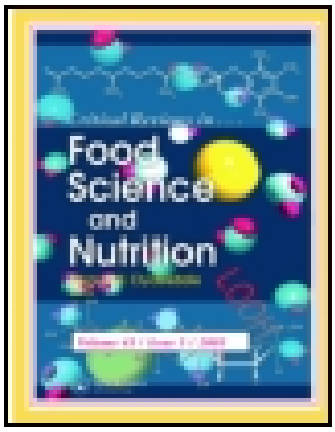

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Categorizing Food Related Illness: Have We Got It Right?

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Categorizing food related illness: Have we got it right?

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INTRODUCTION

National policies on food and nutrition should recognise the interaction between nutrition, food safety and food security (WHO, 2002). Food plays a hugely important role in causing, and also preventing, many human diseases. Eating an inappropriate diet can lead to deficiency related illness, or conversely over-consumption diseases, and contaminated food can cause foodborne disease and harm (WHO, 2002). Malnutrition encompasses the consideration of those that are over nourished (overweight and obese) as well as those who are under nourished. In 2008, there were 1.4 billion people who were overweight, whilst 0.9 billion people were under nourished from an estimated population of 6.7 billion (FAO, 2013; PRB, 2008; WHO, 2013). This represents around 34% of the global population so the issue of inadequate human nutrition is not insignificant. Elia *et al.,* (2010) propose that malnutrition remains an under-recognised and under-treated clinical and public health problem, with adverse clinical and financial consequences. The WHO (2002) suggests that the economic consequences of malnourishment include lower productivity, family distress and costs to national health services. Therefore securing adequate nutrition on a regular basis for individuals and communities at a local, national and regional level lies at the heart of a resilient food supply chain.

Rayner and Scarborough (2005) estimate the cost to the United Kingdom (UK) National Health Service (NHS) of treating diet related ill heath, sometimes known as diet related non-communicable diseases, as being £6 billion per annum. For clarity the term "food related illness" will be used in this paper although a range of terms have been used in the literature. Rayner and Scarborough (2005) propose that there are two main problems in quantifying the burden of ill-

health specifically related to food and in particular when comparing the burden with that related to other causes: measurement (mortality, morbidity and money) and attribution i.e. being able to distinguish between health issues that relate to food and not other risk factors, such as smoking, exercise etc. Elia and Stratton (2009) estimated that in 2007 the UK public expenditure on disease-related malnutrition was in excess of £13 billion, corresponding to about 10% of the expenditure on health and social care with 3 million people being affected. Scarborough et *al.*, (2011) reviewed NHS costs between 2006-2007 and compared poor diet-related illness cost in total (£5.8 billion) to physical inactivity (£0.9 billion), smoking (£3.3 billion), alcohol (£3.3 billion) and overweight and obesity (£5.1 billion). Scharff (2011) estimated the aggregated annual cost of food related illness in 2010 associated with bacteria, viruses and parasites alone in the United States (US) was between $51 and $77.7 billion per annum. In 2008, the estimated annual medical cost of obesity in the US was $147 billion (CDC, 2014). These statistics clearly demonstrate the financial impact of food related illness on two developed national economies.

The developed nation is not alone in needing to deal with the challenges of food related illness. India's National Institute of Nutrition (NIN) published the Second Edition of the Dietary Guidelines for Indians in 2011. The foreword highlights the socio-economic changes in India and the need as a result to update existing nutritional guidelines. These include not only government initiatives in the areas of poverty alleviation, health and nutrition but also changes to diet and lifestyle across India including the introduction in Indian cities of multinational fast food chains. These socio-economic factors mirror developed countries in terms of changing cooking practices, increased intake of processed and ready-to eat foods and a preference for energy-dense foods with a high sugar and salt content (NIN, 2011). Therefore the Guidelines state there is a

growing problem of a "*double burden*" of malnutrition in terms of both *under* and *over* consumption. As a result, what appears on the surface to be a dichotomy is actually a spectrum of food related health issues (low birth weight, micro-nutrient and protein deficiency through to obesity) that need to be addressed and mitigated. Misra *et al.,* (2011) concur determining that India is facing crisis of diet related illness resulting in substantial socioeconomic burden. They conclude that nutrition transition between 1973-2004 resulted in a 7% decrease in energy derived from carbohydrates and a 6% increase in energy derived from fats. Thus a reduced intake of coarse cereals, pulses, fruits and vegetables led to increasing meat and salt consumption, and as a result of rapid urbanization reduced physical activity leading to a rise in levels of obesity, atherogenic dyslipidemia, subclinical inflammation, metabolic syndrome, type 2 diabetes mellitus, and coronary heart disease. To compound this low-birth weight infants, coupled with early childhood "catch-up growth" is leading to obesity in early childhood, thus predisposing to non communicable diseases later in life. The social changes of increasing sedentary life style, altered eating habits and greater fat consumption in India means that hypertension, overweight and obesity are increasing in adolescent groups (Bagudai *et al.,* 2014). Bishwajit *et al.,* (2014) highlight that these food related illnesses are now gaining prevalence not only in India, but also in Bangladesh and Nepal with a clustering of obesity and related diseases among the poor communities as well as the rich in Asia. In China food related chronic illness accounted for 41.6% of all deaths in 1995 and the cost of food related illness is estimated to be 2.1% of annual GDP (Bishwajit *et al.* 2014 citing Popkin *et al.,* 2001). The WHO (2005) estimated for the decade 2005-15, the cost in China of chronic disease such as diabetes and heart disease could be as much as $556 billion (Ellulu *et al.,* 2014*)*. In terms of human health where does the definition

of food safety and harm caused to the individual end and the problems of inappropriate diet and nutrition begin? Are they in fact on the same continuum? By compartmentalizing the spectrum of food safety hazards and wider food related health issues does that in itself form a barrier to overall mitigation at personal and policy level? This is the question that this research sought to answer.

THE ROLE OF FOOD IN HUMAN ILLNESS

Classic food hazard definitions

Humans need nutritious, wholesome and safe food in order to survive. Trench *et al.,* (2011) argue that safe food is not a luxury and identified three factors that drive increasing food health risk: higher demand for "cheap" food thus driving global supply and increased reach for health risks, shifting consumption patterns, and increasing urbanization leading to a reliance on others. This makes the actors in the food supply chain more remote, and anonymous. In the context of classic food safety vocabulary, a food hazard can be defined as "a biological, chemical, or physical agent in, or condition of, food with the potential to cause an adverse health effect." (CAC, 2003:5; BS EN ISO 22000; 2005; Trench et al., 2011:4; Wallace et al., 2011:65). The CBRI (2009) and the BRC Global Standard for Food Safety (2011:117) expand on this tri-categorization to include food allergens as a fourth category and the latter describes a food safety hazard as "a biological, chemical, physical or allergenic agent in food, or condition of food that has the potential to cause an adverse health effect." Mortimore and Wallace (2013) use the CAC (2003) categories, but include allergens within the category of a chemical hazard. Undertaking a food safety hazard risk assessment is usually structured by defining the agent that can cause

harm together with the likely foods in which it could present that harm. The UK Food Standards Agency (FSA, 2011), when determining priorities for food sampling, lists specific hazards and in many instances relate them directly to a foodstuff and a country of origin if of concern when determining the level of risk (Low, Medium, High) e.g. *Listeria monocytogenes* in cooked meats. APA (2002) uses a different approach identifying hazards as a result of specific breakdowns in the food supply chain. FSA (2011) does not address physical hazards and also includes legality issues (Table 1). This body of literature demonstrates that based on underlying principles there is a range of approaches that can be used to categorize food hazards.

