The Singapore Dietitian

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Dietary concerns associated with the use of medications.

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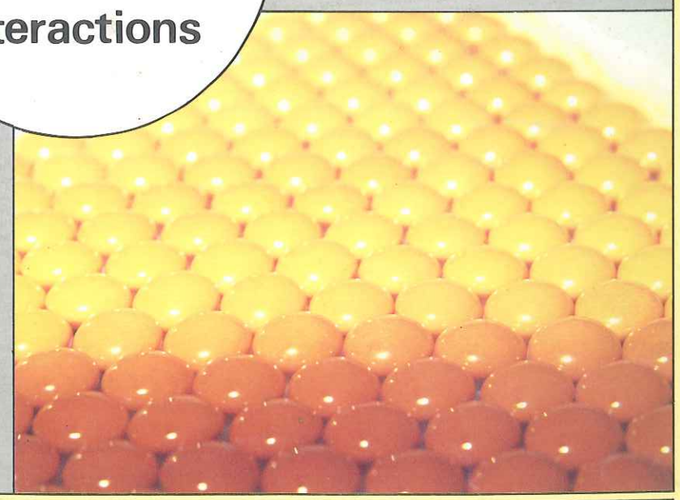
Nutrient and Drug Interactions



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VOL. 1 No. 2 March 1986

From the President .....

Greetings! Recession may be the word everyone is talking about in 1986, but 9 am pleased to say that **growth** *is the theme of my address. The first issue of the journal was a huge success, prompting the Editorial Committee to increase the circulation of the second issue from 1000 to 2500 copies.*

In this issue we feature the interactions of nutrients and drugs, an important overlap area of dietetics and pharmacology often given little attention. We also have two original articles by local authors, and a special report by one of our members who recently attended the Congress of the Asean Federation of Endocrine Societies in Kuala Lumpur.

Since I last wrote, the Association has been growing steadily, with the addition of several full and affiliate members. Our scope of activities has also expanded considerably. One project we will be involved in during 1986 is the production of a series of six articles on therapeutic diets for The Sunday Times. We are also pleased to be joining with fellow-paramedical professionals in the organization of a nationwide careers week, a brainchild of the Singapore Physiotherapy Association. Service to the community is our main aim in these projects, but publicity, and therefore enhanced recognition of our profession, will be a valuable spin-off.

It is my sincere hope that new members will come forward to volunteer their assistance in our various activities. We are a small group, but if we each play our part we can be very effective.

It remains for me to remind all members that our AGM and Annual Dinner will be coming up in April – see you all there!

Mrs Fatimah Lee
President
Singapore Dietitians' Association

CONTENTS

Dietary concerns associated with the use of medications

Summary

Dietary concerns associated with the use of pharmacological agents are many. Drugs can influence nutrient absorption, metabolism, or excretion; these effects may then alter nutritional status. On the other hand, specific nutrients, foods, or beverages may interact with drugs in the gastrointestinal tract or affect drug metabolism, action, or excretion. Such interactions, including dietary changes, may then influence drug response.

The use of alcohol or vitamin, mineral, or other nutritional supplements, as well as drug dosage and duration of therapy, should be examined as part of the potential interactions among food, nutrients and drugs.

The impact of nutritional and environmental substances on the basic processes of drug or nutrient disposition is complex and difficult to predict. Yet, even though many interactions may not be clinically significant at the present time, evidence suggests that nutrition is an important determinant of drug efficacy. Dietary modifications need to be considered for patients whose response to drugs is not within the expected range or who are at nutritional risk.

Drug effects on nutrient absorption

Drugs commonly influence nutrient disposition through their effects on absorption, metabolism, and excretion of nutrients. Table 1 represents a sampling of drug effects on nutrient absorption and includes dietary suggestions associated with the use of specific medications.

The loss of a variety of nutrients may result from prolonged use or overuse of laxatives because of drug-induced hyperperistalsis (bisacodyl, phenolphthalein) or the trapping of fat-soluble nutrients in the laxative itself (mineral oil). Any patient who self-prescribes laxatives for perceived constipation should be informed about the potential of developing laxative-dependent constipation and the importance of diet, fluid intake, and other factors that may aid in the relief of constipation (1).

Drugs can also interfere with the absorption of specific nutrients.

Drugs can influence nutrient absorption, metabolism or excretion; on the other hand, foods or nutrients may interact with drugs affecting their metabolism, action or excretion.

Table 1. Dietary suggestions associated with drugs which alter nutrient absorption*

drug (usage)	nutritional implications: gastrointestinal side/adverse effects	nutritional implications: other reactions	dietary suggestions
aluminum hydroxide gel (antacid, phosphate binder)	bloating constipation fecal impaction nausea/vomiting stomach cramps	phosphate malabsorption hypophosphatemia vitamin A malabsorption thiamin destruction loss of appetite unpalatable (chalky)	Ulcer therapy–Drug: Take between meals. Chew chewable tablets until thoroughly wetted, then follow with 125 ml water. Diet Rx: Teach dietary principles associated with treatment of ulcer disease. Phosphate-binding therapy–Drug: Take at mealtime with 250 ml water or fluids indicated for patients with renal disease. Diet Rx: Dietary phosphate restriction may be prescribed. If permissible, include a high-bulk diet to counter constipation.
bisacodyl (laxative)	belching mild cramping diarrhea nausea	fluid and electrolyte loss hypokalemia (long use)	Drug: Take on empty stomach with at least 250 ml water and at least 1 hr away from milk. (Milk may dissolve enteric coating, causing gastric irritation.) Swallow tablet whole (do not chew or crush). Diet Rx: Drink at least 6 to 8 glasses of 250 ml fluid/day to aid stool softening. Teach the importance of diet, increased fluid intake, and exercise.
cholestyramine (antihyperlipemic, bile acid sequestrant	belching bloating constipation flatulence heartburn nausea/vomiting steatorrhea stomach pain	malabsorption of fat, iron, carotene, vitamins A, D, and K, and folacin hypoprothrombinemia gritty texture unpalatable taste	Drug: Thoroughly hydrate drug with at least 120 to 180 ml water, milk, fruit juice, or non-carbonated or other beverages prior to ingestion; disguise gritty texture and unpalatable taste by mixing with highly flavored liquids, thin soups, milk in cereals, or pulpy fruits (applesauce, crushed pineapple). Diet Rx: If permissible, include a high-bulk diet with increased fluid intake to counter constipation. During long-term therapy, supplementation with a water-soluble (or parenteral) form of vitamins A and D may be prescribed. Parenteral or oral administration of vitamin K may also be considered if hypoprothrombinemia occurs. Folacin supplementation may be prescribed for patients with reduced serum or red cell folacin.
colchicine (antigout)	diarrhea (may be severe) nausea/vomiting abdominal pain	malabsorption of sodium, potassium, fat, carotene, and vitamin B-12 due to altered mucosal function decreased lactase activity loss of appetite	Drug: Take with water immediately before, with, or after meals to reduce gastric irritation. Diet Rx: May indicate increased intake of alkaline-ash foods or beverages and low-purine foods, no alcoholic beverages, and a high fluid intake of > 2,000 ml/day.
mineral oil (laxative)	flatulence indigestion nausea/vomiting (with long use)	malabsorption of carotene, vitamins A, D, E, and K, calcium, and phosphorus tasteless and odorless (when cold) disagreeable consistency loss of appetite, weight loss, and hypokalemia (with long use)	Drug: Take at least 2 hr away from food (to avoid delayed digestion and movement of chyme from stomach). May mix with or follow by orange juice to counter consistency. Diet Rx: See bisacodyl. Concurrent use with fat-soluble vitamins may interfere with vitamin absorption. Do not use in the preparation of low-calorie salad dressings.
phenolphthalein (laxative)	(see bisacodyl)	malabsorption of vitamin D, calcium, and other minerals hypokalemia (long use) may be excreted in breast milk	Drug: Take on empty stomach. Chew gum well. (Do not swallow gum.) Diet Rx: See bisacodyl.
sulfasalazine (anti-inflammatory)	diarrhea gastric distress nausea/vomiting	impaired folacin absorption loss of appetite excreted in breast milk	Drug: Take with 250 ml water or after meals or with food to minimize gastric irritation. Diet Rx: Ensure adequate fluid intake to maintain at least 1,200 to 1,500 ml urine output/day. Encourage the intake of foods high in folacin.

