IMPROVING EARLY NUTRITIONAL INTERVENTION IN HOSPITALISED PATIENTS; LABORATORY TEST (PREALBUMIN) VERSUS ROUTINE CLINICAL ASSESSMENT

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ABSTRACT

Background

Malnutrition in hospitalised patients occurs in 30% of Australasian Hospitals. There are an estimated fifty malnutrition-screening tools currently available, although no single screening tool is universally accepted as gold standard in all settings. The Waitemata District Health Board (WDHB) hospitals: North Shore hospital (NSH) and Waitakere hospital utilise the Malnutrition Universal Screening Tool (MUST). A hospital dietitian audit performed in 2009 at NSH identified as few as 8% of patients were screened using MUST. Universal screening on hospital admission incorporating prealbumin has been proposed as a more effective method for early identification of patients at risk of disease-related malnutrition.

Aims

To evaluate whether universal prealbumin screening increases the number of patients identified and referred to a dietitian for comprehensive disease-related malnutrition assessment.

Method

A two-phase observational cohort study was conducted utilising consecutively admitted patients to: two acute surgical, one acute orthopaedic and two acute medical wards from February to April 2013. Phase I: Observational stage recorded dietetic inpatient referral data for patients screened by MUST triggering a dietetic referral. Phase II: Research protocol, patients were selected using electronic notes programme Concerto^{TM}. Patients that met the research inclusion criteria had admission blood samples tested for prealbumin and C-reactive protein (CRP) within 36 hours post admission. The researcher examined whether abnormal prealbumin level <0.2 g/L triggered a prompt referral to a dietetic assessment. Dietetic inpatient referral data set was repeated for phase II as in phase I.

Results

Phase I, 970 patients were admitted during a 25-day control period. Patient referral pathway was either through MUST or clinical professional referral with 28% of patients having a completed MUST in their clinical notes, a total of 7.8% (76/970) of patients were referred.

Phase II, 776 patients were admitted during a 22-day period, 564 patients met inclusion criteria and were selected for screening. Test results indicated 27% (155/564) had abnormal prealbumin results ranged from 0.03 g/L to 0.54 g/L; these patients deemed at risk of malnutrition. A total of 43 patients were referred to a dietitian through either MUST or clinical professional referral. 30% of those 43 patients assessed by a dietitian had abnormal prealbumin results, although 70% of those did not initiate a dietetic referral. Of 43 patients referred 51% had a completed MUST screen recorded; however nil patients referrals were triggered by abnormal prealbumin results.

Conclusion

In our study prealbumin results were found to be a sensitive marker of malnutrition risk with 27% of screened patients deemed at risk of malnutrition. This is in-line with international prevalence rates of hospital malnutrition set between 20-60%. However, despite ours findings it was identified that the clinicians poorly recognise hospital malnutrition, as patients were not referred based on abnormal pre-albumin levels.

Key words

Prealbumin, Hospital Malnutrition, MUST, malnutrition screening, visceral proteins, protein energy malnutrition, disease-related malnutrition.

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LIST OF ABBREVIATIONS

CRP	C-Reactive Protein
RBP	Retinol Binding Protein
NZ	New Zealand
PEM	Protein Energy Malnutrition
RD	Registered Dietitian
BMI	Body Mass Index
LBM	Lean Body Mass
ESPEN	European Society for Parental and Enteral Nutrition
WDHB	Waitemata District Health Board
NSH	North Shore Hospital
NHS	National Health Service
DM	Diabetes Mellitus
ICU	Intensive care unit
~	Approximately
DNZ	Dietitians New Zealand
NICE	National Institute for Health and Care Excellence
REE	Resting Energy Expenditure
ANCDS	The Australasian Nutrition Care Day Survey
BAPEN	British Association for Parental and Enteral Nutrition
MUST	Malnutrition Universal Screening Tool
NICE	National Institute for Health and Care Excellence
REE	Resting Energy Expenditure
ANCDS	The Australasian Nutrition Care Day Survey
NHI	National Health Index
USA	United States of America
SD	Standard Deviation

STRUCTURE OF THESIS

Chapter 1 comprises of the introduction section, which discusses the rationale supporting this research in New Zealand. Elaborating on the current international research and the meagre New Zealand literature on acute malnutrition in the hospital setting. Furthermore this section states the research study aims, primary objectives and hypothesis.

In Chapter 2 the first stage of the literature review incorporates the search strategy conducted using the Massey University library search programme linked to the electronic databases: Web of Knowledge, Scopus, Cochrane library, PubMed and Science Direct. The terms or phrases used in the search were predominately: prealbumin, transthyretin, albumin, transferrin, retinol-binding protein, hospital malnutrition, disease-related malnutrition, malnutrition screening tools: MNA, MUST, SGA, PG-SGA and DNA, malnutrition and evidence based dietetic practice. The databases were searched for years 1975-2013. Titles and abstracts were examined and when found to be relevant, meeting the inclusion criteria of this literature review then the full article was obtained. The inclusion criteria involved relevant internationally published studies, surveys, population studies and evidence based practice guidelines pertaining to malnutrition/nutrition screening in the hospital based setting. Furthermore evidence from studies was sought in the literature pertaining to the use of validated malnutrition screening tools used in conjunction with biochemical visceral protein analysis to strengthen the malnutrition diagnosis. Within the literature review sections on current use of various biochemical markers of malnutrition risk and malnutrition screening tools is discussed.

A description of the methodology of this research project is discussed in chapter 3 which involved the: research study design, routine clinical nutritional assessment (MUST), inclusion/exclusion criteria, ethical approval and consent,

funding, methods and materials, proposal presentation and the research process in two phases.

The statistical analysis overview is presented in chapter 3 with the remainder of the statistical analysis in the results section in chapter 4.

The results have been statistically conducted using IBM® SPSS® Statistics *20 (*IBM Corp. Released 2011. IBM SPSS Statistics for Mac, Version 20.0. Armonk, NY: IBM Corp) and tabulated into tables and graphs shown in chapter 4. Demographic analysis is presented in mean, range and standard deviation along with the proportion (%) in each age and gender with prealbumin in mean, standard deviation, proportion and correlation between prealbumin and CRP.

The discussion in chapter 5 discusses these results and findings both statistically and in conjunction with national and international literature regarding the results of this research.

The conclusion, chapter 6 examines the strengths and limitations of this research as well as identifying areas for continued research in: malnutrition risk and disease-related malnutrition education as well as the advantages and potential concepts for further research to advance knowledge on disease-related malnutrition in NZ hospitals.

MALNUTRITION RISK IN HOSPITALISED PATIENTS

1.0. INTRODUCTION

Despite the growing body of evidence pertaining to the clinical and financial burden that hospital malnutrition imposes on societal healthcare, little has changed to improve the status quo over the past few decades. Research has consistently shown that malnutrition is often misconstrued, undiagnosed and hence untreated in the hospital setting. This equates to an increased risk of morbidity and mortality (Adams, Bowie, Simmance, Murray, & Crowe, 2008; Parrish, 2006; Stratton et al., 2004). Over the past thirty years an increasing amount of international literature has been published, proposing a broad spectrum of hospital malnutrition rates, estimated anywhere between 20-60% (Almeida, Correia, Camilo, & Ravasco, 2012; Freijer et al., 2013; McWhirter & Pennington, 1994; Norman, Pichard, Lochs, & Pirlich, 2008; Robinson et al., 2003; Schindler et al., 2010; Stratton et al., 2004). Evidence reveals acute malnutrition rates; vary substantially between hospitals and/or the country of origin making it increasingly complex to make exact judgement on international acute care prevalence rates of malnutrition (De Luis et al., 2006; Kubrak & Jensen, 2007; Robinson et al., 2003). The disparity in malnutrition rates has been largely proposed as to the setting in which malnutrition screening is typically performed, notwithstanding the type of malnutrition screening tool and the contradiction of nutrition parameters used internationally to define malnutrition; as the detection of patient malnutrition is well known to have many challenging facets (Alberda, Graf, & McCargar, 2006; Almeida et al., 2012; Freijer et al., 2013). These facets include: in-accuracy of data collection, completed patient information, the patients' level of consciousness and an inability to communicate or be weighed. Furthermore, the malnutrition diagnosis is often confounded by the idiom that denotes malnutrition. These idiom's of

malnutrition may include: sarcopenia (aetiology unknown: age related loss of skeletal muscle mass) starvation, under-nutrition, anorexia and/or cachexia (a complex metabolic syndrome associated with underlying illness and characterised by loss of muscle with or without loss of fat mass and inflammation) rather than simple nutrient deprivation (Dawson, Taylor, & Favaloro, 2008; Evans et al., 2008; Yaxley & Miller, 2011). Although potentially comprehensive in individual aetiology, a generalised clinical differential diagnosis of malnutrition tends to be less frequently defined. This often hinders a focused appropriate nutrition diagnosis and hence the undertaking of a nutritional intervention, which is less than ideal in a patient, centred clinical setting (Baker et al., 1982; Yaxley & Miller, 2011). Widely reported in the literature are the negative outcomes associated with the newer term of secondary malnutrition referred to as disease-related malnutrition. Disease-related malnutrition is defined as malnutrition caused/associated by the severity of illness contributing to under-nutrition or alternatively protein energy malnutrition (PEM) comprehensively defined in the more current research as insufficient protein and/or energy intake (Alberda et al., 2006; Freijer et al., 2013; Jensen et al., 2010; Stratton, Green, & Elia, 2003). A considerable amount of literature has been published on the clinical outcomes associated with disease-related malnutrition which comprise of: impaired wound healing and immune dysfunction, increased rate of pressure sores, muscle wasting and increased susceptibility to further clinical and functional complications (Barker, Gout, & Crowe, 2011a; Jensen et al., 2010; Pennington & McWhirter, 1997; Tappenden et al., 2013). When these negative outcomes are compounded a profound effect on patient outcome often results in increased length of hospital stay, higher treatment costs and re-admission rates with greater morbidity and mortality (Barker et al., 2011; Correia & Waitzberg, 2003). Stratton et al. (2003)

makes specific mention that nutritional intake is potentially the most significant factor contributing to disease-related malnutrition of all age groups.

Increasingly acknowledged is the fast approaching public health challenge of an ageing population affecting economies worldwide, with those of advancing age typically the highest consumers of health and medical expenditure. A key social determinant of good health and wellbeing in the elderly is continued maintenance of a well-balanced nutritional intake and a healthy weight range. A well known longitudinal study conducted in New Zealand (NZ), The LILACS NZ (Life and Living in Advanced Age: A Cohort Study in New Zealand'), recognised that with the burgeoning rates of advancing age adults, those 65+ years transpires the increasing demands on health related needs (Hayman et al., 2012). Increasing literature is emerging that recognises the distinct association of advancing age and the predicted increase in disease-related malnutrition (Vandewoude, Alish, Sauer, & Hegazi, 2012). Although this demographic transition is not unique to NZ, the population projections unveiled that over one third of NZ population will be 65+ by 2030 (Statistics New Zealand, 2000). The number of those people aged 65+ is set to double and likely to be 1.18-1.25 million by 2036, with the greatest growth between 2011 and 2036 (Statistics New Zealand, 2006, 2012). Statistics New Zealand (2013b) state that the 65+ population has increased by 48,200 over the last two years in those born between 1946-1965 and make up 635,200 (14%) of the population. A recently published report: 'Affording our futures' examined this country's long term fiscal position in-line with the inherent costs associated with an ageing population (The Treasury, 2013). One of the two areas of significant growth highlighted was healthcare spending, which is central to increasing government expenditure on healthcare, shown in figure 1.1. (The Treasury, 2013).

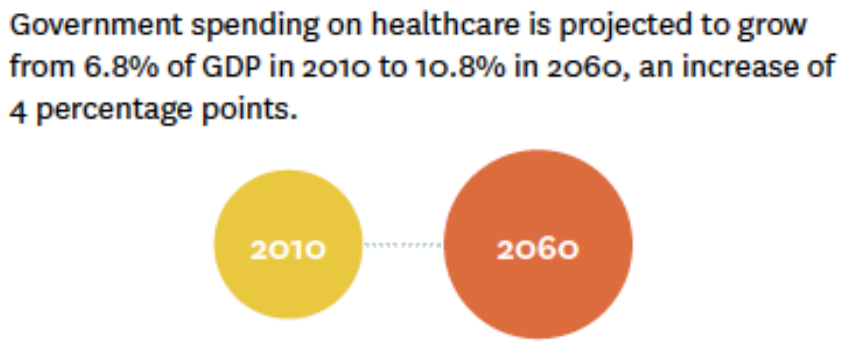

Figure 1.1. Government projection spending on healthcare (The Treasury, 2013).

While the key determinants of insecurity, social issues, health status including oral health and denture care, polypharmacy which potentiates drug/nutrient reactions, disease-related illness and nutrient deficiency influence malnutrition, it is the older age group that are clearly more at risk of malnutrition than any other group. Consequently, older adults often enter the hospital system at lower than optimal nutrition. Sarcopenia is regularly unrecognised as a root cause of adverse health outcomes in the elderly, resulting in increased re-admission rates, length of hospital stay, morbidity and mortality rates, and escalating financial burden (Vandewoude et al., 2012).

The International economics of disease-related malnutrition.

Internationally reported rates of undiagnosed disease-related malnutrition are proposed as largely due to the inadequacy of screening practices at the time of hospital admission. Escalating healthcare expenses can be attributed to the secondary complications of malnutrition including: increased medical complications, length of hospital stay, increased wound cares and infection rates all requiring additional medical intervention and increased pharmaceutical requirements. An example of the significant financial burden was demonstrated by Russell et al. (2011) in Ireland's economic health crisis study related to disease-related malnutrition. Results illustrated the significant financial burden to be estimated at £1.4 billion, representing 10% of their health care budget

(Russell et al., 2011). Furthermore it was found that 70% of the total cost was either in the acute hospital setting or residential care, with as little as 3% spent on nutritional support in both settings (Rice & Normand, 2012). Similar comparative results published in 2010, by the British Association of Parental and Enteral Nutrition (BAPEN) alleged that three million individuals at any one time are undernourished in the United Kingdom (UK) (BAPEN, 2013a). In monetary terms this equates to the estimated financial/economic burden of £13 billion annually (Brotherton, Simmons, & Stroud, 2010). Dr Mike Stroud, chair of BAPEN postulates that a saving of 1% could equate to £130 million saving through identification and an appropriate nutrition intervention (Brotherton et al., 2010). Additionally Stroud suggested that nutrition care is the fourth largest potential source of cost saving in the National Health Service (NHS), stating an improvement in nutrition therapy makes sound financial sense (Brotherton et al., 2010).

A study by Schindler et al. (2010) investigated the nutritional risk in a major European longitudinal study. The 'nutritionDay Survey' examined the malnutrition rates of 21,007 patients at 325 hospitals in 25 countries (Schindler et al., 2010). The study utilised standardised questionnaires and scrutinised the hospital malnutrition screening practices. The examining of the screening practices included: the types of assessment tools used, whether the patients energy requirements and nutrient intake were assessed and monitored at admission and throughout their hospital stay (Schindler et al., 2010). The outcomes suggested that across all of the European hospitals in this study, universally recognised and validated malnutrition screening tools were infrequently used and that locally derived screening was more preferable (Schindler et al., 2010). The criteria of body mass index (BMI <18.5 kg/m^2), age, unintentional weight loss, diminished food intake from the previous week were the main outcome measures used to assess malnutrition risk (Schindler et al.,

2010). Of the patients subjectively measured the results indicated that 27% ($p<0.0001$) were either undernourished or at a higher risk of being malnourished (Schindler et al., 2010). The multivariate analysis identified at risk patients, were more likely: 1) be of more advanced age, 2) have had significant weight loss within the past three months, 3) have had an overall lower dietary intake and 4) had a lower BMI (Schindler et al., 2010). However, it was noted that the nutritional risk was substantially different between the European regions and countries, questioning the variation in the malnutrition criteria of different screening protocols being utilised (Schindler et al., 2010). Results indicated that the nutrition risk was lowest in Hungary, Austria and Germany. Whilst the highest rates were found in the United Kingdom, the rest of the Western and Southern regions, the countries of Central and Eastern Europe (CCEE) and the Nordic countries (Schindler et al., 2010).

The Netherlands 'Cost of illness study 'also considered the economic costs of disease-related malnutrition. The study measured the annual prevalence of disease-related malnutrition in-line with the ESPEN guidelines by combining the Dutch National Prevalence of Health Care Problems (LPZ-Landelijke Prevalentie Zorgproblemen) (Freijer et al., 2013). The cost of illness analysis focused on the direct costs including prevention, diagnoses, intervention, and rehabilitation (Freijer et al., 2013). Results found the estimated expenditure and additional costs associated with the management of disease-related malnutrition, was €1.9 billion in 2011 (Freijer et al., 2013). Furthermore results indicated there was an increase in expenditure by four times for those of at-least 60 years in age, which equated to an additional €1.5 billion in expenditure (Freijer et al., 2013). Evaluating the results found that increased age was associated with an overall rise in disease prevalence (Freijer et al., 2013). Furthermore the cost variation within Europe of disease-related malnutrition was thought to be due to differences in calculation methods of malnutrition. With the

'Cost of illness study' concluding their estimates could be rather conservative (Freijer et al., 2013).

A multisite cross-sectional survey, the 'Australasian Nutrition Care Day Survey' (ANCDS) was conducted in 2010 (Agarwal et al., 2012). The primary aim was to evaluate the Australasian nutrition care practices within participating hospitals, compared to evidence-based nutrition care practice guidelines that both countries endorse (Agarwal et al., 2012). A total of 56 hospitals involving 370 wards participated in the study in Australia (287 wards from 42 hospitals) and New Zealand (83 wards from 14 hospitals) (Agarwal et al., 2012). Surveyed results were recorded from eight speciality wards: Medical, Surgical, Oncology, Neurology, Orthopaedics, Renal/Urology, Gastroenterology, and Cardiology/Respiratory. Results concluded that general malnutrition rates were estimated to be at 30%, with malnutrition risk at 42% in Australian and New Zealand acute care hospitalised patients (Agarwal et al., 2012). Further evaluation of New Zealand weighing and nutrition screening protocols revealed that 53% of patients had nutrition screening performed on admission, 24% had weight only measured and as few as 10% of patients had nutrition screening and weight consecutively measured (Agarwal et al., 2012).

In NZ the 'Evidence-based practice guidelines for the nutritional management of malnutrition in adult patients across the continuum of care', are endorsed by Dietitians New Zealand (DNZ) (Dietitians Association of Australia, 2009). These guidelines suggest that routine screening for malnutrition, diagnosis, intervention, monitoring and evaluation protocols are an integral element of patient centred care (Dietitians Association of Australia, 2009). Associated with the improvement and early identification of malnutrition risk to support the management of malnourished adults (NHMRC Grade of recommendation: B) (Dietitians Association of Australia, 2009). Despite DNZ aligning with the Dietitians Australia Association's (DAA) and the endorsement of these evidence

based nutrition practice guidelines, implementation of these recommendations do not always translate into practice (Agarwal et al., 2012). These guidelines are shown in appendix A.

Comparing malnutrition screening tools

Numerous malnutrition-screening tools have been developed and validated for use in a wide variety of patient care settings. As an introduction into the multitude of nutrition assessment tools, five have been selected for a brief elaboration on their usage. The Subjective Global Assessment (SGA) is configured from patient derived data including: medical history, physical examination, recent weight loss and changes in dietary intake (Almeida et al., 2012). The SGA is simple to implement and allows dietitians to identify the state of malnutrition which can be placed into two categories: well nourished and moderately plus severely undernourished (Almeida et al., 2012). The Mini Nutritional Assessment (MNA) has been developed specifically for geriatric patients. The MNA highlights two nutritional parameters: weight loss and recent appetite that can equate to a total score of ≥2 indicating a risk of malnutrition (Poulia et al., 2012). The Patient Guided Subject Global Assessment (PG-SGA) makes use of the patient's medical history, using relevant diagnosis, any unintended weight changes, dietary intake, metabolic stress and findings of the physical examination including: weight loss, muscle wasting and oedema to give a graded score. A PG-SGA score of A) well nourished, B) moderately or suspected of being malnourished and C) severely malnourished, from which a nutrition intervention can proceed if appropriate (Bauer, Capra, & Ferguson, 2002). An observation study in a private Australian tertiary hospital in 2002 concluded that the PG-SGA allowed for quick and easy identification of malnourished patients (Bauer et al., 2002). However, a major limitation of the PG-SGA is that it must be performed by a trained nutrition-specialist. The Nutrition Risk Screen-2002 (NRS-2002) works on the premise that patients at

increased risk of malnutrition due to varying degrees of disease severity can have malnutrition classified as absent, mild, moderate or severe (Kondrup, Rasmussen, Hamberg, Stanga, & Grp, 2003). The Malnutrition Universal Screening Tool (MUST), developed and released by BAPEN in 2003, involves a scoring system taking into account: BMI, unintentional weight loss and acute disease. Nutritional risk is categorised as: 0 = low risk, 1 = medium risk and ≥2 = high risk (Bapen, 2013d). Results indicating a high-risk MUST score of ≥2 should generate a referral to a dietitian for a nutritional assessment. The MUST is a validated, reliable, simple, evidence-based tool for non-invasive measurement of malnutrition for adults in all care settings and currently used in the Admission to Discharge Planner at North Shore Hospital (NSH). It can be applied to all patients, including those unable to have their weight or height measured and/or may have fluid imbalances. A prospective study by Almeida et al. (2012) demonstrated the MUST to be a highly effective, sensitive and specific tool for recognising patients at risk of malnutrition (Almeida et al., 2012). The authors observed similar performance of NRS-2002 to the MUST in detecting malnutrition of surgical patients at the time of hospital admission (Almeida et al., 2012). These malnutrition risk assessment tools were a brief introduction to the type of tools currently used in clinical practice today.

Laboratory Markers of Malnutrition Risk

Negative acute phase protein such as: Prealbumin, Retinol-Binding Protein (RBP), Transferrin and Albumin have all been historically used as laboratory markers of nutrition status that are all synthesised in the liver (Parrish, 2006). Traditionally albumin was the predominant marker of choice, used as an indicator of acute and chronic malnutrition (Fuhrman, Charney, & Mueller, 2004). However, albumin with a half-life of ~20 days is affected by multiple variables other than nutrition status, which include: impaired liver function, dehydration and oedema, all of which can potentially lead to the

misinterpretation of possible nutritional changes (Gabay & Kushner, 1999; Parrish, 2006). Where as RBP and transferrin have significantly shorter half-life than albumin and are better utilised as markers of nutrition status. Furthermore both are affected by individual micronutrient status; transferrin by iron deficiency and RBP by vitamin A and zinc deficiency, making these markers unreliable if used in isolation (Beck & Rosenthal, 2002; Parrish, 2006). More recently studies have focused on prealbumin (transthyretin), a visceral protein with a small body pool of 0.01g/kg body weight, as a sensitive and accurate marker of current protein-energy status (Cavarocchi, Au, Dalal, Friel, & Mildenberg, 1986; Ingenbleek & Young, 1994; Parrish, 2006). Prealbumin is an hepatic visceral secretory transporter protein for both thyroxine and retinol, through mediation with RBP (Ingenbleek & Young, 1994). Although produced in the liver, serum levels are only decreased in severe liver impairment (Ingenbleek & Young, 1994). With a short biological half-life of ~2-2.5 days, prealbumin rapidly reflects changes in hepatic protein production as an early response to nutritional deficit (Parrish, 2006). Prealbumin has been identified as a cost effective indicator of malnutrition risk: that is not affected by hydration status, responds rapidly to nutrition intervention which correlates well with patient outcomes (Ingenbleek & Young, 1994; Mears, 1999; Robinson et al., 1990; Shenkin, 2006).

In the past decade, studies have researched the usefulness of biochemical analysis using visceral proteins in conjunction with malnutrition screening tool to predict malnutrition risk in hospitalised patients (Devoto et al., 2006; Mears, 1999; Potter & Luxton, 1999; Robinson et al., 2003; Saka et al., 2011). A prospective observational study of 320 admitted patients was conducted by Robinson et al. (2003) at Brigham Women's Hospital in Boston, USA. The research study evaluated the use of visceral proteins: prealbumin, RBP and albumin in malnutrition screening (Robinson et al., 2003). Blood samples were taken within 48 hours of admission and the visceral protein prealbumin (levels

of <20 mg/dL: considered malnourished) was compared to the standard nurse-completed nutritional questionnaire (Robinson et al., 2003). The questionnaire results found 33% (104/320) of patients to be malnourished, where as comparatively the laboratory-based screening of prealbumin found 51% were at risk of malnutrition (162/320). Notably 58% (104/181) who received a registered dietitian (RD) assessment were classified as malnourished. Remarkably 50% of those who were not referred for a comprehensive dietetic assessment as a result of the nurse-completed questionnaire were found at risk of malnutrition on prealbumin tests (Robinson et al., 2003). A close correlation was found between patients that were deemed malnourished by detailed dietetic assessment and the visceral protein laboratory results (Robinson et al., 2003). Additionally the standard nurse-completed nutritional screening process was found to take on average, 1.2 days longer to procure a diagnosis of malnutrition compared to the blood-test ($p=0.0021$) (Robinson et al., 2003). On joint logistic regression only prealbumin was a significant predictor of malnutrition ($p=0.039$) (Robinson et al., 2003).

Interdisciplinary teams

Dietitians are nutrition experts and are acutely aware of the impact of untreated hospital malnutrition, which potentially translates to an increased morbidity and mortality (Walton, 2009). However, there are often practice inhibitors that obstruct assessment in terms of appropriate front-line identification of malnutrition risk. The lack of awareness and education in terms of impact, relating to hospital malnutrition and associated comorbidities is a very real prospect with interdisciplinary teams often compromised by the reality of increasing commitments in a busy hospital (Walton, 2009). Making use of a validated malnutrition-screening tool at the time of admission, as a standard hospital protocol is often not considered in Admission to Discharge Planners, despite the fact that it has been well documented that identifying malnutrition is a fundamental step in patient-centred care (Green & Watson, 2005). More recent literature has proposed that routine clinical nutrition screening should be incorporated into patient care procedures and documentation at time of admission (Adams et al., 2008).

In 2006 the MUST was introduced into the admission to discharge planner documentation at Waitakere and NSH, Auckland, NZ. However, this was not universally adopted. It is still recognised that various alternative clinical screening practices, assessing malnutrition risk still continue, but are not formalised into the patient care pathway (Yovich, 2012). In 2009 the Waitemata District Health Board (WDHB) dietitians performed a malnutrition audit of 634 newly admitted patients. Results indicated that 8% (51/634) of patients had a completed MUST screen in their clinical records with 13% (7/51) of those patients being referred to a RD for formal nutrition assessment. Of those 634 patients followed up by a risk assessment performed by the registered dietitian (RD), the audits conceded 31.5% of these patients were at risk of malnutrition.

Additionally it was found that 31.5% of the at-risk patients were not formally referred to a RD for nutrition assessment (Yovich, 2012).

Summary

In summary the literary evidence revealed that hospital malnutrition prevalence within the western world is unacceptably high and continues to go undiagnosed. Despite the vast array of validated clinical screening tools currently available, of which many are supported by international evidence-based guidelines, screening tools are not being routinely used at hospital admission.

Registered dietitians are specialists in the nutrition field and use comprehensive nutritional parameters to assess patient status comprising of: anthropometric, biochemical, clinical and dietary analysis including clinical expertise as diagnostic tools of nutrition status. Anecdotal evidence does suggest that the facilitation of staff training and buy-in to a standardised validated nutrition-screening protocol that amalgamates the use of a laboratory marker is imperative and could expedite malnutrition risk diagnosis (Mears, 1999; Robinson et al., 2003). Notwithstanding that the literature eloquently states that no single measure of malnutrition assessment has stand alone status to individually assess and diagnose hospital malnutrition (Stratton et al., 2004). A combination of prealbumin and malnutrition screening increases the probability of widening the catchment of hospital malnutrition risk. Enhancing patient centred care by way of improving nutrition diagnosis instigating a nutrition intervention could potentially decrease the length of hospital stay, readmission rates, associated malnutrition comorbidities, mortality and decreased healthcare spending (Baker et al., 1982). Despite these well-documented endorsements, recommendations do not always translate into practice.