In terms of food related illness, it is of value to differentiate between intoxication and infection.

Food related intoxication

BRC (2011:117) defines food safety as the assurance that food will not cause harm to the consumer when it is prepared and/or eaten according to its intended use. The terms "food poisoning", "foodborne illness" "foodborne disease" are used interchangeably in current literature, policy and guidance. For the means of clarity in this paper the terms are redefined as being distinct and mutually exclusive. Sprenger (2014:10) describes food poisoning as an acute illness, usually of sudden onset, brought about by eating contaminated or poisonous food. The use of the word "acute" in this context is concerned with the nature of the symptoms rather than the onset period between ingestion and symptoms. The Food and Agriculture Organization of the United Nations (FAO) reported two decades ago that the ingestion of mycotoxins can produce both acute (short-term) and chronic (medium/long-term) toxicities ranging from death to chronic interferences with the function of the central nervous, cardiovascular and pulmonary systems, and of the alimentary tract. Some mycotoxins are carcinogenic, mutagenic, teratogenic and

immunosuppressive (FAO 1994). Here the term “acute” is used differently to describe the onset period rather than the nature of the illness itself. This divergence of the use of the terms acute and chronic is common across the literature.

Mortimore and Wallace (2013:454) describe a toxin as a chemical or microbiological metabolite that can cause toxic effects when ingested. This term could be extended to define a toxin or toxic agent as being an organic or inorganic chemical, including microbiological metabolites that can cause toxic effects in humans when ingested or synthesized within the human body. This definition will encompass toxins produced as a result of microbiological metabolism, lysis and replication. Food poisoning can, depending on the toxic agent, be both acute and chronic (in terms of onset period), but traditionally the term food poisoning is focused specifically on toxicity i.e. the agent that causes food poisoning being a toxin of either a microbiological origin or other source. This means that food poisoning could be redefined as being a health disorder with symptoms of either of short [acute] or long term duration [chronic] with a specific onset period that is induced by consuming food that is contaminated by biological organisms that have the ability to produce toxins once ingested, or food that contains toxic material at the time of consumption. Examples of the traditionally derived major groups of toxic agents in food have been collated (Table 2).

Being toxic by nature means that the particular toxigenic agent, once consumed, will lead to a health disorder. Some toxic agents may cause illness as a result of a single dose whilst others may be accumulative over time e.g. metals. In the literature, food poisoning has been classified as originating from both toxic, as previously described, and infectious (i.e. through pathogenic invasion of the host) agents. Indeed, some pathogens are identified as having both infectious and

toxic characteristics (Mortimore and Wallace 2013; Sprenger, 2014). Wallace *et al.* (2011) suggest that foodborne illness can be categorized according to the mechanism of the host-pathogen interaction. *Intoxications*, otherwise described elsewhere as microbiological food poisoning, occurs when a pathogen produces toxin whilst growing in a food e.g. *Stapylococcus aureus, Clostridium botulinum*. In contrast, *infections* are caused when viable pathogens contaminating food survive passage through the host's stomach into the intestine (Wallace *et al.*, 2011:69).

Food related infections

Infectious i.e. communicable diseases caused by pathogenic microorganisms (WHO, 2014), can be spread, directly or indirectly via a vehicle, from one person to another and are characterized by having a low infective dose and no requirement for multiplication of the organism within the food to cause illness (Sprenger 2014:382). Examples of foodborne pathogenic viruses that use food as a vehicle and cause infections include influenza viruses (especially avian and swine derived), hepatitis, norovirus and rotaviruses. Newell *et al.*, (2010) argue that the burden of diseases caused by food-borne pathogens remains largely unknown, as quantification across the global population is sporadic by country. Communicable infectious disease includes that caused by bacteria such as *Salmonella, Campylobacter, E.coli*, *Listeria Monocytogenes,* and the aforementioned viruses. Newell *et al.*, (2010) outline that food can be a source of not only antimicrobial resistant bacteria, but also resistance genes leading to food-borne disease that cannot be treated with certain antibiotics. Horizontal transfer of genetic elements can be through a variety of complex routes and it can be difficult to quantify the level of risk of such occurrence in a particular situation. Large outbreaks may occur as a result of contamination of food by a

single foodhandler or source e.g. through Norwalk-like caliciviruses or Hepatitis A virus (HAV), (Koopmans *et al.* 2002). Newell *et al.*, (2010:S9) highlight that food-borne transmission has been documented for viruses belonging to at least 11 known families (Table 3).

There is some debate as to whether proteins derived from viruses can have toxigenic characteristics, but no literature has been identified in this research that states this is the case in food related illness. However as more research is done in this area, toxic agents associated with viruses, especially cytotoxic agents could be identified reducing the power of the intoxication versus infection argument. It is difficult already to make this an "either: or" situation when some organisms as previously explained express both characteristics. Newell *et al.*, (2010:S9) suggest that the probability of the emergence, or evolution, of new communicable food-borne disease viruses is "inevitable given the demographic, economical, and sociological changes that we are now facing". Koopmans and Duizer (2004) propose that in order to control foodborne viral infections there must be more standardized methods of detection and better national and international surveillance systems, greater awareness of the role of food handlers in the spread of the disease, and improved consideration of viral food borne disease during risk assessment and the development of food safety management systems.

Newell *et al.* (2010) determine that approximately 300 species of parasitic worms and over 70 species of protozoa have been described that can infect humans and parasites that can cause food-borne disease. These are draw together (Table 4). The global impact of some food related, especially water related, infectious disease is pronounced. *Ascarius* (giant roundworm) infection

is estimated to affect 1 billion people worldwide i.e. 1 in 7 of the global population and 50 million people are said to suffer from amoebic dysentery at any time (Shaw, 2013).

The difference between pathogens that cause microbiological toxication and those that lead to disease through microbiological infection is of interest, but the wider influence of food intoxication is of focus in this area when considering food related illness. Caffeine is the most frequently consumed substance with centrally stimulating effects (Riesselmann *et al.,* 1999), who determined that caffeine concentrations in plasma above 15 mg/l can cause toxic symptoms while values above 80 mg/l are comatose-fatal. Pohler (2010) concluded that caffeine produces dose-dependent symptoms, and intoxication may develop with overconsumption of caffeine containing drinks. Herbal or fruit derivatives of tea, coffee, soft (soda) drinks and energy drinks can contain high caffeine and sugar and in the case of energy drinks these are particularly marketed at the young (Pohler, 2010; Temple, 2009). There are repeated studies on the ill-effects of alcohol. Alcohol is causally related to over 200 three-digit International Classification of Diseases (ICD) revision 10 codes and is one of the most important risk factors for the burden of disease and injury (Shield *et al.,* 2015) causing 3.3 million deaths and 5.1% of all disability-adjusted life years lost in 2012. This is a global health challenge.