*General references (4, 12-16)

Table 2. Dietary suggestions associated with drugs which alter nutrient metabolism/ excretion*

drug (usage)	nutritional implications: gastrointestinal side/adverse effects	nutritional implications: other reactions	dietary suggestions
hydralazine (antihypertensive)	diarrhea nausea/vomiting constipation (rare)	vitamin B-6 antagonism (may result in peripheral neuropathy) sodium and water retention (long-term therapy) loss of appetite	Drug: Intake with food may increase bioavailability of drug. Consistently take with food. Diet Rx: If appropriate, teach and emphasize the importance of dietary modifications associated with treatment of hypertension. May also indicate a sodium-restricted diet, weight reduction and monitoring, and alcoholic beverage restriction. Vitamin B-6 supplementation may be prescribed if symptoms of peripheral neuropathy develop.
isoniazid (antitubercular)	epigastric distress nausea/vomiting (may be signs of hepatotoxicity)	vitamin B-6 antagonism (may result in peripheral neuropathy) tyramine-type reactions with certain foods dry mouth loss of appetite excreted in breast milk	Drug: Take with 250 ml water on an empty stomach, as food decreases drug absorption. If gastrointestinal irritation occurs, drug may be taken with food to lessen that effect. Diet Rx: May indicate avoidance of alcoholic beverages and foods high in pressor amines. Vitamin B-6 supplementation may be prescribed for the malnourished or for those patients predisposed to vitamin B-6 deficiency or exhibiting signs of peripheral neuropathy.
methotrexate (antineoplastic, antipsoriatic)	abdominal distress diarrhea GI ulceration and bleeding nausea/vomiting	folacin antagonist (irreversibly binds with dihydrofolate reductase) malabsorption of folacin, vitamin B-12, and fat hyperuricemia loss of appetite altered taste acuity sore mouth and lips	Drug: To reduce nausea and to help foster compliance. Absorption may be decreased by milky meals. See Table 3. Diet Rx: May indicate increased intake of alkaline-ash foods and beverages and the ingestion of ~2,000 ml water/day to aid in excretion of uric acid. Patients should avoid the use of alcoholic beverages. Caution patient against self-medication with OTC preparations, especially the use of supplements containing para-aminobenzoic acid and folacin. See Table 5.
phenobarbital (anticonvulsant, sedative-hypnotic)	nausea/vomiting	hepatic microsomal enzyme induction increases inactivation of 25-OH vitamin D and may result in rickets or osteomalacia may decrease serum folacin, vitamin B-12, pyridoxine, calcium, and magnesium appetite changes excreted in breast milk	Drug: Swallow extended-release tablet whole. Take oral solution straight or mix with water, milk, or juice. Diet Rx: Emphasize the importance of good dietary habits with adequate intake of vitamin D-containing foods. Serum folacin, calcium or 25-OH vitamin D levels as well as indexes of bone resorption may be monitored in patients on prolonged therapy (especially children and those concomitantly prescribed phenytoin) prior to vitamin D supplementation. Avoid alcohoic beverages.
furosemide (potassium-depleting diuretic)	constipation (or diarrhea) nausea/vomiting stomach distress	enhances the excretion of potassium, calcium, magnesium, sodium, chloride, and water fluid and electrolyte disturbances dry mouth increased thirst loss of appetite excreted in breast milk	Drug: Intake with food may slow the rate of drug absorption without altering the bioavailability of drug. To minimize the effect of increased urinary output at night, take single daily dose early in the morning. Diet Rx: If appropriate, teach and emphasize the importance of dietary modifications associated with treatment of hypertension. May also indicate the need for a sodium-restricted diet, a high intake of potassium and magnesium-rich foods (especially in patients taking digitalis), weight reduction and monitoring, and alcoholic beverage restriction. Limit the intake of natural licorice. See Table 4.
indomethacin (anti-inflammatory, analgesic)	bloating constipation (or diarrhea) heartburn/ indigestion nausea/vomiting stomach pain ulcerogenic potential	sodium and fluid retention (mild) weight gain (edema) excreted in breast milk	Drug: Even though food may slightly delay or reduce absorption, take drug after meals or with food to reduce gastric irritation. (An antacid may be prescribed.) Diet Rx: Inform patient to avoid alcoholic beverages. Even though salt and fluid retention effects are less pronounced than with phenylbutazone, a sodium-restricted diet may be indicated.

*General references (4, 12-16)

Damage to the intestinal mucosa by drugs is one mechanism that causes nutrient malabsorption. Colchicine, an anti-inflammatory agent used to treat gout, arrests cell mitosis and can result in a loss of lactase activity due to an altered mucosal structure.

Drug effects on nutrient metabolism and excretion

Altered cellular metabolism can result from drug-induced vitamin antagonism or by induction of enzyme systems through which the vitamins are converted to their coenzyme forms and degraded.

Other nutrient-drug interactions worthy of special notice include the effect of diuretics on mineral excretion. Potassium-depleting diuretics (furosemide, thiazide) enhance the excretion not only of potassium but also of magnesium. With either of those drugs, sodium-restricted diets and foods rich in potassium are usually prescribed. (However, caution needs to be exercised in prescribing a high potassium intake for patients with renal impairment.) Patients should also be encouraged to maintain adequate intake of magnesium-rich foods. Similar dietary suggestions are important for patients who are being concomitantly treated with digitalis; either hypokalemia or hypomagnesemia may predispose the patient to digitalis toxicity (2).

However, not all patients treated with diuretics need to consider the importance of potassium-rich foods in their diet; some diuretics (spironolactone) spare potassium. Because of common knowledge associated with diuretic therapy (potassium-depleting), patients treated with other types of diuretics (potassium-sparing) or diuretic combinations may not be aware that mineral requirements vary depending on the action of the drug. Patients need to be counselled about the importance of their personal diet prescription and cautioned about following the dietary advice of others.

Drug-induced fluid or electrolyte disturbances

Sodium or water retention is another undesirable effect associated with the use of various medications, such as steroids and antihypertensive and anti-inflammatory agents. Even though the resulting weight gain (edema) may be treated by reducing dietary intake of sodium, in some instances concomitant diuretic therapy may be administered to counter the effect.

The adrenal corticosteroids have many potential adverse side effects, the incidence of which correlates with duration of therapy, dose, dose schedule, age, underlying illness, and other factors. Fluid and electrolyte disturbances can be treated by a sodium-restricted diet and consumption of potassium-rich foods. Although weight gain may be related to fluid retention, it is important to monitor food intake, since an increased appetite has been associated with glucocorticoid therapy. Long-term use of large doses of glucocorticoids may also cause significant bone loss (osteoporosis), an effect partly related to drug-induced anti-vitamin D activity that affects calcium absorption. Glucocorticoid-induced gluconeogenesis may also result in negative nitrogen balance, an effect that may be somewhat modified by increasing the intake of dietary protein (3, 4)

Varying degrees of fluid retention with weight gain secondary to sodium retention have been associated with several antihypertensive agents (clonidine, guanethidine, hydralazine, methyldopa).

Another aspect of this topic is the interaction between drugs and foods, the effects of which can alter drug disposition.

Food (or nutrient) effects on drug absorption

Examples of foods, beverages, and vitamin, mineral, and other supplements that may affect drug disposition

Table 3. Effects of various foods and beverages on drug absorption*

food or beverage	drug	effect
coffee and tea	neuroleptic agents (fluphenazine, haloperidol)	Mixing drug with coffee or tea can precipitate the drug, prevent absorption, and impede its therapeutic effects.
fiber	digoxin	May reduce drug absorption (17).
bran pectin(?) or high-carbohydrate meal	acetaminophen	May depress rate of drug absorption (18).
food (in general)	chlorothiazide	May increase drug absorption (19).
	propranolol	May increase drug absorption (20).
	nitrofurantoin	Increases bioavailability of the drug.
	cimetidine	Delayed absorption may benefit patient by maintaining blood concentration of drug between meals (21).
	aspirin	May decrease drug absorption and absorptive rate (22).
	antimicrobial agents (cephalexin, penicillin G erythromycin stearate, penicillin V, tetracycline)	May reduce drug absorption.
high-fat meal	griseofulvin	Increases drug absorption.
high-protein diets	levodopa, methyldopa	Amino acids from dietary protein inhibit absorption of drugs.
milk and milk products	tetracycline	Calcium inhibits drug absorption.
milky meal+	methotrexate	May inhibit drug absorption (23).

*Modified from Smith and Bidlack (5).
+Milky meal contained milk, corn flakes, white bread, and sugar.

are shown in Tables 3, 4, and 5. The tables have been modified and updated from a recent review on this topic (5).

It is interesting to note that in some instances, food or a particular component in food enhances drug absorption. Griseofulvin, a drug used to treat fungal infections, is best absorbed after a meal high in fat. Food in general has also been shown to enhance the absorption of chlorothiazide (diuretic, antihypertensive), propranolol (antiarrhythmic, antihypertensive), and nitrofurantoin (antibacterial).

Food, various beverages, and specific nutrients can also have a variety of undesirable effects on drug action (Table 4). Whether or not caffeine can cause a large variation in plasma concentration of neuroleptic agents, as it is thought to do (6), caffeine is a central stimulant which, if ingested in excessive amounts by psychiatric patients, may effect the clinical effectiveness of neuroleptic drugs (7). Also, patients who consume large amounts of coffee or other xanthine-containing beverages while being treated with theophylline (bronchial dilator) may be at risk of developing enhanced drug side effects that apparently result from the similar metabolic actions of those compounds.

Of interest, too, is the potential effect of an excessive intake of alkaline-ash beverages (citrus juices) on quinidine (antiarrhythmic) excretion. The resulting increase in urinary pH is believed to be responsible for increasing the proportion of un-ionized quinidine, thus enhancing renal reabsorption of the drug (8, 9).

Licorice has long been known to cause sodium retention, with edema and hypertension, and has also been shown to cause significant hypokalemia in a patient who had been eating 1.8 kg licorice sweets per week (10). Because of those effects, ingestion of large quantities of natural licorice or licorice extracts containing glycyrrhizic acid may complicate treatment in patients receiving antihypertensive agents. Hypokalemia can also be hazardous in patients treated with digitalis preparations, as concomitant intake with licorice may result in drug intoxication.

When vitamin, mineral, and dietary supplements are indiscriminately used, a variety of undesirable effects on drug disposition can occur (Table 5). Such supplements are widely used by the public with little thought given to potential interactions with drugs. Some of the supplements are complex mixtures that are considered harmless by the public and are many times ingested in large quantities without the knowledge of a physician or a dietitian.

Although nutritional supplements play an important role in the management of debilitated patients, physicians and other medical practitioners need to be aware of the nutritional components in the various products and their potential interaction with drugs. Michaelson et al. (29) reported a case in which the explanation for warfarin resistance was found to be the ingestion of large amounts of a nutritional supplement containing vitamin K.

