

## EDITORIAL

## Requirement for Asian Pacific Collaboration in Assessment of Nutritional Factors Impacting on Cancer Development

Five of the articles in the present issue of the APJCP address aspects of nutrition and cancer development, covering the importance of standardized dietary assessment (Aydemir, 2002), the role of soy foods in colorectal cancer (Toyomura and Kono, 2002), folate and head and neck cancer (Raval et al., 2002), dietary factors and gastric cancer in China (Xibin et al., 2002), and how to use improvement in the diet to control obesity (Hirose et al., 2002). It is obvious that research in nutrition continues to be a major focus within the area of cancer prevention. In this context it is of interest to focus on the volume entitled 'Food, Nutrition and the Prevention of Cancer: a Global Perspective' (1997), jointly produced by The World Cancer Research Fund/American Institute for Cancer Research. This was the result of a gigantic effort to comprehensively review the accumulated data from epidemiological and experimental studies for different food items. However, despite the obvious abundance of findings, and clear protective effects for vegetables across all organs, the conclusions regarding influence of other major food items on cancer risk were severely limited (see Table 1). Thus, for cereals in the alimentary tract, only possible inhibition by the whole grain variety in the stomach and possible enhancement by refined types in the oesophagus, along with insufficient evidence for inhibition in the colon, were decided. No concrete conclusions could be drawn for roots, pulses, nuts and seeds or milk and dairy products. With meat, a probable promoting effect in the colon/rectum was concluded,

while tea may protect in the stomach but increase risk in the oesophagus.

Clearly, given the theoretical protective effects of fibre included in cereals and starches (Ferguson and Harris, 1996; Moore et al., 1998b), a greater influence might have been expected. Indeed, looking at data for incidence of alimentary tract cancers in Globocan, International Agency for Cancer Research, Lyon, for countries across the Asian-Pacific region, along with percentages of cereals, starchy foods and fruit/vegetables in the diet for the same countries from 'Food, Nutrition and the Prevention of Cancer: a Global Perspective' (1997), we can see a significant inverse correlation observed between colon cancer incidence and cereal consumption as a percentage of the diet ( $p < 0.001$ ) (Fig 1), in line with earlier ecological data (Armstrong and Doll, 1975). However, it is of interest that consumption of cereal intake may negatively correlate with vegetable consumption (Fig 2). The fact that cross country comparisons of vegetable and fruit percentages of diet in fact demonstrated no link with any of the cancer sites in the digestive tract, for example in the oesophagus (Fig 3) is interesting in this context. While there is abundant evidence of protective action for vegetables at the individual level within populations, this lack of links suggests that this variable at the population level might be of less importance than cereal consumption.

Such simple comparisons clearly cannot provide detailed information, but they can give pointers to fruitful avenues for future research into nutrition and cancer. For

**Table 1. Consumption of Major Food Items and Cancer Risk in the Alimentary Canal**

| Food Item              | Buccal Cavity | Oesophagus | Stomach | Colon/Rectum | Liver |
|------------------------|---------------|------------|---------|--------------|-------|
| Cereals: Wholegrain    | NE            | NE         | -?      | -??          | NE    |
| Refined                | NE            | +?         | NE      | -??          | NE    |
| Roots/Tubers/Plantains | NE            | NE         | NE      | NE           | NE    |
| Pulses (Legumes)       | NE            | NE         | NE      | NE           | NE    |
| Garlic                 | NE            | NE         | -??     | NE           | NE    |
| Vegetables             | --            | --         | --      | --           | -?    |
| Nuts and Seeds         | NE            | NE         | NE      | NE           | NE    |
| Milk and Dairy         | NE            | NE         | NE      | NE           | NE    |
| Tea                    | NE            | +??        | -?      | NE           | NE    |
| Meat                   | NE            | NE         | NE      | +            | NE    |

Data from World Cancer Research Fund/American Institute for Cancer Research, 1997. 'Food, Nutrition and the Prevention of Cancer: a Global Perspective' NE, no effect concluded; +/- -, convincing evidence of increased/decreased risk; +, probable increased risk; +?/-? possible increased/decreased risk; +?/-?-, insufficient evidence of increased/decreased risk.

