/HPHE diet)	2.4 (1.0 – 5.6)	0.056	2.4 (1.0 – 5.7)	0.049
No intake of ONS (any intake ONS)	5.0 (1.3 – 19.6)	0.021	13.9 (1.7 – 112.4)	0.013
Poor appetite ratings ^b (good appetite ratings)	1.7 (0.6 – 4.8)	0.335	2.5 (0.9 – 7.2)	0.085

^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

Variable (reference value)	Intake <75% EER ^a		Intake <75% EPR ^b	
	Adjusted odds ratio (95% CI)	p value	Adjusted odds ratio (95% CI)	p value
Renal ward (other wards)	4.1 (1.2 – 14.0)	0.027	4.6 (1.3 – 15.6)	0.016
No intake of ONS (any intake ONS)	5.1 (1.2 – 21.2)	0.023	15.5 (1.8 – 132.8)	0.013

^a Regression based on 93 cases. Cox and Snell $R^2 = 0.122$; Nagelkerke $R^2 = 0.163$; Homer and Lemeshow Test: $\chi^2 = 0.005$, $df = 2$, $p = 0.998$. ^b Regression based on 93 cases. Cox and Snell $R^2 = 0.175$; Nagelkerke $R^2 = 0.234$; Homer and Lemeshow Test: $\chi^2 = 1.841$, $df = 2$, $p = 0.398$.

Figure 1 – Flow chart of patient inclusion