Even though mineral (especially calcium) interactions with tetracycline are well known, patients being treated with calcium, magnesium, zinc, or iron supplements need clarification in relationship to time of administration of the drug and the supplement. Such interactions do not imply that drug doses should be missed (or for that matter that milk or milk products should be eliminated from the diet), only that the drug should be taken at different times than the supplements.

The intake of other dietary supplements, such as tryptophan, PABA (para-aminobenzoic acid), protein or amino acid supplements, or yeast concentrates, may cause serious problems in some drug users. Patients prescribed monoamine oxidase inhibitors should be warned to avoid various yeast extracts or products made with them because use of such compounds (as well as foods) with a high pressor amine (especially tyramine) content may cause significant hypertension. A complete list of foods, beverages, or supplements containing pressor amines should be given to any patient for whom monoamine oxidase inhibitors are prescribed. Patients also need to understand the importance of avoiding the use of self-prescribed supplements or other medications.

Table 4. Effects of various foods and beverages on drug action*

food or beverage	drug	effect
beverages		
coffee, tea, and other caffeine-containing beverages	theophylline	Increased intake may enhance drug side effects (nervousness, insomnia).
	neuroleptic agents	Increased intake may result in a large variation in plasma concentration of drug and may reduce its clinical effectiveness.
citrus juices	quinidine	Excessive intake may increase blood levels of drug (alkalinization of urine).
licorice	antihypertensive agents, diuretics	Glycyrrhizic acid in natural licorice tends to induce hypokalemia and sodium retention; ingestion in large amounts may complicate antihypertensive drug therapy.
	digoxin	Licorice-induced hypokalemia may enhance the action of digitalis and result in drug toxicity.
protein or charcoal-broiled meats	theophylline	High protein/low carbohydrate diet or ingestion of charcoal-broiled meats may decrease plasma half-life of drug (24, 25).
salty foods, sodium (salt)	lithium	Increased intake of sodium may reduce therapeutic response to drug. Low-salt diets may enhance drug activity (26).
boiled or fried onions	warfarin	May increase fibrinolytic activity of drug (27).
broccoli, turnip greens, lettuce, cabbage	warfarin	Vegetables rich in vitamin K may inhibit hypoprothrombinemic response to oral anticoagulants.

*Modified from Smith and Bidlack (5).

Table 5. Vitamins, minerals, and other supplements which affect drug action*

supplement	drug	effect
vitamins	alcohol	Hypervitaminosis A may enhance hepatotoxicity of alcohol (28).
vitamin A	isotretinoin	Additive toxic effects may result from combination therapy with vitamin A or other supplements containing vitamin A.
	tetracycline	Combination therapy may enhance drug-induced intracranial hypertension (severe headache).
vitamin D	digoxin	Vitamin D-induced hypercalcemia may potentiate the effects of the drug and result in cardiac arrhythmias (29).
vitamin E	warfarin	May enhance anticoagulant response to warfarin.
vitamin K	warfarin	Vitamin K in liquid food supplements may inhibit the hypoprothrombic effect of drug.
ascorbic acid	fluphenazine	Large doses may interfere with drug absorption and result in a return of manic behavior (30).
	warfarin	Megadoses may decrease prothrombin time.
folacin	methotrexate	Folacin or its derivatives in vitamin preparations may alter responses to drug.
	phenytoin	May decrease anticonvulsant action of drug.
pyridoxine	levodopa	Reverses the antiparkinsonism effect of drug (31).
	phenytoin	Large doses may reduce phenytoin levels (31).
	hydralazine, isoniazid, penicillamine	May correct drug-induced peripheral neuropathy.
minerals		
calcium, iron, magnesium, zinc	tetracycline	Concurrent use may decrease drug absorption.
iron	penicillamine	Concurrent use may decrease drug effectiveness (32).
other supplements		
para-aminobenzoic acid	methotrexate	?May increase toxicity by displacing drug from plasma protein binding (in vitro study).
	pyrimethamine	?May interfere with drug action against toxoplasmosis.
protein or amino acids	levodopa, methyldopa, theophylline	?May inhibit drug absorption. ?May decrease plasma half-life of drug.
tryptophan	monoamine oxidase inhibitors	May cause a deterioration in mental status (33).
yeast extracts	monoamine oxidase inhibitors	Concomitant intake may produce significant hypertension.

* Modified from Smith and Bidlack (5).

Gastrointestinal and other effects

Most drugs at one time or another have probably caused gastrointestinal disturbances, appetite changes, dry or sore mouth, or altered taste perceptions. Dietary suggestions may aid patients experiencing unpleasant drug side effects (12). It is also important to be aware of possible nutritional (and medical) implications for drugs which have unpleasant side effects. Patients in distress may decide on their own to (a) alter dietary habits by reducing or eliminating certain foods from the diet, (b) not eat, (c) use over-the-counter drugs to aid in the relief of the disturbance, or (d) discontinue the use of the drug. Any one of the options can further compromise nutritional (and medical) status or result in altered drug disposition. Either effect poses a risk to the patient.

Conclusion

In the many situations discussed here, various dietary suggestions must be interpreted in relationship to age-related nutrient requirements, dietary habits, sex, reproductive status, need for therapeutic diets, and the underlying illness, which can also alter nutrient requirements. The clinical significance of many of the interactions is obscure, and additional research is needed to clarify drug-induced nutritional changes.

It is important, too, to focus on food habits. Many individuals have strange ideas about the benefits of certain foods, diets, or dietary supplements. Attention must be given to amount and frequency of use of certain foods, beverages, and food or nutritional supplements, as various components in them can affect drug absorption, metabolism, action, or excretion. Also, if patients have been stabilized on a drug while hospitalized, they should be questioned about typical food habits outside the medical setting.

There is generally no justification for assuming that nutrient supplementation is needed on the basis of drug therapy alone. Laboratory data are needed to verify possible changes in nutritional status. A nutritious diet, however, makes an important contribution to the health of drug users and reduces the risk of nutritional disorders or altered drug efficacy. Even though the frequency of adverse effects arising from food-induced drug changes or altered nutritional status is in need of further clarification and consideration, an understanding of reported and potential interactions is a step forward in preventing undesirable and often serious drug effects.

Abstracted from a review by Christine Hamilton Smith, PhD. RD and Wayne R. Bidlack PhD, in J. Am. Diet. Assoc. 84, 8:901 – 914, 1984.

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Are There Carcinogens In The Traditional Singaporean Cuisine?

M. McCabe and Lim Ngah Swan

Singaporeans are justifiably proud of that spicy mix of diet generated originally from Indian, Chinese and Malay traditions of preparing food. Indeed ones only obvious point of criticism of it might be that it is so much the focus of social activities – life here would seem strangely empty if we could not eat, think and discuss the Singapore cuisine. In view of this it seems churlish to raise the issue as to whether or not the various local specialities are as safe as they are tasty.

Concern about the safety of foods is nothing new. Through instinct or knowledge we select for our tables, avoiding as far as possible poisonous or contaminated items, so why should we particularly worry about the essentially Singaporean elements of our cuisine? Perhaps one reason might be the alarmingly high local incidence of a variety of cancers of the digestive tract (See Table). Additionally, while it is fashionable to believe that the high incidence of liver cancers here is due to the prevalence of hepatitis B carriers within the population, there may be other local contributing factors – indeed the multi-hit theory of cancer genesis would seem to require their existence (1).

As industrialisation proceeded in Europe, the United States and Japan, the possibilities for contamination of food grew proportionately larger. This problem was belatedly recognised after a series of disasters and the recognition of several new illnesses. For example, Minimata's disease as a consequence of ingesting mercury-contaminated fish (2) and the "Dutch margarine disease" which occurred in 1960 as a consequence of an anti-spattering additive, while in 1965 there was an outbreak of cardiac failure in the beer drinkers of Omaha which was tracked down to the addition of cobalt sulphate to improve the "head" of the beer (3). These and many other kinds of culinary disasters suggest that we must be constantly on the alert and constantly reviewing food preparation in the light of new knowledge and the changing circumstances of local industrialisation.

As our knowledge of the fine details of cell physiology increases, and as our understanding of the basic molecular events in cancer genesis improves, we are less and less able to be sanguine about the food we eat. Additions to our food are being made all the time during production, storage and cooking – sometimes for purposes of preservation but sometimes inadvertently or for trivial reasons, for example dying rice with saffron. Food can be inadvertently contaminated with industrial pollutants at almost every step of its journey, up to and including the final one of wrapping it in recycled paper (which of course contains printing ink and carbonless "carbon" paper – both rich sources of polychlorinated biphenyls. It has been demonstrated that substantial contamination of food can come about from migration of such contaminants from wrapping materials (4a, b, 5) Additions to food can also be the consequence of spoilage, either of the food or one of its precursors. A degree of spoilage may be too small to be noticed or to give a recognisable change in flavour, but can nevertheless result in dangerously high levels of toxic metabolites. An

Relative Incidence of Cancers of the Digestive Tract in Asia, Europe and America

Site		Singapore	Shanghai	Osaka Japan	Bombay India	Finland	B'ham U.K.	Alberta Canada	Conn. USA	Recife Brazil
Nasopharynx	M	14.8	5.6	0.4	0.4	0.4	0.4	0.5	0.7	0.6
	F	6.2	2.5	0.2	0.3	0.2	0.2	0.3	0.1	0.4
Oesophagus	M	16.5	24.7	9.7	15.2	5.2	5.0	2.6	5.7	5.2
	F	5.1	8.0	2.9	10.8	3.9	2.7	0.7	1.2	1.6
Stomach	M	38.0	55.7	91.4	9.3	37.5	23.3	15.4	13.5	24.3
	F	17.0	21.0	45.1	5.8	19.3	10.6	6.0	5.7	10.8
Colon	M	11.8	6.7	6.3	4.6	7.9	16.5	17.1	30.1	2.8
	F	10.5	6.0	5.0	3.3	8.0	15.0	18.5	26.1	4.1
Rectum	M	11.3	9.0	6.9	4.4	7.7	16.1	10.6	18.2	2.7
	F	7.4	5.7	4.7	2.6	6.1	8.7	6.9	11.1	7.7
Liver	M	28.7	31.7	1.5	1.4	2.1	1.0	1.4	2.0	10.7
	F	7.4	9.1	0.4	0.6	1.1	0.5	0.8	0.7	10.3

Rates per 100,000 persons per year, age-standardised to world population, for years between 1967 – 1977.
M = Male, F = Female.
Taken from : Shanmugaratnam, Lee & Day, 1983 (See ref. 12)

example would be the generation of mycotoxins following some degree of mould or fungal growth. Sometimes the very techniques used to preserve certain foods can generate toxic products.