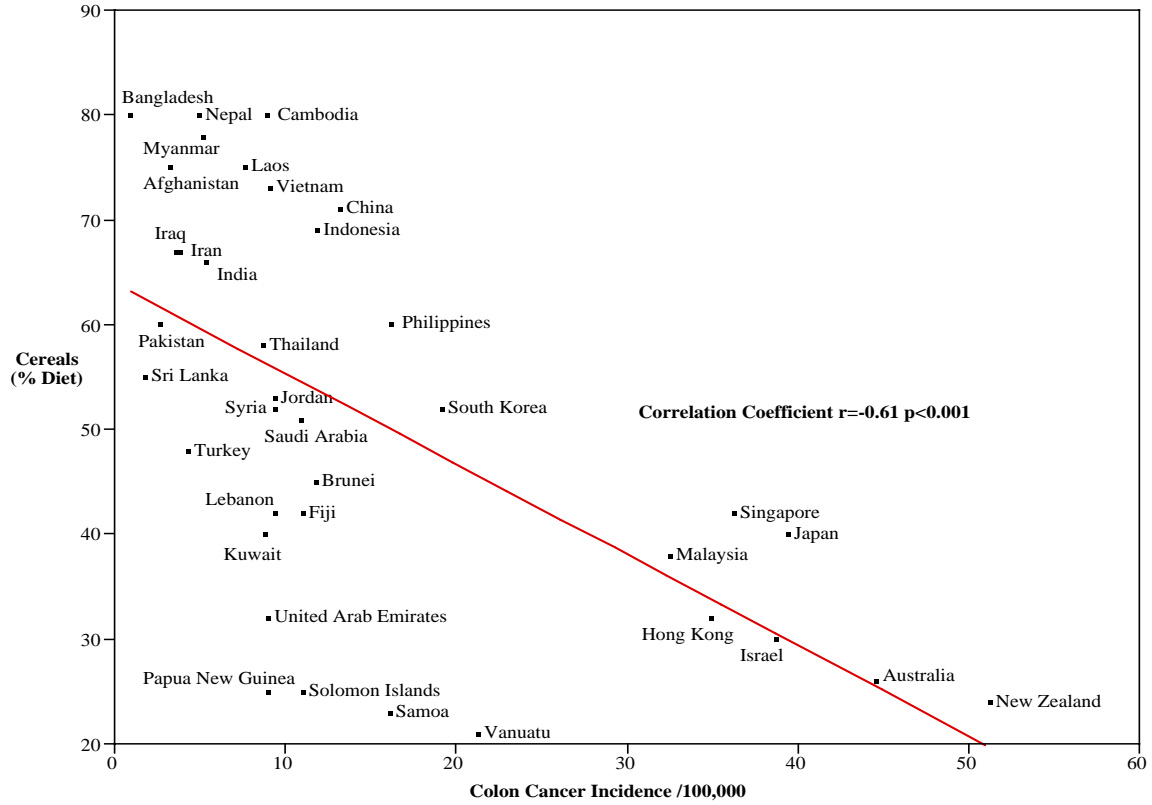


Figure 1. Correlation Between Cereal Percentage of Diet and Colon Cancer Incidence