Salt and sugar are both inexpensive preservatives used widely throughout the food industry. As Nestle (2006:365) explained: *"If you eat snack foods at all, it will not take long for you to eat more salt than is healthy and often much more than is good for you."* Over-consumption of salt has been identified as a significant public health problem, leading to various national efforts (in various countries) to encourage salt reduction (Kenten *et al.,* 2013). Indeed in the UK daily

consumption levels for an adult have been set at a maximum of 6g. Delahaye (2013:325) determines that high salt consumption is a major cause of increased blood pressure, a risk of stroke, left ventricular hypertrophy, renal disease, obesity, renal stones and stomach cancer.

Fructose, glucose and sucrose are all sugars. Fructose, a monosaccharide, is naturally present in fruits and is used in many food products as a sweetener (Keim and Havel, 2013). Rodrigues *et al.,* (2013) argue that fructose intake from added sugars correlates with the epidemic rise in human obesity, metabolic syndrome and cardiovascular diseases. Lee *et al.,* (2009) concur stating that dietary fructose consumption is one of the environmental factors contributing to the development of obesity and hepatic steatosis (fatty liver) and demonstrates cytotoxicity, which is prevented by radical scavengers, lipid antioxidants and reactive oxygen species (ROS) scavengers. Maternal fructose intake during pregnancy led to an impairment in the transduction of the leptin signal in the fetuses. This could be responsible for the development of hepatic steatosis (Rodrigues *et al.,* 2013).

Sucrose occurs naturally and its intake has been associated with adverse health effects such as excess caloric intake, excess weight gain, dilutional effects of essential nutrients, and increased risk for dental caries, type 2 diabetes, and cardiovascular diseases (Caballero, 2013; Steyn and Temple, 2014). Karalius and Shoham (2013) state that research supports a potentially causal role of sugar in chronic kidney disease and more especially several kidney disease risk factors, including increasing serum uric acid levels, diabetes, and obesity. Kendig (2014) goes further extrapolating from an animal model to argue that sugar can induce cognitive dysfunction and impair spatial learning and development as well as altering reward-related behaviour. Francis and Stevenson (2013) concur with Kendig highlighting that while human research data is still at

an early stage, there is evidence of an association between diets high in saturated fat and simple carbohydrates and impaired cognitive function. This diet i.e. the "Western diet" is linked with the development of obesity and Alzheimer's Disease with hippocampal function being particularly susceptible to diet-induced impairment (Kanoski and Davidson, 2011 Knight *et al.,* 2014). Kanoski and Davidson (2011) conclude that such diets may disrupt memory by impairing blood-brain barrier integrity. The impact of a high-fat diet in terms of illness and disease has been well defined in wider literature. The result of such food intake is not just the consumption of fat but also the fact in such diets that there is wider deficiency of essential nutrients and vitamins such as magnesium. There is emerging research of the impact of high fat-high carbohydrate diets (HFCD) and their impact on hippocampal function. Alzoubi *et al.,* (2013a) show that HFCD impairs short- and long- term memory and reduces hippocampal antioxidant mechanisms. The degree of impact depends on other essential vitamins in the diet as Vitamin E can prevent HFCD-induced memory impairment. Thus, Vitamin E and caffeine could be acting through its antioxidant effect on the hippocampus (Alzoubi *et al.,* 2013a; 2013b). As well as food ingredients such as caffeine, alcohol, sugar or salt, Profet (1991:23) proposes that the mammalian immune response known as "allergy" has evolved as a last line of defense against the extensive array of toxic substances that exist in the environment in the form of secondary plant compounds and venoms. Thus by extending this supposition there is an argument that food allergy should be considered within the category of food intoxication.

Immunological and non-immunological reactions to food

Plants commonly synthesize a range of secondary metabolites as part of their protection against attack by herbivores, insects and pathogens or as a means to survive adverse growing conditions

(Khokhar and Apenten, 2003). Profet (1991) argues that the human immune system recognizes as toxic low molecular weight substances that bind covalently to serum proteins (e.g. plant toxins); nontoxic proteins that act as carriers of toxins with low molecular weights (e.g., plant proteins associated with plant toxins); and specific substances of high molecular weight that have harmed individuals for a long period of time and have an influence on natural selection. These substances can be toxic, potentially mutagenic and carcinogenic as well. Thus, by protecting against acute toxicity, allergic reactions may also defend against mutagens and carcinogens. This Profet concludes would explain why allergic cross-reactivity occurs to foods from unrelated botanical families. Wallace *et al.,* (2011:79) also highlight cross-reactivity and the regional associations with allergens e.g. European Union (celery), South-east Asia (buckwheat), Japan (rice). Shaw (2013) outlines cross-reactivity with individuals who appear allergic to latex (from the rubber plant) also being highly sensitive to banana, avocado, shellfish, kiwi fruit, and tomato. The common foods that are associated with allergenic reaction have been reviewed in Table 5 with the specific allergenic protein (where identified in the literature). Subunits of individual allergens have not been included but are detailed in WHO/IUIS (2014).

Mortimore and Wallace (2013:451) define an allergen as a compound capable of inducing a repeatable immune-mediated hypersensitivity response in sensitive individuals. Adverse reaction to a food will include not only allergic reactions to foods that are immune mediated but also non-immune mediated reactions e.g. functional food intolerance due to enzymatic abnormalities in individuals. This demonstrates that there are both toxic and non-toxic sensitivities to food. Alternatively Zopf *et al.,* (2009:359) define food intolerance as a range of food related

complaints of varying etiology e.g. functional disorders such as transport defects, and enzymopathy (absence or poor function of an enzyme) or a structural disorder i.e. "an anatomically and morphologically demonstrable disease involving a structural alteration in the gastrointestinal tract. This results secondarily in food-associated symptoms… [such as] small intestinal diverticula." They embrace the term food allergy/allergen within a wider scope of food intolerance.

Functional disorders can be of toxic or non-toxic origin or as a result of a functional breakdown such as lactase deficiency in the small intestine (Table 6). Non-immunologically mediated reactions account for the majority of all reactions to food i.e. 15% to 20% (Zopf *et al.*, 2009). Examples put forward by Zopf *et al.,* (2009) and Wallace *et al.,* (2011 citing Timbo *et al.,* 2004) include reactions to non-proteinaceous compounds such as salicylates, biogenic amines such as histamine, sulfites, sodium glutamate, colorants and preservatives (such as tartrazine, benzoates, sorbates etc.), and sweeteners (aspartame).

Transport and signaling defects in the gut will influence health status. Zopf *et al.,* (2009) highlight the transport defects that can influence fructose transport (GLUT 5) and glucose, galactase and fructose transport (GLUT 2). Migraine is an allergic disease (Unger and Unger, 1952; Monro *et al.,* 1984). Monro *et al.,* (1980) determine that in their study two-thirds of severe migraine sufferers were allergic to certain foods, shown by dietary exclusion and subsequent challenge. They concluded that the initial specific allergic reaction in the gut could then result in increased mucosal permeability that would allow food antigens, complexes, or mediators to be absorbed and cause symptoms. Ku *et al.,* (2006) suggest that histamine plays a key, triggering role by means of vasodilation and inflammation in the pathogenesis of migraine headaches.