All of this can only generate a feeling of unease and helplessness in us since we do not have the resources as individuals to keep track of the latest findings in toxicology, nor to submit all of our proposed diet for detailed analysis. But it is instructive to see if this unease seems justified when we consider the various items which typify a "Singaporean cuisine".

Traditionally, here, as in other parts of Asia, foodstuffs are preserved by salting, drying, pickling or fermenting. The risks inherent in consuming salt dried fish are now well documented and have been extensively researched by Magee and his colleagues in London (6). Salt preservation is now known to generate nitrosamines in fish, and these nitrosamines are strongly implicated in the genesis of oesophageal and liver tumours. Vanishingly small doses of only a few parts per million may be enough. Nitrosamines are generated not simply in salt preserved fish, however; they are also made *in situ* within our gut when we simultaneously consume nitrite (or nitrate) and amines. Nitrate is a common impurity of the crude commercial salt used for food preservation and nitrite is generated from it by the action of bacteria. In the acid environment of the stomach carcinogenic nitrosamines are formed from these two precursors (7). It is known that many of the spices used in preparing curry powder contain high levels of amines such as piperidine and pyrrolidine (8). As well as salt preserved food, preserved meats such as "Chinese sausages" are rich sources of nitrite, while prawn paste (belachan) may be another source of amines, along perhaps with ikan bilis.

Because the climate of Singapore is always warm and humid, a chronic problem is the tendency for moulds to grow on almost anything left alone for a few days. Of course not all moulds necessarily produce toxic or carcinogenic compounds, but a surprising number do. Many of these mycotoxins are strongly suspected of having a significant role in human carcinogenesis. Those moulds which share our foods here include the common fungus *Aspergillus flavus*. This fungus favours the peanut for its growth and is known to produce several related toxins causing mutations in individual cell lines (the Ames test) (9), and to produce cancers in inbred strains of laboratory animals. What human epidemiological evidence there is, strongly implicates aflatoxins as causes of primary liver cancer (10). Of course peanuts form an important part of our local cuisine so it would be instructive to know the average dose of aflatoxins from this source; one might reasonably suspect that it could be high.

Dried kumquats are popular in Singapore, but housewives often dry them rather slowly in the sun. It is very possible for fungal contamination to occur in this situation, which may not be easily detected. The list of toxic and probably carcinogenic fungal products is already very large but almost certainly far from complete, so housewives should perhaps consider more rapid techniques for drying.

The satay man produces that delightful taste and odour by cooking his slices of meat over an open charcoal fire. In this situation the fat drips down onto the hot coals where it is pyrolysed. Fat pyrolysis is known to produce the premutagen class of benzopyrenes which may then become reabsorbed into the cooking meat (11). If the levels of benzopyrenes should turn out to be unacceptably high in satay as it is usually cooked at present, it might be that simply grilling (i.e. the heat coming from above rather than below) will remove much of the problem with very little, if any, change in flavour.

There are thus substantial reasons for suspicion and concern about those particularly Singaporeans parts of our cuisine. We cannot expect to rely upon overseas research or quality control in this case and so it is up to Singapore to investigate and monitor these products. Such investigation may even ultimately pay off in a reduction in some of the unusually high levels of cancers here.

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Diabetes Mellitus — A review and report adapted from papers presented during the 3rd Congress of the Asean Federation of Endocrine Societies (AFES).

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Prominent diabetologists and endocrinologists from Asean and other Western and Asian countries gathered for the 3rd Congress of Asean Federation of Endocrine Societies held in Kuala Lumpur from 1st – 6th December, 1985. Numerous papers were presented covering a wide range of topics in diabetes mellitus (DM) and other endocrine disorders. The symposia and lectures on DM covered patho-physiology, diagnosis, management and complications associated with the disease. An update on diabetic research in the five participating countries of Asean revealed that although diabetes is not a primary health problem in this region there is reported increased prevalence, especially of Type II (non-insulin dependent) DM among the more affluent population. More concerted effort and cooperation between physicians, health educators and dietitians will bring greater awareness of the disease and contribute to the health and welfare of the diabetics of this region.

Classification Diabetes mellitus is a heterogenous group of diseases characterised by hyperglycaemia and its consequent complications of neuropathies and angiopathies. Most countries adopt the WHO Expert Committee classification for diabetes mellitus and allied categories of glucose intolerance as presented below.

Classification of diabetes mellitus and other categories of glucose intolerance:

1. Idiopathic diabetes
   - Type I/Insulin-dependent diabetes
   - Type II/Non-insulin-dependent diabetes
     - Obese BMI*>25 for women
       BMI >27 for men
       or weight >120% ideal body weight
     - Nonobese BMI < 25 for women
       BMI < 27 for men
       or weight < 120% ideal body weight

2. Diabetes secondary to:
   - Pancreatic disease
   - Hormone excess
   - Drugs or chemicals
   - Insulin-receptor abnormalities
   - Certain genetic syndromes
   - Other types

3. Gestational diabetes

4. Impaired glucose tolerance
   - Obese
   - Nonobese
   - Secondary

* BMI: Body Mass Index, BMI = Weight (kg)/ height^2 (m)

Diagnostic criteria Some current proposals concerning diagnostic criteria are presented in Table 1. The WHO new recommendation for the performance of an OGTT (oral glucose tolerance test) is 75g for a non-pregnant adult and 1.75g/kg ideal body weight to a maximum of 75g for children, and the choice of samples are at fasting and two hours after loading. Division into obese and non-obese subgroups in recommended.

Table 1. Diagnostic criteria for DM

Diagnosis	FPG	mmol/l (mg/dl)	2hr PG	mmol/l (mg/dl)
Normal	<	6.4 (114)	<	7.8(140)
Impaired glucose tolerance		6.4-7.2 (114-130)		7.8-11.1 (140-200)
Overt diabetes	>	7.8(140)	>	11.1(200)

FPG = fasting plasma glucose

2hr PG = 2 hour plasma glucose

All values are expressed as venous plasma glucose concentration.

Type I/insulin dependent diabetes (IDDM)

The basic pathology in IDDM is the destruction of β-cells leading to severe insulinopaenia and ketosis proneness.

Aetiology of IDDM A. Genetic – 50%. Human leucocyte antigens (HLA) in the chromosome 6 region seem to play major role.

B. Enviromental – 50%
   (a) Viral infection – viral and bacteria infection may precipitate the disease, e.g. mumps, rubella, human coxsackie virus B4.
   (b) Drug induced – due to β-cell destruction.
   (c) Food additives – a study in Iceland has shown that food additives comprising nitrosamines may be associated with the disease in the consumers' progeny.
   (d) Breastfeeding – Scandinavian epidemiological studies indicate that breastfeeding leads to a reduced risk of IDDM. Whether it is due to breastfeeding itself or some chemical present in

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milk powder remains unclear and further study is needed.

In the context of how genetic and enviromental factors inter-relate in the genesis of IDDM, Dr Yeo (Singapore), in summing up, suggested that inherited susceptibility to abnormal immunoresponses could lead to increased vulnerability to certain pancreatotoxins and pancreatic damage.

Type II non insulin-dependent diabetes

In contrast to severe insulinopaenia giving rise to ketosis proneness in IDDM, most NIDDM patients are considered to have significant amounts of insulin in circulation, and hyperosmolar non-ketosis is the prominent acute complication.

Aetiology of Type II/NIDDM A. Genetic – concordance rate in identical twins is 90% or more. Multigenic autosomal recessive means of transmission is observed in most communities.

B. Environmental –

(a) Overnutrition and obesity – 60-90% of NIDDM are obese in the West. In Asia approximately 30% of NIDDM are found to be obese.

(b) Physical inactivity – sedentary life style plays an increasing role.

(c) Malnutrition.

(d) Stress – severe or prolonged.

(e) Urbanisation.

(f) Acculturation – e.g. migration.

It is believed that an individual inherits a susceptibility to develop NIDDM and one or more non-genetic factors can eventually precipitate overt clinical disease. Genetic and environmental mechanisms can also contribute to the appearance and severity of diabetic complications.

Malnutrition-related diabetes mellitus (MRDM) MRDM has previously been reported in Africa, rural Malaysia, Indonesia and Thailand although cases are rare in this region nowadays due to improved nutrition. Consumption of certain types of cassava containing cyanogenic glycosides, the subsequent release of cyanide and the deficiency of sulphur-containing amino-acids to help detoxify these cyanide groups are thought to contribute to the pathogenesis of the condition. No new cases have been reported in Malaysia for the past two years.

Gestational diabetes (GDM) This is diabetes appearing during pregnancy and in most cases disappearing after pregnancy. 30–40% become diabetics in 5–10 years.

