Chapter 8
Summary of data

Definitions and classifications for fruit and vegetables

Although botanical definitions for fruit and vegetables are more precise than culinary definitions, the latter are based on cultural uses of foods and are more commonly used by researchers and understood by participants in epidemiological studies. The culinary term *fruit and vegetables* generally refers to edible plant foods with the exclusion of cereal grains, nuts, and seeds. Also excluded are plant parts used to make liquid infusions (tea leaves and coffee and cacao beans) and plant parts used as herbs or spices. The culinary term *fruit* refers to the part of a plant that contains the seeds and pulpy surrounding tissue and has a sweet or tart taste. Fruits are most often used as breakfast beverages, breakfast or lunch side-dishes, snacks or desserts. Plant parts used as *vegetables* include stems and stalks, roots, tubers, bulbs, leaves, flowers, some fruits, and pulses. Vegetables are consumed raw or cooked with a main dish, in a mixed dish, as an appetizer or in a salad.

Subgroup classifications for fruit and vegetables relate to growing conditions, fruit development from flowers, classes used for national food supply or consumption data, botanical families, plant parts and colour. Some aspects of the latter three types of classification have been used to collect and report information in epidemiological studies. Examples of these types of grouping include dark green leafy vegetables (spinach); cruciferous vegetables (cabbage, broccoli); citrus family fruits (orange, tangerine); and *Allium* family bulbs (garlic, onion). The definitions and classification of fruit and vegetables are not precise and differ between dietary assessment instruments (e.g., potatoes or mushrooms may or may not be included), depending on the purposes of the study and the dietary patterns of the population being evaluated.

Measuring intake of fruit and vegetables

Methods for estimating dietary intake of fruit and vegetables include household measures of food availability, questionnaire measures of usual intake and methods for recording actual intake. These methods are used for various purposes including nutritional surveillance, epidemiological research and methodological research for validation of other dietary methods as well as clinical assessment and programme evaluation.

Household measures are used to estimate intake for nutritional surveillance and monitoring and provide data on the availability and per capita consumption of fruit and vegetable intake. Questionnaire methods – food frequency questionnaires (FFQ), and the diet history (DH) – have been the most commonly used methods to assess usual dietary intake at the individual level in cancer epidemiology cohort and case–control studies. Recording methods, 24-h recalls and food records are used in research studies and in national nutrition monitoring and to validate questionnaire methods.

Because of the large intra-individual variation in daily food intake, accurate quantification and classification of individual exposure is complex and susceptible to measurement error. The FFQ and DH are designed to estimate usual intake, to minimize the effect of intra-individual variation and provide a means to rank individuals in epidemiological analyses. In cohort studies, the aim is to assess recent habitual diet. In case–control studies, the aim is to assess habitual diet during a reference period before the onset of disease. There are large differences between epidemiological studies in the FFQ and DH used to estimate fruit and vegetable intake, in terms of (1) the fruits and vegetables included on the questionnaire, (2) how the instrument is structured, (3) the number of questions, (4) the method used to address portion sizes, and (5) the fruit and vegetable categories used in analysis.

Consumption of fruit and vegetables and relevant policies

There is a remarkable scarcity of nationally representative data on fruit and vegetable consumption, especially for developing countries. Also, the data are very diverse in quality regarding the level of representativity of the study groups, the methods used to assess intake and the format of the available data, both in terms of the age groupings and the food categories. Confusion also exists in the classification of the individual food items and the time frames of the surveys are very diverse.

It is clear nevertheless that there is remarkable diversity in the overall amounts consumed and in the proportions of fruit to vegetables. The diversity is at all levels, between individuals, between socio-cultural-economic groups within a given country, and most of all between countries. Some of the most affluent developed countries have relatively low overall intake of fruit and vegetables, such as the European Nordic countries and the USA. An age-associated positive trend in fruit and vegetable consumption seems to exist, but is not seen consistently. There are associations between fruit and vegetable intake and income, as well as with work category, attained level of education and ethnic group. These stratifying parameters are, of course, interrelated, and therefore may confound or magnify relationships to a variable extent.

Information for developed countries derives mainly from the FAO food balance sheets, with additional data from national surveys in a few countries. In general, levels of consumption are strikingly low in sub-Saharan Africa – where a large part of the fruit category is represented by bananas – and in Asia, intermediate in Central and South America, while in North Africa and the Near East, consumption of fruit and vegetables is close to that of the western, industrial areas of the world. The trend in availability of fruit and vegetables over the period 1961 to 2000 shows little change or even a decline in most of sub-Saharan Africa, while elsewhere there have been increases of variable degree.