Histamine rich and histamine releasing foods include wine, beer, cheese, chocolate, dried fruits such as apricots, fermented foods, and processed meats. Many of these foods have been associated with migraine. Zopf *et al.*, (2009) describe migraine as a non-immunological disease although the literature above would suggest that the opposite could be the case or that migraines are the result of multiple factors including gene mutation. This would seem to be the case as Weigand *et al.*, (2014) determine that sporadic hemiplegic migraine type 2 (SHM2) and familial hemiplegic migraine type 2 (FHM2) are rare forms of hemiplegic migraine caused by mutations in the Na^+, K^+-ATPase α2 gene.

According to Pang *et al.*, (2012), the gastrointestinal (GI) tract is a selective nutrient absorption system and the most important signal transduction and information exchange system within the body. Further, it acts as a signal transducer, a neuroendocrine sensor, and an immunological recognition and presentation system and a complex information exchange system comprising a number of signaling networks involving GI cells and cells immobilized in organs or transported in blood (Pang *et al.*, 2012:26). Therefore if the gut is compromised in some way either structurally or functionally this weakness will be highly involved in the mechanisms of illness and disease.

Alkaloids are bioactive natural compounds of many foods and are frequent contaminants of several other foods being present in common foods including potatoes (Table 2), and at higher levels in tea and coffee (Crews, 2014). The term antinutrient or natural toxicant has been widely employed to define plant defense metabolites and legumes prove a rich source of these compounds. Antinutrients commonly found in plant foods have both adverse effects and health benefits (Thompson 1993). Food derived from plants can contain proteinaceous antinutrients

including protease, α-amylase, and lipase inhibitors, phytohaemagglutinins, lectins, and allergens as previously discussed (Khokhar and Apenten, 2003). Thompson argues that whilst phytic acid, lectins, and saponins, among others have been shown to reduce the availability of nutrients and cause growth inhibition, and phytoestrogens and lignans have been linked with infertility problems, phytic acid, lectins, and saponins have also been shown to reduce the blood glucose and insulin responses to starchy foods. In addition, phytic acid, saponins, phytoestrogens and lignans have been related to reduced cancer risks. The observed biological effects vary according to the structure of the metabolite which can range from high molecular weight proteins to simple amino acids and oligosaccharides (Khokhar and Apenten, 2003). Pang *et al.*, (2012) explain that flavones were found to protect against heart disease and soy-based estrogens against cancer. This paints a complex picture of both the pro-nutrient and the anti-nutrient properties of food.

This review has been drawn together to consider where the definition of food safety and harm caused to the individual by a specific food borne agent end and the problems of inappropriate diet and nutrition begins. It is argued that they are they in fact on the same continuum. Research shows that in both developing and developed societies health issues arise from either under or over nutrition. Since the 1950s food safety hazards have been categorized as (micro) biological, chemical or physical hazards. International food safety risk assessment methods, including HACCP use these criteria. However as the body of research into food related illness increases, the spectrum of agents that would be considered as an "agent of harm associated with food" grows encompassing food allergy and intolerance, and the triggers for chronic diseases such as obesity, type 2 diabetes, stroke, heart disease, cancer as equally as the traditional aspects of food poisoning, foodborne illness and food contamination. Therefore over a half-century later is this

the time to redefine the term food related illness and the scope of food safety risk assessment to encompass chronic disease.

The spectrum of food related illness

Rayner and Scarborough (2005:1054) describe food related ill-health as being associated with diseases that are caused by foodborne pathogens, as well as other diseases caused by the over or under consumption of nutrients or other components of foods. Food related illness therefore encompasses aspects of intoxications and infections, but can be described as the illness resulting from consumption of food or water contaminated by pathogenic microorganisms and/or their toxins or other agents or ingredients that impact directly on health and wellbeing. In this paper fat, salt, sugar, alcohol and caffeine are also included in the intoxication aspects of the acute to chronic food safety spectrum. These include viruses, parasites and protozoa, and physical hazards that can cause harm. Physical hazards provide a clear distinction in terms of intrinsic and extrinsic hazards. Intrinsic physical hazards form an integral part of the food component when still living and need to be adequately controlled during harvesting or slaughter, processing and preparation to prevent harm to the consumer when the food is consumed e.g. bones, extraneous vegetable material etc. Extrinsic physical hazards arise from the environment in which the food is grown, prepared or processed and adequate controls need to be in place to prevent such contamination e.g. glass control procedures, wood, metal control and so forth. (Table 7).

Structural and functional issues associated with food allergy and food intolerance need to be understood, and where possible mitigated, both at food technologist level when food specifications including product formulation and product labelling are developed and when there are wider food risk assessment activities undertaken at national policy level, such as planning

annual food surveillance sampling or within the control strategies of the general food supply chain. More research needs to be undertaken especially in the areas of food allergy, food intolerance and the impact of antinutrients in order for applied tools to be developed that can assist those in the supply chain to undertake appropriate risk assessment, develop safe food products and provide adequate information to consumers and the wider food supply chain especially as the body of knowledge on for instance, allergen cross-sensitivity grows.

Government policy with regard to malnutrition in its wider sense has failed to address the rising tide in overweight and obesity levels within the global population and as a result the rise in diet related illness. The literature discussed in this paper has shown this to be the case in both developing and developed countries. Whilst sugar and salt are inexpensive food preservatives, their inclusion in a food product must be undertaken with full consideration of their role in wider food related illness. The corporate social responsibility strategies of food retailers and food manufacturers are developing in this light as they seek to produce lower salt content and lower sugar content foods. However the impact on microbiological food safety, in terms of water activity, and also food quality must be considered fully if these ingredients are reduced in composite food products. By traditionally separately compartmentalizing the spectrum of food safety hazards and wider food related health issues does that in itself form a barrier to overall mitigation at personal and policy level? Communicable disease risk (infection) associated with food has been well defined in traditional food safety risk assessment e.g. with regards to microorganisms and parasites in terms of hazard classification, understanding of potential sources and vehicles for cross-contamination and the means for their control. Indeed risk assessment through the use of binary decision trees, HACCP as a risk assessment approach, or

semi-quantitative risk matrices or risk scoring systems is embedded at organizational, regional and national levels. HACCP as a risk assessment tool was designed over half a century ago, initially to focus on microbiological food safety and has its limits in this approach as the terms and definitions of HACCP do not reflect the wider food related health issues of today. The value of this risk assessment activity is based upon the level of access to pertinent information for those undertaking the assessment and the knowledge of the individuals determining the level of risk. This paper has shown that in some research areas e.g. the intoxication capacity of viruses this knowledge base is limited which ultimately undermines the accuracy of risk assessment activities.

The definition of intoxication has been developed in this paper to include both acute and chronic considerations. Currently intoxication associated with food ingredients, heavy metals in fish, allergens, and illness from foodborne organisms such as *Campylobacter* are treated as discrete policy initiatives but does this approach actually dull the underlying message to the general public with regard to intoxication and food related illness?