Management of DM

Glucose concentration mg/dl (mmol/l)

	normal	acceptable	fair	poor
FPG	<115(6.4)	140(7.8)	200(11.1)	>200(11.1)
2h PG	<140(7.8)	175(9.7)	235(11.7)	>235(11.7)

The major objectives in the treatment of DM have been the alleviation of hyperglycaemic symtoms and the prevention or amelioration of chronic diabetic complications. The success of the therapy depends greatly on the patient's committment and contribution to self-management through the means of diet, excercise and with hypoglycaemic agents or insulin when called for.

In recent years, home blood glucose monitoring (HBGM) has been introduced to supplement urine testing.

Measurement of glycosylated haemoglobin (GHB) can reflect the time-average serum glucose concentration for the preceeding 6-8 weeks. Glycosylated serum albumin (GSA) and protein (GSP) can give further information on glycaemic control during the preceeding short-term span of 1–2 weeks.

Diet Nutrition plays an important role both as a risk factor in the development of diabetes mellitus and also as one of its treatment modalites. All diabetics and relevant family members should, if possible, consult a dietitian for proper dietary instruction. The current recommendations in terms of the composition of diabetic diets (American Diabetes Association, 1979), expressed as % of total caloric intake, are as follows:

carbohydrate	protein	fat
50 – 60%	15 – 20%	20 – 30%

In this region, where high carbohydrate consumption is the common food pattern, a recommendation of

carbohydrate	protein	fat
60 – 70%	10 – 15%	15 – 20%

seems more acceptable according to results of a study by Supartondo *et al.* (Indonesia).

The beneficial effects of a high carbohydrate (CHO), high fibre (HCF), low fat diet are well documented (1,2). It was known that high CHO diet improved glucose tolerance in treated diabetics. This was considered due to increased insulin sensitivity as the fasting glucose levels decreased without an increase in basal insulin levels or an increase of insulin dose. The success of the HCF diet may also be in part due to its low fat content as insulin sensitivity has been shown to be inversely related to the fat content of the diet.

Many studies have shown the value of high fibre in the diet of diabetics. Much work is being done in this region using local high fibre food sources. Baba *et al.* (Japan) in his studies incorporated glucomannan (powder extract of kunig fibre from raw tuber) in the diets of Japanese diabetics to boost their fibre intake to 30g/day. It is suggested that fibre acts as a resin-like binder to decrease the absorption and secretion of bile acids. It has the added advantages of:

(1) inducing satiety due to water-holding capacity.

(2) delaying gastric emptying time.

(3) reducing postprandial blood glucose level.

(4) reducing blood cholesterol level.

(5) increasing intestinal matrix.

He also advised a reduction in salt consumption from the average Japanese intake of 15–16g/day to 7–8g. Alternate seasoning may enhance flavouring and put less demand on salt use. (The average Japanese diet is 2400 kcal/day with 60-65% CHO, 15-20% protein 15-20% fat.) A study by Kurinami *et al* have shown that diabetic diets with added lecithin and EPA (eicosapentaenoic acid) give improved ketone and plasma lipid metabolism. A study by Budhiarta *et al.* (Indonesia) has shown the hypoglycaemic and hypolipidaemic effect of green beans *(Phaseolus vulgaris)* when incorporated to the diabetic HCF diet (3). In a seperate study, Pikir has also shown the hypoglycaemic effect of shallot *(Allium lepa)*. In Thailand, studies with beans and guavas have been carried out with favourable results. More work is being undertaken in Philippines and Malaysia regarding local high fibre food sources.

Table 2. Sample daily menus of standard, adjusted and low glycaemic diabetic diets (2100 kcal)

	Standard	kcal	Adjusted	kcal	Low glycaemic	kcal
Breakfast	rice	350	rice	440	rice	350
	boiled egg	95	meat cooked in curry sauce	250	fried egg	140
	fried tempeh*	125	fried tempeh	125	milk	130
	mushroom stew	30	stewed green bean	75		
Mid-morning snack	papaya	40	lumpia (meat & vegetable savouries)	200	papaya	40
	rice	350				
Lunch	rice	350	–		steamed potato	350
	beef in soy sauce	135	–		fried chicken	140
	tempeh in coconut milk	120	–		mix vegetable in peanut sauce	175
	spinach soup	50	–		banana	40
	papaya	40	–			
Afternoon snack	banana	40	–		pineapple	40
Dinner	rice	350	rice	440	rice	350
	fried fish	140	chicken stew in sweet soya sauce	235	fried fish	140
	boiled tofu	125	fried tofu	125	fried tempeh with spices	125
	vegetable soup	50	mix vegetable in santan	95	sour mixed veg. soup	50
	banana	40	banana	40	orange	40
Supper	–	–			–	

* Tempeh – fermented soya bean cake.

Supartondo *et al.* in their extensive studies in different parts of Indonesia have shown that a diet consisting of 68% CHO, 20% fat, 12% protein within a caloric range of 1900 – 2500 kcal leads to a significant improvement in serum glucose and cholesterol levels as compared to an earlier standard diabetic diet of 50% CHO, 20% protein and 30% fat. He further advocates a diet of 75% CHO, 10% protein and 15% fat for the lower socio-economic group of patients as better compliance and adherence are found, this being more suited to the average Indonesian diet.

Knowledge of the glycaemic index of common foods found in each country could help patients improve their diet planning and glycaemic control. Examples of the glycaemic index of fruit, potato, milk and natural sweetener are as follows:

<20%	20-40%	40-60%	60-80%	100%
fructose	potato (steamed)	papaya pineapple	honey banana orange	glucose

Table 2 shows a sample daily menu comparing an Indonesian standard type diabetic diet, one adjusted for a different meal pattern and one with low glycaemic index.

For the Muslim patients during Ramadan where fasting takes place between sunrise and sunset, Sani *et al.* studied a group of 55 patients following a pattern where meals were consumed at 18 hrs, 22 hrs and 04 hrs (oral hypoglycaemic drugs where needed were taken with meal at

18 hrs). No ill effects were reportedly experienced during fasting; favourable results with significant decrease in body weight, total lipid and triglyceride levels were found.

These studies show the importance of adapting diet to the lifestyle of patients.

For obese NIDDM, a weight reduction diet should be designed so as to reduce the total calories without the expense of protein to achieve a reduction of 0.5 kg/week on a continuous basis until ideal weight is achieved. Obese diabetics tend to lose weight faster on a high protein diet, after which they are shifted to a high CHO diet for maintenance of desirable weight.

Obesity has been shown to cause insulin resistance. The decreased numbers of insulin receptors, intracellular defects in glucose transportation or metabolism may largely account for the resistance to insulin action.

Increase in body fat leads to increased atherogenesis in diabetes mellitus as obesity and hyperlipidaemia are shown to be closely associated.

With weight loss, glucose tolerance and insulin sensitivity often improve with resulting reduction in atherosclerotic risk.

A recent 5-year study of coronary risk factors, Diabetics Intervention Study (DIS), carried out by the European Atherosclerosis Group, has yielded some preliminary results as summarised in Table 3 and compared with the general population (Dresden study).

Table 3: Prevalence of coronary risk factors (%) among (I) DIS patients (n = 1139) on admission to the study and (2) the general population (Dresden study) (n = 1216).

Risk factors	DIS	Dresden Study	Limits
Hyperlipoproteinaemia	17.6	7.6	triglyceride≥250 mg/dl and or cholesterol≥300 mg/dl
Hypertension	53.0	17.3	blood pressure≥160/95 mmHg and or antihypertensive drug
Smoking	34.0	30.3	tobacco consumption ≥ 1g/day
Obesity	49.0	8.2	ideal weight index M > 1.2 F > 1.3
Hyperuricaemia	22.5	3.8	serum uric acid M > 7.0 mg/dl F > 6.0 mg/dl

The object of DIS was to document the most important risk factors in Type II diabetics and to influence them specifically through combined preventive measures. The results of the latter part of the study are not yet available. When known, however, they will no doubt help to show that the reduction of lipids is an important preventive measure in the treatment of diabetes.

It is apparent that diet therapy plays a pivotal role in the management of DM.* Many factors influence the implementation of the diabetic diet. Diabetologists and dietitians must find ways to educate and motivate the patients. Adaptation and modification of diet to the individual patient's life pattern, work schedule, socio-economic conditions etc. may help to improve adherence, hence better hyperglycaemic control with reduced risks of diabetic complications.

Exercise Regular exercise is widely believed to be beneficial to health, and in diabetics has the added advantage of accelerating weight loss in the obese, boosting psychological well-being and improving insulin sensitivity in IDDM. There is also the possibility of lowering cardiovascular risk factors as it has been shown to lower LDL and VLDL cholesterol, increase HDL cholesterol, improve hypertension and reduce cardiac work load (4, 5).

With Type II NIDDM, patients on diet alone should be encouraged to excercise regularly as there is no hypoglycaemic risk: Those on hypoglycaemic drugs may sometimes need to adjust diet and drug to avert hypoglycaemia during vigorous excercise.

The essence for excercise in diabetes is adaptation, in the words of Dr Gill (UK). Learning to cope with the necessary modifications the patient can become more knowledgeable and proficient in management of his own diabetes – a highly desirable educational process.

Hypoglycaemic agents For NIDDM, if diet and exercise alone should prove ineffective for metabolic control, addition of hypoglycaemic agents is indicated particularly in those without serious complications.

Dr de Fronzo (USA) suggested that disordered and increased hepatic glucose production is the major cause of fasting hypoglycaemic state in NIDDM.