Within NZ the discharge rate per annum at the NSH in Auckland, was estimated at approximately ~42,318 in 2012, with the average length of stay (LOS) ~4.8 days (Ayar, 2013). When considering the availability of a validated clinical

screening tool (MUST) and the mediocre rate of malnutrition screening performed in practice. The extent to which disease-related malnutrition goes undiagnosed and the number of patients potentially at risk of malnutrition can only be postulated. The unknown burden and associated comorbidities equating to escalating societal healthcare costs by way of increased LOS, clinical complications, morbidity and mortality within the hospitals, substantiates the rationale for implementing a laboratory marker notably prealbumin in conjunction with a universally approved nutrition screening tool (Robinson et al., 2003). With this in mind the primary basis for this research was to increase the NZ evidence based knowledge on malnutrition risk in the hospital setting. Secondly to advocate and strengthen the evidence for the combination of a malnutrition screening protocol including laboratory screening (Prealbumin) to complement nutrition screening practices and improve referral rates to a dietitian. Finally to enhance patient centred care through early assessment, diagnosis and treatment of hospital based malnutrition. However, in NZ the literature pertaining to hospital based prevalence and the financial impact of hospital malnutrition is sparse.

1.1. AIM

The aim of this research was to investigate whether the introduction of routine prealbumin screening was more effective than routine clinical screening for identifying patients at nutrition risk, when admitted acutely to North Shore Hospital, Auckland.

1.2. PRIMARY OBJECTIVES

The objectives of this research are:

1. To examine whether more patients are identified at risk of malnutrition before and after the introduction of an obligatory Prealbumin test in research test wards 3,4,7,8 and 10 at North Shore Hospital.
2. To determine whether more patients were referred for registered dietitian nutrition assessment after the introduction Prealbumin from test wards at North Shore Hospital.

1.3. HYPOTHESIS

H_1: It is hypothesised that mandatory laboratory testing of prealbumin will increase the detection of disease-related malnutrition in acutely hospitalised patients at North Shore Hospital, Auckland

1.4. TABLE OF CONTRIBUTIONS

Table of Contributions	
Task	**Contributors**
Research Planning and design Design planning Funding	Dr Russell Walmsley (Clinical Research Supervisor) conceived the study design to align similarly to the literature from Robinson (2003). Design collaboration with Tracey Eccles, Russell Walmsley and Franica Yovich. Dr Russell Walmsley procured funding through Siemens New Zealand and New Zealand Society of Gastroenterology and WDHB.
Data Collection:	Tracey Eccles (Lead Researcher) Collection all patient admission data required for analysis. Transfer to North Shore Hospital Laboratory for prealbumin analysis. Collated all results in excel from the dietetic team. Franica Yovich (team leader) and the Dietetic team at North Shore Hospital performed phase I and II of patient demographic data collection and dietetic referral pathway information.
Laboratory Testing	Tracey Eccles collated all patient information presented to the laboratory daily throughout the study period. Angela Pountney, lead biochemist (WDHB). Co-ordinated all laboratory testing for prealbumin and C-Reactive protein.
Introduction/ Literature Review	Tracey Eccles (Lead Researcher). Performed a comprehensive literature review on hospital malnutrition nationally and internationally.

	Reviewed: Professor Gil Hardy (Academic Supervisor).
Statistical Analysis:	Welma Stonehouse supplied initial advice for statistical analysis, with additional guidance from Cheryl Gammon from Massey University. Statistical Analysis was conducted by Tracey Eccles (Lead Researcher). Reviewed: Professor Gil Hardy (Academic Supervisor).
Results	Results write up conducted by Tracey Eccles (Lead Researcher). Reviewed: Professor Gil Hardy (Academic Supervisor)
Discussion and Conclusion	Write up conducted by Tracey Eccles (Lead Researcher). Reviewed: Professor Gil Hardy (Academic Supervisor).
Presentation	Research findings presented by Tracey Eccles (Lead Researcher). The New Zealand Society of Gastroenterology Conference, Wellington 21st November 2013.

2.0. LITERATURE REVIEW

Nutrition is a fundamental pillar of human life with nutritional well-being dependant upon many critical factors including: food security, good health, wellness and the appropriate environment enabling sustainability of healthy living (World Health Organization, 2000). The hedonic pleasure evoked by the intake of food often diminishes markedly during hospital stay and critical illness however; nutrition is critically essential to recovery. Inadequate food intake alters the degree and prevalence of disease-related malnutrition (Dupertuis et al., 2003). The purpose of this literature review was to evaluate the phenomenon of disease-related malnutrition whilst acknowledging the numerous pathophysiological pathways that lead to malnutrition risk. Malnutrition is without question not a new phenomenon in the public health arena. This review examines the published national and international research pertaining to the clinical use and limitations of laboratory measures and available screening tools used most commonly to assess malnutrition risk. The NZ published literature that critically examines the impact of hospital malnutrition is meagre despite this limitation, all specific literary transcripts pertaining to the prevalence in NZ hospital malnutrition was reviewed.

2.1. AETIOLOGY OF MALNUTRITION

The World Health Organization (WHO) cites malnutrition as the single greatest public health threat in the world, markedly increasing the rates of morbidity and mortality (Blossner, 2005). Despite the fact that malnutrition is not rare in the undeveloped world, it often seems incomprehensible in an era of scientific advancement that malnutrition currently exists in the developed world. The aetiology of malnutrition is multifactorial and historically malnutrition research was based on two well-documented categories of malnutrition marasmus and kwashiorkor (Fuhrman et al., 2004). An insight into this domain of

malnutrition/semi-starvation was revealed in the outstanding published research of the first clinical semi-starvation trial more than sixty years ago. The first serious discussions and analyses of malnutrition was performed in America by Ancel Keys and his team in the 1945 study, which is commonly referred to as 'The Minnesota Experiment' (Franklin, Schiele, Brozek, & Keys, 1948). This famous study's main objective was to gain insight into the physiological and psychological effects of semi-starvation and refeeding of 36 previously healthy young men aged between 20-33 years (Franklin et al., 1948). During the starvation period the 36 men had reduced caloric intake from ~3210 calories per day to ~1600 calories/days for six months and experienced extreme weight loss of ~24% (Franklin et al., 1948). Within the first twelve weeks the men experienced fatigue, extreme weakness, muscle wasting, neurological deficit, reduced co-ordination and peripheral oedema (Franklin et al., 1948). When comparing the results of this trial against the criteria of multiple malnutrition screening tools, the defined criteria of both decreased caloric intake and ≥10% weight loss are highlighted as both key indicators of malnutrition risk. However, it was acknowledged that the weight loss demonstrated in the semi-starvation trial far exceeded the ≥10% as a common variable stipulated in the malnutrition screening tools and was noted at ~24% (Franklin et al., 1948). The body's metabolic alteration to combat starvation is to predominantly use the body's muscle protein as a fuel during the transition between well fed and starvation. Significant finding of this research found that the physiological and psychological impairment was evident with: fatigue, depression and mental apathy being greatly diminished, as was the desire to get well, often seen in hospitalised malnourished patients especially the elderly (Blossner, 2005; Franklin et al., 1948). Increased infection rates, oedema, depressed immune function, increased length of healing time and medical complications were all key features of malnutrition in both this landmark study and more recent

research into disease-related malnutrition (Barker et al., 2011; Franklin et al., 1948; Frew, Cant, & Sequeira, 2010). The increased knowledge obtained through Keys et al. (1945) research has given the medical world an insight into the measurable and consequential effects malnutrition imposes on the human body (Jensen et al., 2010). Therefore when considering these finding of nearly 65 years ago, it is somewhat surprising that the metamorphosis in disease-related malnutrition screening to decreased malnutrition prevalence has not progressed.

2.2. MALNUTRITION DIAGNOSIS

Numerous studies have attempted to elucidate malnutrition through a broad spectrum of disordered nutrition. When extrapolating the clinical term of malnutrition the synonymous relationship of simple undernutrition is quintessentially defined as an imbalance of nutrients. However, if proposing that malnutrition is primarily an imbalance of nutrients, due to the insufficient intake of quality nutrients, then the consideration must also apply to the overconsumption of energy dense, nutrient poor foods that equally play a part in malnutrition due to the lack of nutrient quality. Traditionally primary malnutrition definitions have encompassed those individuals that have exhibited physical deprivation or wasting with two principal malnutrition classifications of marasmus and kwashiorkor. However, in the developed world of the twenty first century, nutrient quality and nutrient deprivation all equally add to the defines of the malnutrition burden leading to overweight and obesity. Therefore appreciating that the recognition and diagnosis of secondary malnutrition is complex and multifactorial. Table 2.1. Highlights the diversity and enormous scale of malnutrition definitions within a small catchment the literature.

Table 2.1: Malnutrition Definitions

Author	Definition
Jensen et al. (2012)	'Newly proposed malnutrition syndromes includes: 'starvation-associated malnutrition', chronic starvation without inflammation; 'chronic disease-associated malnutrition', when inflammation is chronic and of mild to moderate degree; and 'acute disease or injury-associated malnutrition', when inflammation is acute and/or severe (Jensen & Wheeler, 2012. pp.206-211).
Barker (2011)	'Malnutrition is a broad term that can be used to describe any imbalance in nutrition; from over-nutrition often seen in the developed world, to under-nutrition seen in many developing countries and in hospitals and residential care facilities in developed nations. Malnutrition can develop as a consequence of deficiency in dietary intake, increased requirements associated with a disease state, from complications of an underlying illness such as poor absorption and excessive nutrient losses, or from a combination of these aforementioned factors' (Barker et al., 2011a, p.515).
Elia et al. (2004)	'Malnutrition can be defined as: 'a state of nutrition in which a deficiency, excess or imbalance of energy, protein, and other nutrients causes measurable adverse effects on tissue/body form (body shape, size, composition) and function and clinical outcome'(BAPEN, 2013a; Stratton et al., 2004, p.799).
WHO (1998)	'Malnutrition is the cellular imbalance between supply of nutrients and energy, and the body's demand for them to ensure growth, maintenance, and specific functions' (World Health Organization, 1998, p.1).
NICE guidelines (2012)	Malnutrition is a state in which a deficiency of nutrients such as energy, protein, vitamins and minerals causes measurable adverse effects on body composition, function or clinical outcome (NICE National Institue for Health and Care Excellence, 2012).

A possible causality of malnutrition misdiagnosis and the resulting confusion in malnutrition definition is there is not one universally defined criteria of secondary malnutrition. Jensen et al. (2010) agrees that the pathophysiology of disease-related malnutrition includes a myriad of characteristics. It could be proposed that encompassed in the aetiology of disease-related malnutrition which occurs in multiple divergent settings are the clinical comorbidities ranging from: chronic starvation e.g. anorexia nervosa, chronic disease with moderate of mild inflammation e.g. organ failure various palliative cancer states to acute disease or injury e.g. acute trauma, septic shock, fistulas and open wounds (Jensen et al., 2010). With systemic inflammation in patients predicting a significant increase in the risk of precipitating anorexia directly resulting in malnutrition (Jensen, Bistrian, Roubenoff, & Heimburger, 2009; Stratton et al., 2003). Whether these factors are present or related to under or over-nutrition, acute or chronic inflammation, preclinical or subclinical malnutrition all result in the metabolic dysfunction of the body and alter physiological function (Jensen et al., 2010). ASPEN's International Guidelines committee agreed through a consensus approach to define malnutrition through way of extrapolating a classification of malnutrition syndrome (2010). These malnutrition syndromes consisting of: starvation-related malnutrition, chronic disease-related malnutrition, and acute disease or injury-related malnutrition (National Alliance for Infusion Therapy and the American Society for Parenteral and Enteral Nutrition Public Policy Committee and Board of Directors, 2010). Stratton et al. (2003) provides evidence that early detection of preclinical and subclinical malnutrition can have a definitive advantage in slowing the progression and improving patient outcome. Additionally, Mears (2007) does raise the point that the prevalence of subclinical malnutrition is significantly higher than symptomatic malnutrition and also acknowledges the importance of a clear distinction in terms to enhance the recognition of subclinical malnutrition at

hospital admission. The conundrum of defining malnutrition criteria is somewhat divergent. A possible causality in the disparity in malnutrition rates is the various settings of which assessment is undertaken. In defining malnutrition criteria a myriad of malnutrition parameters are proposed as diagnostic indicators, which include: BMI, anthropometric measurements, unintended weight loss and dietary alterations. Within all these definitions the changing variables used for screening and diagnosis of malnutrition risk can be typically categorised into: a) the setting in which the assessment is performed, b) whether the assessment was performed at the time of hospital admission, c) the procurement and definition of malnutrition assessment i.e. how unintended weight-loss is measured, d) notwithstanding the type of screening tool used i.e. Malnutrition Universal Screening Tool or Subjective Global Assessment. According to the definitions provided by Mears (2007) and Stratton et al. (2003) which suggest that malnutrition can be split into two categories: a) primary which refers to non-disease related malnutrition, relating more simply to nutrient intake inadequacy or b) secondary malnutrition more recently referred to as disease related malnutrition that is associated with the patients disease status. Well documented is that hospital malnutrition procures the necessity for an increased demand of energy/nutrient intake whilst healing, which implies that those who are malnourished have impaired healing ability that can result in an extension in hospital stay (Alberda et al., 2006; Norman et al., 2008).

2.3. AETIOLOGY OF DISEASE-RELATED MALNUTRITION

Disease-related malnutrition has superseded more common terms of secondary, protein energy or calorie malnutrition. Consensus among nutrition specialists and clinicians is that a state of under-nutrition is primarily due to physiological and metabolic alterations influenced by the disease state, of which nutrient intake often is substantially reduced whilst energy requirements are increased (Freijer et al., 2013; Jensen et al., 2010; Norman et al., 2008). The prognostic impact of disease-related malnutrition cited by Norman et al. (2008) describes the pathophysiology as attributable to a metabolic alteration of the human body shown in figure 2.1. Norman el al. (2008) propounds a vicious circle in the progression of disease-related malnutrition and the associated malnutrition comorbidities which can include an increased infection rate and depressed immune function often resulting in extended length of hospital stay. As Tappenden (2013) points out, adverse nosocomial infection rates occur more readily in those entering the hospital system in a malnourished state. A study by Schneider et al. (2004) found nosocomial infections rates were highest in severely malnourished patients 14·6% ($p = 0.009$) compared to 7·6% in moderately malnourished patients and 4·4% in those who were not malnourished. Concluding that nutritional status, nosocomial infections and readmission rates have a strong positive correlation with disease-related malnutrition (Schneider et al., 2004).

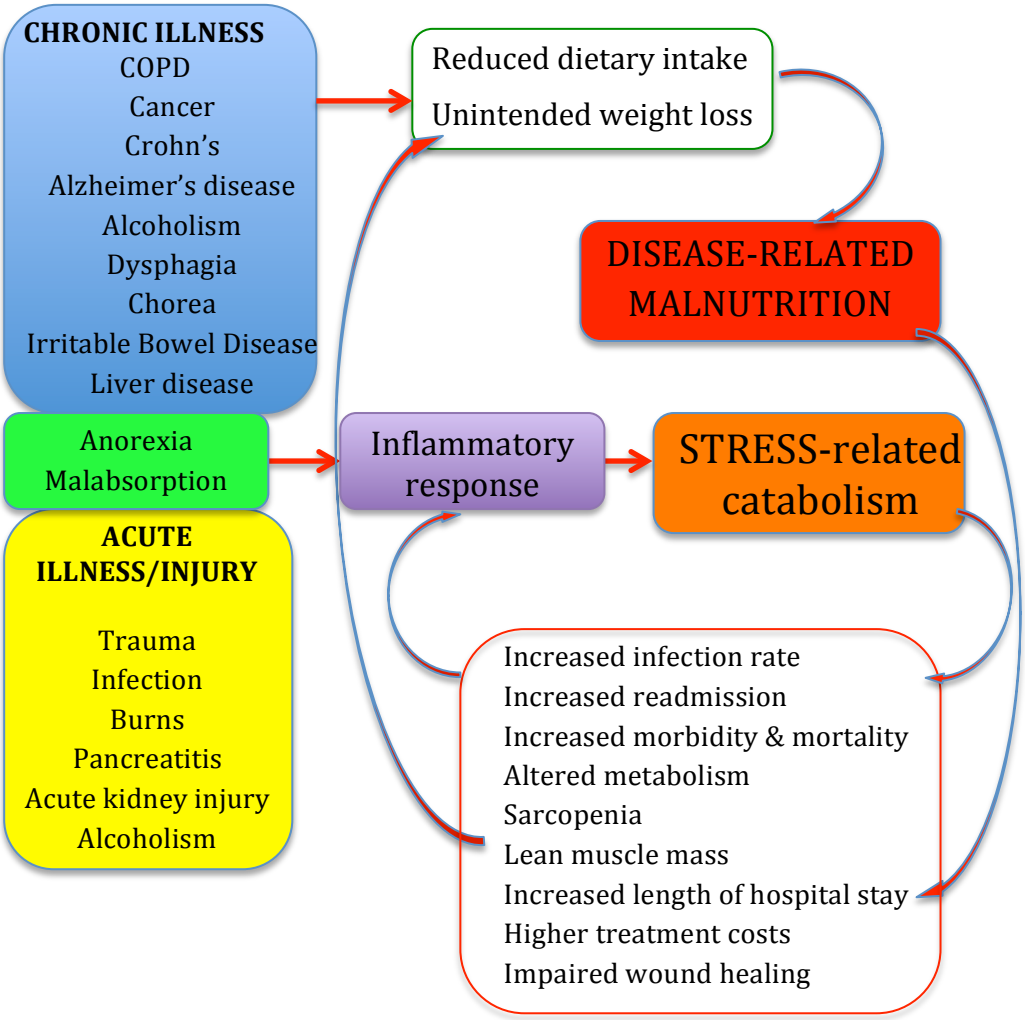

Figure 2.1: Vicious cycle in the progression of Disease-related malnutrition adapted from Norman (2008).

According to Stratton et al. (2004) and Freijer et al. (2013) secondary malnutrition as a condition originating from disease-related factors of malabsorption, increased demands on nutrients or insufficient nutrient utilisation having consequential adverse effects on the body functionality and clinical outcome. Whereas Barker et al. (2011) proposes that recent evidence suggests that disease-related malnutrition comprises of both cachexia (multifactorial syndrome) and malnutrition (inadequate oral intake of nutrients). This notion is

also supported by the European Society of Parenteral and Enteral Nutrition (ESPEN) Barker et al. (2011) and ASPEN the American Society of Parenteral and Enteral Nutrition (Kondrup, Allison, Elia, Vellas, & Plauth, 2003; National Alliance for Infusion Therapy and the American Society for Parenteral and Enteral Nutrition Public Policy Committee and Board of Directors, 2010). Alberda et al. (2006) and Jensen et al. (2010) corroborate the aetiological theory that disease-related malnutrition encompasses a hypermetabolic stress state, induced by illness or injury that is strongly influenced by hormone and inflammatory mediation which correlates to lean muscle mass wasting. Notably impaired nutrient absorption or utilisation during this hypermetabolic or hypercatabolic state during critical illness potentiates malnutrition and/or cachexia or simply a temporal decrease in nutrient intake. Despite these definitions, all have measurable and consequential effects on clinical outcome (Barker et al., 2011; Russell, 2007). As the acute phase inflammatory response increases the nutritional requirements through an elevated in resting energy expenditure (REE), reprioritisation of protein synthesis and nitrogen excretion that occurs elevating nutrient requirements (Jensen et al., 2010). Jensen and Wheeler (2012) strongly endorses that a preliminary nutritional assessment is crucial at the time of admission. They claim that although critically unwell patient's may not be acutely malnourished at the time of admission, although the metabolic dysregulation that is initiated via the catabolic response of illness requires adequate replacement of macro and micronutrients to prevent disease-related malnutrition (Jensen & Wheeler, 2012). To elaborate further, the utilisation of metabolic substrates e.g. carbohydrate and protein via gluconeogenesis in times of markedly reduced nutrient intake, involves a cascade of mediated hormones that initiate an inflammatory response which directly corresponds to disease-related malnutrition and/or acute injury (Alberda et al., 2006).

These biochemical responses manifest lipolysis and protein catabolism, which directly facilitates skeletal muscle atrophy and hence greatly reduces lean body mass (LBM) (Alberda et al., 2006). Unintended weight loss and muscle atrophy are symptomatic of malnutrition and are often used as distinctive markers in malnutrition screening tools. Jensen et al. (2009) adds that the skeletal muscle is the largest reservoir of protein that is primarily influenced by protein energy malnutrition. McCarthy and Esser (2010) attest to skeletal muscle atrophy as a result of malnutrition, greatly influences the quality of life in patients with chronic disease especially affecting the elderly and delaying patient recovery time. In addition the contributing factor of fluid resuscitation treatment performed in specific cases at times of acute injury and illness can precipitate fluid overloading and oedema. This poses additional challenges for medical staff often leading to the misdiagnosis of disease-related malnutrition, as patients have the potential to look well nourished when fluid overloaded. Notably disease-related malnutrition does not discriminate and impacts patients despite: age, gender, ethnicity, socioeconomic status, educational level or culture. Norman et al. (2008) reviewed the prevalence of disease-related malnutrition and reported sick elderly patients and patients with severe disease were the most at risk of disease-related malnutrition. Disease-related malnutrition prevalence habitually occurs in various chronic diseases with foremost reference given to gastrointestinal dysfunction inhibiting normal nutrient utilisation predominantly in those with liver disease, pancreatitis and inflammatory bowel diseases (Alberda et al., 2006; Norman et al., 2008). Alternatively, additional physiological conditions that attributed to disease-related malnutrition are: mechanical obstruction either oropharyngeal or gastrointestinal tract obstructions resulting in a significantly reduced dietary intake (Alberda et al., 2006; Norman et al., 2008). In addition to clinical treatment/drug therapy that induces side effects precipitating anorexia e.g. pain

medication, chemotherapy, inflammatory bowel diseases, neurodegenerative conditions such as Alzheimer's and Parkinson's disease all exacerbate the condition of disease-related malnutrition (Alberda et al., 2006; Norman et al., 2008). Research has provided evidence that individuals with disease-related malnutrition specifically protein-energy malnutrition (PEM) have more problematic wound cares increased risk of pressure ulcers, which results in detrimental effects on surgical incisions and impacts on clinical outcome (Alberda et al., 2006; Allison, 2000). Additionally there are increased metabolic demands required to produce new biological proteins and collagen to procure a new matrix synthesis for effective wound healing which requires an adequate intake of dietary protein and nutrients (Alberda et al., 2006; Norman et al., 2008). With the exceeding degeneration of protein in times of often high demand for protein synthesis, the atrophy of skeletal muscle can be found in chronic disease states including: cancer, renal failure, chronic obstructive pulmonary disease (COPD) and sepsis and ageing (sarcopenia) which have escalating nutritional demands (McCarthy & Esser, 2010).

Similarly Jensen et al. (2010) and Stratton et al. (2003) concur that the aetiology of disease-related malnutrition's clinical diagnosis as occurring in multiple divergent settings from chronic starvation e.g. anorexia nervosa, chronic disease with moderate of mild inflammation e.g. organ failure various palliative cancer states to acute disease or injury e.g. acute trauma, septic shock, fistulas and open wounds. With systemic inflammation in patients predicting a significantly increased risk of precipitating anorexia resulting in malnutrition. Malone's et al. (2013) analysis of this insidious problem provides us with a multiplicity of rationale leading us to identify individual disease-related malnutrition burden including: mastication issues and dysphagia, advanced age, polypharmacy and nutrient/drug reactions and disease burden. Whereas Kubrak and Jensen's (2007) logical analysis is that disease-related malnutrition

both pre and post admission have the potential to be inextricably linked often exacerbated during the hospital stay, with more complex reciprocation between disease effect and the intricacy of metabolic nutrient modification during time of acute or critical illness. Furthermore, the intricate balance between well-nourished and disease-related malnutrition prior admission is often tipped during the hospital stay due to: extended periods of nil by mouth, unassisted meal times, loss of appetite and/or nausea, or simply poor assessment of malnutrition status on admission (Kubrak & Jensen, 2007). Jensen et al. (2010) therefore proposes that adequate nutrient feeding may favourably alter the hospital outcome for many patients, by inhibiting the loss of skeletal muscle mass and ceasing or slowing functional impairment thereby improving clinical outcome and potentially decreasing the length of hospital stay. Tappenden et al. (2013) eloquently details their call to action in addressing adult hospital malnutrition, stating that an enormous opportunity exists on numerous levels to improve the clinical outcome of hospital patients, through the early identification and treatment of malnutrition resulting in a cascade of effects both through benefitting society by reducing the economical burden that disease-related malnutrition poses.

2.4. SCOPE OF THE DISEASE-RELATED MALNUTRITION PROBLEM

Despite the existence of knowledge regarding hospital malnutrition reported in the literature at least thirty years ago, little has changed apart from a name change to ‘disease-related malnutrition’. Disease-related malnutrition or simply hospital malnutrition related to illness/disease is definitely not a newly uncovered medical phenomenon, having been acknowledged by Florence Nightingale in 1859 of the hospitalised soldiers during the Crimea war (Barker, Gout, & Crowe, 2011). More recently published research continues to demonstrate the prognostic impact imposed by disease-related malnutrition on

the clinical health outcomes, patient recovery times and alarming increases in associated health-care costs (Jensen et al., 2010).

In 1974, Butterworth's landmark study potentiated the enlightenment of many clinicians as to the influence hospital based malnutrition has on patient outcome (Butterworth, 2005). Recently, researchers of multiple organisations including large networks of experts have researched, audited and surveyed hospitals in relation to disease-related malnutrition in community hospitals in an attempt to highlight and address the issue of hospital malnutrition. Several studies have revealed the lack of clinical recognition in disease-related malnutrition including the IBRANUTRI study a cross-sectional, multicentre epidemiologic study of 4000 patients in Brazil, which found the malnutrition awareness by physicians was limited and nutrition therapy was under-prescribed (Waitzberg, Caiaffa, & Correia, 2001). Additionally the nutrition screening survey in the UK and Ireland in 2011, found malnutrition affected 1 in 3 adults on admission with nutrition screening policies and nutrition practices under-recognised and un-treated (Russell et al., 2011). Not to mention newly released and published data from the 'Nutrition Care Day Survey 2010' found an unacceptably high rate of malnutrition in acute care hospital wards in Australia and New Zealand (Agarwal et al., 2012). The survey found of 3122 patients surveyed one in three (32%) were malnourished, 41% were at risk of malnutrition, mortality at 90 days was twice that of malnourished compared to well nourished, the average stay was 5 days longer stay and readmission rates 6% higher in malnourished patients (Agarwal et al., 2013). The Consensus statement from the Academy of Nutrition and Dietetics and the American Society for Parenteral and Enteral Nutrition, recommends the identification and documentation of adult malnutrition to adult starvation and disease-related malnutrition. A proposal for Aetiology-Based Diagnosis in the Clinical Practice Setting endorsed by the British Association for Parenteral and Enteral Nutrition (BAPEN) recommends that all patients should

be routinely screened on admission and throughout their hospitalisation (White et al., 2012). More recently the NICE Quality Standard 24, Nutrition Support in Adults highlighted again the importance of screening and effectively managing malnutrition in clinical practice (NICE National Institue for Health and Care Excellence, 2012).

All of these experts in the field of adult malnutrition ascribe to the prognostic impact of disease-related malnutrition and the imperativeness of screening, documentation and intervention at the time of hospital admission. However, despite multiple research studies and reviews that estimate the prevalence of disease-related malnutrition, it continues to elude detection predominantly due to the criteria used to define malnutrition prevalence. International studies have suggested that one third of all admitted patients are either malnourished or at significant risk of malnutrition post hospital admission, with many of those patients going undiagnosed and rapidly declining during their hospital stay (Barker et al., 2011; Robinson et al., 2003; Tappenden et al., 2013).

2.5. PREVALENCE OF HOSPITAL MALNUTRITION

Despite the large volume of published studies over the past quarter of a century alluding to the negative impact associated with hospital-based malnutrition little has changed internationally to alter the prevalence and recognition. Kubrak and Jensen (2007) have stated in a review that the malnutrition rates in the literature ranged from 13–78% among acute care patients. Table 2.2. Illustrates the significant variation in malnutrition prevalence rates of international studies post 1990.

