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POINTS OF VIEW: NUTRITION

Trace elements

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SUMMARY

Trace elements are essential micronutrients. They are at present seldom considered in the routine management of critical care patients but are immensely important for redox balance and antioxidant function as well as the prevention of clinical deficiency states. Intensive care patients are at risk of both overt and sub-clinical trace element deficiencies and studies show that supplementing critically ill patients with trace elements may offer them a mortality benefit. This article reviews those trace elements relevant to critical care and the research that has been performed on them to date.

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1. What are trace elements?

Trace elements are dietary minerals required in minute quantities for normal physiological function. They are mostly structural components of enzymes or cofactors whose roles include the prevention of nutritional deficiencies, immune functions, regulation of gene expression, antioxidant defence, and prevention of chronic diseases. They are generally defined as each constituting less than 0.01% of body mass and collectively comprise <1% of total body mass. These essential micronutrients are absorbed from the gastro-intestinal tract and stored in the liver and include: chromium, cobalt, copper, fluorine, iodine, iron, manganese, molybdenum, selenium and zinc (see Table 1).

2. Why are they important for normal health?

Specific micronutrient deficiencies cause reproducible structural or biochemical deficits corrected by supplementation of the relevant element. However, overt nutritional deficiencies are rare in developed countries, even in the context of critical care.

The Food Standards Agency UK Dietary Survey 2003 found the intake of some trace elements (iron, copper, iodine) were well below the RNI (Reference Nutrient Intake) for a number of demographic groups and when compared with the population survey from 1986/7 there had been a significant reduction in consumption of copper, iodine and zinc from foods.[1] Studies have also demonstrated that the EU population is selenium deficient, with an insufficient estimated daily intake and reduced plasma levels.[2–5]

Case reports indicate that overt trace element deficiencies occur due to malabsorption syndromes, poor diet (e.g. alcoholics), long-term TPN administration, or genetic predisposition (e.g. inborn errors of metabolism). The UK general population is at risk from sub-clinical trace element deficiencies, especially the elderly, those institutionalised and patients with chronic health problems; it is particularly common in, and relevant to, critical care and maybe masked by co-existing disease.

Table 1
Trace elements.

Trace Element	Essential to man	Risk of deficiency in critical care and with long-term TPN	Risk of toxicity with organ dysfunction and with long-term TPN	Interest in supra-physiological dosing in critical care
Arsenic (As)	?			
Bromine (Br)	??			
Cadmium (Cd)	??			
Chromium (Cr)	✓	✓	✓	
Cobalt (Co)	✓			
Copper (Cu)	✓	✓	✓	✓
Fluorine (F)	✓			
Iodine (I)	✓			
Iron (Fe)	✓		✓	
Lead (Pb)	??			
Lithium (Li)	?			
Manganese (Mn)	✓		✓	
Molybdenum (Mo)	✓	✓		
Nickel (Ni)	?			
Selenium (Se)	✓	✓		✓
Silicon (Si)	?			
Tin (Sn)	??			
Vanadium (V)	?			
Zinc (Zn)	✓	✓		✓

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Table 2
Reference Nutrient Intake (RNI).

Trace element		selenium	copper	zinc	chromium	molybdenum	iodine	manganese
ASPEN: JPEN[6]	Enteral	55 mcg	0.9 mg	11 mg	30 mcg	45 mcg	150 mcg	2.3 mg
	Parenteral	20–60 mcg	0.3–0.5 mg	2.5–5 mg	10–15 mcg	not routinely added	not well defined	60–100 mcg
FSA/COMA[7]		60–75 mcg	1.2 mg	5.5–9.5 mg	25 mcg	not set	0.07–0.14 mg	Not set
WHO[8]		40 mcg	Not set	4.2–14 mg	Not set	0.1–0.3 mg	150 mcg	Not set
US[9]		40–55 mcg	0.7–0.9 mg	12–15 mg	25–35 mcg	34–45 mcg	150 mcg	1.8–2.3 mg

3. How much do we normally need?

The Reference Nutrient Intake (RNI) for a number of trace elements are presented in Table 2.