In basal state, the rate of body tissue uptake of glucose for metabolism should be the same as the hepatic output since plasma glucose concentration is held constant in normal subjects. In NIDDM, the rate of endogenous glucose production is increased significantly. It is this excess glucose that is responsible for the hyperglycaemic state. He envisages that in future new hypoglycaemic drugs that block gluconeogenesis and those that promote glycogen uptake will help alleviate this disorder.

Insulin The aim of therapy is to achieve normo-insulinaemia in IDDM, in order to:

(1) Avoid acute symptoms and hypoglycaemia.

(2) Reduce development and onset of the long term complications retinopathy, neuropathy, microvascular diseases.

These goals can best be attained by insulin replacement in such a way as to mimic physiological insulin secretion.

Complications associated with diabetes mellitus

Ketoacidosis and comas Fluid, insulin, potassium and alkali are the common therapy used for ketoacidotic coma. Location and treatment of infection, if present, must be carried out.

Diabetic retinopathy (1) background retinopathy – microaneurysms.

(2) pre-proliferation – venous dilatation, beading, loops; retinal haemorrhage, retinal oedema, lipid exudates.

Early treatment is called for; photocoagulation is the best method to date.

** Supartondo mentioned that in the hospital he works with, there are close to 20 dietitians attached to different wards and units. This has enabled them to participate widely in research and patient-education work – a policy most dietitians would surely want to endorse and encourage.*

Diabetic nephropathy 30-35% of Type I IDDM develop diabetic nephropathy and it signifies a serious prognosis. It is less of a problem in maturity onset NIDDM.

Albuminurea, increased glomerular filtration rate (GFR) and increased renal plasma flow (RPF) are the early signs. Hyperglycaemia seems to be associated with these changes.

Treatment (1) Hypertension – reduction of blood pressure in hypertensive patients with diabetic renal disease lowers the rate of progression of renal impairment (6).

(2) Improved hyperglycaemic control may reduce development of diabetic nephropathy.

(3) Management of end-stage renal failure in diabetics should follow the same principles as in non-diabetic renal failure.

Restriction of protein intake, chronic dialysis and transplantation have markedly improved the management of these patients.

Cardiovascular complications Approximately 50% of mortality in diabetics in the West can be attributed to cardiovascular complications. A close relationship between cardiovascular disease and carbohydrate and lipid metabolism has been established. The contributing risk factors are:

hyperglycaemia, hypertriglyceridaemia, hypercholesterolaemia, insulin resistance.

The therapeutic aim is to normalise both lipid and carbohydrate metabolism as far as possible by diet and

Update of diabetes mellitus in ASEAN

A. Indonesia:

Epidemiologic studies showed a prevalence of 1.46% to 2.3% in different parts of Indonesia as of 1975. Juvenile diabetes is rare – 0.26% in population study (below 15 years) while hospital studies revealed 1.4 – 4% of diabetics were below 24 yrs. Slight male preponderance (similar to findings found in other Asean countries) was observed in contrast to that of the West where female preponderance is generally found. Mortality rate of diabetic ketoacidosis is still high. The prevalence of diabetic complications from surveys done is 12-27% hypertension, 25-27% CHD, 10-32% retinopathy, 9-55% nephropathy. Increased awareness within the last ten years has prompted more studies and with the setting up of diabetic clinics over all the major cities the future seems brighter for diabetics in Indonesia.

B. Philippines:

National prevalence shown to be in the region of 4.1% from epidemiological studies (4% in male and 42% in female), with a greater prevalence in the urban areas (6.8%) as compared to the rural areas (2.5%). While acute complications are rare with majority being NIDDM, chronic complications follow similar patterns as other countries, with neuropathy being the highest ranking among the diabetic triopathy. Much research is being carried out on the different aspects of diabetes mellitus in the Philippines. On the dietotherapy front, glycaemic indices of Philippine food items and combinations of such items are currently being undertaken together with studies on high fibre diabetic diets.

C. Thailand:

The statistics of diabetes mellitus in Thailand were not made available during the symposia. Recent studies on Thai elderly (60 yrs and over) show a high prevalence rate of 13%, with 28% of the tested population being hypertensive (Bunnag). A high proportion of those found gouty have high incidence of carbohydrate intolerance. It was suggested that improper dietary intake, low physical activity, reduced lean body mass to store glucose load and reduced normal insulin receptors are all contributing factors to diabetes in this population group.

D. Malaysia and Singapore:

The prevalence of DM among different ethnic groups of the population is as follows:

	Chinese	Malay	Indian
Singapore	1.6%	2.4%	6.1%
Malaysia	1.4%	3.9%	3.5%

In Singapore, population studies show a prevalence of 2% in the adult population with a male preponderance of 2.4% and female 1.6%. Recent studies indicate incidence increasing with age group (15 – 39 yrs, 0.4% and over 40 yrs, 5.1%). Juvenile diabetes from the same study shows 0.04/1000 under the age of 12 yrs. The prevalence of diabetic complications are hypertension 27%, nephropathy 9%, retinopathy 10%, CHD 6%. Research has shown 71.5% of diabetics to be on oral hypoglycaemic agents, 15% on diet only, 4.5% on insulin and 9% on traditional herbal therapy.

In Malaysia, hospital based studies have shown a prevalence of 2.1% in 1983 compared to the 1.05% shown in 1978 and 0.65% in 1960. A population based study in 1984 showed a prevalence of 3.9% among Malays. 3.5% among Indians and 1.4% among Chinese. 7.7% of total diabetic cases are IDDM, of which, 4.1% are in their twenties (Mustaffa 1984), as compared to 2.4% shown in the 1978 study.

All studies indicate marked increased prevalence of diabetes mellitus within the last ten years. In both Singapore and Malaysia, male and female Indians have the highest prevalence among the three ethnic groups, the majority in the age group of 40 – 60yrs. Comparing the prevalence rate of 1.1% of Indians in India and the 0.6 – 0.7% of Chinese in China, the environmental factors such as migration, increased affluence, etc. might have contributed to the increased prevalence. More studies need to be undertaken on this aspect.

From available data, the last few years have seen a consistent increase in prevalence of diabetes mellitus among the general population in the ASEAN countries, with the onset age getting younger. Better health education is called for in the hope to retard this undesirable process.

if necessary lipid-lowering drugs.

Juvenile diabetes

A recent study from Sweden of children up to the age of 14 years suggested an annual incidence of 22.7 per 100,000 and a prevalence of 1.48/1000. The incidence in this region is rare but there are increased reports of cases especially with the Mason Type of NIDDM seen in young, obese persons. The average age of onset for juveniles is from 11 years (later than Caucasians). Young IDDM here usually are underweight and highly ketoacidotic, often presenting with acute symtoms (33%) due to poor glycaemic control.

With children, appropriate growth velocity coupled with satisfactory GHB & HBGM results indicates good control. The insulin regime best followed is the twice daily injection and/or intermediate insulin before bedtime.

Gestational diabetes

Management of impaired GT during pregnancy should be the same as for diabetics. The greatest hazard of hyperglycaemia occuring in early pregnancy is its contribution to congenital malformation. This is reported to occur 2-4 times more frequently in infants of diabetic mothers. Later on in pregnancy, fasting and post-prandial hyperglycaemia increase foetal risk for intrauterine death or neonatal mortality.

Diet is prescribed at 35 kcal/kg body weight with a distribution of 50-60% CHO, 15-25% protein and 25-30% fat. For NIDDM, 3 meals with a late evening snack and IDDM, 3 meals and 3 snacks are suggested. The diet should not make pregnant diabetics lose weight due to the danger of ketosis to the foetus. Excessive weight gain is discouraged, no more than 3 kg/month in the 1st trimester and a progressive linear rate of about 350-400 g/week in the last trimester. Glycaemic control is considered adequate if normal blood glucose is around 105 mg/dl in fasting state and 120-135 mg/dl in the 2hr postprandial state.

CONCLUSION

Much progress and advancement has been made towards understanding diabetes mellitus and its pathogenesis. Hope for the future includes prevention and the possibility of early curative treatment. For the patients of today, the management of diabetics must depend on a team approach – that of the diabetologist, the dietitian, and the specialist diabetic nurse and nurse educator. The success of the therapy requires intensive education particularly that of the patient so that he himself is in charge. In the words of Dr Albert; "It is not good medicine nor education if the patient ends up with a very rigid life; he must educate himself so as to be able to adapt his diet, medication and/or insulin to coincide with his own lifestyle, but with normoglycaemia and ultimately normoinsulinaemia". This is a challenge for all concerned.

PAPERS PRESENTED:

Plenary Lectures:	Insulin Therapy in Diabetics – K.G.M.M. Alberti
	Immunotherapy in Diabetics – S.Baba
Symposia:	
Pathophysiology of Diabetes	Gentics/Environment – P.B.B. Yeo
	Abnormalities of Insulin Secretion – L. Heding
	The Insulin Receptor and Insulin Resistance – R.G. Larkins
Laboratory Tests in Diagnosis and Management of Diabetes	Laboratory Tests in Diagnosis of Diabetes – J.M. Bumin
	Long Term Monitoring – A. Vichayanrat
	Home Monitoring – A.C. Thai
Management	Interorgan Exchange of Glucose: Implications for Future Therapy – R.A. de Fronzo
	Diet for Diabetes – A. Supartondo
	Diabetes and Exercise – G.Gill
	Childhood Diabetes – J. Nabarro
	Diabetes in Pregnancy – A.D. Litonjua
	Use of Insulin Pump in Clinical Practice – H. Susetrunk *et al.*
Type II Diabetes	Presentation and Diagnosis – R.E. Fernando
	Obesity and Hyperlipidaemia – T.C. AW
	Role of Oral Agents – S.C. Bunnag *et al.*
Diabetes Complications	Ketoacidosis and Comas – K.G.M.M. Alberti
	Diabetic Retinopathy – Hardeep Singh
	Diabetes and the Kidney – R.G. Larkins

UPDATE on DIABETES RESEARCH in ASEAN:

Indonesia	– U. Sukaton
Malaysia	– B.E. Mustaffa
Philippines	– A.D. Litorjua
Thailand	– S.C. Bunnag
Singapore	– P.B.B. Yeo

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SHORT COMMUNICATION:

Anthropometry – Body Composition Versus Weight

Barbara Burlingame and Dr Ang Kong Ee

Overweight and obesity are often incorrectly used as synonymous terms. However, it is essential to remember that obesity indicates excess fatness, independent of body weight. It is possible for a person to be overweight and not be obese, and it is very common for people to be obese and not be overweight. In addition to the inherent problems in assessing obesity on the basis of body weight, the most widely used standards are biased toward Western populations, making the norms additionally unreliable for Asian populations. As the incidence of degenerative diseases is on the rise, and obesity is a significant contributing factor, a reliable measure of obesity is essential.