Nutrition and health research, food policies and nutrition programmes have changed focus in the last hundred years. The early 1900s focused on identifying and preventing nutrient deficiency diseases and determining nutrient requirements. More recently, investigations have turned to the role of diet in maintaining health and reducing cancer, heart disease, osteoporosis and other noncommunicable diseases. During the past 25 years, international and national health agencies have established priorities for diet and cancer research and prevention efforts. These in turn influenced development of international and national recommendations for dietary intake and dietary guidance. The World Health Organization recently concluded that fruit and vegetables are important in health maintenance and nutrition security and recommended for adults an intake of at least 400 grams of fruit and vegetables per day. Concordant recommendations for fruit and vegetable intake have been published by several organizations recommending that at least five servings or 400 grams of fruit and vegetables be consumed per day.

National and regional health organizations have translated these international policy statements into food-based dietary guidelines that reflect the cultural food patterns and the prevalence of noncommunicable diseases in individual populations. Food guidance recommendations have led to policies and programmes for public education, nutritional surveillance, nutrition campaigns, labelling of foods and food safety. Globally there have been many campaigns and initiatives aimed at increasing fruit and vegetable intake. Some 200 nations have established food and nutrition plans and many have food-based dietary guidelines that include recommendations for fruit and vegetable intake. Strategies for increasing fruit and vegetable intake include efforts at the levels of health facilities, schools, workplaces and commercial activities.

Cancer-preventive effects

Human studies

Studies were included in the evaluation if the reports provided estimates of risk for total fruit or for total vegetable consumption, and 95% confidence intervals were available; measurement error, confounding, and selection and recall bias in case–control studies were also considered. Ecological studies were not considered in the evaluation as they were deemed to be insufficiently informative.

Estimates of a weighted mean of the reported relative risks are provided. If a study report included estimates for different sub-groups, e.g., males and females, these were both included. These weighted means must be interpreted recognizing that they do not represent the result of a formal meta-analysis, and that the contrasts of high versus low consumption are not consistent between studies.

A minority of the epidemiological studies also investigated associations between combined fruit and vegetable intake in relation to cancer risk. Overall, this did not alter the conclusions.

Oral cavity and pharynx

Most studies conducted on oropharyngeal cancer risk in relation to fruit and vegetable consumption have been hospital-based case–control studies. For the 10 evaluable case–control studies of fruit consumption, the mean relative risk for high versus low consumption was 0.45 and the range 0.10–0.70. Despite the relatively good agreement between the results, doubt remains as to whether residual confounding due to smoking habits and alcohol drinking or socioeconomic factors, recall bias among the cases, and selection bias in the control group might account for these findings.

Vegetable intake was also consistently inversely associated with risk of oropharyngeal cancer. For the seven evaluable case–control studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.49 and the range 0.19–0.80. As for fruit, the possibility remains that these results are due to residual confounding by smoking and alcohol drinking as well as socioeconomic status, or to recall or selection bias.

There are no consistent findings of an inverse association of salivary gland and nasopharynx cancer with fruit or vegetable consumption.

Oesophagus

In one cohort study, an inverse association between fruit consumption and mortality from oesophageal cancer was reported. Among 16 evaluable case–control studies of fruit consumption, the mean relative risk for high versus low consumption was 0.54 and the range 0.14–1.50.

Vegetable intake was also often significantly inversely related to risk for this cancer site. For 10 evaluable case–control studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.64 and the range 0.10–0.97.

A recent meta-analysis also found inverse associations for fruit and for vegetables. The set of studies used in the meta-analysis was not completely identical with the studies evaluated here.

The studies did not indicate gender-specific effects of fruit or vegetable consumption. The studies used for evaluation were underpowered to detect effect modification by strata of smoking and alcohol consumption. Thus specific effects on smokers or alcohol drinkers could not be evaluated.

It remains possible that some or all of the observed associations resulted from selection bias, recall bias or residual confounding due to insufficient control for smoking history, history of alcohol drinking, or other factors associated with the occurrence of oesophageal cancer.

Stomach

The association between intake of total fruit and risk of gastric cancer was evaluable in 10 cohort and 28 case–control studies. The mean relative risk for high versus low consumption was 0.85 and the range 0.55–1.92 in cohort studies and 0.63, range 0.31–1.39, in case–control studies.