Concluding remarks

This research argues that food related illness should be redefined to include both acute and chronic health issues that arise on the basis of food choice and the behavior of the general public in terms of food. Appropriate risk assessment activity should include this wider spectrum of food related activity. Lebel (2003) proposes a holistic or ecosystem approach to health with consideration given to economic, environmental and community (social) drivers. Figure 1 outlines a risk assessment approach with the spectrum of food related illness considerations worthy of assessment. Using the multidisciplinary approach put forward by Lebel to identify

realistic agents of harm, the means for their control and then the level of risk associated with each agent as a result will help to prioritise national, regional, local and individual business risk assessment activities. The steps in this type of risk assessment are as follows:

1. Determine the realistic agent(s) with the potential to cause harm and then assess each agent in turn.
2. Identify the likelihood, and the severity of the agent should it arise. **Likelihood:** consider potential for occurrence with current controls and management in place validating these controls where necessary, and consider whether this is frequency of occurrence is acceptable to stakeholders; and **Severity:** associated potential to cause death, hospitalisation, disability, limited or extreme incapacitation, or economic cost.
3. Determine whether additional controls need to be put in place to reduce likelihood of occurrence, and implement if required.
4. Redefine the likelihood of the agent should it arise based on additional controls that have been put in place.
5. Rank risks according to importance: high risk, to medium risk, to low or negligible risk and these will identify the priority agents of concern by product, by sector or by country.
6. Verify that the controls are effective through specific verification activities then at a defined frequency revisit steps 1-5 again.

Take in Figure 1

Food choice by consumers especially as urbanization increases needs to be underpinned by appropriate corporate social responsibility strategies by those organisations from whom food is

purchased especially where personal choice is limited by socio-economic factors. The current global health care costs of food related illness is high and ever growing. A combined focus with regard to non-toxic illness, physical food contamination, infection and food intoxication in all its forms would assist in delivering key health messages both on pack at the point of purchase and in wider communication campaigns. As globalization of the food supply chain continues, emerging and reemerging hazards need to be considered where there is little evidence/data available for those undertaking assessments, at national or corporate level. Thus it can be difficult to draw appropriate conclusions on risk to a population or target group e.g. regional issues with food allergy or intolerance. Therefore new decision-making tools need to be developed that recognise the evolving spectrum of food related illness so the government and the industry can deliver more tailored solutions in order to safeguard health and wellbeing of the global population.

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Table 1. Potential food safety hazards of concern (Manning et al., forthcoming)

APA (2002)	FSA (2011)
• Naturally occurring toxins (cyanogenetic glycosides, scombrotoxin, paralytic shellfish toxin, etc.); • Contamination from a polluted production environment or contaminated animal feed (heavy metals, dioxins etc.); • Contamination through poor hygiene (bacteria, viruses); • Contamination by metabolites from micro-organisms (mycotoxins etc.); • Use of unsuitable ingredients (e.g. non-food ingredients such as diethylene glycol etc.); • Poor control over the levels of additives (e.g. excessive levels of preservatives, colors etc.); • Contamination from the harvesting process (insects, foreign plants etc.); • Contamination arising from the production process (residues from pesticides, veterinary treatments, fungicides, foreign objects, contact with machinery etc.); • Deterioration during production or subsequent storage (migration from packaging material, protein breakdown, insect infestation); and • Failure to declare the presence of ingredients to which a consumer individual may be allergic (nuts, fish by-products, milk products etc.)	• ***Microbiological*** – raw/lightly cooked read to eat foods (medium); *Listeria Monocytogenes* in cooked meats (high); *Salmonella* in sprouted seeds and herbs and spices (high); *Campylobacter* in poultry (high); aflatoxins in groundnuts, brazil nuts, maize and cereals (high); aflatoxins in pistachio nuts (medium); ochratoxin A in spices (high); OTA in cereals and bakery products (high); other mycotoxins e.g. citrinin, moniliformin, cyclopiazonic acid, sterigmatocystin, alternaria toxin (low); OTA in dried fruit, fresh produce (high); patulin in pressed apple juices (medium); • ***Chemical*** - primary aromatic amines from migration in kitchen utensils (high) formaldehyde from migration from melamine ware (high); 3-mcpd in non-naturally fermented soy sauce (low); non dioxin like PCBs in meat, fish and dairy products (high); PAH in herbal supplements (low); dioxin in clay food supplements (medium); heavy metals e.g. tin, mercury, cadmium, arsenic in imported fish, aluminum in noodles from China and Hong King (medium); irradiated products (low) chloramphenicol and nitrofurans in casings (high); aminoglycoside in farmed fish (medium); carbendazim thiophanate-methyl and tubuconxole in yams, okra, salad crops and herbs(medium); Bt63 rice from China (low); radioactive caesium in mushrooms and berries from eastern Europe (low); undeclared allergens in pre-packed foods (high); • ***Legality*** – speciation – fish (low); Quantitative ingredient declarations (QUID) (low)

Table 2. Toxic agents in food (Adapted from INFOSAN, 2011; Wallace et al., 2011; Bad Bug Book, 2013; Mortimore and Wallace 2013; Shaw, 2013; Sprenger 2014; WSDH, 2014)

Toxic agent	Traditional classification	Examples
Bacterial endotoxins	Biological hazard	Endotoxins are released from the cell but are cell-associated substances lipopolysaccharides (LPS) i.e. an integral component of the outer membrane of the bacteria. Food examples – *Shigella spp. Salmonella spp.*
Bacterial exotoxins	Biological hazard	Highly toxic proteins (polypeptides) produced by living bacteria during growth, multiplication or sporulation and secreted – extracellular, soluble and diffusible. Often produced in food. Enterotoxins are exotoxins that affect the gastrointestinal tract. The botulinum toxin is an example of an exotoxin that acts as a neurotoxin. Food examples - *Stapylococcus aureus, Bacillus cereus, Clostridium botulinum,*
Marine and fish toxins	Biological hazard	**Shellfish:** amnesic shellfish poisoning [ASP] (domoic acid); azaspiracid shellfish poisoning [AZP] (azaspiracid); diarrhetic shellfish poisoning [DSP] (okadaic acid); neurotoxic shellfish poisoning [NSP] (brevetoxin); paralytic shellfish poisoning [PSP] (incl. C-toxins, gonyautoxins and saxitoxins) **Finfish:** ciguatera poisoning (ciguatoxin)**,** escola, oilfish poisoning (gempylotoxin)**.** puffer fish poisoning (tetrodotoxin)**; s**combrotoxic fish poisoning (histidine to histamine)**; v**enomous fish e.g. lionfish
Mold and fungal toxins	Biological hazard – some carcinogenic	Metabolites of molds and fungi that can cause illness and death. Species that produce mycotoxins include *Aspergillus*, *Cladosporium, Fusarium,* and *Penicillium.* Major toxins associated with them include aflatoxins (B1, B2, G1, G2, M1, M2), ochratoxins, patulin, trichothecenes, (such as diacetoxyscirpenol (DAS), deoxynivalenol (DON), nivalenol (NIV)), zearalenone (ZEA) and fumonisins (B1, B2, B3 in maize)
Plant toxins	Biological hazard – some carcinogenic; phytoestrogens endocrine disruption chemicals	Many plants are toxic to humans and can cause acute poisoning e.g. with alkaloids - cassava (linamarin - cyanogen); cereals, cucumber, beans, peas, soy (phytoestrogens such as coumestrol and genistein); cucumbers, courgettes/zucchini (cucurbitacins); cycad (cycasin); millet (goitrogens); mushrooms (phenylhydrazine amanitin, gyromitrin, orellanine, muscarine, ibotenic acid, muscimol, psilocybin, coprine); parsnips, parsley, carrot, celery and limes (furocoumarins); nightshade family including aubergine, green pepper, potato green sprouts and green peel (glycoalkoloids e.g. solanin); red kidney beans (lectin