Table 2.2: Hospital malnutrition prevalence by country in studies post 1990.

Author	*Country*	*n*	*Prevalence (%)*
(Coats, Morgan, Bartolucci, & Weinsier, 1993)	USA	228	38
(Frew et al., 2010)	Australia	501	17
(Devoto et al., 2006)	Italy	108	60
(McWhirter & Pennington, 1994)	Scotland	500	40
(Waitzberg et al., 2001) -IBRANUTRI study	Brazil	4000	48
(Potter & Luxton, 1999b)	Canada	147	36
(Robinson et al., 2003)	USA	320	51
(Kruizenga et al., 2003)	Netherlands	7606	25
(Edington et al., 2000)	U.K	850	20
(Agarwal et al., 2013) Nutrition Care Day Survey 2010	Australia/ NZ	3122	32
(Elia, Jones, & Russell, 2008b) Nutrition screening survey	UK	9336	27

The estimated prevalence of disease-related malnutrition in the hospital setting is internationally recognised at between 20-60%, with some studies giving consideration to seasonal variation at the time of the conducted research. In recent years, there has been an increasing agreement that seasonal variation cannot be discounted in the hospital setting (BAPEN, 2013a; Elia, Zellipour, & Stratton, 2005; O'Flynn, Peake, Hickson, Foster, & Frost, 2005; The Patients Association, 2011).

The following studies unveiled a wide variation in the hospital malnutrition rates and proposed different reasoning. Edington et al. (2000) scrutinised the prevalence of hospital malnutrition in four English hospitals. The study was based on the evaluation of 850 patients, from 1611 admissions in four separate hospitals: two district and two teaching hospitals (Edington et al., 2000). The primary objective was to estimate the prevalence of malnutrition within the hospitals, which was found to be at 20%. However, what the study concluded was that despite the estimated malnutrition rate, the true prevalence was

questionable. It was thought that a more realistic estimate would far exceed the proposed 20%, the explanation provided was that 761 patients did not participate in the study because they were: unable to be weighed (*n*=256), too ill for a nutrition assessment (*n*=269) or just declined inclusion in the study (*n*=236) (Edington et al., 2000). McWhirter and Pennington's (1994) prospective study of an acute teaching hospital in Scotland, sought to determine the incidence of malnutrition among 500 patients, admitted to five speciality wards: general surgery, general medicine, orthopaedic surgery, respiratory surgery and medicine for elderly. Findings of this research concluded that malnutrition rates on admission were at 40% however, it was additionally noted that of the 200 patients found to be undernourished as few as 96 had nutrition status documented in their patient notes (McWhirter & Pennington, 1994). Walton's study (2009) examined the malnutrition prevalence rates in Australian hospitals and found the results ranged between 6% to 53%, this disparity was considered to be largely due to: the time frame of whether the assessment was performed at time of admission or later in hospital stay or alternatively the type of screening tool used. A cross-sectional epidemiology study of 4000 hospital patients from 25 Brazilian hospitals, was one of the largest hospital malnutrition prevalence study's published to date (Waitzberg et al., 2001). The study's primary objective was to examine the prevalence rates of malnutrition, determined to be at 48%, although as little as 19% had nutrition status documented in patient records and 15.5% had weight taken on admission (Waitzberg et al., 2001). A major nutrition screening survey performed in the UK in 2010, found malnutrition risk of 34%, with the breakdown by medical category of: gastrointestinal disease (48%) and neurological disease (34%) versus cardiovascular disease (23%) and musculoskeletal conditions (24%) (Russell et al., 2011). Finally the Dutch Dietetic Association in 2003 conducted a national malnutrition screening assessment and found ~25% of 7606 patients across all

medical fields were moderately to severely malnourished with approximately half of these patients not assessed by a dietitian for nutrition intervention (Kruizenga et al., 2003). The themes that emerged from these studies were that a) screening was inconsistently performed, b) weight was not often taken on admission and c) nutrition documentation was sorely lacking from patient's clinical notes. A conjecture that often emerges as to the reason for inconsistent or difficulty in nutrition evaluation the two main reasons are 1) unable to be weighed and 2) too ill for a nutrition assessment.

2.6. INCREASING GERIATRIC POPULATION

The evolutionary changes that have occurred in medical science in the developed world over the past century are well documented. We are now living healthier, longer lives than any other historical point in time. The latest census report 2013, state the number of those 50-69 (*n*=607,032) has shown the largest increase with over 73,000 people aged 85 years and over, a 29.4% increase since the last census in 2006 (Statistics New Zealand, 2013a). At no other time in history has the population prediction in age structure set to significantly change so dramatically with predictions of 1 million 65+ by the late 2020s and the 65+ age group set to exceed the population of children aged 0-14 years (Statistics New Zealand, 2009). Figure 2.2. Illustrates the New Zealand population pyramids in 1981, 2013 and the predicted change in the ageing population pyramids in 2061.

Figure: 2.2. Population age pyramid projections from 2009-2061 (Statistics New Zealand, 2009, 2013a).

Numerous research has been published regarding the inevitability regarding the transition to an ever increasing ageing population and the inescapable effects this poses on worldwide economies (Bryant, Teasdale, Tobias, Cheung, & McHugh, 2004; Ministry of Health, 2006). With increasing life expectancy the elderly poised to infiltrate the hospital system, as the inevitability of increased prevalence in chronic disease envelops adults of advancing age. The NZ Ministry of Health reports the prevalence of hospitalisation and mortality rates for COPD, all cancers, ischemic heart disease (IHD), cardiovascular disease (CVD) are significantly higher in adults of advancing age (Ministry of Health, 2006). Studies have postulated whether the age-related inflammatory process could be the rationale behind the manifestation of cachexia or failure to thrive in the geriatric communities, which is often misrecognised as naturally occurring sarcopenia (Jensen et al., 2009; Wells & Dumbrell, 2006). Elderly are the largest most vulnerable group for nutritional inadequacy and malnutrition is shown to be detrimental to cognitive competence (Wells & Dumbrell, 2006). Wells and Dumbell (2006) agreed that malnutrition in the elderly is frequently undiagnosed. Furthermore, studies have revealed that elderly hospitalised

patients are 20-65% more prone to suffering nutritional deficiencies including malnutrition, with numerous comorbidities linked to poor nutrition status (Wells & Dumbrell, 2006). A calculation based in the UK during the national screening weeks from 2007-2011, has estimated that 33% of patients aged over 65 years are malnourished at the time of hospital admission with those 75+ years at an even higher risk of malnutrition and being more likely than any other age to be admitted to hospital (BAPEN, 2013; Health and Social Care Information Centre, 2012; Office of National Statistics, 2011). Poor recognition of malnutrition in the geriatric hospital community is often exacerbated during hospitalisation, with the deterioration of malnutrition related comorbidities; whether due to feeding assistance requirement, physiological decrease in appetite oral health complications (ill-fitting dentures) or multiple periods of nil by mouth (Adams et al., 2008; Visvanathan, 2003). A Melbourne study in Australia conducted in a tertiary teaching hospital in 2006, assessed 100 elderly patients of ≥70 years for malnutrition using the Mini Nutrition Assessment tool (MNA) with admission nutrition status noted within the first 24-48 hours. Results concluded that 9 of 100 participating patients were not at risk of malnutrition, 61 patients had significant weight loss and 97% were either at risk of malnutrition or malnourished at time of assessment with only 19% identified and 7% had an actioned dietetic referral (Adams et al., 2008).

Significant association has been found within the geriatric population with malnutrition strongly correlated with anorexia of ageing, depression, and a significant decline in the will to recover with age. Muscle weakness tends to be prominent with associated PEM reducing cardiac and respiratory function. As with all disease-related malnutrition that goes undiagnosed and untreated, elderly are at increasing risk which is of even greater concern due to the well documented ageing population in the western world (Statistics New Zealand, 2000). Worth consideration are the hospitalisation statistics from the NZ Ministry

of Health, which stated inpatient hospitalisation statistics during 2010/11 were at 1,052,870 annually an increase of 1.6% from 2009/10, inpatient hospitalisations (672,171) were 63.8 percent of total discharges with 85+ year olds being 65,861 or 6.3% of total discharges (Ministry of Health, 2013b). When taking into account the increasing magnitude of potentially malnourished patients that will enter the hospital system within the next few decades and the increasing burden imposed on healthcare the justification and prioritisation of malnutrition screening protocols at the time of hospital admission in everyday practice is becoming increasingly more evident.

2.7. MALNUTRITION ASSESSMENT

Multiple nutritional variables have been used to assess malnutrition and are largely based on an array of anthropometry, biochemical, and clinical and dietary assessments. Regardless of the large number of available tools and multiple anthropometric variables to guide and quantify malnutrition risk, malnutrition still eludes detection. Two of the most common variables have historically been unintentional weight loss and BMI. Unintentional weight loss is a well-known constant quantifiable measure and excellent predictor of malnutrition risk. Despite this myriad of malnutrition definition criteria, the National Institute for Health and Care Excellence (NICE) have implemented care guidelines for malnutrition, defined as being:

1. A BMI of less than 18.5 kg/m^2.
2. Unintentional weight loss greater than 10% within the last 3-6 months.
3. A BMI of less than 20 kg/m^2 plus unintentional weight loss greater than 5% within the last 3-6 months.
4. Additionally those individuals who have not eaten for 5 days or longer are at greater risk of malnutrition (National Institute for Health and Clinical Excellence, 2006).

However, the challenge of gaining an accurate patient weight at the time of

hospital admission can have multifactorial opposition. As current medical status can prohibit weight attainment e.g. the patient being in an unconscious state or too medically unwell to be weighed. Careful consideration should also be taken when estimating a patients weight, current hydration status can alter and influence the diagnosis of nutrition status whether at risk or not at risk, therefore a patient's euvolemic weight in those patients with ascites or having recently received fluid resuscitation is more advantageous as a diagnostic tool. The use of BMI and unintentional weight loss are used as parameters to define nutritional status, lower BMI values tend to correlate more with chronic malnutrition whereas more recently unintentional weight loss is indicative of acute malnutrition (Neelemaat, Meijers, Kruizenga, van Ballegooijen, & van Bokhorst-de van der Schueren, 2011). BMI measurement has it's own challenging facets, as the debate continues to rage over BMI cut-off interpretation as within BMI cut-off there are ethnic-specific BMI ranges (Dietitians NZ, 2011). A BMI of ≤18.5 kg/m^2 is thought to be an indicative sign of underweight or at greater risk of malnutrition. However, compelling evidence suggests that when using BMI in isolation as a predictive marker nutrition status we often overlook obese and/or overweight individuals when assessing malnutrition risk. Due to the increasing preconception that an increased BMI of greater than 25 kg/m2, invariably signifies an individual who is over-nourished with seemingly no possible risk of malnutrition (Dietitians NZ, 2011; Ministry of Health, 2013). Despite predetermined conceptualisation, obese and/or overweight individuals are at equal risk of malnutrition as any other individual, due to the propensity for reduced food intake due to acute illness or an acute traumatic incident (Tappenden et al., 2013; White et al., 2012). Adams et al. (2008) examined the identification and recognition of disease-related malnutrition by health professionals, including their perceptions and awareness of clinical signs and symptoms and the appropriate risk management of

disease-related malnutrition. Results indicated that nurses focused on physical assessment whilst doctors focused on the biochemical parameters e.g. albumin, despite the knowledge and awareness of factors contributing to malnutrition risk, only a small number of patients were referred for dietetic assessment (Adams et al., 2008). Adams et al. (2008) attests that their study is definitely not the first study to show evidence of low rates of nutrition risk documented in clinical notes. Lazarus and Hamlyn (2005) found malnutrition risk documentation was completed in 1 out of every 137 patients in Australian hospitalised patients with only 15% ever referred for nutrition intervention. The IBRANUTRI study concluded that most doctors were unaware of patient's nutrition status despite the high prevalence of malnutrition (Waitzberg et al., 2001). These studies have illustrated a small catchment in the considerable amount of literature has been published regarding the lack of awareness, recognition and referral of patients at risk of malnutrition.

2.8. MALNUTRITION ASSESSMENT TOOLS

A trained professional can divide malnutrition assessment tools into those that are simple to use, quick and require no additional training and those that are more comprehensive, complex and require clinical knowledge. The consensus of multiple research studies is that there is no universally accepted gold standard for malnutrition diagnosis, criteria or documentation criteria pertaining to accurately delineate adult malnutrition in all settings (Stratton et al., 2004; Velasco et al., 2011; White et al., 2012). The current literature attests to the significant dilemma malnutrition imposes through adverse metabolic and functional derangement on organs systems that can result in long lasting consequences on the individual physiologically and on society financially (Alberda et al., 2006; Almeida et al., 2012; Stratton et al., 2004). Diverse literature reviewing the detection of preclinical and subclinical malnutrition has harmoniously agreed that appropriate recognition and treatment is

advantageous on multiple levels. Whether that is at an individual level of improving physiological function and enhancing the speedy return to good health or at a commercial level of decreased expenditure of healthcare.

Stratton et al. (2003) suggests there are currently well in excess of 50 published nutrition screening tools currently in use. Whether these screening tools are valid in the setting of choice does raise the point, that without a standardised protocol for malnutrition screening and diagnosis there is always the potential for misdiagnosis and multiple screening tools do not allow for the inflammation response (White et al., 2012). Elia et al. (2005) published that the prevalence of malnutrition has been found to vary according to assessment criteria. To make effective use of malnutrition screening tools in the hospital setting the malnutrition screening tool must first be validated, reliable, relatively simple to perform and sensitive in the appropriate setting of choice. Additionally it must be endorsed by evidence-based practice and performed appropriately and completely at the point of admission. Although weight is a predictive parameter of malnutrition in multiple screening protocols, estimated weight is also often required to determine medication dosage as well as anaesthesiology requirement during hospitalisation (Elia et al., 2005). Despite weight procurement at admission being advantageous on multiple levels, weighing is often missed and absent in clinical documentation (Agarwal et al., 2012; Barker et al., 2011). Case and point was demonstrated by Agarwal et al. (2012) whose study found that in fourteen NZ hospitals as little as 12% of admissions had both nutrition screened and weighing measured. The *American Society for Parenteral and Enteral Nutrition (*A.S.P.E.N) looked at the aetiological basis of malnutrition definition and provided guidelines for diagnosis with six identifying physiological characteristics with two equating to moderate to severe malnutrition risk (White et al., 2012). These six characteristics included: insufficient energy intake, weight loss, loss of muscled mass, loss of

subcutaneous fat, localised or generalised fluid accumulation that may sometimes mask weight loss and diminished functional status as measured by hand grip strength, identification of two or more of these assessment in context of acute illness or injury is recommended for diagnosis (White et al., 2012). There is a wide variety of screening tools available, a prospective study in two Sydney teaching hospitals was undertaken to determine the prevalence of malnutrition. Of the 2194 eligible patients 819 were systematically selected, with 36% determined as malnourished using SGA protocol (Middleton, Nazarenko, Nivison-Smith, & Smerdely, 2001). The SGA was noted as a reliable, validated assessment method to indicate the prognosis of risk of malnutrition (Barbosa-Silva & Barros, 2006). Neelemaat et al. (2011) when comparing five malnutrition screening tools in an inpatient setting in one hospital found that both the MUST and the NRS-2002 demonstrated the highest percentage rates of malnutrition risk whereas the MST had the lower percentage. However, MST, MUST and NRS-2002 all showed sensitivity and specificity of at-least 70% (Neelemaat et al., 2011). A study conducted by Devakonda et al. (2008) that compared nutrition screening tools to laboratory markers, suggested that although the SGA is highly substantiated assessment tool, a limitation is that it must be performed by a trained professional. Finally Kruizenga et al. (2005) remarks that the utilisation of nutrition screening tools at the time of hospital admission has the potential to improve recognition from 50 to 80%, resulting in a reduced length of hospital stay.

2.8.1. Limitations of malnutrition screening in the hospital setting.

It has been proposed that nutrition screening should be able to be performed by any trained health professional (Barker et al., 2011). However, nurse assessment is preferable due to their high patient contact times (Green & Watson, 2005). One requirement that must be adhered to is that the terms of screening and assessment for malnutrition risk must not be interchangeable.

Screening tends to be a simple quick process usually by way of a questionnaire format to screen for malnutrition risk that requires minimal training, whereas the assessment process is more complex requiring clinical training and judgment typically performed by a trained nutrition specialist e.g. dietitian. Despite multiple researchers concurring that malnutrition risk screening at the time of hospital admission should be documented as part of an admission to discharge planner, many studies agree malnutrition screening does not routinely occur (Elia, Jones, & Russell, 2008; Gout, Barker, & Crowe, 2009).

Additionally Nursal et al. (2004) concurred with other research, claiming that nutritional assessment techniques often require specially trained personnel and that some of the measurement tools take a considerable amount of time (15-30 minutes) and require regular calibration not always readily available (Nursal, Noyan, Atalay, Koz, & Karakayali, 2005). The IBRANUTRI study concluded that less than 20% of patients had nutritional status information charted in patient records (Waitzberg et al., 2001). With many clinicians unaware of the prevalence of malnutrition and subsequently the impact malnutrition has on patient outcome, LOS and readmission rates and increased hospital cost (Nursal et al., 2005).

2.9. MALNUTRITION UNIVERSAL SCREENING TOOL (MUST)

The Malnutrition Universal Screening Tool (MUST) for adults was established by the standing committee of the Malnutrition Advisory Group, of the British Association for Parental and Enteral Nutrition (BAPEN) launched in 2003 (Bapen, 2013). The MUST was initially developed for use in the community and then extended for use in all health settings including the hospitals (BAPEN, 2013). A clear benefit of this tool is the extension to multiple settings from community to hospital without loss of consistency or alteration of underlying principles. The MUST is simple to apply, is expeditious, valid and reliable in identifying risk of adult malnutrition. The tool comprises of a five-step process of

which the clinician measures the following three main parameters: BMI score, unintended weight loss score within the last three to six months and acute disease effect score: likely or no dietary intake for more than five days. The malnutrition overall malnutrition risk, is stratified into three categories: 0 = Low Risk (Routine clinical care), 1 = Medium Risk (Observe) and 2 or more = High Risk (Treat). Based on the score of ≥2: high risk (treat) the dietitian develops and initiates a nutrition intervention plan where appropriate.

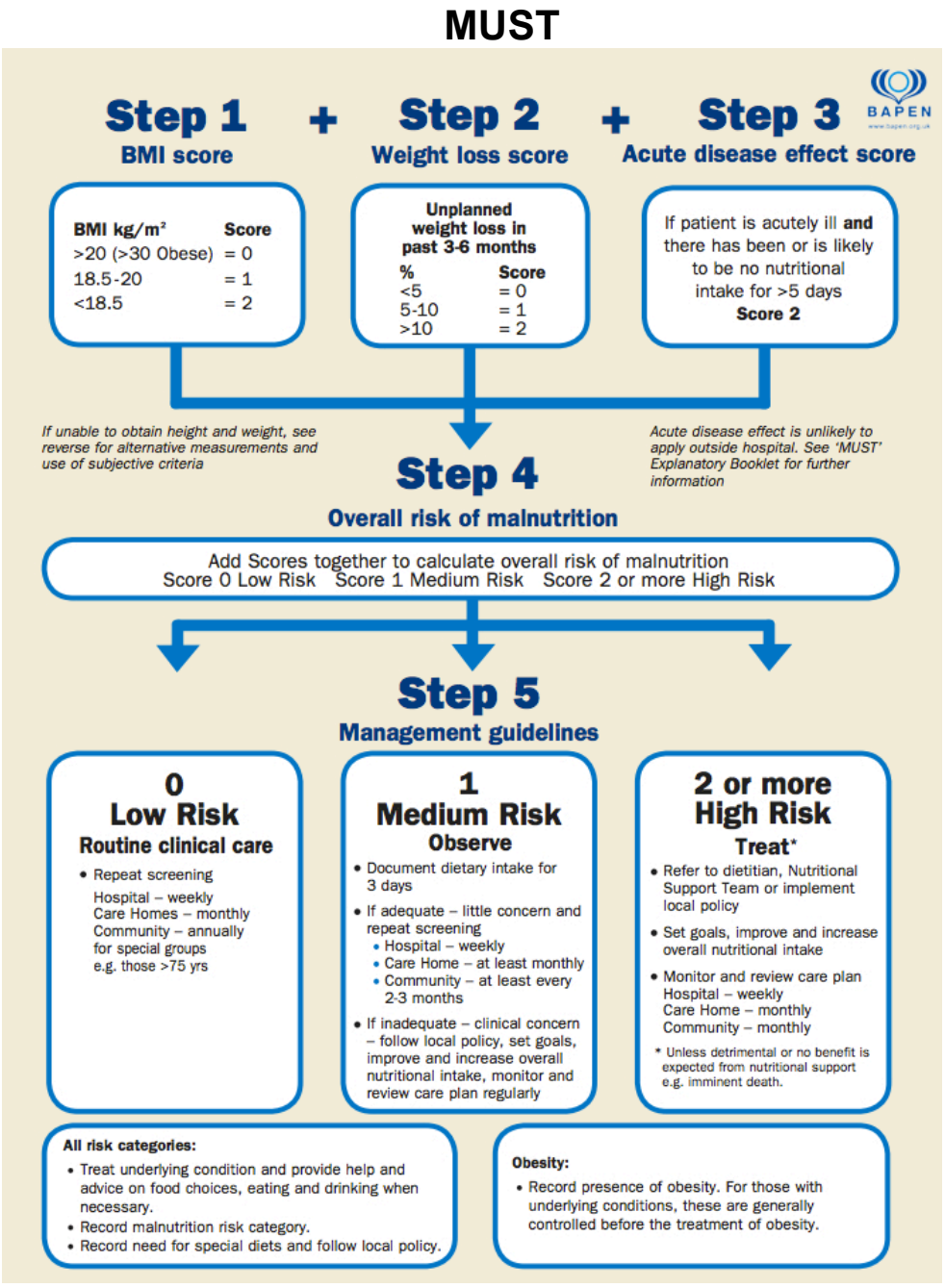

Figure: 2.3. MUST (2003).

Stratton et al. (2004) eloquently suggests these three parameters combined can independently predict clinical outcome, whilst individually these parameters reflect the patient's journey with weight loss and a predictive component of BMI to present clinical status of disease effect. Investigators such as Rice and Normand (2012) make mention of the MUST criteria and the operational functionality of the tool, which was used in the largest survey of disease-related malnutrition in Ireland in conjunction with the Nutrition Screening week 2010 (Elia et al., 2008; Freijer et al., 2013; Russell et al., 2011). Lamb's et al. (2009) study applied the MUST to all: medical, surgical, orthopaedic and critical care in-patients, 44% of the 328 patients who met the criteria in an North-East England acute hospital were found to be malnourished. These studies illustrate how the MUST has been used in clinical research, to demonstrate how effective this tool is in identifying malnutrition risk in all care settings. Finally hard copy version of the MUST tool has been traditionally used however, more recently a newly developed APP has been made available to increase usage of the MUST tool in all settings, shown in appendix B.

2.9.1. MUST limitations

MUST screening involve weight and height measures for the BMI and calculation this can pose a challenge in immobile patients. However, the MUST does allow for this with suggested alternative measures i.e. forearm length (ulna length). A limitation this creates is the continuous requirement mandating the use of a paper tape measure as many patients are at high infection risks or in isolation and this requirement prevents inadvertent spread of infection. A study by Kelly et al. (2000) concluded a practical issue of obtaining accurate measurement of height and weight for BMI calculation, requires regularly calibrated measurement equipment which is not always available or in good working order. Lastly Barker et al. (2011) cites a limitation of MUST is that is that it has not been validated for those patients with renal impairment (Barker et

al., 2011). The limitations of the MUST screen are minimal, like all nutrition screening they take time, which in a busy hospital is seen as a luxury.

2.10. LABORATORY MARKERS OF MALNUTRITON RISK

Nutritional assessment includes various nutritional parameters to assess malnutrition including: anthropometric and biochemical analysis. The clinical use of biological proteins as laboratory markers of malnutrition risk is due to the acute phase response to nutrient deprivation and disease-related malnutrition. To clarify, this acute phase response is explained as a prominent systematic biological reaction, with both positive e.g. CRP and negative e.g. prealbumin protein synthesis (Gruys, Toussaint, Niewold, & Koopmans, 2005). Which can alternate in synthesis during injury and/or illness and nutrient deprivation, often seen with prealbumin decreasing whilst CRP increases (Gruys et al., 2005). Visceral proteins are increasing in popularity as an assessment parameter of nutrition in the clinical practice setting, used often in conjunction with anthropometric measures. These proteins can be expeditiously evaluated and interpreted as nutritional deprivation and protein energy malnutrition are known to acutely alter biological protein synthesis (Bae et al., 2011). It has been suggested the quintessential nutritional laboratory marker should ideally have a: small body pool, short half-life, respond predictably and quickly to the incremental changes in protein catabolism and anabolism and be influenced by dietary intake (Beck & Rosenthal, 2002). Additionally they should not be majorly influenced by non-nutritional factors such as disease status e.g. liver and renal disease, be relatively inexpensive, readily available and simple to use. The most widely used laboratory indicators of malnutrition risk are the visceral proteins: Prealbumin, Albumin, Retinol Binding Protein (RPB) and Transferrin (Spiekerman, 1995). Four common visceral proteins synthesised by the nutrition organ the liver are discussed in table 2.3. in slightly more detail.

Table 2.3. Visceral Markers of Malnutrition Risk.

Retinal Binding Protein (RBP)	Transferrin	Prealbumin (Transthyretin)	Albumin
Half-life: *t½* ~12 hours	***Half-life:*** *t½* ~8-10 days	***Half-life:*** *t½* ~2.5 days	***Half-life:*** *t½* ~14-20 days
Increased by: Renal failure Dependent: On normal levels of Vitamin A & Zinc	***Increased by***: Iron deficiency Chronic blood loss Chronic renal failure Dehydration	***Increased by***: Severe renal failure Corticosteroids Oral Contraceptives	***Increased by***: Blood transfusions Severe renal failure Dehydration
Decreased by: Pernicious anaemia Anaemia of chronic disease.	***Decreased by:*** Pernicious anaemia Anaemia of chronic disease Folate deficiency Anaemia Over-hydration Chronic infection Acute catabolic states	***Decreased by:*** Inflammation (CRP ⇧) Hyperthyroidism Dialysis Severe liver disease Infection, stress, inflammation Decreased protein intake	***Decreased by:*** Inflammation, Infection, Metabolic Syndrome Burns Over-hydration Trauma, Post op, Corticosteroids Hepatic failure
Advantages: Short half life	***Advantages:*** Levels decrease in severe malnutrition	***Advantages:*** Relatively in-expense and simple to measure Excellent sensitivity Responses quickly to nutrition deprivation Short half-life. Tryptophan rich	***Advantages:*** Relatively in-expense and simple to measure Good measure of mortality
Reference range: 3.0-6.0 mg/dl (0.03 – 0.06 g/L)	***Reference range:*** 2.4-3.6 g/L	***Reference range:*** 0.2 0.3 g/L	***Reference range:*** 38-50 g/L (1-70 years) 36-50 g/L (>70 years)

(Mears, 2007; Parrish, 2006).

Although all of these visceral proteins have limitations of effective use, prealbumin appears to have the least limitations; each visceral protein and their individual limitations will be discussed in subsequent sections.

More recently concerns have been raised regarding the limitation of use of biological proteins for nutrition status due to the reprioritisation of negative acute phase proteins e.g. prealbumin, albumin, RBP and transferrin toward the positive acute phase protein CRP in times of acute metabolic stress response. Raynaud-Simon et al. (2011) provides statement regarding this physiological acute metabolic phase response during times of physical stress and/or injury that potentiates an inflammatory and endocrine reaction. This acute innate metabolic response to tissue injury alters the reprioritisation and synthesis by the liver of these visceral proteins e.g. prealbumin can potentially decrease as CRP increases (Shenkin, 2006; Whicher, Bienvenu, & Price, 1991). This metabolic alteration significantly increases the need for nutritional/energy input to prevent hyper-catabolism and a negative nitrogen balance (Raynaud-Simon et al., 2011). Gruys et al. (2005) states that the acute phase response can be used to assess reactive inflammatory process in times of nutritional deficits. In a state of acute or chronic disease, inflammation often increases whilst acute phase proteins: prealbumin and albumin diminish in conjunction with skeletal muscle proteolysis (Jensen et al., 2009). Additionally the anorectic effects of pro-inflammatory cytokines in the brain have a substantial effect on the neuroendocrine system that modulate intermediary metabolism of carbohydrate, fat, and protein substrates (Gruys et al., 2005; R. W. Johnson, 1997). Additionally implicated in this regulation of hypothalamic-pituitary outflow is the result of the brain sending signals to reduces food intake (Gruys et al., 2005; R. W. Johnson, 1997). Concluding that those with acute injury or acutely illness have subsequently reduced oral intake that is either patient directed or at times

clinician directed, as an example of this is in situations of bowel rest with Crohn's disease or other inflammatory bowel conditions.