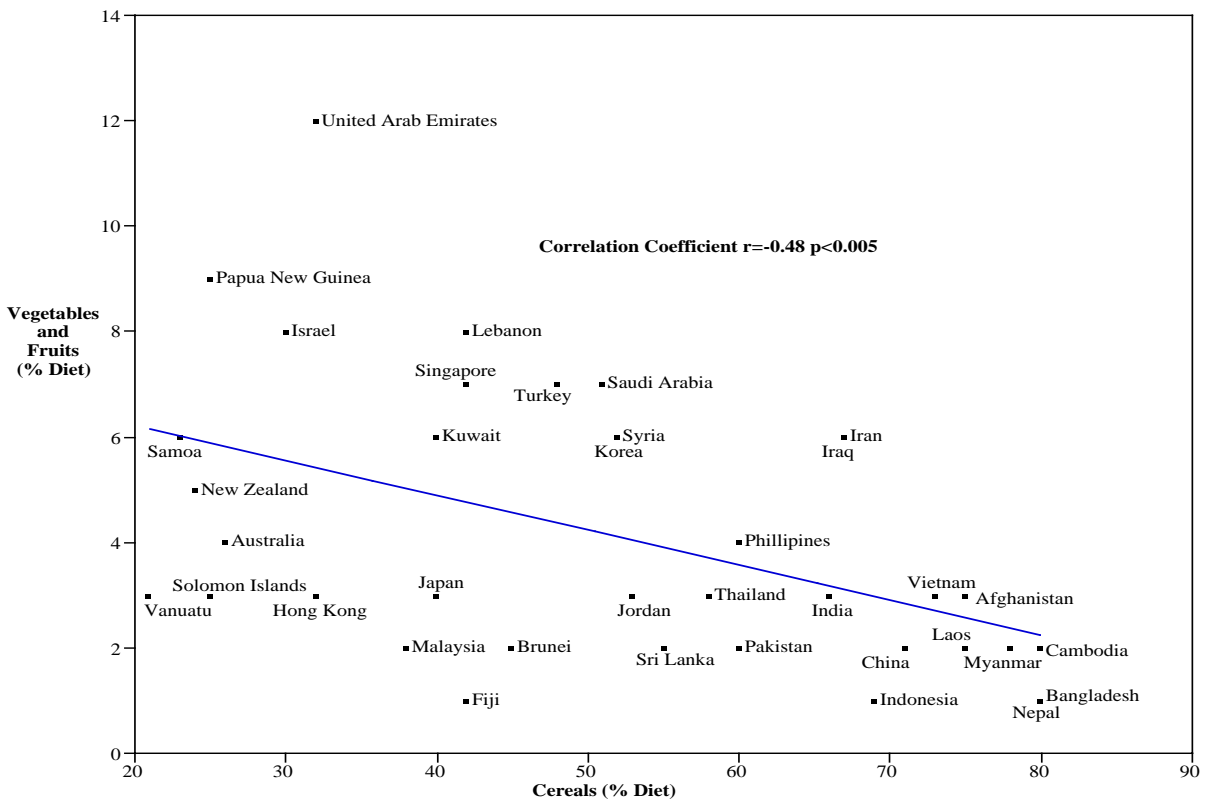
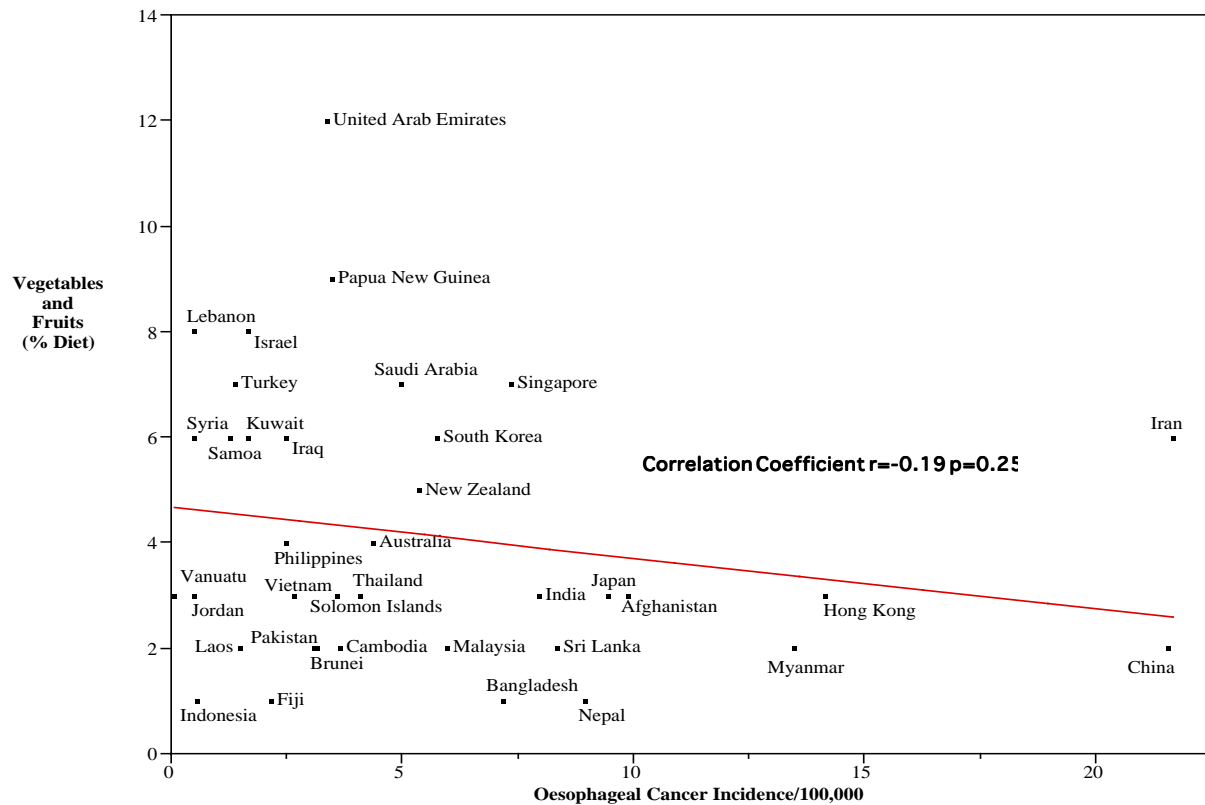