4. What are normal levels?

The measurement of trace elements is complicated and interpretation of results should be done in conjunction with consideration of the clinical, nutritional and other biochemical information available. Trace element assays are susceptible to contamination and there are uncertainties in selecting the component to measure; laboratory tests lack sensitivity and specificity and there is often a long turnaround time (approximately 1 week). Measuring enzyme function maybe a more clinically useful tool for some trace elements in critical care as blood levels of trace elements vary with the acute phase reaction due to redistribution, changes in protein binding, as well as utilisation. Trace elements can be measured from whole blood or serum, urine, and a variety of tissues. Reference ranges will to some extent depend on the specific laboratory (Table 3).

4.1. Monitoring guidelines

Trace element requirements in critical illness are unknown. The National Institute for Clinical Excellence (NICE) recommend measuring selenium levels if there is a risk of depletion and subsequent testing depending on that result. Baseline copper and zinc levels are also recommended followed by levels every 2–4 weeks, as deficiency is common especially when there are increased losses. NICE also recommend manganese levels to be measured every 3–6 months in patients on home TPN due to the risk of toxicity.[11] Sequential testing with the results reviewed in conjunction with inflammatory markers (e.g. CRP) will be most meaningful on the ICU and will prevent extremes of provision.

Table 3
Trace element reference ranges.[10]

Trace Element	Serum level	Measurement method	Other	Acute phase response
Selenium	0.9–2 μmol/l	Atomic absorption spectrometry	Whole blood glutathione peroxidase 20-70U/g Hb	Levels fall
Copper	10–22 μmol/l	Inductively coupled plasma/emission spectrometry	Caeruloplasmin 0.2–0.6 g/l	Levels rise
Zinc	12–18 μmol/l	Inductively coupled plasma/emission spectrometry	–	Levels fall
Chromium	–	Graphite furnace/atomic absorption spectrometry	Urine <6 nmol/mmol creatinine. Improvement in glucose tolerance test after supplementation	–
Manganese	–	Graphite furnace/atomic absorption spectrometry	Whole blood 70–280 nmol/l	–

5. What happens in critical illness?

In practical terms for critical care the trace elements can be divided into 3 groups:

1. Risk of nutritional deficiency with critical illness and/or long-term TPN e.g. selenium, chromium, copper, zinc, molybdenum.
2. Risk of toxicity with TPN e.g. manganese (especially in liver disease).
3. No identified concerns e.g. iodine - neither deficiency nor toxicity reported.

5.1. Deficiency

Critical illness is a hypermetabolic state and trace element requirements are increased especially for use in redox reactions. Patients are at risk of deficiency for a number of additional reasons:

- Reduced trace element status on admission to critical care due to the background deficiency of our general population, chronic illness, alcohol abuse, and the elderly.
- Increased losses especially through burns, trauma and haemo- or peritoneal dialysis; studies on trace element kinetics in haemofiltration have demonstrated significant losses of selenium and copper, and losses to a lesser extent of chromium, manganese and zinc.[12–14] Gut losses also occur through gastric aspiration, diarrhoea and fistulae and critical illness per se is associated with increased urinary losses of zinc, copper and iron and this maybe influenced by drug choice and preparation.[15]
- Reduced provision due to an inability to receive adequate calorific intake (poor enteral absorption, drug therapy, surgery and procedures requiring starvation periods) and inadequate prescriptions.
  - o Enteral: Most enteral feeds in common usage in the UK[16] adequately provide the majority of essential trace elements if the feed is able to be given at sufficient calorific rates (Appendix A.1). The bioavailability of trace elements from feed is influenced by other feed-related factors e.g. the presence of fibre reduces zinc absorption.
  - o Parenteral: Although there are Total Parenteral Nutrition (TPN) formulations available which contain added trace elements the majority of formulations used "off the shelf" in current clinical practise[16] do not and require the prescription and addition of specific preparations (e.g. Additrace® or Decan®) (Appendix A.2). There are clearly problems with this process as trace element deficiencies have been reported in patients on long-term TPN identifying a failure of care. This is unacceptable and clinicians must recognise the potential for acquiring a clinical deficiency and supplement trace elements before one occurs.

5.2. Toxicity

- Manganese and copper can both be toxic in liver dysfunction. This is a particular problem with manganese and TPN-associated cholestasis. Specific trace element deficiencies should be supplemented individually to prevent over-supplementation of

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