2.11. PREALBUMIN (Transthyretin)

Prealbumin also known as Transthyretin is a biochemical marker of protein energy malnutrition. First identified in 1942, prealbumin is a visceral dual transport functional protein of retinol and vitamin A (Ingenbleek & Young, 1994; Myron Johnson et al., 2007; Robbins, 2002; Spiekerman, 1995). Prealbumin was found to have one of the four subunits and binding sites specifically allocated for retinol binding protein and thyroid hormones (Ingenbleek & Young, 1994; Myron Johnson et al., 2007; Robbins, 2002; Spiekerman, 1995). Illustration 2.1. Displays the protein structure showing the four subunits in separate colours for distinction.

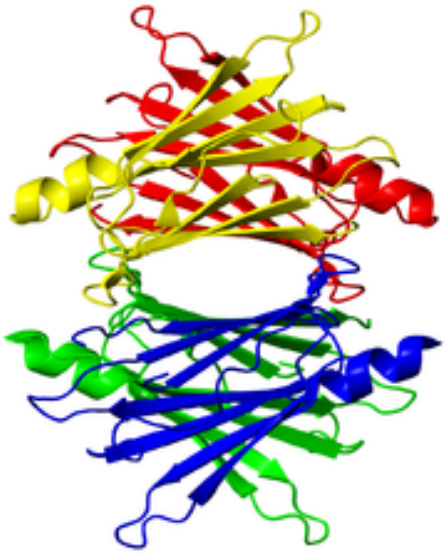

Illustration 2.1. Prealbumin Protein Structure (Wikimedia, 2005).

The hepatic synthesis of prealbumin necessitates a high concentration of the essential amino acid tryptophan, which plays a major role in protein synthesis initiation and is eminently sensitive to recent changes in nutritional status both protein and energy depletion (Bae et al., 2011; Ingenbleek & Young, 1994; Spiekerman, 1995). With a short half life of ~2-2.5 days the quantitation of

prealbumin is sensitive to changes in nutritional status due to its anabolic response to nutrient consumption and responds well to nutritional supplementation (Spiekerman, 1995). Prealbumin laboratory method is typically nephelometric or turbidimetric assay, which are more sensitive and precise than alternative methods including radial immunodiffusion assays (Potter & Luxton, 1999). There is a large volume of published studies describing the role of prealbumin in the detection of malnutrition risk (Devakonda et al., 2008; A. M. Johnson, Merlini, Sheldon, & Ichihara, 2007; Potter & Luxton, 1999; Robinson et al., 1990; Shenkin, 2006; Spiekerman, 1995). Several in-depth research studies into the role of prealbumin as a protein marker of nutritional status was first introduced by Ingenbleek in 1974 and 1975, he proposed the index of protein and energy malnutrition characterised by serum prealbumin changes and a faster response to refeeding (Ingenbleek, Vandenschrieck, Denayer, & Devisscher, 1975). In 1975 Ingenbleek, assessed malnutrition by comparing the response of four hepatic proteins: transferrin, albumin and prealbumin-retinol binding protein complex, findings suggested that albumin had low sensitivity, transferrin intermediate and prealbumin-retinol binding protein complex the highest sensitivity (Ingenbleek et al., 1975). Compelling evidence of a strong correlation between morbidity and mortality with hepatic protein synthesis, suggest that hepatic proteins help to identify malnutrition risk even if well nourished prior to onset of acute illness or injury (Fuhrman et al., 2004). However, as with RBP, Transferrin and Albumin, Prealbumin has its limitations although they are significantly less than the formentioned proteins.

2.11.1. Limitations of Prealbumin

The criticism and limitations suggested of prealbumin in regard to malnutrition risk assessment is largely due to the reprioritisation of prealbumin to CRP during acute inflammation, with CRP taking priority over prealbumin in hepatic synthesis and thereby limiting prealbumin specificity. However, other nutrition and non-nutritional components have received bad press as affecting prealbumin synthesis those being: zinc deficiency, acute alcohol intoxication, corticosteroid use and age. Several studies have examined the relationship between transport proteins: albumin, prealbumin biological zinc levels (Bates & McClain, 1981; Koch et al., 1996). Firstly the hepatic synthesis of prealbumin metabolism requires adequate zinc levels and may be depressed in times of deficiency or advanced states of malnutrition (Ingenbleek & Young, 1994; Koch et al., 1996). However, a documented complication of these studies is that the prevalence of malnutrition is a limiting factor on the severity of the relationship between transport proteins and zinc deficiency (Bates & McClain, 1981). Despite the known fact that zinc levels are necessary for prealbumin synthesis, it is worth pondering the multifactorial nature of zinc and whether the zinc supplementation or parental nutrition are fundamental factors of prealbumin increase (Bates & McClain, 1981). In respect to NZ the 1997 Ministry of Health (MoH) performed the National Nutrition Survey (NNS_{97}) which found that less than two percent of the New Zealand population were are risk of having inadequate zinc intake as zinc is widely available in the food supply (Ministry of Health, 2011/2012). Another noteworthy consideration is vitamin deficiency e.g. vitamin A does not affect prealbumin synthesis (Beck & Rosenthal, 2002; Ministry of Health, 2011/2012). The limitation of acute alcohol intoxication or binge drinking being potentially linked to a rise in prealbumin levels is due to the leakage of proteins caused by hepatic cell damage, worthy noting is that those individuals with chronic alcohol intoxication often supplement alcohol for

nutrition and are often malnourished (Bates & McClain, 1981). Finally prealbumin levels are thought to decrease after the age of 50 years in both men and women, this decrease is more pronounced in males but levels out for both sexes after the seventh decade of life, considering this limitation the thought that elderly are more at risk of malnutrition is additionally an important point to consider (Ingenbleek & Young, 1994). In conclusion despite these limitations, if these multitude of caveats are at least considered, the use of prealbumin as a predictive variable in nutrition assessment has been shown in the literature to be advantageous.

2.12. ALBUMIN

The prognostic value of the visceral protein albumin, has historically been used as a measure of acute and chronic malnutrition in hospitalised patient's and was traditionally the most frequently used laboratory marker of choice (Bae et al., 2011). However, with a half-life of ~14-20 days, albumin is relatively insensitive to rapid changes in nutritional status taking approximately 14 days to return to normal status (Beck & Rosenthal, 2002). Optimum albumin status occurs in an environment where nutritional adequacy relating to a sufficient supply of amino acids, hormonal and osmotic balance and the biological energy ATP and/or GTP is appropriate for synthesis (Nicholson, Wolmarans, & Park, 2000). Albumin is produced exclusively in the liver and is the most abundant visceral protein, despite as little as 5% being synthesised and degraded daily (Marshall, 2008; Nicholson et al., 2000). With five complex diverse functions albumin has a) a principal role as a carrier protein for thyroid hormones, steroids and fatty acids b) drug binding and transference c) is a main driver that influences and maintains colloid oncotic pressure, d) anti-thrombotic and platelet effects and lastly e) vascular permeability (Don & Kaysen, 2004; Nicholson et al., 2000; Parrish, 2006). The re-apportioning of albumin between intravascular and extravascular space occurs during times of acute and chronic stress on the

body as does the catabolism and anabolism of albumin (Bae et al., 2011; Nicholson et al., 2000). With a comparatively large body pool of approximately 3.5-5.0 g kg^{-1}, albumin concentration in the average healthy 70 kg adult equates to 250-300 g (Nicholson et al., 2000; Parrish, 2006). Albumin has a half–life of ~14-20 days with minimal daily urinary losses of roughly 10-20 mg. Additionally albumin passes through the kidneys with minimal loss and loses approximately 1 g through gastrointestinal loss (GI), as albumin is continuously reabsorbed by the body (Nicholson et al., 2000; Parrish, 2006). Composed of a single polypeptide chain of 585 amino acids, fasting can markedly reduce albumin synthesis, with dietary protein restriction having the most pronounced effect (Nicholson et al., 2000). Research has suggested that albumin is a preferable indicator of chronic rather than acute nutrient deficiency (Spiekerman, 1995). As Bae et al. (2011) suggests the sensitivity of albumin in acute protein malnutrition is more difficult to perceive because of the large half-life and body pool. A shift to low serum albumin concentration has been shown to be an excellent independent predictor of morbidity, stated by Ingenbleek and Young (1994) and Schneider et al. (2004) they also make mention that hypoalbuminaemia is a valid negative prognostic factor associated with increased mortality. Vincent et al. (2003) conducted a meta-analysis of 90 cohort studies relating to hypoalbuminaemia as predictor variable of poor outcome in 291,433 patients. Results demonstrated that for each 10 g/L decline in albumin, the odds of mortality increased by 137%, morbidity increased by 89%, prolonged Intensive Care Unit (ICU) stay increased by 28%, LOS by 17%, and increased resource utilization by 66% (Vincent et al., 2003). Nicholson et al. (2000) suggests there is evidence that each 2.5 g $litre^{-1}$ decrease in serum albumin is associated with a 24-56% risk of mortality with clear correlation between increased LOS, infection and complication rates of hospitalised

patients. Concluding that albumin is a better predictor of morbidity and mortality than an aberration in nutrition status.

2.12.1. Albumin Limitations

Fraught with limitations albumin as a marker of nutrition status is less sensitive marker primarily due to its long half-life (14-20 days), large body pool, reduced synthesis rate for metabolic recovery status and the inability to recognise short term changes in nutritional status (Ingenbleek & Young, 1994). Both nutritional factors and inflammation are contributory factors associated with low serum albumin levels. The decreased rate of albumin synthesis can be effected by several causal factors of illness through to a decrease in substrate availability or liver failure (Marshall, 2008). Evidence suggests the usefulness of albumin as a prognostic indicator of malnutrition could be questionable as it is highly influenced by the cytokine mediated inflammatory response (Covinsky, Covinsky, Palmer, & Sehgal, 2002; Jensen et al., 2009). Serum albumin levels are affected by multiple variables other than nutrition status including: impaired liver function, impaired kidney function through damage to glomeruli, dehydration and oedema, potentially leading to misinterpretation of possible nutritional changes (Nicholson et al., 2000; Potter & Luxton, 1999). In times of oedema and fluid overload, albumin transitions from intravascular compartments to extravascular compartments which may be precipitated by resuscitation fluids or drug reactions mediated in the hospital (Parrish, 2006). Additionally serum albumin may increase during times of dehydration and give a false negative result (Parrish, 2006). Baker et al. (1982) states that the prolonged half-life of serum albumin poses a potential issue in distinguishing alterations within nutritional status. Although albumin may not be a reliable marker of short-term changes in nutritional status, it is more likely a predictive marker of morbidity and mortality.

2.13. RETINOL BINDING PROTEIN

Retinol binding protein is collectively placed in the same category as the visceral proteins discussed previously. Similarly RBP is synthesized by the liver and catabolized in the kidneys. With a half-life of 12 hours RBP is circulated in a complex with prealbumin at a 1:1 ratio (Cavanna et al., 1985; Spiekerman, 1995). Used primarily as a metabolic marker of nutrition status, due to its biological plasma half-life and small body pool (Marshall, 2008). Like prealbumin, RBP responses quickly to positive and negative protein energy balance however, like the aforementioned visceral proteins discussed so far, RBP has its limitations. (Spiekerman, 1995).

2.13.1. Retinol Binding Protein Limitations

A significant potential drawback of RBP as a nutritional marker of malnutrition risk is that RBP is notably affected by renal failure. Excreted by the kidney RBP concentrations are elevated in those patients with chronic renal insufficiency due to decreased catabolism (Spiekerman, 1995). Additionally RBP is dependent on sufficient Vitamin A and zinc levels as deficiency inhibits liver production and movement of RBP, limiting its usability in isolation as a marker of malnutrition risk (Raguso, Dupertuis, & Pichard, 2003).

2.14. TRANSFERRIN

Influenced by iron metabolism, transferrin is an iron-transport protein synthesized in the liver (Spiekerman, 1995). Transferrin is a transport protein of ferric iron through blood to iron depot: liver spleen, bone marrow and organs that require iron, specifically haemopoietic tissue (Parrish, 2006; Spiekerman, 1995). Transferrin has a biological half life of ~9 days and a smaller body pool than albumin that responds to dietary changes quicker than albumin (Spiekerman, 1995).

2.14.1. Limitations of Transferrin

The usefulness of transferrin as a marker of malnutrition is questionable due to its role as an iron-transport protein directly affected by iron status (Parrish, 2006). Spiekerman (1995) suggests that although directly affected by dietary changes in protein and iron deficiency, transferrin is equally affected by non-nutritional factors such as liver disease, anaemia, hepatitis, pregnancy and some medications e.g. tetracycline and aminoglycosides and thereby limiting its usefulness. Although iron deficiency evokes the hepatic response for transferrin synthesis, synthesis is proportional to iron deficiency and low iron stores thus increasing serum levels, whereas on the flip side iron overload decreases transferrin synthesis (Parrish, 2006).

2.14.2. Summary of Laboratory markers of malnutrition risk

In conclusion, transferrin, RBP, prealbumin and albumin all have their limitations when used in isolation as laboratory markers of malnutrition risk. However, in conjunction with each other and/or with anthropometric and clinical judgement made by a nutrition specialist, they may all have a place in increasing malnutrition risk identification. Prealbumin was the laboratory marker of choice in this research due to the clear advantages discussed previously in this literature review.

2.15. C-REACTIVE PROTEIN

CRP is the final protein under discussion. Whereas the aforementioned proteins: prealbumin, transferrin, albumin and RBP are referred to as negative acute phase proteins, CRP is a positive acute phase protein. CRP is traditionally the most common laboratory indicator of acute and chronic inflammation, as its concentration increases within 6-12 hours post an inflammatory incident with initiation reflecting de novo synthesis (Husebekk & Hansson, 2009). CRP's ability to rise under acute conditions of inflammation is

dependant on the type of tissue injury e.g. wounds, burns or sepsis. However, CRP does not rise in alternative inflammatory conditions e.g. ulcerative colitis (Parrish, 2006; Spiekerman, 1995). Although other medical conditions including acute tissue trauma, head injury and acute episodes of Crohn's disease symptoms can all exacerbate proportional elevations in CRP (Jensen et al., 2009). It is worth noting the catabolism of skeletal muscles in times of acute inflammation, generates the building blocks to synthesise CRP in a cytokine medicated response (Jensen et al., 2010). As in a malnourished state, muscle proteolysis means amino acids transition from the muscle to the liver and these amino acids aid in gluconeogenesis production for energy requirements (Jensen et al., 2009). During the inflammatory response cytokines are released from cells and muscle proteolysis that occurs initiates a decrease prealbumin and albumin (Desborough, 2000; Whicher et al., 1991). An outstanding example of the relationship between inflammation and malnutrition was demonstrated in a study of 33 maintenance haemodialysis (MHD) patients, where malnutrition is more commonly referred to as malnutrition-inflammation complex syndrome (MICS) (Kalantar-Zadeh, Block, McAllister, Humphreys, & Kopple, 2004). Results from this study found the odds ratio for diminished versus normal appetite was significant for those patient's with increased CRP with the hazard ratio of death for diminished appetite at 4.74 (95% CI: 1.85, 12.16; P=0.001), confirming the relationship between diminished appetite, increased pro-inflammatory cytokines and inflammation and poorer clinical outcome (Kalantar-Zadeh et al., 2004). Acknowledgment that pro-inflammatory cytokines correlate closely with decreased appetite posing a risk of malnutrition (Devoto et al., 2006; Myron Johnson et al., 2007; Shenkin, 2006). Furthermore research has shown that the acute-phase metabolic response effects nutrition status through the elevation of the body's resting energy expenditure level and hence contributes to increased energy requirement (Jensen et al., 2009). With

increasing levels of inflammation the potential for anorexia occurs, which is often accompanied by a diminution of lean body mass (Jensen et al., 2009). These significant findings from these research studies shows hepatic production of prealbumin may decline in favour of CRP increasing, as a result of inflammation. Finally the conundrum still remain is that at times of inflammation the increase in inflammatory markers could potentiate a false negative assumption in regard to malnutrition status e.g. correlation between increased CRP as prealbumin decreases in volume. However, in times of acute/chronic inflammatory responses, linked to acute or chronic illness and/or injury the potential impacts on nutritional intake still exists and hence a decrease in prealbumin. Table 2.4. Illustrates the biological value of CRP.

Table 2.4. Visceral Protein: Positive acute phase protein CRP.

Visceral Protein	C-Reactive protein
Phase of Protein	Positive acute phase protein
Half life	t½ ~19 hours
Increased by	Increased by: onset of inflammation, infection, or tissue trauma
Reference range	<5 mg/L normal 10-40 mg/L mild inflammation 40-200 mg/L Acute inflammation, bacterial infections >200 mg/L Severe bacterial infection, malignancy, pancreatitis, extensive trauma etc.

(Gillanders, 2010; Parrish, 2006).

2.15.1. Limitations of CRP

Perhaps the most serious disadvantage cited in the literature is the complex enigmatic relationship between the two laboratory measures i.e. prealbumin in nutritional status and CRP which is and indicative measure of inflammatory response (Gallo et al., 2011). Although the inflammatory marker CRP can increase significantly during critical illness or acute major trauma, CRP values are non-specific and should not be interpreted without a complete clinical history. When utilising an marker for bacterial infections, the acute phase

protein CRP is well respected however, in the event of viral infection CRP is not a reliable measure (Whicher et al., 1991). The reviewed literature acknowledges that at times of acute physiological stress, illness or injury patients often suffer from shock, anorexia, vomiting or nausea, all of which effect dietary intake potentiating a consequential decrease in oral intake resulting unintentional weight loss and malnutrition, which can coincide with inflammation a which can be signified by a decrease in prealbumin and concurrently elevation in CRP (Spiekerman, 1995). Devakonda et al, (2008) conclude there is ample justification for the emergence of a combination approach to nutrition assessment using concurrent objective and subjective measurement of laboratory and clinical parameters.

2.16. RESEARCH STUDIES: Nutritional Screening: Anthropometric vs. Biochemical Markers

Robinson's et al. (2003) research used the nutrition screening protocol consisting of the laboratory markers: prealbumin, retinol-binding protein and albumin taken in conjunction with a nurse-completed nutritional questionnaire. The screened medical wards included: general medicine, general surgery, medical ICU, surgical ICU, bone marrow and transplant. The average age of the population was 56 ± 16 years with 59% (188/320) men and 41% (132/320) women participating in the study (Robinson et al., 2003). As previously reported 104 out of 320 (33%) of the study patients were found to be malnourished: with standard nutrition assessment protocol not triggered in 139 (43%) of patients who were not assessed by a RD, results are illustrated in figure 2.4. (Robinson et al., 2003).

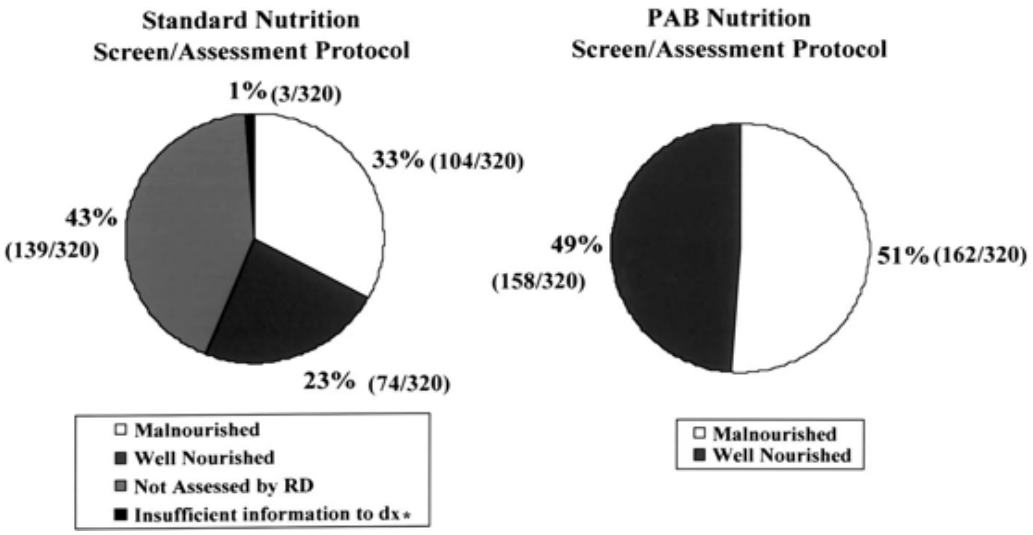

Figure 2.4. Results from Robinson et al. (2003) showing the nutritional screen and assessment using standard and prealbumin protocols.

Robinson et al. (2003) stated that ideally a nutrition expert should evaluate all hospital admissions as they are the expert in nutritional assessment who can utilise and interpret medical and nutrition histories, physical finding and laboratory data to quantify malnutrition risk. The final conclusion of this study was that a combination of screening and assessment techniques could more appropriately identify malnutrition, stating prealbumin is an important adjunct in overall nutrition management (Robinson et al., 2003, p.394). Another researcher Mears (1996) conducted three consecutive studies and while the third was similar to Robinson et al. (2003) one included a total of 177 patients randomly selected over a three month period who had prealbumin values recorded within 48 hours of admission. Prealbumin results that indicated a moderate to severe risk of malnutrition were referred to a RD, who performed a more comprehensive nutrition assessment. The quality improvement model describes the clinical pathway Mears used in this research shown in figure 2.5.

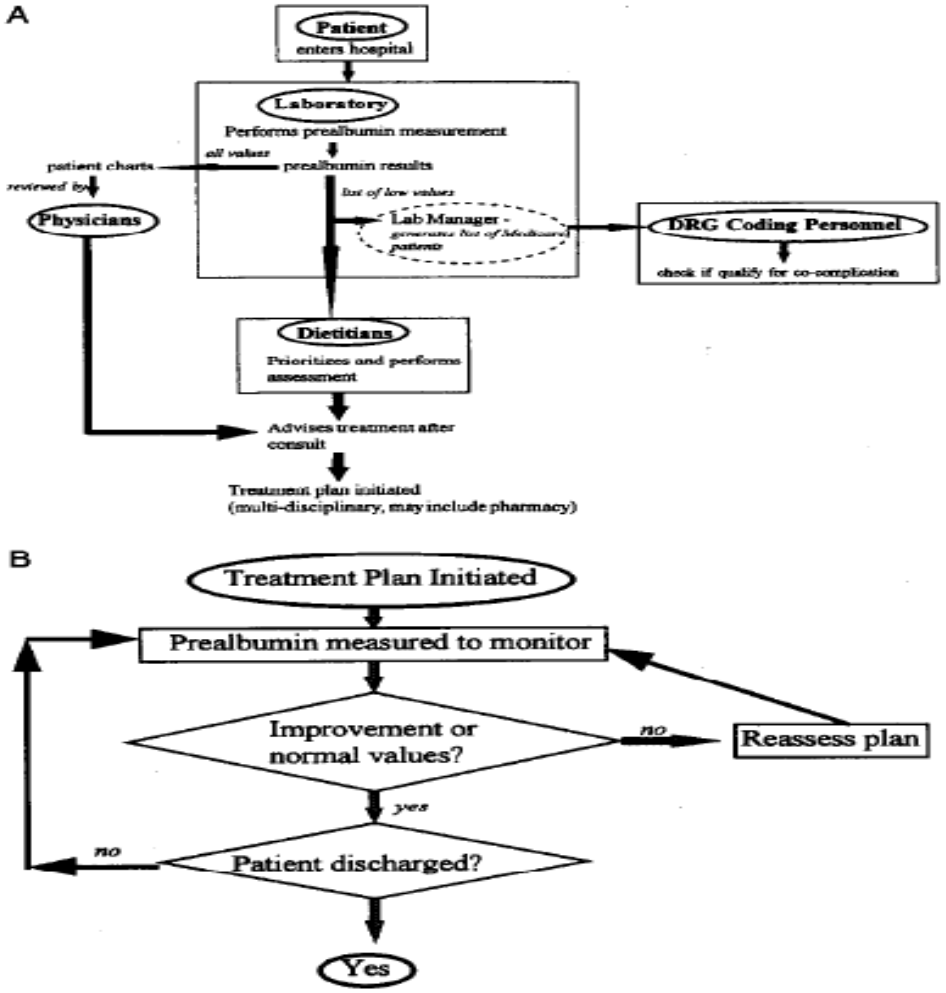

Figure 2.5. Mears (Mears, 1996) Quality improvement model describing the clinical pathway.

Of the 177 patients 79 (45%) were found to be at risk of PCM and were referred for nutrition treatment and monitoring. Additionally the LOS for malnourished patients who were nutritionally treated was significantly lower than patients not treated with the estimated daily saving projected to be $500 daily (Mears, 1999). The most striking result to emerge from this data was that as a result of this study the medical committee decided to incorporate prealbumin testing into the nutritional treatment plan for all new patients to improve patient care (Mears, 1999). In another study Bae et al. (2011) performed an observation study at Seoul National University Hospital which examined the effects of preoperative nutrition status on postoperative complications of 183 patients that had undergone gastric surgery. Their complication rate in those patient's with abnormal prealbumin was 52%, suggesting that prealbumin had exhibited

higher sensitivity when compared to albumin in regard to predicting postoperative complications after gastric surgery (0.235 vs. 0.139) and infection rate complications (0.278 vs. 0.167) (Bae et al., 2011). Furthermore these authors found that by excluding patients with inflammation and on corticosteroid therapy to improve reliability of results, preoperative abnormal prealbumin status was a sensitive predictor of postoperative complications (Bae et al., 2011). Devoto et al, (2006) published a research paper on 108 hospitalised patients at day three post admission which compared prealbumin to two nutritional assessment methods, the Subjective Global Assessment (SGA) and the Prognostic Inflammatory and Nutritional Index score (PINI), with the reference method of the Detailed Nutritional Assessment (DNA). The results of this study showed prealbumin had the best sensitivity of 83.7% and SGA the best specificity of 83.1% compared to the reference method DNA (Devoto et al., 2006). Demonstrating that prealbumin is a feasible and reliable tool in the evaluation of malnutrition, especially in situations where it is difficult to do a comprehensive nutrition assessment with prevalence data showing 60% of patients had various degrees of malnutrition (Devoto et al., 2006). A serious weakness in Devoto's argument was reported by Shenkin (2006) who proposed that the correlation between DNA and prealbumin with or without an increase in CRP maybe in-fact be questionable. Shenkin's (2006) rational for disputing this claim was that DNA unlike many alternative screening and assessment tool e.g. MUST, DNA is affected by multiple non-nutritional factors, additionally it does not contain nutritional risk markers of unintentional weight loss and/or decreased oral intake. A cohort study by Potter and Luxton (1999b) performed a pilot study to examine the effectiveness of routine prealbumin testing on emergency hospital admissions for protein calorie malnutrition (PCM) and concluded 24% had at least mild PCM, those with PCM had LOS 16 (SD: 18) days compared to 8 days (SD: 12) for those without PCM, in hospital mortality

was 17% compared to 4% consecutively with as few as 42% patients with PCM receiving nutritional supplementation. Additionally the cost analysis performed using prealbumin as a marker of malnutrition risk at the time of admission, was projected to save $414 per patient (Potter & Luxton, 1999). When considering this research was performed fourteen years ago and with the substantial increases in daily living expenditure, the projected cost saving for today could feasibly be considerably higher. As mentioned multiple studies have examined the prevalence of malnutrition in the hospital setting using a variety of screening and assessment tools with some showing interesting results.