Figure 2. Correlation between Cereal Percentage of Diet and Values for Vegetable/Fruit



**Figure 3. Correlation between Vegetable/Fruit Percentage of Diet and Oesophageal Cancer Incidences**

example, while starch intake appears to be associated with oral cavity cancers, especially high levels being found in both the Solomon Islands and Papua New Guinea, the other two communities in the Pacific with very high starch intake, Vanuatu and Samoa, have relatively low incidences, indicating that comparison of other factors in these communities is warranted.

The great variation in cancer prevalence across the Asian Pacific can clearly be of great assistance regarding assessment of interactions between risk factors. It may be that the geographical links for specific cancers, like squamous cell carcinomas of the upper alimentary and respiratory tracts, will allow descriptive epidemiological comparisons of populations to give clues to the operation of risk or protective influence (Moore et al., 1998a; 1999a; 1999b) but these run the risk of being criticized as too simplistic or on the basis of the 'ecological fallacy' (Greenland and Robins, 1994). However, as pointed out by Cohen (1994) this latter only appertains to situations where average exposure to a group of people does not determine their average risk. When comparing populations for incidences of a particular cancer we are viewing the aggregate effects of a large number of factors. Obtaining proof of one shared risk factor under these conditions is clearly impossible but the approach may offer the same capacity for generating circumstantial evidence as other epidemiological exercises. To paraphrase Cohen (1994), automatic rejection of ecological studies without due attention to their individual merits and demerits is not defensible. The 'eco-epidemiology'

proposed by Susser and Susser (1996) with its emphasis on many levels of organization and interplay of molecular, cellular, tissue and societal factors, is worthy of particular attention. As argued by Pearce (1996) and more recently by McMichael (1999), epidemiology needs to evolve to provide insights into the complex social and environmental systems that are the background to disease. Comparisons of different countries and cultures in the Asian Pacific area, possibly in conjunction with data collected by the IARC/WHO (Parkin et al., 1997), could clearly be of great assistance in this endeavour, facilitating understanding of neoplasia and how it is to be prevented. In this context the call by Aydemir (2002) for standardized dietary assessment is of particular importance.

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## CORRIGENDUM

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The Managing Editor would like to extend sincere apologies to Drs Yuji Kurokawa and Dai Nakae, respectively President of the Sasaki Institute and Chief of its Department of Pathology, for the unfortunate misspelling of their names in the Cancer Research Institute report which appeared in APJCP Volume 2, pages 3-4.