		- phytohaemagglutinin); rhubarb leaves (oxalic acid);
Prion	Biological hazard	Protein that can be reproduced in cells and cause of spongiform encephalopathies such as *Creutzfeld-Jakob disease* (CJD)
Chemicals	Chemical hazard – linked to behavoral change, functional change and some suggested to be carginogenic	**Additives,** (excess foaming agents, gels, etc), **cleaning chemicals, drugs and veterinary medicines** (antibiotics, growth promoters e.g. clenbuterol, chloramphenicol, nitrofurans, hormones etc.), **crop protection products** (fungicides, herbicides, insecticides, pesticides, e.g. organophosphates) **fertilizers** (nitrates, phosphates), **food contact materials and packaging chemicals** (bisphenol A (BPA), formaldehyde, melamine and cyanuric acid, placticizers), **industrial chemicals** (e.g. benzene and other polycyclic aromatic hydrocarbons (PAHs), dioxin, furans, hexachlorobenzene (HCB), mineral oils polychlorinated biphenyls (PCBs) refrigeration fluids), **masonry biocides and treated paints**, **food preservatives** (sulphites – some allergenicity, nitrites, nitrates), **rodenticides, synthetic food colorants** (e.g. Brilliant Blue FCF, Erythrosine, Sudan 1, Tartrazine), **synthetic sweetners** (aspartame and phenylketonuria (PKU)) **wood treatment chemicals**
Metals	Chemical hazard	Metals including aluminium, antimony, arsenic, bismuth, cadmium, copper, lead, mercury, tin and iron, uranium and zinc
Radiological hazards	Chemical hazard	Examples: americium (^{241}Am), caesium (^{134}Cs, ^{137}Cs), carbon (^{14}C), cerium (^{103}Ce), cobalt (^{60}Co), hydrogen (^{3}H), iodine (^{131}I, ^{129}I), iridium (^{192}Ir), plutonium (^{238}Pu, ^{239}Pu, ^{240}Pu), potassium-40 (^{40}K), radium (^{226}Ra), ruthenium (^{103}Ru, ^{106}Ru), strontium (^{89}Sr ^{90}Sr), sulphur (^{35}S), technetium (^{99}Tc), and uranium (^{235}U, ^{236}U, ^{238}U) Some such as ^{40}K are naturally occurring.

Table 3. Families of food-borne viruses with associated clinical symptoms (Adapted from Bad Bug Book, 2012; Newell *et al.*, 2010; Bajolet and Chippaux-Hyppolite, 1990)

Genome	Family	Clinical symptoms	Notes
Double stranded DNA (dsDNA)	*Adenoviridae*	Vomiting and diarrhoea	Enteric adenoviruses cause 5% to 20% of gastroenteritis in young children. By 4 years of age, 85% of children have developed immunity. Can be transmitted by respiratory route (Bad Bug Book, 2012).
Single stranded DNA (ssDNA)	*Parvoviridae*	Vomiting and diarrhoea	Human bocavirus can cause gastroenteritis. Porcine and bovine parvovirus cause problems in the supply chain.
Double stranded RNA (dsRNA)	*Reoviridae*	Vomiting and diarrhoea	Rotaviruses cause 140 million cases of diarrhoea a year (Bajolet and Chippaux-Hyppolite, 1990)
- strand RNA	*Orthomyxoviridae*	Influenza-like illness, diarrhoea	Avian influenza e.g. H5N1, H7N9
	Paramyxoviridae	Influenza-like illness and neurological symptoms	Nipah virus (NIV) through eating raw date palm sap. Human-to-human infection has been identified.
+ strand RNA	*Astroviridae*	Vomiting and diarrhoea	
	Caliciviridae	Vomiting and diarrhoea	Norwalk-like viruses e.g. norovirus
	Coronaviridae	Respiratory disease, diarrhoea	
	Flaviviridae	Influenza-like illness, rash and neurological symptoms	Milk as been identified as a food source.
	Hepeviridae	Hepatitis	*Hepatitis E*
	Picornaviridae	Diarrhoea, rash, neurological symptoms	*Hepatitis A*

Table 4. Parasites in different foods (Newell et *al.*, 2010)

Foods	Protozoa	Nematodes	Cestodes	Trematodes
Beef	*Toxoplasma gondii* *Cryptosporidium parvum*		*Taenia saginata*	*Fasciola hepatica*
Pork	*Toxoplasma gondii*	*Trichinella* spp.	Taenia solium/asiatica	
Other meat	*Toxoplasma* *Cryptosporidium* (sheep/goat)	*Trichinella* spp. (horse, wild boar bear, walrus, crocodile, *Gnathostoma* (frogs)	*Alaria alata* (wild boar)	*Paragonimus* (wild boar)
Milk	*Toxoplasma* *Cryptosporidium*			
Fish/squid		*Anisakis spp.* *Gnathostoma*	*Diphyllobothrium*	*Clonorchis* *Opisthorchis*
Crabs, shrimps		*Gnathostoma*		Paragonimus
Shell fish	*Cryptosporidium spp.* *Giardia lamblia* *Toxoplasma gondii*	*Gnathostoma*		*Echinostomes*
Snails/slugs		*Angiostrongylus*		*Echinostomes*
Fruit/vegetables (raw)	*Cyclospora* *Cryptosporidium spp.* *Giardia lamblia* *Toxoplasma gondii* *Entamoeba histolytica* *Balantidium coli* *Trypanosma cruzi*	*Angiostrongylus* *Ascaris* *Toxocara* *Baylisascaris spp* *Trichuris trichiura*	*Echinococcus* *Taenia solium*	*Echinostomes* *Fasciola hepatica* *Fasciolopsis*
Water	*Cyclospora* *Cryptosporidium* *Giardia lamblia* *Toxoplasma gondii* *Balantidium coli*	*Ascaris*	*Echinococcus*	*Fasciola* *Fasciolopsis*

Table 5. Common foods and associated allergens (Adapted from Walsh *et al.*, 1988; Maleki et al., 2003; Caubet and Wang, 2011; Mortimore and Wallace 2013; Shaw 2013; WHO/IUIS, 2014)