2.17. BENEFITS OF NUTRITION ASSESSMENT & INTERVENTION

The benefits of nutrition screening, assessment and intervention have been studied over a number of years. Findings of these studies have unearthed multiple significant positive parameters in postoperative outcomes with oral nutrition support. A financial analysis undertaken in the United Kingdom of the current health expenditure, found that annual savings of £266 million, could be attained if 10% of hospital patients received nutritional support with a consequential reduction in the length of patients hospital stay by five days (Russell, 2007). Nemayer et al. (2001) performed a Clinical Practice Improvement (CPI) study and found early and sufficient nutrition resulted in a shorter LOS 11.9 days compared to 14.8 days for those patients who did not receive nutrition intervention. A randomised control trial study performed by Beattie et all (2000) examined the use of enteral nutrition supplementation compared with no supplementation on screened postoperatively in malnourished surgical patients in Ninewells Hospital and Medical School, Tayside University Hospitals in Dundee, UK Results concluded that those who received supplementation compared with the control group lost less weigh 3.4kg (SD:0.89), anthropometry and grip strength were similarly significantly different between groups ($p<0.001$) with fewer patients in the nutritional support

group (7/52) requiring antibiotic prescriptions compared with (15/49) control group (Beattie et al., 2000). Nutrition intervention studies have consistently found those given nutrition intervention have a better clinical outcome than malnourished patients who received no nutritional intervention.

2.18. SUMMARY

Disease-related malnutrition in the hospital setting is generally under recognised, unacknowledged, undiagnosed and hence untreated. Thus a vicious cycle potentially ensues with those malnourished and acutely or chronically ill as illness often threatens the normality of food intake potentially for a extended period of time (Allison, 2000; Desborough, 2000). Individuals that are critically ill, in immense pain, unconscious, or suffering from a stroke having inhibited speech or dementia may not always be able to provide sufficient accurate information for nutrition assessment (White et al., 2012). The classification of malnourished compared to those, at risk of malnutrition needs to be definitively quantified as disease-related malnutrition is often missed or unappreciated by clinicians. Those already malnourished require immediate nutrition intervention to prevent further clinical deterioration and decrease the risk of morbidity and mortality. Whereas those at risk of disease-related malnutrition having increased metabolic demands and nutrient requirements require more detailed clinical assessment performed by a dietitian with an appropriate intervention to prevent further deterioration.

The diagnosis of malnutrition can be objectively constructed in a tiered approach using equitable measures including various anthropometric measurements, clinical, dietary intake assessment and biochemical measurement of visceral proteins. A combination of these measurement variables for malnutrition risk assessment may have epidemiological foundation that correlates with the improvement in disease-related malnutrition diagnosis and treatment. Shenkin (2006) raises the point that although prealbumin as a

marker of change in hepatic protein synthesis has been known for decades, it has only recently been recognised and considered for the inclusion into clinical practice. Additionally they suggest that the most valuable interpretation of biochemical assessment would be better achieved through multiple prealbumin screenings in conjunction with CRP (Shenkin, 2006). The studies that examined prealbumin suggest a strong correlation between increasing prealbumin and the effectiveness of nutritional intervention with decreased length of hospital stay (Gallo et al., 2011; Mears, 1996; Potter & Luxton, 1999b). As the results of the research study in prealbumin screening was discussed previously from Mears (1996) the findings of the research, resulted in prealbumin screening being introduced as a new protocol for their institution to determine nutrition status.

This literature review has endeavoured to reveal the broad spectrum of malnutrition prevalence internationally, screening and valid assessment tools, potential laboratory markers of malnutrition risk and as well as the rational behind these rates of disease-related malnutrition risk from: disparity in nutrition knowledge and education, locality and timing of malnutrition assessment. There is agreement that the management of disease-related malnutrition through screening assessment and intervention is interdisciplinary through allied health professionals.

Dietitians are nutrition experts that specialise in scientifically sound nutrition advice, based on the latest evidence based practice in nutrition assessment and intervention and adhere to a professional code of practice with patient centred care a primary focus. Although dietitians acknowledge that malnutrition recognition is paramount especially at time of admission, interdisciplinary team management often hinders screening. Through a variety of reasons e.g. lack of time, un-calibrated or unavailable weighing tools, inconsistent nutrition beliefs, training and staffing levels of nutrition providers notwithstanding the importance placed on malnutrition acknowledgement, all of which diminish patient's

standards of care (Walton, 2009). Well recognised is the need for an interdisciplinary approach to identify and treat malnutrition at admission. With on-going research which endeavours to unveil a most accurate biochemical marker with the least limitations, that assimilates closely to anthropometric markers of malnutrition risk.

Undeniably whilst the cost of daily life and health care are escalating exponentially, available resources are diminishing. The associated comorbidities of disease-related malnutrition often result in increased treatment costs which are ultimately thrust upon community and national healthcare systems (Correia & Waitzberg, 2003). It could be argued that research has already shown the immense potential for a vast reduction in hospital based disease-related malnutrition. The leading question as to whether these rapidly escalating costs associated with the adverse effects of undiagnosed disease-related malnutrition, are sustainable long-term. It is proposed that as a direct reflection of early referral, diagnosis and treatment of disease-related malnutrition the increasing cost to the health care budget will be limited. This research aims to provide evidenced based rationale for the inclusion of prealbumin into nutrition screening protocol at the time of patient admission, that initiates an automatic referral to a dietitian when abnormal results are indicated.

3.0 METHOLOGY

3.1. RESEARCH DESIGN INTRODUCTION

This chapter contains an overview of the research process. The primary aim was to investigate whether the introduction of an obligatory test of the visceral protein prealbumin, would improve the number of patients identified at risk of malnutrition. However, as this was an observational study research supervisors and academic advisers agreed that prealbumin education to clinicians' prior the routine introduction of prealbumin screening could potentially bias results, therefore this was not pursued.

It is anticipated that by increasing the number of those referred for more in-depth nutrition assessment by a registered dietitian employed at NSH will ultimately improve clinical outcome. Currently at North Shore Hospital the standard clinical documentation which is completed at first by the admitting doctor, encompasses the MUST score as integral component of the Admission-to-Discharge Planner. Once transferred to the ward the nursing process theoretically involves the completion of a second MUST score sheet and weighing of the patient. Each ward has its own allocated dietitian, who assesses nutrition status and the patient's individual requirements using a myriad of clinical judgement and assessments including the use of the PG-SGA.

3.2. ROUTINE CLINICAL ASSESSMENT

As an integral component of clinical assessment the Malnutrition Universal Screening Tool (MUST) for adults was adopted as a routine clinical nutrition assessment integrated into the Admission to Discharge Planner at NSH in 2006. The 'MUST' is a five-step screening tool developed by BAPEN to identify Adults, who are malnourished or at risk of malnutrition in a wide range of clinical settings (BAPEN, 2013b). A key step in the MUST screening is unplanned weight loss over a 3-6 month timeframe. The practice of measuring unplanned

weight loss is a quantifiable well published predictive parameter of malnutrition risk (Norman et al., 2008). On completion of the MUST assessment, those patients that have a MUST screen score of 2 or greater are required to be referred to the Nutrition Services Department for dietetic assessment. It is noted however that at NSH various alternative clinical assessment practices for malnutrition risk assessment are used as part of the referral process, but are not formalised into the patient care pathway (Yovich, 2012).

Table 3.1. MUST Measurement Criteria

Steps	Measurement	Score
Step 1:	**BMI Kg/m2**	**Score**
BMI	>20 (>30 Obese)	0
	18.5-20	1
	<18.5	2
Step 2:	Unplanned weight loss in 3-6 months	**Score**
Weight loss	**%**	
	<5	0
	5-10	1
	>10	2
Step 3:	If patient is acutely ill and there has been or is likely to be not nutrition intake for >5 days.	**Score**
Acute disease effect		2
Step 4:		
Overall risk of malnutrition	Add scores together to calculate overall risk.	If score equals 2 or more refer
Step 5:	**Guidelines**	**Score**
Management Guidelines	1 Lower Risk: Routine Clinical Care	0
	2 Medium Risk: Observe	1
	3 High Risk: Treat	2 or more

3.3. PARTICIPANTS

It was calculated that a minimum of 240 patients would be required for phase two in order to demonstrate a difference of 20% in the proportion of patients diagnosed as malnourished using prealbumin (50%) compared to routine malnutrition screening (30%) a minimum of 120 patients are needed per phase at $\alpha=0.05$ and $\beta=0.1$ with a power of 90% (Robinson et al., 2003). The power calculations were based on data retrieved from Robinson et al. (2003) with the addition of the pilot data recorded in November 2012, where 214 referrals were received due to a high MUST score/week/ward at NSH. On average the test wards 3,4,7,8 and 10 admit and discharge 350 patients per month each. It was estimated that 25% of admitted patients would be discharged before the results are placed on Concerto^{TM}. Acting on this estimate, a minimum of 700 patients was needed for prealbumin screening tests to ensure an adequate comparative sample was assessed. On admission the patient gives verbal consent to admitting doctor for the extraction of bloods for sampling. Therefore patient's permission was not sought directly to participate in this study all publishable information was made unidentifiable. Due to not obtaining a contractual agreement to collect extra bloods, any patient who had an insufficient blood sample on admission was excluded from the study. Consequently no additional consent was required. Those patients admitted to the wards from Sunday to Thursday were chosen for the study. This time frame was due to the dietitians limited availability to assess referred patients after Friday midday, or on Saturday and Sunday. Some referrals sent on Friday afternoon were potentially included if seen early Monday morning.

3.3.1. Inclusion Criteria

Eligibility criteria required individual patients either male or female admitted acutely on the study days, Sunday to Thursday of each week to wards: Ward 3 Acute Medical, Ward 4 Acute Surgical, Ward 7 Acute Orthopaedic, Ward 8 Acute Surgical and ward 10 Acute Medical at NSH, Auckland. Because NSH is an adult hospital, the age was restricted to men and women 16 years and older. A total of 77 patient referrals were recorded for phase I and a total of 564 patients, 283 men and 281 women were included in phase II of the study.

3.3.2. Exclusion Criteria

There were no specified exclusion criteria for participation in this research. The exclusion of any patient results was primarily due to the insufficient amount of blood sample remaining for visceral protein analysis. The reason for this was potentially due to a recent blood transfusion or the large number of blood testing requirements on admission. Alternatively whether patients had been recently transferred from another ward or hospital decreased the availability and time frame of available blood samples for testing. Admission bloods were often not required for day stay patients or alternatively blood sampling had been undertaking at community laboratories prior admission e.g. for gynaecological or urological investigation.

3.4. ETHICAL APPROVAL AND CONSENT

The design of this study used only admission blood samples, which meant that patients did not require any additional blood sampling. Furthermore as consent is given for admission bloods to be extracted on hospital admission no secondary consent was sought. Patient information collected from the patient hospital records (Concerto^{TM}) was made unidentifiable with NHI numbers and clinical information not drafted within this research documentation. The sole

demographic information used for statistical analysis was age and gender all other personal information was excluded.

A Human and Disability Ethics Committee (HDEC) Full Review pathway was conducted with a review meeting attended by the Main Researcher and WDHB Supervisor with the Human and Disability Ethics committee in Auckland.

- Application Submitted on 30/01/2013.
- Review completed on February 15/02/2013.
- Approved by the HDEC Northern A Health and Disability Ethics Committee, approval received on the 15th February 2013 (Ref: 13/NTA/16).

The main issues raised and discussed at the HDEC meeting in Auckland were as follows:

- The Committee noted that no participant information would be provided in this study. That as researchers we had adequately explained that formal written consent was not obtained routinely to take blood samples, and that the tests were largely a matter of clinical judgement. Additionally the proteins to be tested as part of this study did not have a genetic component and fell within the range of laboratory test that it was reasonable for people to expect to be tested in the hospital setting. The testing was additionally an alternative to a nurse-lead nutrition screening that was routine protocol for the hospital, for which written consent was not normally obtained. No additional tissue/blood sample would be obtained as part of the study, should there be inadequate remaining admission blood serum.
- The Committee additionally noted that people who would be unable to give written consent due to plausible explanations, one being current medical condition on admission. These participants may be over

represented in terms of malnutrition, and that including these individuals in the study would increase the power of the study.

- The Committee also discussed the possibility for the study to generate benefits for Māori, and suggested that there be greater engagement with Māori on the study.
- The committee requested clarification as to whether the recommendation in Massey University's peer review letter had been accepted. The peer review letter is attached in Appendix C. The minor recommendations that were made and discussed with the committee. Included whether teaching the house officers of North Shore Hospital regarding prealbumin in reference to malnutrition risk, prior to the laboratory marker being placed on the hospital patient results portal "Concerto"; would prejudice the observation criteria of this study. The conclusive agreement was made that yes any additional prealbumin training would bias the study and that training would not take place. The HDEC committee were in agreement with the decision. Information pertaining to the study was kept in either a locked drawer or remained at NSH and all electronic files were password protected.

  A copy of the ethics approval letter is attached in Appendix D.

Additionally this research was registered through the Australian New Zealand Clinical Trials Registry.

Trial ID ACTRN12613000154707

Trial Status: Registered

Date Submitted: 7/02/2013

Date Registered: 8/02/2013

3.5. FUNDING

Siemens NZ supplied partial funding by way of a half price reduction in prealbumin testing kit costs. Additionally one of the small grants was allocated by the New Zealand Society of Gastroenterology, which covered the laboratory costs. The grant approval letter is provided in Appendix E.

3.6. METHODS AND MATERIALS

Admission blood samples were collected via venepuncture by a trained WDHB phlebotomist. Blood samples were then sent via a pneumatic tube system that transports specimens from the wards directly to the laboratory. The sample tubes were then accurately bar coded with patient details and specimen specifications for sampling were entered into the laboratory computer system. The blood samples undergo the biological process of complete clot formation. Once clot formation is completed the sample is centrifuged to separate the plasma component from the red bloods cells. Plasma samples are typically held in the laboratory cold storeroom in heparinised tubes at 4° for up to one week. According to manufacturers instructions the reagent is stored at 2-8°. Prealbumin testing was performed with 38 hours post admission. The reagent used is PREALB Flex® reagent cartridge, Cat. No. K7064. Sampling, reagent delivery, mixing, and processing are automatically performed by the Dimension Vista® System (Siemens, 2012). All study samples were analysed on a Dimension Vista® analyser (Siemens Healthcare Diagnostics, Australia and New Zealand) at NSH Laboratory. Figure 3.2. shows Angela Pountney the WDHB laboratory service lead biochemist with the Dimension Vista® Analyser at NSH laboratory (Siemens, 2012).

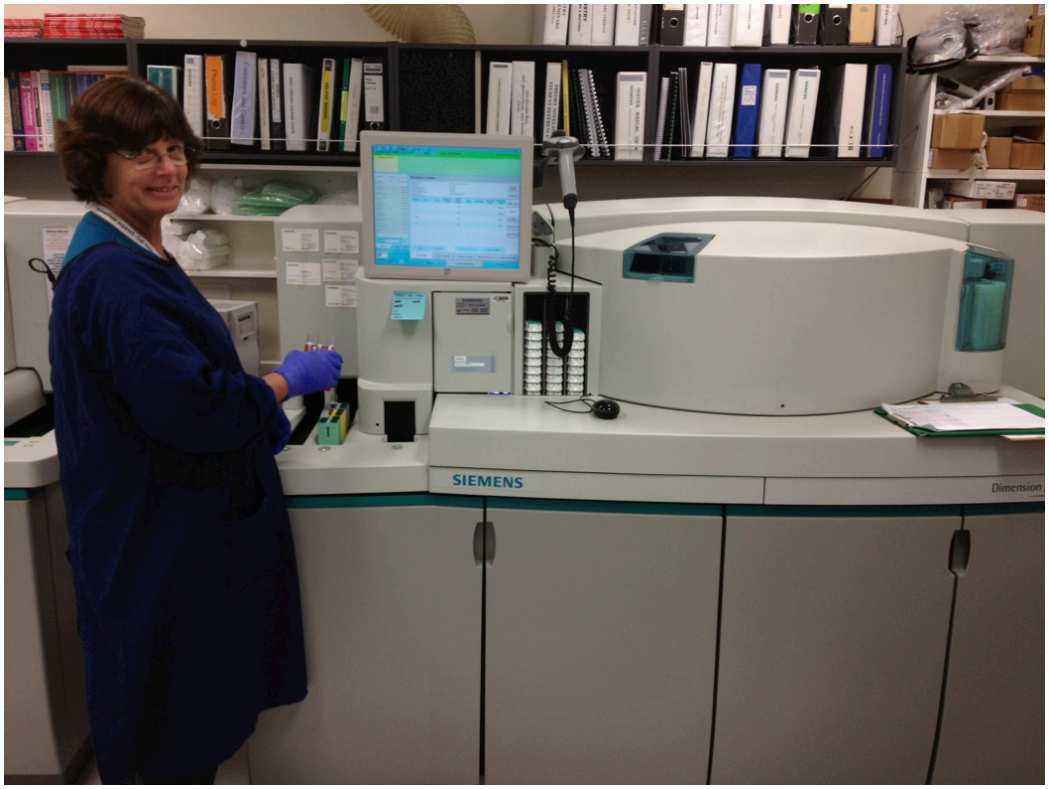

Figure 3.1.: Angela Pountney (Service Lead Biochemistry, WDHB) and the Dimension Vista® analyser

Using an in-vitro diagnostic test the setting for the nephelometric test is set at 840 nm and requires 6 minutes to perform a quantitative measure of prealbumin. The serum parameters of prealbumin reference range for healthy adult is: 0.2 - 0.3 g/L with an uncertainty of measurement of 11% at 0.35 g/L and 7% at 0.1 g/L calculates the concentration of prealbumin in mg/dL [g/L] using the calculation scheme (Pountney, 2013). Prior to the commencement of the research an alert was added to the concerto. All prealbumin results were made available immediately through being posted on the WDHB computer patient result portal, Concerto^{TM}. Abnormal results of <0.2 g/L were indicated by a green 'NUTRITION' on the laboratory page in Concerto^{TM} of each patient which activated an automatic alert in red which stated.

"*A low prealbumin may indicate a failure of hepatic synthesis as a result of malnutrition, hepatic injury or inflammation, or be due to loss of protein as in protein losing enteropathy. A referral to the ward dietitian is appropriate".* An example of this page is illustrated in figure 3.2.

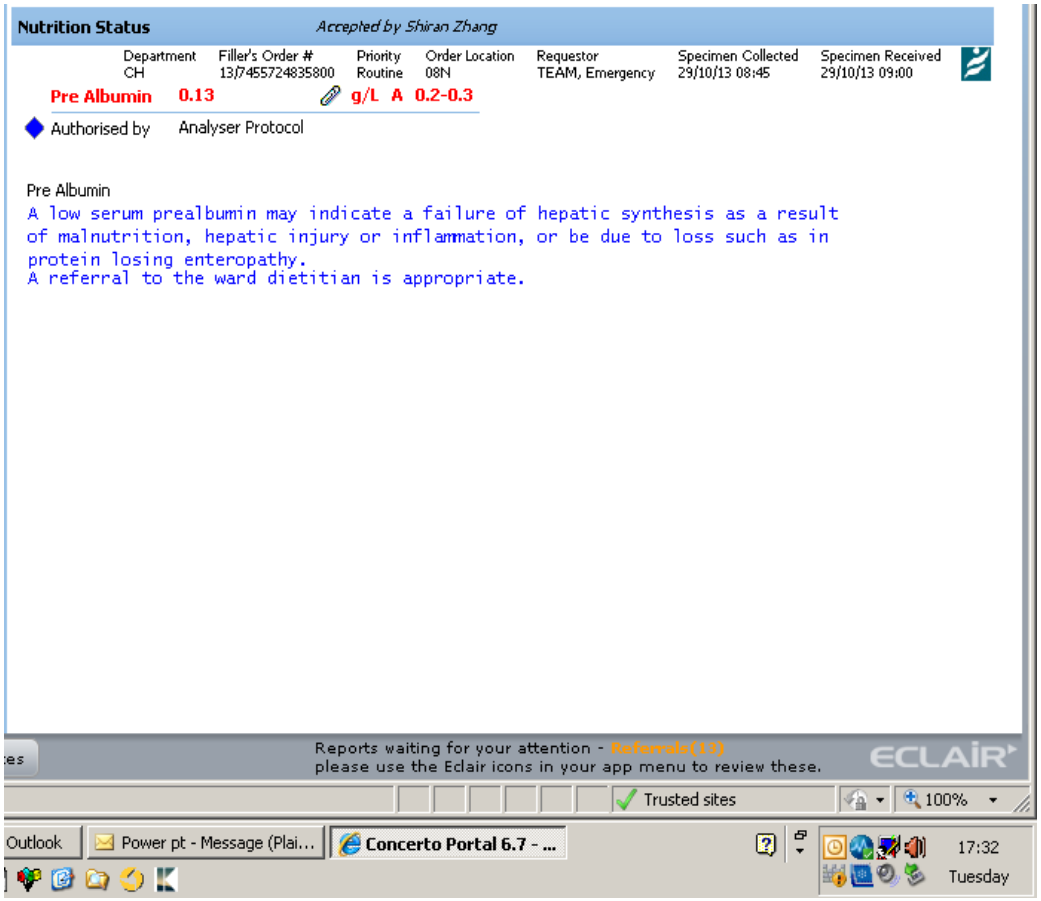

Figure 3.2. Concerto™ patient portal laboratory example.

CRP testing was undertaken through in-vitro diagnostic testing with the sampling, reagent delivery, mixing, and processing automatically performed for the quantitative measurement in human serum and heparinised plasma. The nephelometric test is set at 840 nm testing required a reaction time of 5 minutes and 50 seconds. CRP is a marker of inflammatory status and in this research the measurement was used to assess any inverse correlation with prealbumin results. The statistical analysis is available in the results section below. All patient lists were summited to the laboratory for testing no later than 7.30am had prealbumin and CRP results were placed on concerto prior most wards rounds each morning at 8.30am.

3.7. STUDY DESIGN

This study was conducted at NSH in Auckland, a 480-bed hospital facility that serves the entire Waitemata District of Auckland. The WDHB is the largest and second fastest growing district health board in NZ (Ministry of Health, 2010). Servicing the healthcare requirements of 560,000 residents of the North Shore including: North Shore City, Waitakere City and the Rodney District (Ministry of Health, 2010).

The study design consisted of a two-phase prospective observational cohort study (Figure 3.3.). The inclusion criteria were consecutive patients admitted to two acute surgical, one acute orthopaedic and two acute medical wards. Phase I was carried-out Monday to Friday from the 11th of February until the 8th of March 2013. Included primary data collection for the control period's statistical analysis, which is discussed in more depth in section 3.11.

Throughout the study the ward dietitian record the following data set during phase I and 2: NHI number; date admitted, ward, date referred to nutrition services, who referred, what screening tool triggered referral (MUST, prealbumin or other); MUST score, prealbumin level, PG-SGA score; subjective assessment whether malnourished or not; degree of malnutrition (subjective) mild/moderate/severe; whether discharged before being seen. An in-depth analysis of both phases of this research will be discussed further in the methods and materials section below.

Study Design: Improving Early Assessment of Malnutrition in Hospitalised Patients; Prealbumin versus Routine Clinical Assessment

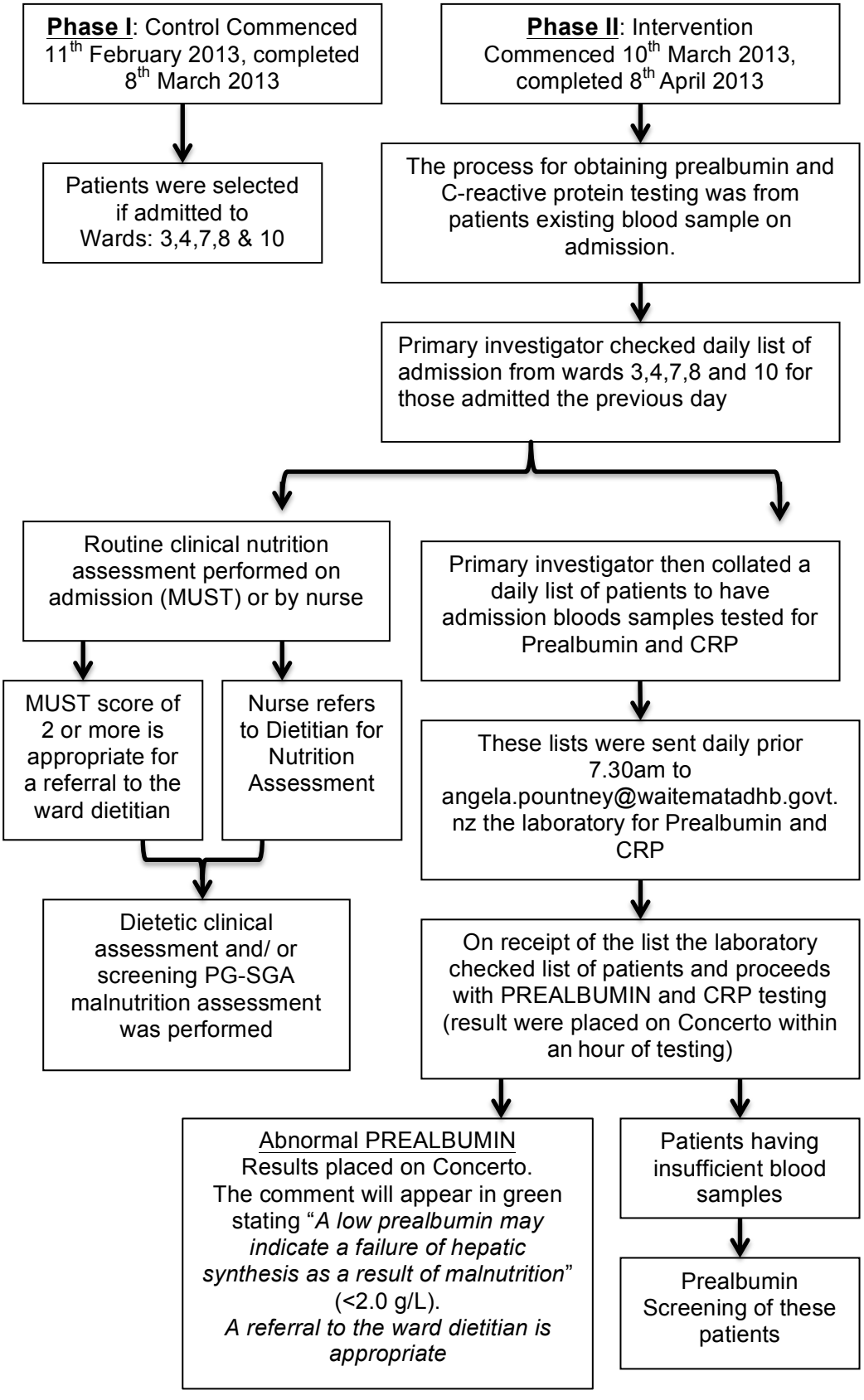

Figure 3.3. Study Design

3.8. RESEARCH STUDY METHODOLOGY

Presentation of the research proposal to the NSH Dietitians.

The research proposal was presented by power point to the NSH dietitians prior the commencement of phase two on February 2013. The presentation unveiled the meagre amount of literature pertaining to the estimated disease-related prevalence in NZ. In comparison with the significant amount of literature pertaining to the estimated prevalence internationally. The main concerns outlined are the growing trend of hospital malnutrition. The results of the MUST audit performed by NSH Dietitians in 2009 was presented to the newer dietitians and reviewed by the more experienced dietitians. The outlined method of data collection and collation of results was proposed and the desired results and additional workload discussed. The dietitians would be required to complete the Dietetic Inpatient Referral Data Collection Form each day and perform a PG-SGA on appropriate patients as agreed and signed off by the nutrition department of NSH. The dietitians raised concerns regarding one of the test wards, which have a large number of bariatric patients. It was pointed out by one of the dietitians that most of these patients were unlikely to have dietary protein deficit and hence low visceral protein or require blood tests on admission. All concerns raised were discussed in-depth with suitable measures to prevent confounding factors applied e.g. patients without admission bloods would not be eligible for this study. Additionally these patients are predominately elective patients with some patient's part of a concurrent bariatric research project being undertaken at NSH. The dietitians proposed that the PG-SGA are not performed routinely and that clinical judgement is within their protocol as to whether to use this form of assessment which is often not applicable for all patients. It was therefore agreed that where appropriate, dietitians would continue to use their clinical judgement when screening using

the PG-SGA. Additionally there were no specific exclusion criteria for these predefined research wards.