Many methods are available for determining the percentage of body fat. Some of the most valid methods include underwater weighing, radiographic analysis, isotopic dilution and ultrasound techniques. Because of time constraints, expense of sophisticated equipment and space requirements, these methods can not be used in widescale screenings and in most clinical settings. Skinfold fat measurements are much more practical for general usage. The technique correlates highly with underwater weighing, it is easy to learn and the information obtained is much more useful than the measurement of body weight (1) (See Table 1).

In a fitness evaluation programme held at Dynami, the Total Fitness Club, men and women of all ages and races had their body fat measured with Harpenden skinfold calipers, with an accuracy of ±0.1 millimetres. The procedure for measuring skinfold fat is outlined below and the Jackson and Pollock protocol (3, 4) was used, as follows:

Assessing Body Composition (Jackson & Pollock, 1978)

Percent Fat: Men

For men, the sum of four:

1. Chest
2. Ilium
3. Abdomen
4. Axilla

$\%\ fat = 0.27784\ (X_1) - 0.00053\ (X_1)^2 + 0.12437\ (X_2) - 3.28791$

where: X_1 = sum of 4 skinfolds
X_2 = age
R = 0.892 SE = 3.63% fat
Obesity = greater than 20% fat

Percent Fat: Women

For women, the sum of three:

1. Triceps
2. Abdomen
3. Ilium

$\%\ fat = 0.41563\ (X_1) - 0.00112\ (X_1)^2 + 0.03661\ (X_2) + 4.03653$

where: X_1 = sum of 3 skinfolds
X_2 = age
R = 0.825 SE = 3.98% fat
Obesity = greater than 30% fat

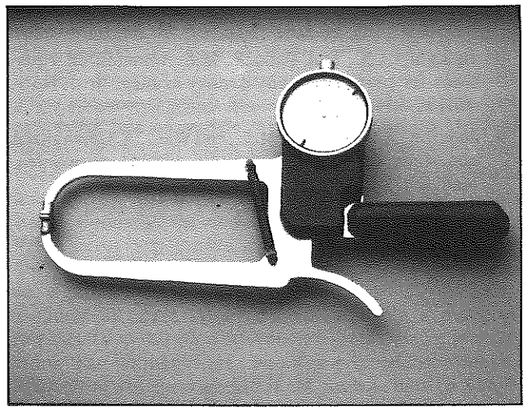

Fig 1. The Harpenden skinfold calipers

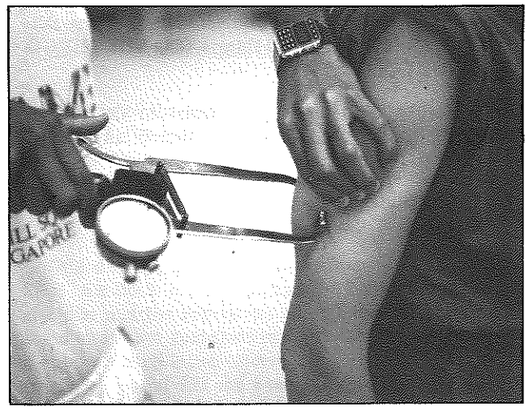

Fig 2. Measurement of the triceps skinfold.

Table 1. Correlation between hydrostatically determined body density and anthropometric variables

Anthropometric Variable	Female Sample (n=249)		Male Sample (n=308)	
	r	SE	r	SE
Age	–0.35	0.015	– 0.38	0.017
Height	– 0.08	0.016	0.01	0.018
Weight	– 0.63	0.012	– 0.62	0.014
Body Mass Index[a]	– 0.70	0.011	– 0.69	0.013
Sum of 7 Skinfolds	– 0.85	0.008	– 0.88	0.009

[a] Body Mass Index = weight/height^2; weight in kilograms and height in metres

(Source: Pollock, M.L., et.al. Comprehensive Therapy, Vol. 6, No. 9, pp. 12-27, 1980)

In addition, the same subjects were measured for body weight and height on a balance beam scale with an accuracy of ±100 grams. Weight was evaluated using the 1959 Metropolitan Life Insurance Company standards (2). Many of the subjects were found to have a weight in the "ideal" range, and yet they were found to be obese based on skin-fold fat measurements. A formal study of the correction between body composition and weight is now in progress.

PROCEDURE FOR MEASURING SKINFOLD FAT

1. Testers should trim their fingernails
2. All measurements must be taken when the skin is dry.
3. All measurements must be taken on the right side of the body.
4. Firmly grasp the skinfold between the left thumb and four fingers.
5. Pinch and lift the fold several times to make certain that no musculature is grasped.
6. Continue to hold the skinfold firmly with the thumb and fingers and place the contact surface of the calipers below the thumb and fingers. (DO NOT LET GO OF THE FOLD)
7. The caliper must be perpendicular to the fold at approximately 1 cm from the thumb and finger. (Refer to the attached figures).
8. While maintaining a grasp of the skinfold, allow the caliper grip to be released completely so that the full tension is exerted on the skinfold.
9. Read the dial to the nearest 0.1 mm (Harpenden) and 0.5 mm (Lange) approximately 1 to 2 seconds after the grip has been released.
10. Take two measurements at each site. Take a 3rd measurement if the first two measurements vary by more than 1 to 2 mm.
11. If consecutive fat measurements become increasingly smaller, it means that the fat is being compressed; this occurs mainly with fleshy people. If this occurs, proceed to the next site and return to the trouble spot after finishing the other measurements.
12. Take the average of the two skinfold values that agree most closely.

Sites of measurement

Chest: a diagonal fold taken one half of the distance between the anterior axillary line and the nipple for men and one third of the distance from the anterior axillary line and the equivalent position for women.

Axilla: a vertical fold on the midaxillary line at the level of the xiphoid process of the sternum.

Triceps: a vertical fold on the posterior midline of the upper arm (over triceps muscle), halfway between the acromion and olecranon processes; the elbow should be extended and relaxed.

Abdominal: a vertical fold taken at a lateral distance of approximately 2 cm from the umbilicus.

Suprailium: a diagonal fold above the crest of the ilium at the spot where an imaginary line would come down from the anterior axillary line. It should be noted that many recommend that the measure be taken more laterally at the midaxillary line. Data for generalized equations of Jackson and Pollock were determined at the anterior axillary line.

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BOOK REVIEW:

The Complete Asian Health & Diet Plan

by Mary Trevelyan Hodder
Times Books International: Singapore, 1985, price S$24.50

It should be noted that this book is intended as an easy-reading guide for the layman and not as a reference for health and medical professionals.

This book is an easy-to-read, understandable, enjoyable and fairly thorough book on the basic fundamental principles of nutrition and dieting for the layman. However, if your expectation is to gather greater insight into the Asian dietary pitfalls and problems, you will be disappointed. It had the potential of making a significant contribution to our understanding of local dietary patterns; unfortunately it falls short of this mark.

The title is somewhat misleading. What is so Asian about this book? Certainly not the body of the book which describes a very international health and diet plan. It is the appendices A – E which are the only uniquely Asian aspect of the book. The author must be applauded for her work on these sections; it is about time we had some information, any information, on the nutrient content of our local foods. It is a pity, however, that no indication is given as to how the compositions of hawker dishes were arrived at. Without this vital information it is impossible for local dietitians to evaluate and therefore use this data. It is also unfortunate that there is at least one gross error in the Food Composition Table (cod-liver oil supposedly is 100% protein and contains 18,000 mg calcium) (p. 226).

The dieting section outlines a sensible and sound approach to reducing body fat; better than most diet books on the bookstore shelves. Unfortunately, there are some factual errors and technical flaws. These do not detract significantly from the overall good qualities of the book, but they should be addressed to prevent any misconceptions on the part of the readers. The section entitled "Overweight: the disease of plenty" is very good and loaded with important information. However, there are a few statements which are misleading. While a poor diet can cause loss of muscle, a good diet **does not** lead to deposit of muscle tissue (in the adult). Smaller, more frequent feedings **do not** lead to greater gains in muscle mass (p.16). The advice is good, but not for the reasons stated. Similarly, the advice to avoid alcohol is good, but it has never been shown to be a carcinogen (p.18), it is only associated with cancer in epidemiological studies (an important difference).

Other incorrect statements are probably a result of relying on pop-nutrition information for reference material, rather than textbooks and up-to-date professional journals. Fibre **does not** increase intestinal passage time (p.53), it increases the movement of food through the body thereby decreasing the time. Singaporean children do have a bigger build now than children of generations before, and they do eat much more sugar, but the sugar intake has never been related to the larger stature of the Singaporean children (p.46). The general consenus reported in international health and medical journals is that lecithin does help to bring down blood cholesterol levels (see p.40). This, however is more a subject of pharmacology than nutrition. The recommended diet consists of about 20-25% of kilocalories from fat, 10-15% from protein and 65-70% from carbohydrate, not 20-25% fat etc per se (p.33). And finally, the diet is good and sensible, but it will not necessarily take fat off the stomach first (p.119); no diet can promise that.