In 25 studies (five cohort and 20 case–control), the association between intake of total vegetables and risk of gastric cancer was evaluable. The mean relative risk for high versus low consumption was 0.94 and the range 0.70–1.25 for cohort studies and 0.66 (range 0.30–1.70) for case–control studies. Most of the case–control studies adjusted for more potential confounders than the cohort studies, but many did not provide data on total fruit and total vegetable consumption.

The reason why case–control studies were more likely to show inverse associations is not clear. Case–control studies may be affected by recall bias; further, people with preclinical symptoms of stomach carcinoma or stomach disorders may have changed their dietary habits before the diagnosis.

Colon and rectum

For the 11 evaluable cohort studies of fruit consumption, the mean relative risk for high versus low consumption was 1.00 and the range 0.50–1.60. For the nine evaluable case–control studies, the mean relative risk for high versus low consumption was 0.87 and the range 0.30–1.74. A recent meta-analysis showed a small statistically significant reduction in risk across case–control studies and a small non-significant reduction across cohort studies. Among the cohort studies, the small reduction in risk was restricted to women.

For the 10 evaluable cohort studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.94 and the range 0.72–1.78. For the 13 evaluable case–control studies, the mean relative risk for high versus low consumption was 0.63 and the range 0.18–1.29. A recent meta-analysis showed a substantial reduction in risk across case–control studies, but only a small non-significant reduction in risk across cohort studies.

It is not possible to rule out the possibility that bias affects the results in two ways. Recall and selection bias in the case–control studies and confounding in both cohort and case–control studies could be producing artefactual inverse associations.

Liver

One cohort study in Japan considered liver cancer mortality and fruit consumption and found no evidence of an inverse association. Only one case–control study was evaluable and showed no effect.

The only evaluable cohort study on vegetable consumption and risk of liver cancer found significant inverse

associations. The evaluable case–control study showed no association. One case–control study found a significant inverse association for fruit and vegetables combined.

Biliary tract

One cohort study showed no significant effect of fruit consumption on risk of gallbladder cancer. One case–control study showed a significant association between fruit and vegetable consumption and risk of gallbladder cancer.

Pancreas

In all three evaluable cohort studies of fruit consumption, inverse associations were found, but none were significant. For six evaluable case–control studies, the mean relative risk for high versus low consumption was 0.72 and the range 0.07–0.92.

In two evaluable cohort studies of vegetable consumption, non-significant inverse associations were found. For five evaluable case–control studies, the mean relative risk for high versus low consumption was 0.80 and the range 0.32–1.03.

There is concern over studies in which large numbers of proxies of cases were interviewed, as well as those that excluded deceased cases. Further, many of the inverse associations were found in studies where the response rate for controls was low.

Larynx

Studies on larynx cancer were conducted in Europe, Asia and South America. For four evaluable case–control studies on fruit consumption, the mean relative risk for high versus low consumption was 0.63 and the range 0.38–0.80. For six evaluable case–control studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.49 and the range 0.17–1.1.

The majority of the studies were hospital-based, but there was one large population-based case–control study. Control for smoking was rather crude and incomplete in the early studies; more recent studies have used more elaborate models and also observed inverse associations with fruit and vegetable intake. Only one study addressed associations between fruit and vegetables and larynx cancer in subgroups of smoking and alcohol intake. Odds ratios for fruit became weaker in these subgroups, which might indicate residual confounding by smoking and alcohol. The possibility of recall and selection bias in these case–control studies cannot be excluded.

Lung

Studies were conducted in North America, Europe, Australasia, Japan and South America. For 13 evaluable cohort studies of fruit consumption, the mean relative risk for high versus low consumption was 0.77 and the range 0.26–1.22. For 21 evaluable case–control studies, the mean relative risk for high versus low consumption was 0.70 and the range 0.33–2.04.

For 11 evaluable cohort studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.80 and the range 0.47–1.37. For 18 evaluable case–control studies, the mean relative risk for high versus low consumption was 0.69 and the range 0.30–1.49.

The latest results from cohort studies and a recent meta-analysis suggest that the inverse association is stronger for fruit than for vegetables. Studies vary in the number of items included in the 'total' fruit or vegetable group. There was no clear difference in results between men and women, between hospital- and population-based case–control studies, nor between morphological categories of lung cancer. The strength of the association was smaller for cohort studies than for case–control studies, leaving the possibility of recall and selection bias in the case–control studies.