Food	Animal or plant species	Molecule (Allergen)
Celery	*Apium graveolens*	(Api g 1); (Api g 2); (Api g 3); Profilin (Api g 4); (Api g 5); (Api g 6)
Cereal (e.g. wheat)	*Triticum aestivum*	Profilin (Tri a 12); (Tri a 14); Agglutinin isolectin 1 (Tri a 18); Omega-5 gliadin (Tri a 19) Gamma gliadin (Tri a 20); Thioredoxin (Tri a 25); (Tri a 26); (Tri a 36)α-purothionin (Tri a 37)
Cow's milk	*Bos domesticus*	α-Lactalbumin (Bos d 4); β-Lactoglobulin (Bos d 5); Serum albumin (Bos d 6); Immunoglobulin (Bos d 7); Caseins (Bos d 8); α-S1-casein (Bos d 9); α-S2-casein (Bos d 10); β-casein (Bos d 11); K-casein (Bos d 12)
Egg	*Gallus domesticus*	Ovamucoid (Gal d 1); Ovalbumin (Gal d 2); Ovatransferrin (Gal d 3); Lysosyme C (Gal d 4) α-Livetin (Gal d 5) – can also cause a cross reaction with poultry meat; Phosvitin (Gal d 6); Apovitellenins I (Gal d Apo I); Apovitellenins VI (Gal d Apo VI)
Fish/Shellfish (some examples)	*Metapenaeus ensis* (shrimp) *Gadus callarius* (Baltic cod) *Gadus morhua* (Atlantic cod) *Salmo salar* (Atlantic salmon) *Penaeus aztecus* (brown shrimp) *Charybdis feriatus* (crab) *Todarodes pacifus* (squid)	Tropomyosin (Met e 1); β-parvalbumin (Gad c 1); β-parvalbumin (Gad m 1); β-enolase (Gad m 2); Aldolase A (Gad m 3); β-parvalbumin (Sal s 1); β-enolase (Sal s 2); Aldolase A (Sal s 3) Tropomyosin (Pen a 1) Tropomyosin (Cha f 1) Tropomyosin (Tod p 1) Chitinase may be an allergen
Legumes	*Glycine ussuruensis* (soy)	Glycinin (Gly m 1); Defensin (Gly m 2); Profilin (Gly m 3); (Gly m 4); Vicilin (β-Conglycinin); (Gly m 5); Glycinin (Gly m 6); (Gly m 7); 2S albumin (Gly m 8)
Lupin	*Lupinus angustifolius*	Conglutin beta (Lup an 1)
Mustard	*Sinapis alba*	2S albumin (Sin a 1); 11S globulin (Sin a 2); (Sin a 3);

		Profilin (Sin a 4)
Peanut	*Arachis hypogaea*	Cupin Vicilin like (Ara h 1) causes severe reaction in those with a peanut allergy including anaphylactic shock; Conglutinin (Ara h 2) inhibits digestive enzyme trypsin; Cupin Legumin-type (Ara h 3); (Ara h 4) renamed Ara h 3.02; Profilin (Ara h 5); Conglutin (Ara h 6) (Ara h 7); (Ara h 8); (Ara h 9); (Ara h 10) (Ara h 11); Definsin (Ara h 12) ;(Ara h 13)
Tree nuts (some examples)	*Juglans regia* (walnut) *Anacardium orientale* (cashew)	2S albumin (Jug r 1); Vicilin (Jug r 2); (Jug r 3); 11S globulin (Jug r 4); Vicilin (Ana o 1); Legumin (Ana o 2); 2S albumin (Ana o 3)
Apple	*Malus domestica*	(Mal d 1); Thaumatin-like protein (Mal d 2); (Mal d 3) ; Profilin (Mal d 4) – Profilin (Bet v 2) is a birch derived allergen – thus suggesting the link with cross-sensitivity as identified in the text.

This table is not designed to be an exhaustive list, but to give an indication of the complexity of food allergen classification.

Table 6. Functional and structural food intolerance (Adapted from Zopf *et al.*, 2009 and Wang *et al.*, 2011)

<table>
<tr><th colspan="3">Functional food intolerance</th><th>Structural food intolerance</th></tr>
<tr><th>Non-toxic</th><th colspan="2">Toxic</th><th rowspan="2">Non-toxic</th></tr>
<tr><th>Non-immunological</th><th>Immunological (allergy) with immune response</th><th>Toxin</th></tr>
<tr><td>Enzymatic e.g. Lactase (lactose), galactase (galactase), diamine oxidase (histamine)</td><td>Immunoglobulin E (IgE) mediated food allergy e.g. peanut, tree nut, soy, minutes to hours before symptoms start can lead to anaphylaxis</td><td rowspan="5">See Table 2 for agents that cause intoxication</td><td>Oesophagus: Achalasia, stricture</td></tr>
<tr><td>Transport defect e.g. GLUT 5 transport defect (fructose) or GLUT 2 (glucose, galactase, and fructose transport)</td><td rowspan="2">Non IgE antibody related food allergy symptoms take longer to develop e.g. symptoms such as atopic eczema, gastro-oesophageal reflux disease, colic, constipation</td><td>Stomach: resection</td></tr>
<tr><td>Pharmacological – related to a specific food ingredient or food – often with a particular trigger dose e.g. migraine</td><td>Gallbladder: fat malabsorption, obstructive jaundice</td></tr>
<tr><td rowspan="2">Pseudo-allergies - activation of inflammatory or anaphylactic mechanisms independent of antigen-specific immune responses.</td><td rowspan="2">IgE and non IgE related food allergy – some allergens can cause either or both allergic reactions e.g. cows milk</td><td>Pancreas: chronic pancreatitis</td></tr>
<tr><td>Small intestine: infection, diverticulum, small intestine bacterial overgrowth (SIBO)</td></tr>
<tr><td>Idiosyncrasy (food additives) – abnormal response to a food additive that resembles</td><td>Other single or combination of antibody types e.g. celiac disease - tissue transglutaminase antibodies (tTGA), endomysial antibody (EMA) and</td><td>Large intestine: chronic inflammatory bowel disease (CIBD), diverticulum, surgical sequelae</td></tr>
</table>

hyperactivity e.g. tartrazine, sunset yellow	Immunoglobulin A (IgA) Immunoglobulin G (IgG)		
	Infections such as lambliasis, enterohaemorrhagic Escherichia coli (EHEC) enterohaemorrhagic Escherichia coli;		**Vascular and lymphatic**: abdominal angina, lymphangietasia, right ventricular failure

Table 7. Food related illness (Adapted from Wallace *et al.*, 2011; Bad Bug Book, 2012; Caballero, 2013; Mortimore and Wallace, 2013; Shaw 2013; Liu *et al.*, 2014: Sprenger 2014)