The nutrition chapters are short, but very thorough in the coverage of important material. However, the section on vegetarian diets is disappointing in that trivial information is given and important information is omitted. Much more significant than vitamin D intake for vegetarians is adequate intake and absorption of essential trace elements. Iron is given all too brief a mention and other essential trace elements are not considered. The fact is, these trace elements are present in vegetarian foods, but they are not well absorbed unless taken with foods (or supplements) which contain high levels of ascorbic acid (vitamin C) or certain other acidic compounds. Deficiencies of essential trace elements are much more frequent in vegetarians than vitamin D and protein deficiencies.

The scope of the exercise section is wide, addressing the most recent topics in the health and exercise fields, including exercise and pregnancy, exercise and cardiovascular disease, diabetes, and the interactions of drugs and exercise. Although the author seems informed on the topics, many of the references that the author has chosen to cite have little scientific credibility.

Because of the wide range of health books on today's market, if the readers' main purpose is to learn more about nutrition and exercise, it would be advisable to choose a book written by an author with a formal academic background in nutrition and fitness.

In summary, aside from the aforementioned problem areas, the book is a well-packaged introduction to health and diet and the appendices will provide the reader with some important guidelines in the choice of local foods. As diet books go, this one offers more than most – education on the subject of diet and a sensible approach to correcting the problem of obesity which is the major health risk factor among the Singapore population.

Sources of Nutritional Reference in Singapore

Last issue, as a service to our members and other interested readers, we published a list of nutrition-related journals and periodicals currently available in the Medical Library of the National University of Singapore. We are following this up in the present issue with a compilation of book titles on nutrition and dietetics currently in the medical Library. We have included the authors' credentials where possible, since quite often this gives a clue as to the angle the book has been written from. The list is not exhaustive, and books published before 1970 have generally been excluded. There is no Nutrition section in the library and so the books are to be found under various other subject headings such as Biochemistry, Pharmacology and Paediatrics. For ease of reference these different sections are indicated below by their library classification code letters (QP, RM etc.)

RM

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24. Milk Intolerances and Rejection. J. Delmont, ed. Karger: Basel, 1983.

Cont'd on page 5

In Brief ...

Thermogenic Drugs in the Treatment of Obesity.

Thermogenesis induced by cold stress or ingestion of food may contribute significantly to energy expenditure.

In the 70's, discovery of the thermogenic potential of brown adipose tissue (BAT) in infants and cold-stressed animals was hailed as a possible solution to the puzzle of why some people remain lean for years with no effort to control food intake.

We now know that evidence for the existence of BAT in humans after early infancy is rather slight. The proposal, however, that postprandial thermogenesis is reduced in obesity may still have some mileage. Thus the possibility is raised that thermogenic drugs could be of value in treating obesity.

Researchers Brooke and Abernethy (Hum. Nutr: Appl. Nutr. 39A: 304-314, 1985) report preliminary trials in obese children with one such drug, ephedrine. Results so far are not encouraging. Other beta-adrenergic stimulants produce weight loss in animals but may have side effects in the doses required by humans. Thyroid hormones also produce increased thermogenesis, so may prove useful. The researchers stress, however, that long-term results of thermogenic drug therapy are no more satisfactory than dietary treatment alone.

Polyunsaturated Fats Lower Blood Pressure

Nutrition researchers recognize that the relationship between diet and blood pressure is far more complex than the amount of salt in the diet. Recent findings indicate that hypertension may stem from too little of other substances in the blood. An important factor affecting blood pressure is now suggested to be the amount of polyunsaturated fat in the diet.

According to James. M Iacano, Director of Western Human Nutrition Research Centre, California, Americans typically consume 3-4% of their total calories as polyunsaturated fat, while saturated fats account for 4.5 times that amount. He says that one has to consume 6-7% polyunsaturated fats to lower blood pressure. In studies conducted on 40-60 year old men and women, increased polyunsaturates significantly lowered both systolic and diastolic blood pressure although meat, dairy products, eggs and salt intakes were maintained at normal levels. Increasing polyunsaturates in the diet also improved clotting time.

The polyunsaturated fatty acids in food comprise mainly linoleic acid, which the body requires to synthesise prostaglandins. Prostaglandins regulate blood clothing, increase muscle tone of blood vessels and regulate blood pressure through the kidneys.

USDA Study Shows Sodium and Potassium Intake Askew in Adult Diets.

A one year study of 28 human subjects has shown that they eat too much table salt – sodium chloride – and not enough potassium. The research suggests that an adult may risk developing high blood pressure if the sodium – potassium ratio stays askew. People who consume a high level of potassium excrete more sodium in the urine, but a higher level of sodium intake in relation to potassium causes retention of excess sodium in fluids surrounding cells of the body. The Food and Nutrition Board of the National Academy of Sciences recommends the ratio of 0.6g for every gram of potassium. In the study, the subjects consumed 1.3g sodium for every gram of potassium. The average intake of potassium was 2.8g per day, within the range of 1.9 to 5.6g per day considered safe for adults. A safe range of potassium may be misleading if the amount of sodium exceeds the recommended ratio. The best sources of potassium are fresh vegetables and fruits.

Abstracts

USE OF XANTHAN GUM IN DIETARY MANAGEMENT OF DIABETES MELLITUS *Odutola Oselesi, David L. Trout, Esther E Glover, Sarah M. Harper, Eunsook T. Koh, Kay M. Behall, Thomas M O' Dorisio and June Tark. Am. J. Clin, Nutr. 42: 597-603, 1985.* Xanthan gum (12g per day) was fed in muffins, during the first or second half of a 12 week period, to free-living subjects. Nine were diabetics managing without insulin or hypoglycaemic drugs and the rest were non-diabetics. The xanthan gum lowered fasting and post-load serum glucose and reduced fasting levels of total plasma cholesterol in diabetic subjects.

EFFECT OF EXERCISE ON FOOD INTAKE IN HUMAN SUBJECTS. *F. Xavier Pi-Sunyer and Rosy Woo, Am. J. Clin. Nutr. 42: 983-990, 1985.* In this review of studies on the effect of physical activity on food intake in human subjects, lean persons seemed to regulate and maintain weight well, but studies on obese subjects are inconclusive. In metabolic ward studies, exercise neither enhanced nor inhibited food intake in obese individuals, but it created a negative energy balance. Thus energy output does not regulate energy intake closely in the obese.

THE ENERGY COST OF AEROBIC EXERCISE IN FED AND FASTED NORMAL SUBJECTS. *Paul J. Pacy, Nicola Barton, Joan D. Webster and John S. Garrow. Am. J. Clin. Nutr. 42: 764-768, 1985.*

The thermogenic effect of moderate exercise in the fasted and fed state in four lean subjects during weight maintenance was studied. Oxygen intake increased 22% over the 165 mins. after the meal. There was a significant but similar elevation of mean oxygen uptake during 40 mins. post exercise by 13.6% in the fasted and fed state. Sixty minutes after ceasing exercise mean oxygen uptake was not different from pre-exercise levels Thus there is no prolonged thermogenic effect of moderate repeated exercise in weight maintanance of lean subjects.

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Meetings

May 21-23, 1986

SYMPOSIUM ON SWEETNESS
International Conference Centre of Geneva, Switzerland

This symposium has been organized by the International Life Science Institute

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"Dietetics – the next 50 years"

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5th INTERNATIONAL CONGRESS ON OBESITY
Jerusalem, Israel

Contact: S.H. Blondheim, M.D.,
Chairman
P.O. Box 983
Jerusalem 91009, Israel

September 15-18, 1986

2nd INTERNATIONAL CONFERENCE OF DIET AND NUTRITION
Jerusalem, Israel

Contact: C. Horwitz, Ph.D.
Chairlady
P.O. Box 983
Jerusalem 91009, Israel

Abstracts

DIGESTION OF POLYSACCHARIDES OF SOME CEREAL FOODS IN THE HUMAN SMALL INTESTINE.

Hans N. Englyst and John H. Cummings. Am. J Clin Nutr 42: 778 – 787, 1985. The digestion and absorption of dietary starch and non-starch polysaccharides (NSP) in the small intestine of man from oats, cornflakes and white bread has been determined by feeding 7 ileostomists test meals and estimating carbohydrate recovery in the effluent. NSP was almost completely recovered from all three test meals. 5.8% of carbohydrate in white bread, 5.3% in cornflakes and 11.7% in oats was recovered. This study supports the view that human digestive enzymes do not break down dietary NSP and identifies a "resistant starch" present in processed foods which resists breakdown by amylase in vitro and in the small intestine.

Cont'd from page 24

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RJ

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Announcing ...

LOGO COMPETITION

All members are invited to participate in a competition to design a logo for our Association.

A prize of $50.00 will be awarded to the winning entry. A panel of judges will be appointed and the results will be announced at the Annual General Meeting in April.

Rules

1. All entries are to be presented in the finished art form and should be no larger than A4 size.
2. Rationale for the logo is to be given.
3. Any number of entries may be submitted by one person.
4. Each entry should have name and address clearly stated.
5. All entries are to be received by March 31, 1986, and are to be sent to:

   The Hon. Secretary
   Singapore Dietitians' Association
   Blk 78, Yong Siak Street
   #01-06
   Singapore 0316

6) The judges appointed are not eligible for the competition.

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