3.9. RESEARCH PROCESS PHASE I: (Control phase)

Phase I comprised of 76 patients and ran consecutively over the time period of the 11th of February until the 8th of March 2013. The first was solely the observational phase with no alteration to nutrition assessment including MUST screening and routine clinical care. The five study wards: Ward 3, Acute Medical, Ward 4 Acute Surgical, Ward 7, Acute Orthopaedic, Ward 8 Acute Surgical and Ward 10 Acute Medical were selected for the inclusion into this study. The patients that were referred either by MUST or an alternative method for a nutrition assessment were assessed by a registered dietitian inline with WDHB protocol.

Data collection for phase I, was recorded on a '*Dietetic Inpatient Referral Data Collection Form'* by the appropriate ward dietitian for each ward. The recorded data included: Patient NHI number; date admitted, ward, date referred to nutrition services, who referred, what screening tool triggered referral (MUST, prealbumin or other); MUST score, prealbumin level, PG-SGA score; subjective assessment as to whether malnourished or not; degree of malnutrition (subjective) moderate/severe; whether discharged before being seen. This form is available for viewing in Appendix E. This data was then collated in a spreadsheet where demographic information was appended which included: age and gender.

3.10. RESEARCH PROCESS PHASE II: (Prealbumin testing)

Phase II ran over consecutive weeks over a timeline of Monday to Friday from the 10th March until the 8th April 2013.

During phase II the protocol of routine clinical screening for malnutrition risk that resulted in a dietetic referral for the assessment and/or intervention as performed appropriately was identical to phase I. The data also recorded on '*Dietetic Inpatient Referral Data Collection Form'* for the preselected research Wards 3, 4, 7, 8 10 by the ward dietitian as in phase I.

Routine clinical care plus access to prealbumin results were included. During phase II, the admission bloods that requested by the admitting doctor were extracted. Patients who were newly admitted to the test wards were identified for the addition of prealbumin testing. Selection of these newly admitted patients were through the daily ward lists. These daily ward lists were procured through the WDHB computer system; printed and all new admissions were highlighted for prealbumin testing by the lead researcher. Once completed this lists were then delivered each morning to the laboratory for prealbumin and CRP testing and placement of results on Concerto TM prior ward rounds each morning.

Due to insufficient data an audit of patient records was required. A request was provided to Phillip Roxborough at WDHB clinical records department at NSH. The request was approved and a total of 78 records were gathered. A list of NHI numbers was presented to the clinical notes room where Jasmin O'Sullivan (WDHB) collected the relevant patient records. The information required was the confirmation regarding the presence of a MUST screening in the patients clinical notes and confirmation of dietetic input.

3.11. STATISTICAL ANALYSIS

All statistical analysis was conducted using IBM® SPSS® Statistics *20 (*IBM Corp. Released 2011. IBM SPSS Statistics for Mac, Version 20.0. Armonk, NY: IBM Corp). Prior to the commencement of statistical analysis the data was cleaned and checked for coding errors. The sample size power calculation was calculated at $\alpha=0.05$ and $\beta=0.1$ with a power of 90%.

The statistical analysis of the variables is shown in table 3.2. The central tendency of: mean, range and standard deviation was deemed appropriate for the demographic of age. With central tendency: mean and standard deviation calculated for prealbumin, using Pearson's correlation to examine the relationship between prealbumin and CRP.

Table 3.2. Table of

Variables	**Analysis**
Age	Central tendency: Mean, Range and SD (±) Proportion (*n*,%)
Gender	Proportion (*n*,%)
Prealbumin	Central tendency: Mean ± SD Proportion (*n*/%) Correlation: CRP/Prealbumin

4.0. RESULTS

4.1. INTRODUCTION

Review of patient selection: phase I on the 11th of February and concluded on the 8th of March 2013. The first phase was solely the observational phase with no alteration to routine clinical assessment for nutrition status. The clinical dietitians recorded data using the '*Dietetic Inpatient Referral Data Collection Form'*. Phase two of the research protocol ran consecutively from 10th March until the 8th April 2013. All potential participants were selected through the use of the electronic notes programme Concerto™. Patients admitted daily to each of the five wards had their NHI number highlighted on the daily ward lists. These lists were then presented to the laboratory each day no later than 7.30am. Results were made available before 8.30 enabling clinicians to view prior ward rounds. After cleaning up the data as previously described, the following results were made available.

4.2. DEMOGRAPHIC ANALYSIS

The demographic results of the study population referred for dietetic assessment in phase I revealed the age range of the patient population was between 16 and 96 years (mean age: 76 years: Standard Deviation (SD±19).

During phase I, 76 patients from a total of 970 patients admitted to five speciality wards were referred for dietetic assessment. The majority of the patients 71% were aged ≥65 years 54/76), with the acute orthopaedic ward having the oldest representative population at 80 (SD±9) years. However, this ward generated the lowest number of referrals (n=3). The highest number of referrals came from ward 10 an acute medical ward (n=22), with the mean age of 75 (SD±19) years. The gender representation was unevenly split with 62% (n=47) male and 38% (n=29) females.

The demographic results differed in phase II with a total of 776 patients admitted over the study period and put forward for prealbumin testing. The data was cleaned for statistical analysis and 212 patient NHI numbers were removed.

Results for 564 patients were available to be statistically analysed. The gender representation in phase II of those tested for prealbumin was more evenly split with males representing 24 (56%) of the 43 referrals. The highest number of admissions came from ward 10 acute medical 137 (24%). In comparing the information from this study to the data from Robinson et al. (2003) their study examined the malnutrition risk of a total of 320 patients. Moreover, in this study the patients were admitted predominately to an acute surgical ward when compared with the findings from Robinson et al. (2003) whose patients were admitted to an acute medical ward. The average age of the population in Robinson et al. (2003) was 56 years with more men than women (59% vs. 41%) whereas while the average age of those screened in phase II in this study was 66 years, the male ratio however was similar (56% vs. 44%). Table 4.1. depicts the admission-related characteristics including the demographic results of age and gender for both phase I and phase II.

Results have been stated in range, mean and standard deviation to depict the average age of the study population and standard deviation showing the variability and spread of age from the mean in table 4.1.

TABLE 4.1. Primary and Secondary Endpoint Referral Demographics

Characteristic[1]	**Phase I** [2]	**Phase II**[3]
1° End Point: Referrals[4]	76 (7.8%)	43 (97.6%)
Age Range (years)[7]	16-96	26-92
Mean Age (years)[7]	76 ± 19	66 ± 17
Male (*n*[8], %[9])	47 (62%)	24 (56%)
2° End Point: Referrals[5]		
Malnutrition (Prealbumin <0.2g/L)	N/A	155 (27%)
Average age each study ward[6]		
Ward 3 Acute Medical (yrs.)[7] SD[10]	74 ± 16	69 ± 19
Ward 4 Acute Surgical (yrs.)[7] SD[10]	62 ± 21	57 ± 20
Ward 7 Acute Orthopaedic (yrs.)[7] SD[10]	80 ± 9	55 ± 22
Ward 8 Acute Surgical (yrs.)[7] SD[10]	64 ± 15	60 ± 18
Ward 10 Acute Medical (yrs.)[7] SD[10]	75 ± 19	72 ± 17

Key

[1] Characteristics
[2] Phase I: Control period
[3] Phase II: Study Period
[4] 1° End Point: Referrals
[5] 2° End Point: Referrals
[6] Ward
[7] Years
[8] Number
[9] Percentage
[10] Standard Deviation

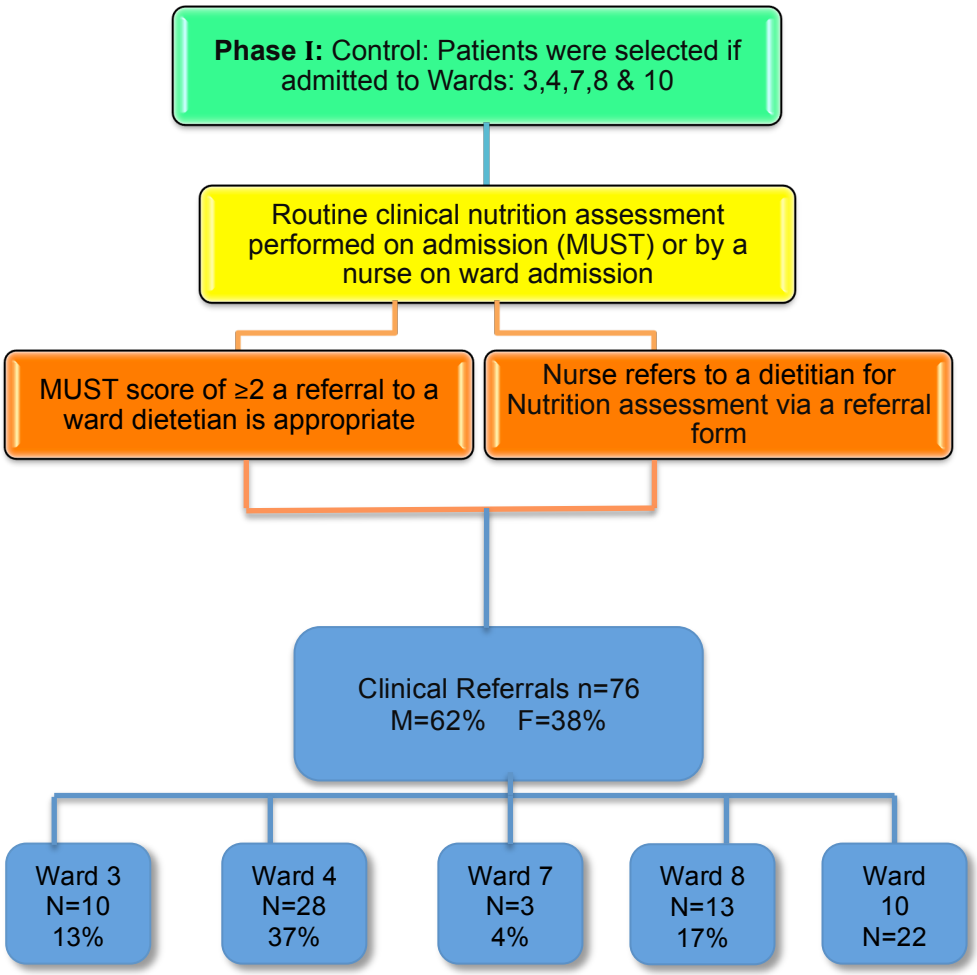

Figure 4.1. Phase I: Participant selection pathways.

In phase II the demographic results of the study population referred for dietetic assessment was 43. Compared with phase I these results show a relatively even representation between men and women (56% vs. 44%) and are made up of 24 male and 19 female patients. The age range of the referred patient population was 26 to 92 years (mean age: 66 (SD± 17) years, with 53% (23/43) of those referred ≥65 years. The oldest population groups were in the two acute medical wards (3 & 10). Within these five study wards of: acute medical, surgical and orthopaedic wards the breakdown of patient numbers tested for prealbumin came from the following wards. Acute medical ward 3, *n*=106 (20%), which incorporates a coronary care unit, cardiac investigation and palliative care. Ward 10 the second acute medical ward specialising in a wide range of

medical conditions that includes complex infection control had *n*=137 (24%) patients tested for prealbumin. Women's health and urology, hepatobiliary, bariatric, and upper gastrointestinal acute surgery is covered by ward 4 acute surgical, which had *n*=109 (19%) patients tested. Ward 8, the second acute surgical ward specialising in colorectal, breast and endocrine surgery had *n*=114 (20%) patients tested. Finally ward 7 an acute orthopaedic ward which provides care to patients with complex orthopaedic injuries had *n*=97 (17%) patients tested. With figure 4.2. demonstrating the number of patients per research ward selected for prealbumin testing. Figure 4.2. is a visual illustration of the selection pathway of the five study wards in phase two of this study.

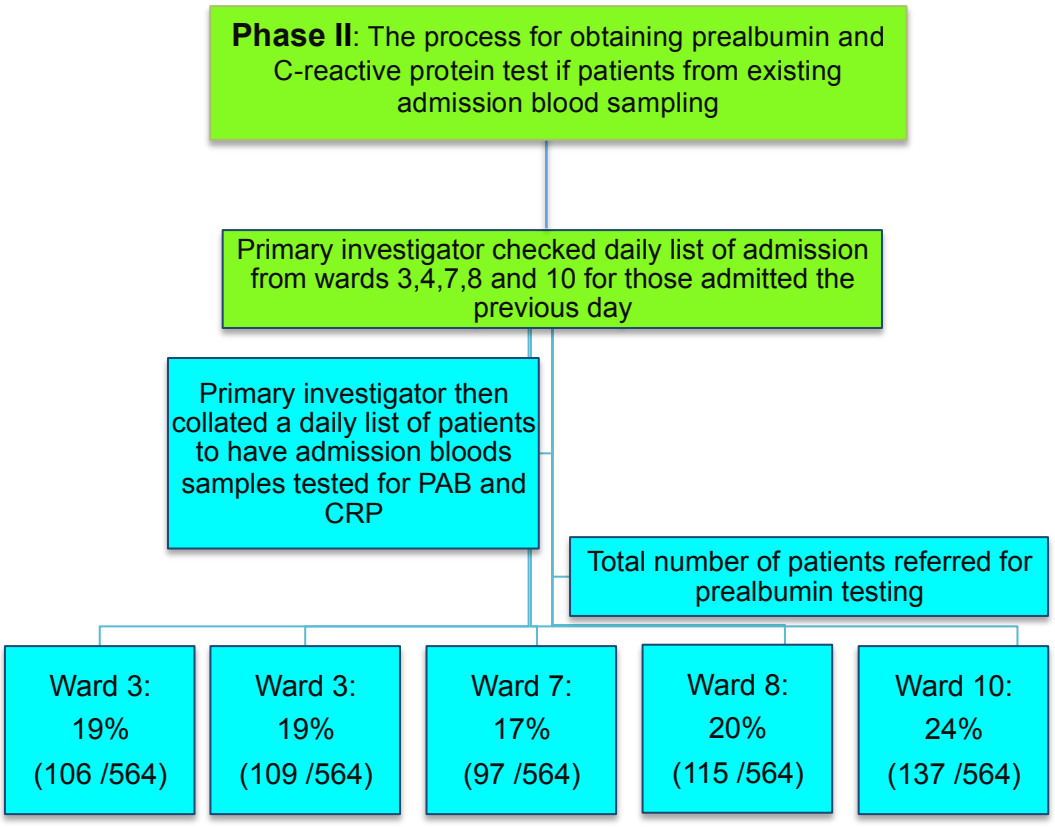

Figure 4.2. Study phase II: Participant selection pathway.

4.3. MUST PROTOCOL RESULTS

Phase I shows the total of 7.8% (76/970) patients received a dietetic referral from the 970 admissions to the five speciality wards. A total of 28% (21/76) patients had a completed MUST in their clinical records remembering for the MUST to initiate a referral score must be ≥2. The results also indicated that a completed MUST was not performed on 72% (55/76). The referrals were initiated by one of the medical team completing a dietetic referral form as shown in appendix G. However, what remains uncertain is the total number of patients that had a MUST completed at the first stage of malnutrition screening protocol of NSH in the Admission to Discharge Planner. Table 4.2. illustrates the MUST screening data available in clinical notes over the five study wards from phase I.

Table 4.2. MUST Results/Ward: Phase I.

		Ward 3: *Acute Medical*	Ward 4: *Acute Surgical*	Ward 7: *Acute Orthopaedic*	Ward 8: *Acute Surgical*	Ward 10: *Acute Medical*	Total number
MUST	Yes	3	2	0	2	14	21
	No	7	26	3	11	8	55

Table 4.3. Shows the number of patients from the five study wards that had a completed MUST screen documented in their clinical records. As seen from the tabulated results only 22 patient records from the total of 43 patients in phase II that were referred for dietetic input contained a completed MUST screen.

Table 4.3. MUST Results/Ward: Phase II.

		Ward 3: *Acute Medical*	Ward 4: *Acute Surgical*	Ward 7: *Acute Orthopaedic*	Ward 8: *Acute Surgical*	Ward 10: *Acute Medical*	Total number
MUST	Yes	3	3	1	1	14	22
	No	1	17	0	1	2	21

Interestingly table 4.4. illustrates the number of referrals generated 7.6% (43/564) during phase II by means other than abnormal prealbumin e.g. doctor, nurse, consultant or MUST screen, compared to the number of completed

MUST screens 51% (22/43). This confirms that abnormal prealbumin levels (n=155), did not procure any additional referrals from clinicians. Section two of this table demonstrates the number of patients at risk of malnutrition in each ward which equated to a total of 27% (155/564). Results indicated that of the total 155 patients that had abnormal prealbumin <0.2 g/L as few as 13 had been referred to a dietitian through an alternative pathway. This translates to mean 142 patients with abnormal prealbumin were not seen by a dietitian on admission.

Table 4.4. MUST/Referral/ Prealbumin <0.2 g/L per 5 Study Wards.

	Ward 3: *Acute Medical*	Ward 4: *Acute Surgical*	Ward 7: *Acute Orthopaedic*	Ward 8: *Acute Surgical*	Ward 10: *Acute Medical*	Total (%/*n*)
Referral[1]	4	20	1	2	16	7.6% (43/564)
MUST completed[2]	3	3	1	1	14	51% (22/43)
Prealbumin <0.2 g/L[3] Malnutrition Risk	27	33	19	39	37	27% (155/564)

Key

[1] Total number of patient's referral during Phase II.

[2] Number of patients referred that had a MUST screen in clinical notes.

[3] Total number of patients with abnormal prealbumin results.

[4] Total numbers of patients referred by means other, than abnormal prealbumin.

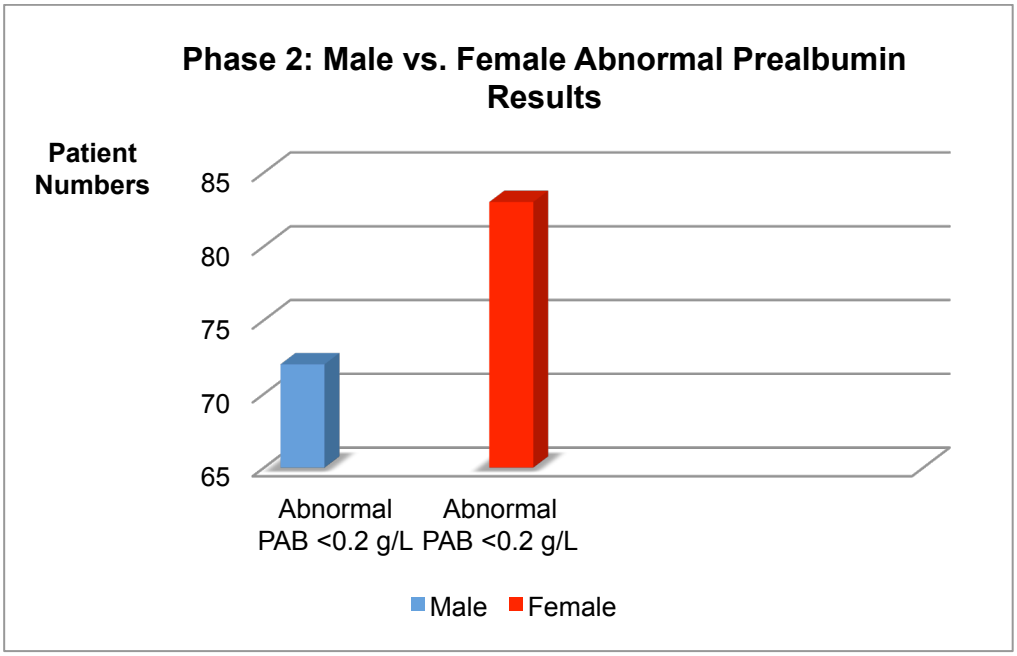

Figure 4.3. Phase II: Male vs. Female Abnormal Prealbumin Results.

This graph shows that more women (*n*=83) than men (*n*=72) were found to have abnormal prealbumin levels and to be at risk of malnutrition. The spread of patients with abnormal prealbumin in phase II, came from the following ward breakdown: Ward 4 Acute Surgical (*n*=33), Ward 8 Acute Surgical (*n*=39) and ward 10 acute medical (*n*=37) had the highest number of patients with prealbumin <0.2 g/L indicating malnutrition risk. In ward 4, acute surgical there were a total of 20 referrals over this study period. Three of those referrals had a completed MUST screen and 33 of the patients had prealbumin <0.2 g/L. Of those 33 that had low prealbumin <0.2 g/L as few a 7 received a referral for dietetic assessment. The referral, however, was not made because of the low prealbumin results. An additionally significant finding was that in ward 8 which had the highest number of patients (n=39) with prealbumin levels <0.2 g/L, only 2 patients had referrals for dietetic assessment. The results indicate that while one patient had had a MUST completed none of the patients with low prealbumin had been referred for a dietetic comprehensive assessment.

MUST compared to total number of referrals per ward: Phase II

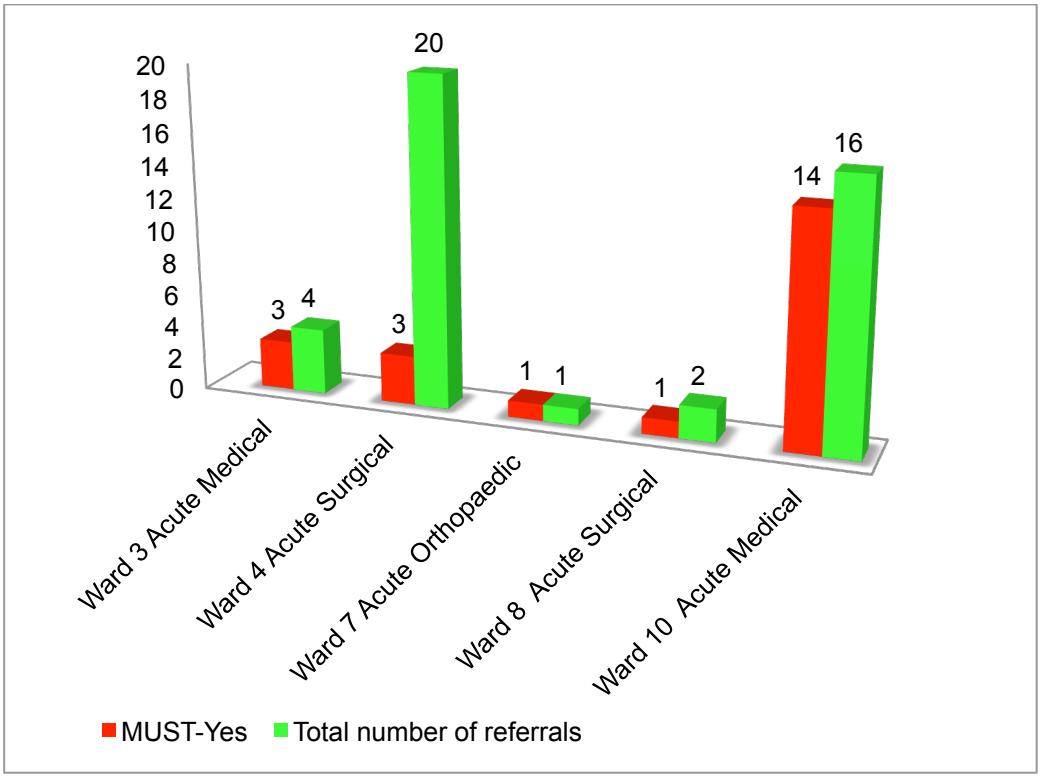

Figure 4.4. MUST screen/referral compared to total number of referrals per speciality ward: Phase II

4.4. PRIMARY ENDPOINT- MORE PATIENTS IDENTIFIED AT RISK OF MALNUTRITION; PREALBUMIN RESULTS

The first set of analyses examined the patient's prealbumin levels to compare the proportion of abnormal to normal prealbumin results. Analysis was performed within 48 hours of admission and although a comprehensive list of patients was given daily to the laboratory for prealbumin testing, it was inevitable that some patients would not qualify for inclusion. The data was cleaned appropriately to reflect patient lists that excluded approximately 212 patients. Patient's were excluded for the following reasons: having insufficient admission blood samples to procure the prealbumin test, were only an overnight stay, had received a recent blood transfusion which inadvertently increases prealbumin (Parrish, 2006), were discharged within 24 hours, did not have

admission blood tests or blood tests were taken at a community laboratory and tested prior admission, were transferred from another hospital or ward or lastly were gynaecological and urological patients not requiring blood tests on admission.

The outcome data of prealbumin tests was available for 564 patients admitted to the five test wards during phase II. Using prealbumin standard reference range 0.2-0.3 g/L supplied by Siemens NZ, Ltd (Siemens, 2012). Malnutrition risk was defined as <0.2 g/L, with patient's readings >0.2 g/L considered not at risk of malnutrition at the time of admission (Siemens, 2012). Once the NSH laboratory had completed the prealbumin analysis, results were placed on Concerto ™, with abnormal results those being <0.2 g/L placed in green under nutrition, a total of 155 patients were confirmed with prealbumin results <0.20 g/L and 409 with normal results ≥0.2 g/L. The alert was activated for 155 (155/564) patients with abnormal prealbumin results.

Blood was successfully tested for 564 patients in phase II, the test results indicating abnormal serum prealbumin levels of <0.2 g/L. A total of 155 patients had low prealbumin levels below <0.2 g/L. The range of prealbumin results is illustrated in table 4.5. there is a significant difference between the lower limit of patient prealbumin level of 0.03 g/L and the upper limit of 0.54 g/L.

TABLE 4.5. Prealbumin characteristics Phase: 2

Prealbumin	**g/L[1]**	
Mean (SD)[2]	0.24 (±0.83)	
Range	0.03 – 0.54	
Patients Prealbumin Results	**g/L[1]**	***n*[3]**
Prealbumin	<0.2	55
	0.2	25
	≥0.2	384

Key
[1] Grams per litre
[2] Standard Deviation
[3] Number

Abnormal prealbumin levels are presented for each ward and include: combined acute medical wards with 42% (64/155), combined acute surgical

wards at 46% (72/155) and the acute orthopaedic ward at 12% (19/155). The prevalence of malnutrition risk between the five test wards was significantly different. Ward 7 acute orthopaedic ward had the lowest number of patients with abnormal prealbumin levels (<0.2 g/L) at 19/124 (12%) patients and the highest was ward 8 acute surgical with 39/115 (figure 4.5.). Of the five test wards low prealbumin when compared with the total number of prealbumin tests per ward was shown to be more evident in wards 8 and 4 both surgical wards had the highest number of abnormal prealbumin followed by 3 and 10 both medical wards and finally ward 7 that had the lowest percentage of patients with low prealbumin <0.2 g/L. The acute orthopaedic ward also had the lowest total admissions *n*=124 compared to ward 4 acute surgical with the highest number of admissions *n*=181.