Disease or health-related agent	Traditional classification[1]	Examples
		Infection
Viruses (see Table 3)	Biological hazard	Examples: Norovirus, Hepatitis A, Rotavirus, Astroviruses, Sapoviruses, Enteric adenoviruses, Bocaviruses, Aichi virus – some viruses can produce toxin like proteins but in the research no literature found to associate viral toxins with food borne illness.
Parasites and protozoa (see Table 4)	Biological hazard	Parasites have complex life cycles and are all zoonoses. Parasite food-borne illness is common in developing countries and ranges from single-celled to highly adapted animals. Protozoa include, *Cryptosporidium parvum, Cyclospora cayetanensis, Entamoeba histolytica*, (causes dysentery) *Giardia spp., Sarcocystis, Toxoplasma gondii* **Flatworms (Platyhelminthes including cestodes (tapeworms), Trematodes (flukes))** Fish tapeworms *Diphylloborthrium spp.* Beef tapeworm *Taenia sagitata*, Pork tapeworm *Taenia solium*, Liver fluke, *Fasciola hepatica* cause fascioliasis **Nematodes –thread or round worms** Fish nematodes – *Anisakis sp.* causes anisakiasis**;** Giant roundworm *Ascarius lumbriciodes***;** *Trichinella sp.* causes trichinosis associated with pork; Whipworm –*Trichuris trichhiura* **Trematodes** Fish fluke *Clonorchis sinensis*
		Intoxication
Toxic agents in food (see Table 2)	Biological and Chemical hazards	**Biological hazard** including bacterial endotoxins, bacterial exotoxins, marine and fish toxins, mould and fungal toxins, plant toxins and prions. **Chemical hazards** including acute and chronic agents such as metals, radiological hazards, and chemicals linked to behavoral change, functional change and some potential carcinogens.
Toxic functional food intolerance (see Table 6)	Biological and chemical or as a sole category	Immunological (allergy) with an immune response. These can be IgE mediated, Non IgE antibody related food allergy or single or combination of antibody types. Examples of foods that give rise to this response include celery, cereals, cow's milk, egg, fish, shellfish, legumes, lupin, mustard, peanut, tree nuts and apple.
Salt	Not classified	Adverse health effects include impaired cognitive function, enhanced oxidative stress in the hippocampus which can lead to

		neurodegenerative disease such as Parkinson's Disease, Alzheimer's disease, blood pressure, heart disease, stroke.
Sugar	Not classified	Adverse health effects include excess caloric intake, dental caries, excess weight gain, dilutional effects of essential nutrients, and increased risk for dental caries, type 2 diabetes, and cardiovascular diseases.
Fat	Not classified	Adverse health effects include overweight, increased risk of coronary heart disease, high blood pressure
Alcohol	Not classified	Accumulative toxin that can cause increased risk of heart attack and cancer, depression and addiction, liver problems, reduced fertility, high blood pressure
Caffeine	Not classified	Adverse health effects include headache, nausea, anxiety, addiction,
Foreign bodies		
Intrinsic or extrinsic foreign bodies	Physical hazards	Examples: bones, ceramic, glass, metal (jewellery, bolts, packaging, nails, wire etc.), pest material (insects, body parts), plastic (hard and soft), seeds, shells, soil, stones, stalks, wood
Non-toxic intolerance		
Non-toxic functional food intolerance (see Table 6)	Not classified	Examples include enzymatic deficiency, transport defect, pharmacological, pseudo-allergies and idiosyncrasy e.g. an abnormal response to an additive that causes hyperactivity.
Nont-oxic structural food intolerance (see Table 6)		Examples can be associated with the oesophagus, stomach, gallbladder, pancreas, small intestine, large intestine or vascular and lymphatic systems.

[1]Biological, chemical and physical hazards as per CAC (2003)

Steps of risk assessment

1: Determine the realistic agent(s) and assess each in turn.

2: Identify the likelihood, and the severity of the agent should it arise.

3. Determine whether additional controls need to be put in place to reduce likelihood of occurrence, and implement if required.

4. Redefine the likelihood of the agent should it arise based on additional controls that have been put in place.

5. Rank risks according to importance and these identify the priority agents of concern

6. Verify that the controls are effective then at a defined frequency revisit steps 1-5 again.

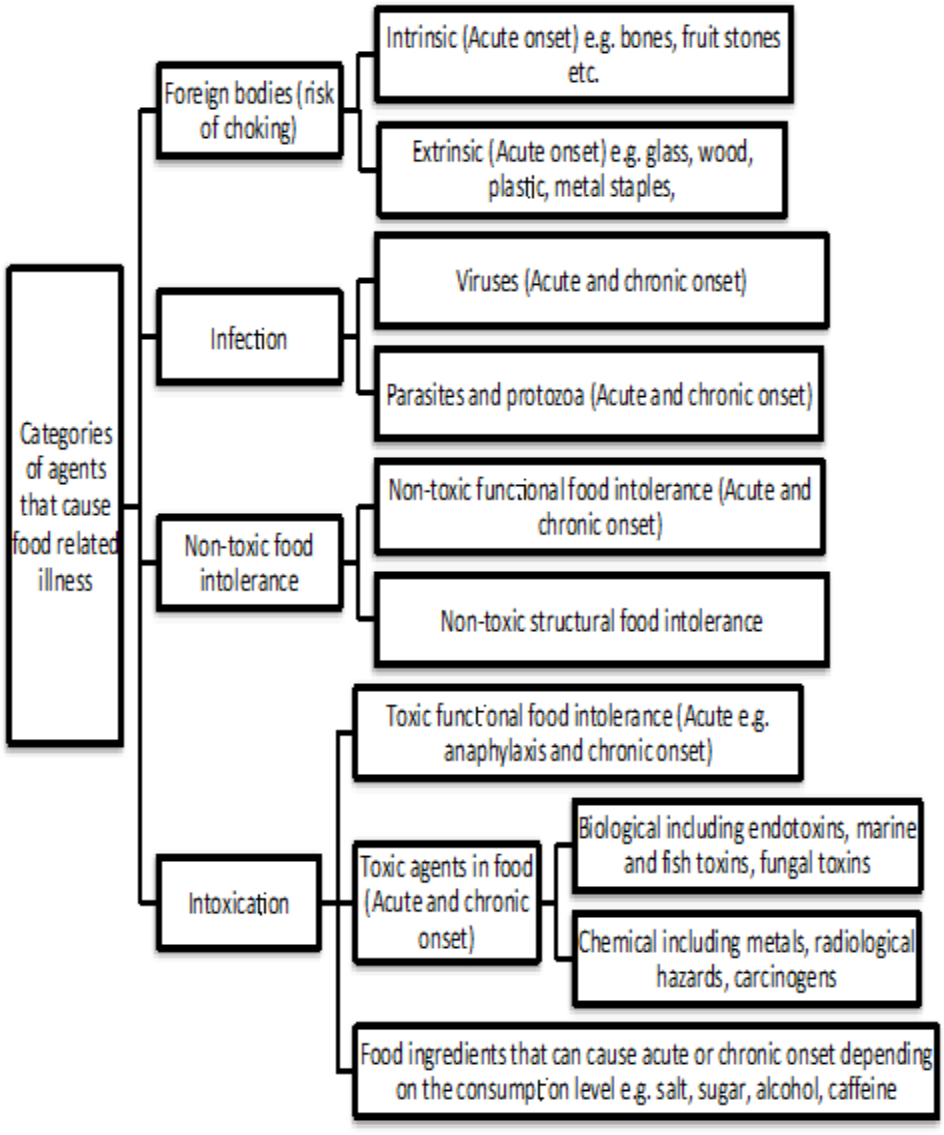

Figure 1. Food related illness risk assessment