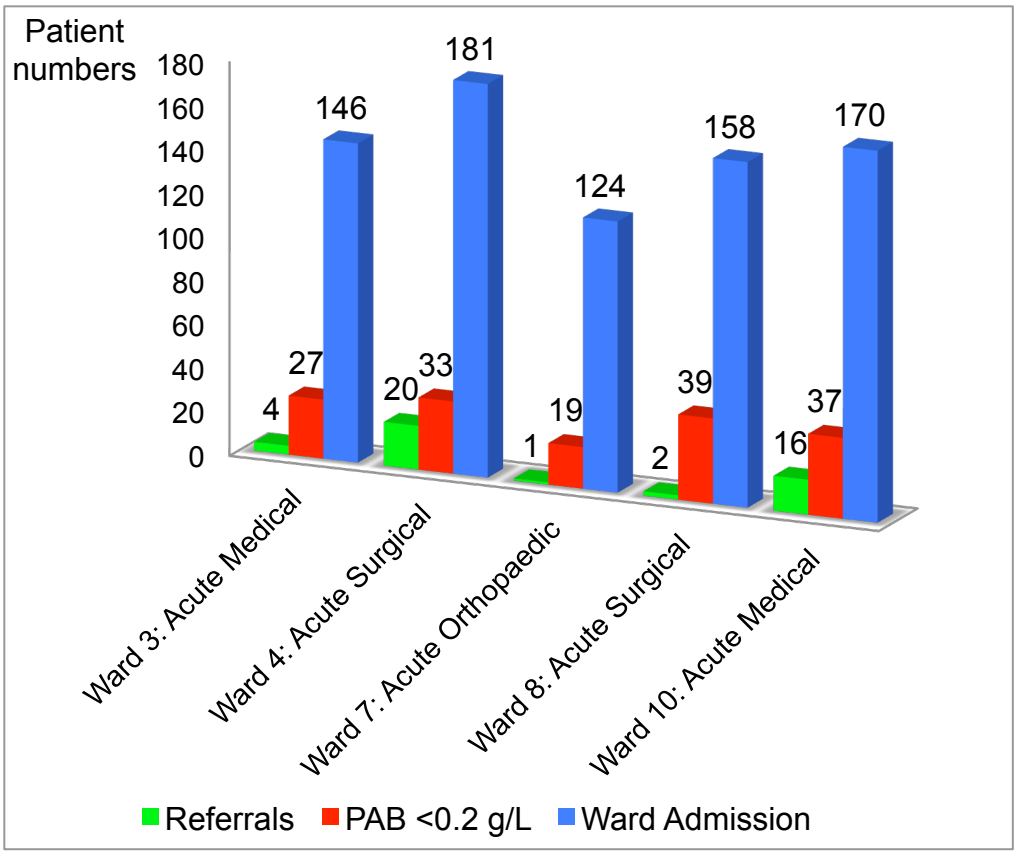

Figure 4.5. Results of Phase II by Speciality Ward.

Within this hospital setting the medical staff that would check daily blood results would typically be: house officers, registrars, consultants and dietitians. The abnormal prealbumin results <0.20 g/L, were intended to trigger additional referrals sent from this list of clinicians. Dietitians were excluded in this instance as they typically check the blood results of patients that have already been referred to them, checking laboratory blood results is an integral component of their comprehensive clinical assessment. Abnormal prealbumin results were intended to initiate new referrals to the dietitians from clinicians having read the alert on Concerto ^{TM} of their patients with abnormal prealbumin levels. However, despite the introduction of the obligatory test prealbumin test in the test wards, which identified 155 patients were at risk of malnutrition no additional referrals were generated by the clinicians in the research test wards 3,4,7,8 and 10 at NSH.

Additionally the majority of abnormal patient results did lie in the range of 0.1-0.15 g/L which was well below the normal reference range of 0.2-0.3 g/L. Interestingly 25 patients prealbumin tests results were border-line indicated with levels of 0.2 g/L. The cut off value for the normal prealbumin range in this study was 0.2 g/L, patients whose results indicated 0.2g/L could also potentially be at risk of malnutrition. As when these patients at borderline risk first enter the hospital system, a decreased oral intake is a reality and often transpires depending on disease-related effects of their condition. The decline in oral/nutritional intake on admission is well documented with the potential for prealbumin levels *(t½*=~2-2.5 days) to fall dramatically within the first few day post admission (Norman et al., 2008). However, this research only examined the prealbumin levels on admission and did not follow up with prealbumin testing at days 2,4,6 and 8 post admissions to identify changes in prealbumin levels (prealbumin t½ 2-2.5 days).

4.5. PREALBUMIN/CRP CORRELATION

Within the literature the inverse correlation between acute phase protein CRP and prealbumin is frequently scrutinised. All participants were screened for CRP in conjunction with prealbumin to extrapolate a significant inverse correlation. Table 4.5. has grouped patients by CRP value into CRP stratified range of severity as proposed by Dietitians NZ. The CRP measurements were then compared with abnormal and normal prealbumin levels. Results indicated 72% of the patients with mild inflammation (CRP 10-40 g/L) had normal prealbumin levels, 39% of patients with normal prealbumin had acute inflammation 40-200 g/L, 33% of patients with CRP >200 g/L had normal prealbumin levels, In correlation with the literature these findings support the a significant inverse relationship between prealbumin and CRP at the $p<0.01$ level (2-tailed).

Figure 4.6. is a visual representation of the CRP stratification compared to both abnormal and normal prealbumin values.

Table 4.6. Analysis of CRP stratification vs. prealbumin

	CRP (*n*)	Prealbumin <0.2 g/L (*n*/%)	Prealbumin ≥0.2 g/L (*n*/%)
<5 normal	217	17 (8%)	200 (92%)
5-9	59	7 (12%)	52 (88%)
10-40 Mild inflammation.	134	37 (28%)	97 (72%)
0-200 Acute inflammation, bacterial infections.	134	82 (61%)	52 (39%)
>200 Severe bacterial infection, malignancy, pancreatitis, extensive trauma etc.	18	12 (67%)	6 (33%)
Total	564	155	409

(Gillanders, 2010).

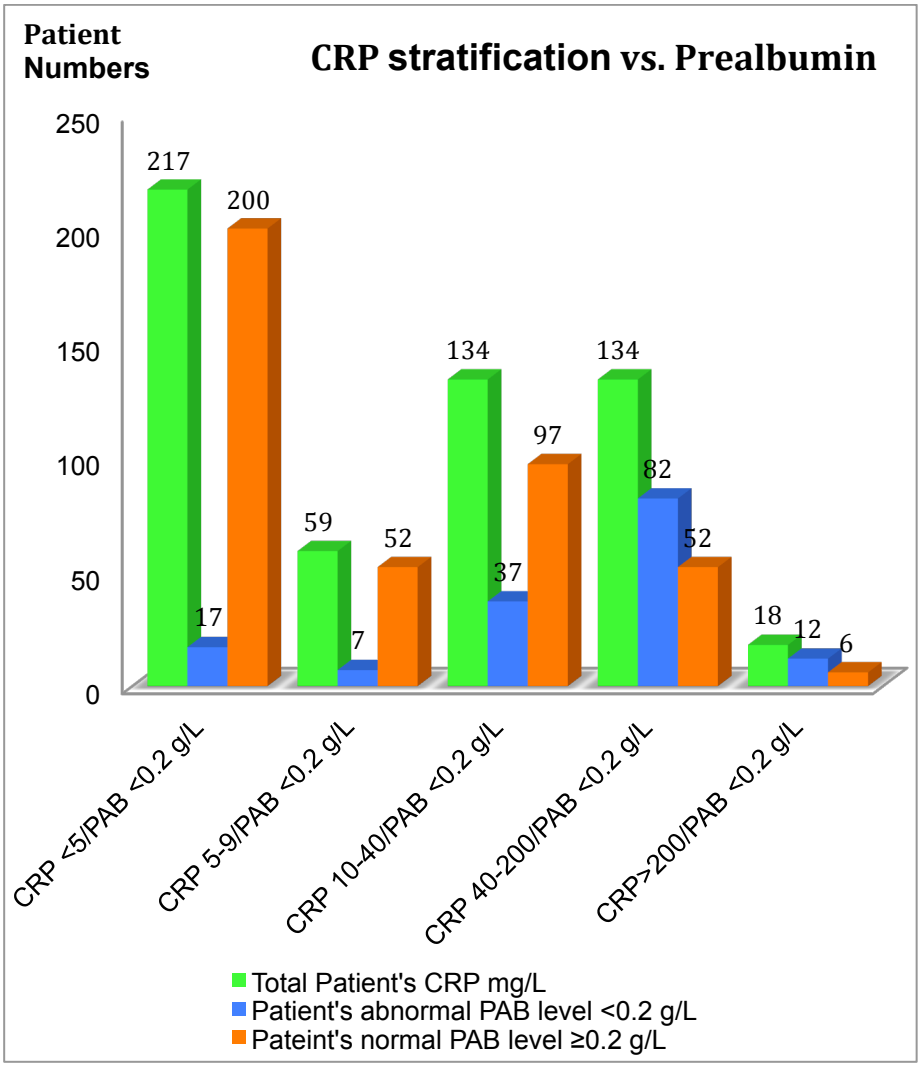

Figure 4.6. CRP stratification vs. Prealbumin

4.6. SECONDARY ENDPOINT-REFERRAL TO A DIETITIAN

In phase I the number of referrals made to a dietitian that were selected for inclusion into the study were as follows: ward 3: Acute Medical (n=10), Ward 4: Acute Surgical (n=28), Ward 7: Acute Orthopaedic (n=3), Ward 8: Acute Surgical (n=13) and Ward 10: Acute Medical (n=22). The two wards showing the highest number of referrals during this period were acute surgical ward 4 at 37% (28/76) and acute medical ward 10 at 29% (22/76). A total of 970 patients were admitted during phase I with 76 patients referred to a dietitian during the study period. During phase II, as discussed in the prealbumin results section, 776 patients were admitted with 564 tested for risk of malnutrition using prealbumin as the laboratory marker. Concurrently referrals were also received by either nurse and/or medical staff was via a different pathway e.g. MUST. A total of 43 patients were referred, ward 4 had the highest referrals at 20 with 33 patients having abnormal prealbumin. Whereas ward 7 had 1 referral although 19 out of 124 patients had abnormally low prealbumin. Significantly there was no increase in the rate of referrals from the obligatory testing of prealbumin despite 155 abnormal prealbumin results.

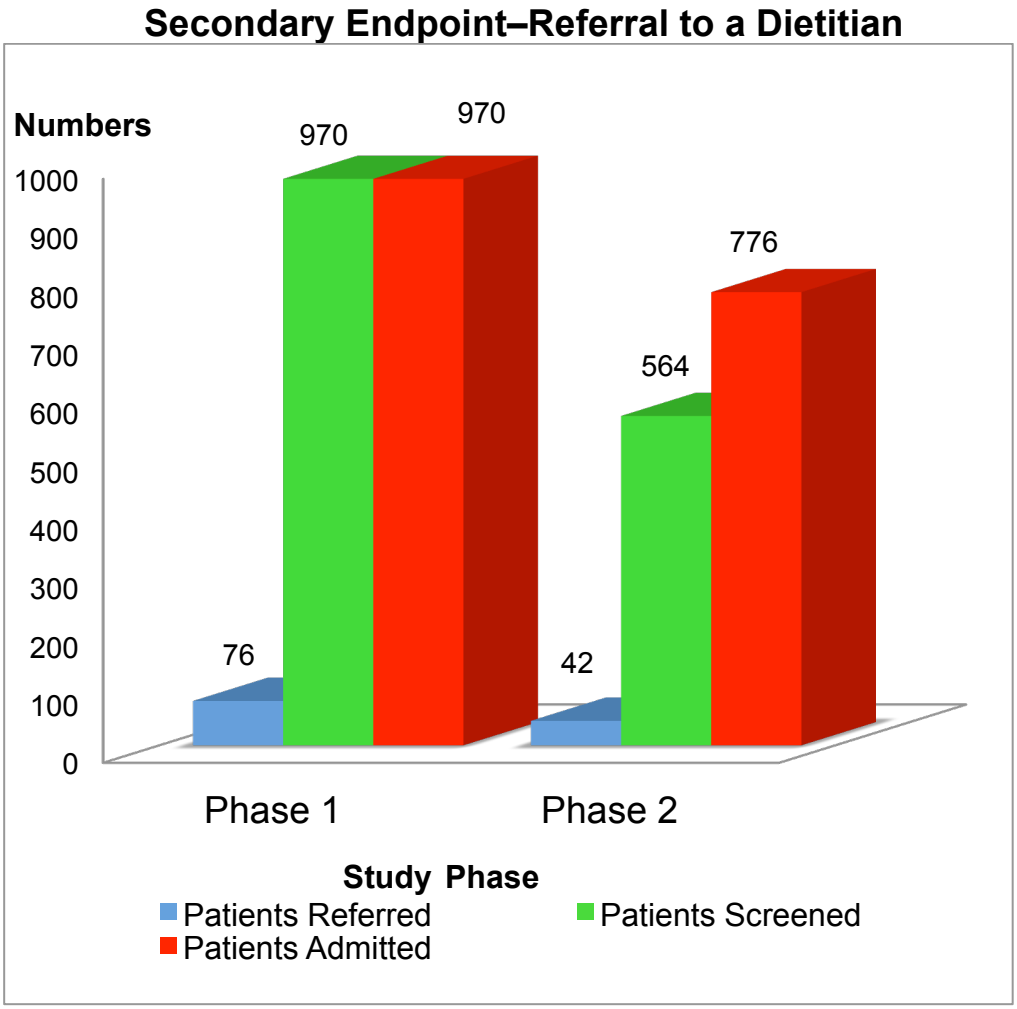

Figure 4.7. Secondary Endpoint–Referral to a Dietitian

4.7. SUMMARY

In the research phase of this study, 564 patients were screened for risk of malnutrition using the laboratory test: prealbumin. A total of 155 patient's results were noted as being abnormal. The intended research protocol was for patients whose laboratory results indicated abnormal levels of <0.2 g/L, a referral form would be generated and faxed to the appropriate ward dietitian in the Nutrition Services Department. Of the 155 patient confirmed with prealbumin results <0.2 g/L, it was found that no referrals were generated by the prealbumin alert placed on Concerto™. In phase II, routine clinical screening occurred concurrently to the prealbumin testing segment of this research and referral protocols remained unchanged. Of the total 776 patients admitted to the five

study wards, 43 patients were recorded by the dietitian as having received an assessment and referral. Of the 155 patients with abnormal results, thirteen patients were referred through alternative means of either a MUST generated referral due to a score of ≥2 or by a medical staff member. Results indicated the remaining 142 patients were not evaluated for malnutrition risk and at the time of collating the referrals had not been visited nor referred to a ward dietitian on admission.

5.0. DISCUSSION

5.1. INTRODUCTION

This research study has given an account of the disease-related malnutrition risk at an acute care hospital, NSH in Auckland. Disease-related malnutrition continues to prevail despite the vast array of international literature alluding to the commonality and the poor clinical outcome associated with disease-related malnutrition present in the hospital setting.

This research study set out to determine whether the introduction of prealbumin, as an obligatory test would identify more patients at risk of malnutrition. With the second aim to investigate whether patients that presented with a prealbumin serum level <0.2 g/L would instigate more patient referrals to a RD for a more comprehensive nutrition assessment. The results of this study found 27% (155/564) of patients were at risk of malnutrition which aligned with national and international prevalence statistics of between 20-60% disease-related malnutrition risk.

5.2. PARTICIPANT DEMOGRAPHICS

The sample of 43 participants that were referred to in this study had an average age of 66 years with a gender ratio of 44% females. When compared to the literature our study's participant demographics sat midway between Devoto's et al. (2006) study's average age of 75 years and 63% females and Robinson et al. (2003) average age of 56 years and 41% females.

5.3. PREALBUMIN SCREENING

The aim of this study was to determine whether more acutely hospitalised patients could be identified at risk of malnutrition due to the introduction of obligatory laboratory test of prealbumin. In phase II, 564 patients were selected from five acute specialty wards. The results over the five speciality wards for abnormal prealbumin levels were reasonably well spread; the largest disparity

of patient numbers was between acute surgical ward 8 (*n*=39) and acute orthopaedic ward 7 (*n*=19). Similar malnutrition risk has been shown in other studies such as Russell and Elia (2008) who found that the incidences of malnutrition per department was: medical, surgical and orthopaedic consecutively. In line with the literature more women than men were found at risk of malnutrition 83 (54%) to 72 (46%) (Elia et al., 2008). In comparison with current literature the results of this study were slightly lower than expected. According to Russell et al. (2003) the standard nutrition screening indicated 33% of the study population was malnourished, whilst 51% of patients were considered malnourished using the prealbumin screening. The nutrition audit performed at NSH in 2009 found 31.5% at risk of malnutrition which was similar to our results of 27% (Yovich, 2012). The Australasian Nutrition Care day survey in 2010 found 30% malnourished and 42% were at risk of malnutrition (Agarwal et al., 2012). However, when comparing results of malnutrition risk of 9936 patients in the Nutrition screening survey in the UK in 2007 (BAPEN), 28% were at risk of malnutrition (Elia et al., 2008). The results of this research were also comparable with the research study by Potter et al. (1999) which examined the effectiveness of prealbumin as a routine diagnostic test for protein calorie malnutrition (PCM) in emergency admissions, they found 24% of the 147 patients had mild PCM and 12% severe PCM as little as 25% of patients with PCM received dietetic consultation ($p < 0.01$). Overall our results indicated that 27% (155/564) patients were at risk of malnutrition with abnormal prealbumin results of <0.2 g/L which was in-line with both national and international results of disease-related malnutrition risk.

The second aim of this study was to determine whether more patients were referred for RD nutrition assessment after the introduction of prealbumin screening. During Phase I (control period) the standard nutrition screen/assessment protocol generated referrals for 7.8% (76/970) of patients.

These patients were referred to a dietitian for a full dietetic assessment and interventions were put into place where the dietitian deemed appropriate. Despite prealbumin laboratory results placed on Concerto^{TM} with a clear specific alert advising when prealbumin at a level of <0.2 g/L a referral to a ward dietitian is appropriate, no referrals were generated from the abnormal prealbumin laboratory results. All referrals made were through a divergent pathway of either a nurse completed referral or a MUST score of ≥2. This also meant the actual nutritional status of the 155 (27%) patients deemed at risk of malnutrition by abnormal prealbumin results were never formally evaluated using the standard nutrition assessment protocol by a RD, which included a PG-SGA.

When carefully scrutinising the type and number of referrals initiated during this study period, compared to the abnormal prealbumin results as few as 43 patients (7.6%) were referred by medical staff to a dietitian for a comprehensive dietetic assessment through a divergent pathway. Additionally the results unveiled that 70% (30/40) of patients referred for dietetic input, had normal prealbumin ≥ 0.2 g/L whereas 30% (13/43) of the total referrals had abnormal prealbumin levels <0.2 g/L. Significantly 8% (13/155) of these patients with an abnormal prealbumin level were formally referred for dietetic input and comprehensive assessment, although it was noted that this was through an alternative pathway to the prealbumin laboratory tests and the referral noted no acknowledgment of prealbumin results. This left 92% (142/155) of patients with abnormal prealbumin results without a referral to a dietitian at admission. However, it is noteworthy to mention that this study only examined referrals on admission and not past the predefined study period, which meant potentially these patients may have been referred and seen by a dietitian at a later date. Potentially there are several consequences as a result of clinicians not generating referrals for patients whose prealbumin results <0.2 g/L. These may

include a) not being assessed by a dietitian b) not having their prealbumin results substantiated as to the degree of malnutrition risk e.g. mild, moderate or severe using the validated PG-SGA tool and lastly c) if these patients were in fact at risk of malnutrition or malnourished only 30% of these patients were seen due to an alternative referral pathway. Furthermore, of the 70% of patients with abnormal prealbumin results that were not assessed by a dietitian, those patients deemed at risk of malnutrition were potentially suffering from disease-related malnutrition. As the literature eloquently states that not only is there the potential for these patients to have increased LOS and complication rates up to 20 times higher than well-nourished patients the risk of morbidity and mortality may be increased exponentially (Robinson et al., 2003). The findings of this study regarding referral and acknowledgement of malnutrition risk are consistent with findings in the literature. Lazarus and Hamlyn (2005) in an Australian study found poor documentation of malnutrition in 137 (42%) hospitalised patients, with only 21 (15%) of these patients referred to a dietitian. Adams et al. (2008) found that although there was good knowledge of the medical risk factors of malnutrition there was poor knowledge of the major malnutrition risk factors with 30% of medical professionals unaware that recent unintended weight loss was a marker of malnutrition risk as is decreased appetite. Additionally Gout et al. (2009) who examined the adequacy of dietetic referrals for treatment of malnutrition in the acute care wards of a metropolitan tertiary teaching hospital, found 23% (63/275) prevalence of malnutrition. Of the 63 patients identified as malnourished as few as 15% were correctly diagnosed and documented, 45% of the malnourished were seen by a dietitian and of those correctly identified 29% of cases were documented in the notes. Gout's et al. (2009) findings were consistent with numerous studies discussed in the paper and that conclude despite the evidence eluding to the prevalence rates of malnutrition, little had altered to make a significant difference Their study

provided a unique insight into the inadequacy of dietetic referral and medical staff clinical documentation of malnutrition.

The plausible causality of a nil increase in referral rates directly attributed to abnormal prealbumin analyses could be potentially due to a clinical knowledge gap regarding malnutrition risk and the associated outcome of untreated malnutrition. Furthermore, although a clear alert appeared on Concerto™ whenever patients' laboratory results were abnormal, a proposed explanation could be the uncertainty of responsibility regarding the referral pathway initiation for these patients in each ward as the dietitian did not receive the alert directly. As the protocol for dietitians at NSH to attend to the speciality nutritional needs of a patient is through a referral it is proposed that a direct alert should automatically initiate a referral to a dietitian, more patients at risk of malnutrition could be assessed.

It is presumed that should a more comprehensive dietetic assessment ensued, a significant number of patients potentially confirmed at either at risk of malnutrition, malnourished and/or requiring further nutrition assessment, nutrition intervention and appropriate monitoring could have been effectively instigated. The literature does agree with the findings of this research that highlight the need for education in the field of disease-related malnutrition risk identification and the results and fall out of associated comorbidities of undiagnosed malnutrition, should malnutrition continue to elude detection. McWhirter and Pennington (1994) concluded from their research study, the need to broaden the educated requirement of clinical nutrition and disease-related malnutrition risk, Nightingale and Reeves (1999) agree claiming poor knowledge regarding the assessment and management of malnutrition amongst hospital doctors, medical students, nurses and pharmacists.

A variable not tested for that has the propensity for alteration in the malnutrition rate with the additional non-nutritional variable, i.e. seasonal variation. The

slightly lower than expected rate of 27% of patients at risk of malnutrition found in this study, rather than the higher rates found in some of the literature, could have been due to the seasonal variation in this study was performed in summer. It could be contended that had this research been conducted in autumn and/or winter the rates of malnutrition could have been significantly higher. Previous research, including a report by BAPEN confirms that the prevalence of malnutrition in the community with seasonal variation in malnutrition is a real concern with more patients suffering in the winter months (BAPEN, 2013c; Elia et al., 2005; O'Flynn et al., 2005). Robinson et al. (2003) does not mention the time of year their study was conducted and hence no comparisons regarding seasonal variation could be made. What's more there seems to be sparse research that has examined a universally agreed stratification and reference range for prealbumin in the assessment of malnutrition risk that would potentially close the gap of malnutrition prevalence variation. Within the literature few studies have stratified prealbumin levels that identify mild, moderate or severe risk of malnutrition. Table 5.1 demonstrates an example of a Prealbumin risk stratification initially described by Bernstein et al.(1995) & then adapted by Beck and Rosenthal (2002).

Table 5.1. Prealbumin Risk Stratification

Prealbumin level	
<5.0 mg/dL (<50 mg/L)	Poor prognosis
5.0 to 10.9 mg/dL (50 to 109 mg/L)	Significant risk: aggressive nutritional support indicated
11.0 to 15.0 mg/dL (110 to 150 mg/L)	Increased risk: monitoring status biweekly
15.0 to 35.0 mg/dL (150 to 350 mg/L)	Normal

Currently because NZ does not stratify prealbumin levels the normal reference range is between 0.2-0.3 g/L. Authors such as Potter et al, (1999) claim that the use of stratified prealbumin levels is an important indicator of mild PCM with prealbumin levels of <160 mg/L and severe PCM <107 mg/L. Multiple studies conclude that prealbumin is a useful additional tool in identifying malnutrition risk rather than a single diagnostic tool of malnutrition (Mears, 1996; Potter & Luxton, 1999b; Saka et al., 2011). As previously acknowledged, although prealbumin is affected by non-nutritional factors these are minimal when compared to alternative biochemical markers of malnutrition risk such as albumin, transferrin and RBP. Prealbumin is the most sensitive to dietary changes in nutrition/protein intake and has the potential for measurement in all individuals who traditionally may not have been measured e.g. obese/overweight in-ambulatory or unconscious individuals. Well acknowledged in the literature is that the body composition of an obese person is often presumed as being well nourished with PEM being unlikely (Jensen et al., 2010; Spiekerman, 1995). However, such explanations tend to overlook the fact that obese individuals are just as likely to suffer acute and chronic disease and injury, to have inadequate dietary or nutritional intake for five days or greater and to have had significant weight loss and still be overweight on visual assessment. Prealbumin is a sensitive indicator for PEM and could quickly and effectively determine any biochemical changes within the body of all patients without prejudice and could potentially acknowledge more individuals at risk of further clinical complications imposed both functionally and physiologically due to disease-related malnutrition. A case in point is demonstrated by one result in this study, where it was found that a bariatric patient's prealbumin results were abnormal <0.2 g/L. A laboratory marker of malnutrition risk that is sensitive to dietary changes, expeditious, simple to perform and minimally altered by non-

nutrition factors that could be positively integrated into the overall assessment programme, could minimise the occurrence of such anomalies.

Further rationale for the introduction of prealbumin to assess malnutrition risks into the assessment plan and to improve patient care, is economically based. Mears (1996) claim that found at the time of this research study that a cost saving estimated at $500 per day with LOS reduced by 2 days for moderately malnourished risk and 12 days for severe risk with the introduction of nutrition supplementation of malnourished patients. As a direct result of Mears (1996) research study findings, the hospital medical committee implemented prealbumin laboratory testing into the multidisciplinary nutrition care programme which saved the hospital of $600,000 per year. This programme included a prealbumin training module for all staff using prealbumin as a tool in defining nutritional status. The nutritional treatment plan met the 1995 Joint Commission on Accreditation of Healthcare Organizations' Nutrition Care Standards (Mears, 1996).

5.4. PREALBUMIN CORRELATION WITH CRP

Previous studies have reported the relationship between malnutrition and inflammation, which was common in the patients of this research study (Jensen et al., 2010; Xie et al., 2011). The negative acute-phase protein prealbumin is inversely associated with inflammation, as both prealbumin and CRP has the potential to alter in value during acute disease, injury or illness as both are synthesised by the liver. The reprioritisation of protein synthesis towards CRP at times of acute and chronic inflammation has opened up the question of whether decreased prealbumin synthesis in the liver is in-fact an accurate representation of malnutrition risk. Although the literature eloquently states that in times of acute inflammation, muscle proteolysis occurs in this reprioritisation of protein synthesis and the relationship between diminished appetite when an increase in pro-inflammatory cytokines initiates increased synthesis of CRP (Jensen et al.,

2010). Several other studies have acknowledged the importance of the biochemical changes proposed by this relationship between CRP and prealbumin (Jensen et al., 2010; Myron Johnson et al., 2007). The statistical correlation in this study agreed with the literature, concluding an inverse correlation between CRP and prealbumin. As the results of this study indicated there was individual variation amongst patients with abnormal prealbumin, showing that not all patients with abnormal prealbumin had significantly high CRP. The variation of individual CRP/prealbumin inverse correlation is illustrated in table 4.6 of the results section. Results of CRP showed that 134 patients had CRP ranging from 10-200 (deemed acute inflammation) however, of those 134 patients, 82 of them had abnormal prealbumin <0.2 g/L. These results are substantiated by Spiekerman (1995) who agrees with the conclusion that inflammation and hence CRP increases significantly in times of acute sepsis, trauma, burns and inflammation with onset increasing as much as 1000 times after tissue injury. Consistent with previous studies Spiekerman (1995) and Jensen et al. (2010) concur that the complex pathophysiology of disease-related malnutrition can include varying degrees of inflammation and a merger between under and over nutrition directly attributed to acute and chronic disease effect, infection and/or trauma. Furthermore inflammation is recurrently associated with acute and chronic disease states and trauma. Consequently many of these patients are often too unwell to maintain sufficient dietary intake to prevent catabolism of skeletal muscle, resulting in a decrease of lean body mass; a clinical sign of malnutrition (Jensen et al., 2010). Finally as the results of this study have acknowledged, the referral for dietetic intervention was poor for all patients having abnormal prealbumin (<0.2 g/L). As a direct result of poor referral for dietetic intervention the malnutrition status of patients was never formally quantified in terms of malnutrition risk with or without raised CRP levels.

5.5. DISEASE-RELATED MALNUTRITION SCREENING & ASSESSMENT

In an ideal world, all new patients should be evaluated at the time of hospital admission. International nutrition care guidelines advocate for nutrition assessment to enable diagnosis of disease-related malnutrition with a strong recommendation to undertake screening at the time of admission (Agarwal et al., 2012; Kondrup, Allison, et al., 2003). Robinson et al. (2003) eloquently states that nutrition specialists namely dietitians, are the most appropriately trained clinical professional to elucidate a detailed nutritional assessment including: past and present medical histories, interpret laboratory biochemical markers, undertake anthropometric measurement to accurately quantify malnutrition risk of all individual patients.

This research study was conducted at NSH in Auckland, NZ. Introduced in 2006, the MUST was identified & used as the standardised protocol for malnutrition screening. The protocol states the MUST ideally should be performed at the time of admission and is an integral component of the Admission to Discharge Planner. A dietetic assessment is based on a formalised referral structure through the MUST pathway or clinical referral. The MUST was developed for use of nutritional screening in all health care settings specifically for adult patient groups (Stratton et al., 2004). Stratton et al. (2004) concluded in their study that the MUST has a fair to good to excellent concurrent validity.

Results from this study revealed a total of 43 patients in phase II were referred through the standard nutrition protocol but as few as 22 patients had MUST screen recorded in their patient's clinical records. As discussed previously in the introduction to this paper these findings were consistent with the 2009 Waitemata hospital audit of the MUST, which found a total of 31.5% of patients were at risk of malnutrition. Additionally the results of this research collaborated

the findings of the ANCDS in 2010 showing 64% of patients were not nutrition screened at the point of admission and 95% were not rescreened during hospital stay (Agarwal et al., 2012). The lack of screening was also evident in the methodology where 78 records were requisitioned to either confirm whether patients already identified as part of the study had a completed MUST screen in their clinical file. Other patient files were also examined to identify whether there was sufficient referral information and dietetic input to be included in the referral data. A review of the referral documentation found it incomplete which resulted with the patients not being included in this study. As a consequence the nutritional status of patients with abnormal prealbumin levels was unable to be formally evaluated due to no referrals being initiated.

In summary, it is well known that hospitals' and medical professionals have to deal with many competing priorities. Initial disease-related malnutrition risk screening protocols are often inhibited by the lack of risk management identifying the implications of malnutrition risk, adequate time availability, use of validated screening tools, available resources and patient compliance, not underestimating the challenges of patient access and the degree of cultural diversity and language barriers in NZ hospitals. It order to have buy-in from medical staff to perform nutrition-screening protocols at admission, communication between allied staff is crucial with adequate training and appropriately allocated timeframe for screening. Regardless of the reasoning behind the inadequacy of disease-related malnutrition screening at the time of admission or in many cases on admission to the ward, what is clear is that a more effective protocol is required to combat the prevalence of disease-related malnutrition in NZ hospitals.

5.6. COSTS AND LENGTH OF STAY

A strong theme emerged when conducting the literature review that malnutrition is costly in multiple ways whether with higher risk of complication, poor outcomes, increased morbidity and mortality or financially on individual hospitals, nationally or economically on governmental healthcare budgets. A positive correlation has been found with an increased length of hospital stay and patients admitted with malnutrition, and this has been cited in multiple literature ranging with the average LOS being 4-12 days longer in malnourished compared to well-nourished patients (Mears, 2007; Saka et al., 2011). Although this study did not directly study LOS in malnourished compared with well-nourished patients, evidence in the literature suggests a substantial increase in the average daily expenditure for malnourished compared to well-nourished patients (Correia & Waitzberg, 2003; Russell, 2007). Correia and Waitzberg (2003) performed an analysis of costs to demonstrate the mean daily expense of malnourished patients US $228.00/patient compared to well nourished patients US $138.00/patient.

Currently the average length of stay at NSH is 4.38 days with 42,318 discharges between 2012-2013 (Ayar, 2013) and the cost of prealbumin screening set a $4.87 (Pountney, 2013). When consideration is given to the vast array of speciality care each individual admission requires i.e. varying levels of medical expertise, pharmaceutical input, medical/disease status of the patient, food and all other essential care items calculated for one day's stay (Thomson, 2013), the cost of $4.87 for prealbumin screening seems negligible.

It is unclear whether it is the level of complacency; lack of awareness, appropriate nutrition education or knowledge gaps regarding the major risk factors associated with malnutrition, that influences disease-related malnutrition rates remaining high. However, improving patient outcome and reducing the escalating healthcare costs could all be achieved through early identification if

improved assessment, diagnosis and nutrition intervention and treatment protocols were in place.

5.7. SUMMARY

Disease-related malnutrition continues to elude detection which causes substantial follow on effects in the complex interplay implicated in the meagre malnutrition screening protocol currently undertaken in hospitals worldwide. Although there are inhibitory non-nutritional factors that influence the use of laboratory markers, prealbumin has been proposed as the preferred laboratory marker of malnutrition risk assessment, Prealbumin screening has the least limitations, can be used in a variety of settings, does not discriminate, is quick, simple and relatively inexpensive.

The known complex reciprocation between inflammation and serum prealbumin levels is not completely understood, but what is known is that there in an intrinsic link in clinical outcome with the improvement of the patient's metabolic status, by improving inflammation and nutritional status. Hospital care is expensive and with the right level of disease-related malnutrition screening, assessment, nutrition support and intervention the result of improved outcome and quality of care which can equate to decreased LOS and reduced hospital costs is plausible (Mears, 1996, 1999). Despite the fact there is no gold standard measurement for nutrition assessment including the use of prealbumin, studies have shown prealbumin to be a useful malnutrition risk screening parameter in conjunction with anthropometric assessment (Robinson et al., 2003; Devoto et al., 2006).

6.0. CONCLUSION

6.1. STRENGTHS AND LIMITATIONS OF THIS RESEARCH

6.1.1. Strengths

This study was the first NZ study to evaluate whether universal prealbumin screening increases the number of patients identified and referred to a dietitian for comprehensive disease-related malnutrition assessment. The study enrolled a wide variety of acute medical, surgical and orthopaedic speciality wards. Results from this study were compared to current evidence based practice guidelines for the management of patients at nutritional risk as well as malnutrition screening studies performed nationally and internationally. The data provided strong evidence that is accessible for use by dietetic managers across NZ that could potentially provide useful information on malnutrition risk screening protocols at the time of admission. Furthermore it is hoped that through the results of this study, the evidence has shown that an increased number of patients were identified at risk of malnutrition and that prealbumin is a useful tool in disease-related malnutrition screening. In contrast to prealbumin screening many other malnutrition screening tools have limitations' in the assessment of in-ambulatory, obese/overweight and unconscious or patients that are either too unwell or cognitive to verbalise themselves adequately to answer any questions pertaining to malnutrition risk screening. Therefore it is proposed that NZ hospitals may consider the implementation of prealbumin in conjunction with nutrition screening tools, currently in use to provide increase care in the risk management of disease-related malnutrition and hence improve patient centred care.

While the results are obviously relevant for the development and/or improvement of malnutrition screening protocols, it is important to acknowledge several limitations that could be addressed in future studies.

6.1.2. Limitations

A number of important limitations manifested during this research that require due consideration. The current research was not specifically designed to evaluate the level of knowledge medical professionals have relating to disease-related malnutrition risk and the variables associated with malnutrition risk assessment. In particular unintentional weight loss or the range of malnutrition related comorbidities that can accrue with untreated malnutrition are not included. Another limitation of this study is the lack education provided at the beginning regarding the laboratory marker prealbumin The reason for this was to test the effect of a single intervention – the use of an 'obligatory' blood test: prealbumin.

Therefore as a result the knowledge barriers pertaining to the identification and significance of malnutrition risk was perhaps the most serious disadvantage in the methodology as it was revealed through an appreciable lack of patient referrals via abnormal prealbumin levels. Additionally as the abnormal prealbumin levels of 155 patients did not result in appropriate clinician instigated referrals to a dietitian, these patients were unable to be formally assessed for disease-related malnutrition. Therefore the secondary major limitation of this study was that the malnutrition risk was never quantified to expose accurate levels of malnutrition e.g. mild, moderate or severely malnourished at NSH over this study period.

Currently NZ does not stratify prealbumin reference ranges and therefore another issue of malnutrition risk stratification was additionally not addressed. Potentially this is an area hospital laboratories could define more clearly as: not at risk, mildly, moderately or malnourished to ensure the effective detection of malnutrition. Finally the MUST screening of all admissions in phase I and II was not recorded and therefore comparisons of the percentage of those patients that were accurately screened for malnutrition risk using the MUST could not be

documented. Only the patients that were referred were recorded as having or not having a MUST screen in their clinical records. Despite these limitation, the results of 27% (155/565) patients deemed at risk of malnutrition was a good result.

6.2. RECOMMENDATIONS FOR FUTURE RESEARCH

This study set out to examine the prevalence of hospital malnutrition on admission, and to attempt to validate the use of a simple laboratory test measure to determine those at risk of malnutrition. This research unveiled several areas that could potentially be reviewed to increase the use of prealbumin screening and identification of malnutrition risk in NZ.

Future research could potentially follow the trends of prealbumin as 25 out of the 564 patients prealbumin tested had marginal results of 0.2 g/L. Levels which are marginal between risk and not at risk can tip the balance in succeeding days after admission. Some reasons for tipping the balance include extended periods of nil by mouth (NBM) or alternative clinical inhibitors e.g. dysphagia. Further research could be performed to establish whether a stratification system of prealbumin levels could enhance the diagnosis of severity or degree of malnutrition risk within NZ acute care hospitals. Whether the severity is borderline, mild, moderate or as in examples of some results in this study, potentially severe malnutrition with several patients prealbumin levels below 0.05 g/L.

Further NZ based research might also explore factors such as LOS in those patients with prealbumin levels <0.2 g/L compared to patients with prealbumin >0.2 g/L. with either similar medical conditions or within the same wards.

Clinical knowledge pertaining to the identification of disease-related malnutrition and related comorbidities in the acute care setting was a stand out area that could be examined. Examining the effect of nutrition and disease-related

malnutrition risk education on the improvement of early identification of disease-related malnutrition in the acute care setting could be explored in more depth.

Another recommendation would be to investigate if prealbumin testing on admission and at two-day intervals would increase the number of patients being referred to a RD, receiving treatment and decreasing LOS and readmission rates. Another area of worth examining is whether universal malnutrition screening using prealbumin might result in improved rates of malnutrition risk detection if abnormal levels automatically triggered dietitian assessment. Finally some research exploring if the prevalence rates of disease-related malnutrition and related comorbidities decline, would the enormous expense of disease-related malnutrition imposes on societal healthcare, complication rates, morbidity, LOS and readmission rates also decline.

6.3. CONCLUSION

This study was the first two-phase observational cohort study performed in NZ, that increased scientific based knowledge pertaining to the introduction of a laboratory test to screen for adult disease-related malnutrition risk in a large district health board hospital.

The hypothesis of this study was H_1: It is hypothesised that mandatory laboratory testing of prealbumin will increase the detection of disease-related malnutrition in acutely hospitalised patients at North Shore Hospital Auckland.

The addition of malnutrition screening using prealbumin did increase the number of patients identified at risk of malnutrition 27% (155/564). However the introduction of the obligatory test of prealbumin during this study period did not improve recognition by clinicians and hence increase the number of referrals to a dietitian.

There is a substantial amount of evidence that recognises the appreciable burden and adverse clinical outcomes of disease-related malnutrition in hospitals internationally. Multiple studies have cited 1 in 3 hospitalised patients

are malnourished on admission and if left untreated continue to deteriorate nutritionally, which impacts significantly on recovery and outcome (Agarwal et al., 2012; Tappenden et al., 2013). Although the data on hospital malnutrition varies across studies, it is acknowledged that more than 30 years ago hospital malnutrition was unveiled as highly prevalent (Butterworth, 2005). Conjointly the new millennium of disease-related malnutrition continues to elude detection and the occurrence rates remain unacceptably high. With the continued prevalence and poor detection and suboptimal referral rates, disease-related malnutrition places an increasing burden on societal healthcare facilities. Although medical advancement has profoundly improved the clinical outcome for many patients, malnutrition has the potential to simply be corrected by nutrition intervention is still often overlooked. There is an enormous potential to decrease health expenditure and improve clinical outcome, through the prevention and treatment of hospital malnutrition for the multiple thousands of patients that pass through the hospital system annually.

The rationale behind increasing accuracy and early detection of disease-related malnutrition screening with assessment performed post admission and follow up with a referral and comprehensive dietetic assessment, is without question. Nutrition professionals base the assessment on multiple variables including: disease type and progression, patient's age and physical assessment (ADIME) of patients referred to their care. There is considerable scope that exists to decrease the knowledge gap of malnutrition that incorporates risk factors and comorbidities of malnutrition amongst all allied health professionals.

Furthermore the interdisciplinary team approach promoted within the district health boards in Auckland, advocates all allied health staff working as a team for all aspects of patient centred care. This development of innovative strategies using the interdisciplinary team approach to hospital malnutrition risk include: the facilitation of nurses to screening and encourage patient compliance,

dietitians to complete detailed nutrition assessment with the incorporation of prealbumin, diagnosis and develop evidence-based intervention(s) and monitoring/evaluation and pharmacists to manage patient drug-nutrient interactions with all completing clear precise nutrition documentation in all clinical records (Agarwal et al., 2012; Tappenden et al., 2013). To strengthen the argument the Dietitians Association of Australia Best Practice Guidelines advocates for the implementation of routine nutrition screening for all patients in the acute care hospital setting (Evidence based practice guidelines for the nutritional management of malnutrition in adult patients across the continuum of care, 2009).

Research has strived to identify a single biological marker to assess malnutrition risk and although anthropometric markers including current weight compared to unintended weight loss are closely associated with malnutrition risk, the use of anthropometric measurements can often be hindered by nutrition, poor screening, patient compliance, cognition and conscious states. These anthropometric markers can be compounded by the interdisciplinary team members misunderstanding the importance of malnutrition.

Prealbumin has been proposed as a laboratory marker that can identify malnutrition risk even when clinical signs are not immediately evident. Used in combination with anthropometric measurement prealbumin testing has the potential of early identification of patients at risk of malnutrition, that elude detection. Instead of reflecting overall nutritional status, a low serum prealbumin is a valuable laboratory marker that is simple to use, expeditious, readily available and increases the early identification of malnutrition risk that may pre-empt a nutritional assessment.

Multiple studies recommend the endorsement that nutrition screening on admission is a critical component of patient centred care in an attempt to reduce the number of nutrition-related complications and improving patient outcome

(Correia & Waitzberg, 2003). Ultimately accurate nutrition screening is essential to improve the detection of disease-related malnutrition and malnourished hospitalised patients through refining nutrition-screening practices. Numerous research studies have conveyed findings that nutrition intervention and sufficient feeding can improve patient outcome, lessen complications, and decrease LOS and readmission rates. In combination these factors can contribute significantly to financial savings within hospitals and significantly improved patient outcome.

6.4. RECOMMENDATION

A proposal is recommended that universal malnutrition screening using prealbumin may potentially result in improved rates of malnutrition risk detection if abnormal levels were to automatically trigger a direct dietitian assessment.

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8.0 APPENDICES

APPENDIX A: Evidence based guideline for nutrition screening

Criteria	Evidence based statement	Grade (NHMRC[11])	Grade (NCCAC)	Grade (ADA EAL)
Nutrition screening	i Screening for malnutrition and the risk for malnutrition should be carried out by health care professionals with appropriate skills and training.	–	D(GPP)	–
	ii All hospital inpatients on admission should be screening. Screening should be repeated weekly for inpatients.	–	D (GPP)	
	iii Screening should assess BMI, percentage unintentional weight loss and should also consider the time over which nutrient intake has been unintentionally reduced and/or the likelihood of future impaired nutrient intake.	–	D (GPP)	
	iv Implementation of a nutrition risk screening program:	–	–	
	a Improves the identification of individuals at risk of malnutrition;	B	–	
	b Facilitates timely and appropriate referral for nutrition	B	–	
Nutrition screening tools	Valid nutrition risk screening tools include:		–	
	i MST	B		II
	ii MUST	B	–	II
	iii NRS-2002	B		I
Nutrition Interventions	i Dietary counselling by a dietitian may improve outcomes such as:		–	–
	a Weight status and physical function	C	–	–
	b Weight status and body composition	C		
	i Oral Nutritional Supplements may improve outcomes such as:		–	–
	a Weight status, body composition, complications, pressure ulcers, life expectancy (evidence of an effect)	A	–	–
	b Energy and protein intake, global nutritional status, mood	A		
	iii Individually prescribed nutritional support (including high energy diets ± ONS) may improve outcomes including:		–	–
	a Energy intake and wound healing	C	–	–
	b Weight status and nutritional biochemistry	C		
	iv Feeding assistance may improve outcomes including energy intake, body composition, life expectancy and use of antibiotics	C	–	–
	v "Protected" Meal times	No evidence located	–	–

NHMRC: National Health and Medical Research Council; NCCAC: National Collaborating Centre for Acute Care; ADA EAL: American Dietetic Association Evidence Analysis Library®, BMI: Body Mass Index; Aus: Australia; MST: Malnutrition Screening Tool; MUST: Malnutrition Universal Screening Tool; NRS-2002: Nutrition Risk Screening-2002; ONS: Oral Nutritional Supplements.
NHMRC: Grade A: Excellent level of evidence; Grade B: Good level of evidence; C: Satisfactory level of evidence.
NCCAC: Grade D (GPP): A good practice point (GPP) is a recommendation for best practice based on the experience of the Guideline Development Group.
ADA EAL: Grade I: Good strength of the evidence; Grade II: Fair strength of the evidence.

APPENDIX B: MUST APP

BAPEN 'Malnutrition Universal Screening Tool' ('MUST') 'MUST' App for the iPhone.

Scan this QR code with your iPhone camera to download the 'MUST' app

With the growth of the use of mobile technology and Apps in particular in healthcare, BAPEN has converted its popular and validated screening tool for identifying malnutrition into an electronic version that can be used across all health and care settings by front line staff.

APPENDIX C: Massey University Peer Review letter

MASSEY UNIVERSITY
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25 January 2013

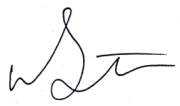
To whom it may concern

Peer review of research proposal: Improving early assessment of malnutrition in hospitalised patients; prealbumin *versus* routine clinical assessment

This letter is to confirm that the above research proposal has been presented to and peer-reviewed by a group of lecturers and scientists in Human Nutrition and Dietetics from Massey University as well as the Dietetic Professional leader from Waitemata District Health Board at a symposium held on 30 November 2012 at Massey University, Albany campus.

The proposal was found to be scientifically sound and some minor recommendations were made.

Kind regards



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APPENDIX D: HDEC Approval letter 2013

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hdecs@moh.govt.nz

15 February 2013

Mrs Tracey Eccles
136 Blackbridge Road
RD4 Albany
0794

Dear Mrs Eccles

Re:	**Ethics ref:**	**13/NTA/16**
	Study title:	Improving Early Assessment of Malnutrition in Hospitalised Patients; laboratory test for risk of malnutrition versus Routine Clinical Assessment

I am pleased to advise that this application has been *approved* by the Northern A Health and Disability Ethics Committee. This decision was made through the HDEC-Full Review pathway.

The main issues considered by the HDEC in giving approval were as follows.

- The researchers briefly explained the study, which aimed to look for pre-indicators of malnutrition in persons admitted to hospital.
- The Committee noted that no participant information would be provided in this study. The researchers explained that formal written consent was not obtained routinely to take blood samples, and that the tests were largely a matter of clinical judgement. The proteins that would be tested as part of this study did not have a genetic component and fell within the range of things that it was reasonable for people to expect to be tested for. The testing was an alternative to a nurse-led questionnaire on nutrition, for which written consent was not normally obtained. No additional tissue would be obtained as part of the study.
- The Committee noted that people who would not be able to give informed consent might be overrepresented in terms of malnutrition, and that including them would help power the study.
- The Committee discussed the possibility for the study to generate benefits for Māori, and suggested that there be greater engagement with Māori on the study.
- The Committee asked for clarification as to whether the recommendations in Massey University's peer review letter had been accepted.

Conditions of HDEC approval

HDEC approval for this study is subject to the following conditions being met prior to the commencement of the study in New Zealand. It is your responsibility, and that of the study's sponsor, to ensure that these conditions are met. No further review by the Northern A Health and Disability Ethics Committee is required.

Standard conditions:

APPENDIX E: NZSG Small Research Grant 2013 letter

New Zealand Society of Gastroenterology Inc

PO Box 10-601, Wellington, 6143 New Zealand lily.brown@racp.org.nz Tel: (04) 460 8127 Fax: (04) 472 6718

4 April 2013

Dr Russell Walmsley
Dep. of Gastroenterology
North Shore Hospital
Private Bag 93503
Takapuna
Auckland

By email: Russell.walmsley@waitematadhb.govt.nz

Dear Russell

NZSG Small Research Grant 2013 – Round 1

Thank you for applying for the NZSG Small Research Grant. The applications were considered by the NZSG Scientific Committee as well as an external reviewer, Stephen Gerred. I am writing to advise you that you were successful on your application for "Improving Early Nutritional Intervention in Hospitalised Patients; Laboratory Tests versus Routine Clinical Assessment". The amount awarded was $5,000.

We are pleased as a Society to be able to assist you in your research. We believe this to be an important project and look forward to hearing some results.

Some of the conditions of this grant are:

- It is expected that results from this research will be presented at the ASM of the NZSG and a one page report should also be forwarded to the Secretary of the NZSG within 12 months of receipt of the grant.
- NZSG must be acknowledged in any publications that are published as a result of the grants.
- The research must be conducted in New Zealand.
- The grant will be only be forwarded into a research account and not directly to an individual.

Please provide Lily Brown, Executive Officer (lily.brown@racp.org.nz), with an invoice for payment into the designated research account.

Yours sincerely,

Alan Fraser
President

APPENDIX F: Dietetic Inpatient Referral Data Collection Form)

NHI Number	Date Admitted	Ward	Date Referred	Referrer (MUST, Nurse, Other)	MUST Score	PREALBUMIN level	PG-SGA score	Malnutrition Y = Yes N = No	Degree of Malnutrition S = Severe M = Moderate

APPENDIX G: WDHB Dietitian Referral Form

Waitemata District Health Board
Best Care for Everyone

[PLACE PATIENT LABEL HERE]			
First Name:	_____	Gender:	_____
Surname:	_____		
Address:	_____		
Date of Birth:	_____	NHI#:	_____
Ward/Clinic:	_____	Consultant:	_____

Nutrition Services

Dietitian Referral – Inpatient (North Shore Hospital)

Please attach patient label. Please completed all sections.
Please file completed form in the patient's clinical notes, after it's been faxed.

Referrals should be faxed to Nutrition Services ext. 3940 (North Shore Hospital)

Date	
Referrer	
Malnutrition Screening (MUST) Score	
Diagnosis / active problems	
Reason for referral	

For more information see *Dietitian Referrals – Inpatients* (Quality Documentation, Clinical Practice, Waitemata DHB Intranet) or visit the dietitian's page on the intranet.

NUTRITION SERVICES ONLY

Date received	Date screened
Accepted / Declined (complete below)	
Reason ☐ There is insufficient information on the referral form. Please complete the form and re-fax. ☐ This referral is not appropriate for dietetic assessment because: ☐ Other reason Name: Designation: Ext:	

Trail Date: mmm yyyy
Review Date: mmm yyyy
(***Note:*** *Trials must be no longer than 3 months. Complete Authorisation Form* ***after*** *the trial period*)

Dietitian Referral – Inpatients (North Shore Hospital)

Trial

APPENDIX G: Evidence based guideline for nutrition screening

Criteria	Evidence based statement	Grade (NHMRC[11])	Grade (NCCAC)	Grade (ADA EAL)
Nutrition screening	i Screening for malnutrition and the risk for malnutrition should be carried out by health care professionals with appropriate skills and training.	–	D(GPP)	–
	ii All hospital inpatients on admission should be screening. Screening should be repeated weekly for inpatients.	–	D (GPP)	
	iii Screening should assess BMI, percentage unintentional weight loss and should also consider the time over which nutrient intake has been unintentionally reduced and/or the likelihood of future impaired nutrient intake.	–	D (GPP)	
	iv Implementation of a nutrition risk screening program:	–	–	
	a Improves the identification of individuals at risk of malnutrition;	B	–	
	b Facilitates timely and appropriate referral for nutrition	B	–	
Nutrition screening tools	Valid nutrition risk screening tools include:		–	
	i MST	B		II
	ii MUST	B	–	II
	iii NRS-2002	B		I
Nutrition Interventions	i Dietary counselling by a dietitian may improve outcomes such as:		–	–
	a Weight status and physical function	C	–	–
	b Weight status and body composition	C		
	i Oral Nutritional Supplements may improve outcomes such as:		–	–
	a Weight status, body composition, complications, pressure ulcers, life expectancy (evidence of an effect)	A	–	–
	b Energy and protein intake, global nutritional status, mood	A		
	iii Individually prescribed nutritional support (including high energy diets ± ONS) may improve outcomes including:		–	–
	a Energy intake and wound healing	C	–	–
	b Weight status and nutritional biochemistry	C		
	iv Feeding assistance may improve outcomes including energy intake, body composition, life expectancy and use of antibiotics	C	–	–
	v "Protected" Meal times	No evidence located	–	–

NHMRC: National Health and Medical Research Council; NCCAC: National Collaborating Centre for Acute Care; ADA EAL: American Dietetic Association Evidence Analysis Library®, BMI: Body Mass Index; Aus: Australia; MST: Malnutrition Screening Tool; MUST: Malnutrition Universal Screening Tool; NRS-2002: Nutrition Risk Screening-2002; ONS: Oral Nutritional Supplements.

NHMRC: Grade A: Excellent level of evidence; Grade B: Good level of evidence; C: Satisfactory level of evidence.

NCCAC: Grade D (GPP): A good practice point (GPP) is a recommendation for best practice based on the experience of the Guideline Development Group.

ADA EAL: Grade I: Good strength of the evidence; Grade II: Fair strength of the evidence.

APPENDIX H: Nutrition Study Data

NUTRITION STUDY DATA
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(for Thu 14 Mar 13)

PreAlb (0.2-0.3) g/L	RBP (30-60) mg/L	Transfern (2.0-3.6) g/L	CRP (0-5) mg/L	Albumin (38-52) g/L
0.31	89.6	3.1	3.00	36
0.26	46.4	2.1	6.80	
0.29	55.6	2.4	3.00	37
0.10	13.6	2.0	151.00	39
0.24	48.1	2.4	3.00	40
0.32	56.2	2.3	6.53	36
0.26	44.1	2.3	3.00	35
0.24	50.5	2.3	3.00	34
0.33	127.0	1.9	59.80	25
0.37	61.0	2.5	3.00	41
0.18	44.7	2.6	3.58	34
0.28	45.0	2.4	3.00	34
0.28	56.2	2.2	6.29	40
0.16	23.6	1.6	98.70	27
0.22	41.8	2.2	3.00	34
0.25	52.3	2.0	3.00	32
0.21	41.5	2.7	3.00	38
0.15	63.9	2.3	20.40	30
0.20	34.0	1.9	77.10	32
0.17	35.8	1.6	187.00	30
0.32	75.7	1.9	3.09	36
0.34	73.7	4.1	3.00	41
0.24	30.5	3.0	16.40	36
0.14	24.6	3.1	36.00	29
0.15	24.6	2.9	3.00	37
0.24	64.9	2.3	3.38	37
0.36	81.0	2.2	3.00	43
0.24	40.7	1.8	3.00	36
0.19	29.5	2.8	3.00	
0.32	43.6	2.5	3.00	42
0.18	46.8	1.8	109.00	31
0.09	16.6	1.6	27.00	27
0.13	25.5	1.6	148.00	26
0.26	69.0	2.6	3.00	41
0.24	78.9	2.3	19.80	36
0.25	31.6	1.5	140.00	28
0.33	47.8	2.5	3.00	41
0.37	74.9	2.6	3.00	
0.55	123.0	3.4	3.00	40
0.27	39.7	3.1	3.00	35
0.35	63.6	2.0	42.90	41
0.25	48.9	2.2	3.00	36
0.42	70.6	3.2	3.00	47
0.27	58.9	2.7	3.00	36
0.24	102.0	1.3	246.00	23
0.25	48.1	2.2	3.36	39
0.10	31.1	1.1	145.00	22
0.27	40.0	2.6	38.80	36
0.20	32.2	2.0	84.90	32
0.24	46.2	2.2	72.60	33