Because smoking is a strong risk factor for lung cancer, and smoking and fruit (and, to a lesser extent, vegetable) consumption are inversely associated, appropriate control for confounding by smoking is crucial. While the newer cohort studies have attempted to control for confounding by smoking much better than earlier cohort studies, residual confounding by smoking cannot be excluded and cohort studies often fail to capture changes in smoking and diet after the baseline measurement. Subgroup analyses among categories of smoking showed inverse associations in never-smokers (often non-significant) in the cohort studies. However, case–control studies among never- or non-smokers were not entirely consistent in showing an inverse association with fruits or vegetables.

Breast

About 30 epidemiological studies have examined the association between total fruit and total vegetable consumption during adulthood and the risk of breast cancer in women.

For six evaluable cohort studies of fruit consumption, the mean relative risk for high versus low consumption was 0.82 and the range 0.74–1.08. For 12 evaluable case–control studies, the mean relative risk for high versus low consumption was 0.99 and the range 0.57–1.82.

For five evaluable cohort studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.94 and the range 0.64–1.43. For 12 evaluable case–control studies, the mean relative risk for high versus low consumption was 0.66 and the range 0.09–1.40.

A pooled analysis of eight cohort studies which included some of the

studies considered above found non-significant weak inverse associations between either fruit or vegetable consumption and the risk of breast cancer. In contrast, two meta-analyses of case–control studies (some studies were included in both meta-analyses) found approximately 10–20% reductions in the risk of breast cancer with increasing vegetable consumption; however, in both meta-analyses there was significant heterogeneity across the studies. There was little suggestion that associations differed by menopausal status. Because positive associations have been reported rarely for high fruit and vegetable consumption, and fruit and vegetable consumption is measured with error in epidemiological studies, the Working Group could not exclude the possibility that fruit and vegetable consumption may be associated with a slight decrease in risk of breast cancer. In addition, few studies have evaluated the influence of fruit and vegetable consumption during childhood and adolescence on the subsequent risk of developing breast cancer and of effect modification by other risk factors.

Associations between fruit or vegetable consumption and the risk of breast cancer in men have rarely been examined.

Cervix

There have been no cohort studies of fruit and vegetable consumption and risk of cervix cancer.

The case–control studies were not completely consistent and there is little evidence for a strong effect of either fruit or vegetable consumption.

Because of the strong relationship of human papillomavirus (HPV) with risk for this disease, there is concern about appropriate control for possible confounding or modifying effects of this infection. Only one study has examined risk restricted to women who were HPV-positive; results were similar when both HPV-positive and -negative controls were included or when controls were limited to women with HPV infections.

Endometrium

The associations between intake of fruit and vegetables and risk of endometrium cancer have been studied only in case–control studies.

For seven evaluable case–control studies of fruit consumption, the mean relative risk for high versus low consumption was 1.03 and the range 0.67–1.97. For five evaluable case–control studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.75 and the range 0.65–1.00.

Fruit and vegetable intake combined was inversely associated in one cohort study, and in three case–control studies. Body mass index is an important known risk factor for endometrial cancer which was adjusted for in most, but not all, studies.

Ovary

The number of studies available on fruit consumption was limited and the results were inconsistent.

For vegetable consumption, an inverse association was found in two cohort studies and in five (three of which significant) out of six case–control studies.

In one case–control study, there was an inverse association with combined fruit and vegetable intake.

Prostate

For this site, there are no established risk factors other than age, family history and ethnic group. Hence generally confounding by non-dietary factors is not an issue. There is a possibility of detection bias, due to the use of PSA testing, but this would not have affected the majority of the studies reviewed.

For eight evaluable cohort studies of fruit consumption, the mean relative risk for high versus low consumption was 1.11 and the range 0.84–1.57. For nine evaluable case–control studies, the mean relative risk for high versus low consumption was 1.08 and the range 0.40–1.70.

For six evaluable cohort studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.95 and the range 0.7–1.04. For nine evaluable case–control studies, the mean relative risk for high versus low consumption was 0.90 and the range 0.6–1.39.

The results for fruit are consistent and suggest that high fruit consumption does not reduce prostate cancer risk. The increased risk seen in some studies could be due to bias associated with detection in health-conscious men. For vegetables, the majority of studies have reported a slight, not significant lower risk for high consumption; vegetable consumption is measured with substantial error in epidemiological studies, so the Working Group could not exclude the possibility that vegetable consumption may be associated with a slight decrease in the risk of prostate cancer.

Testis

There were no cohort studies of testis cancer, and the two available case–control studies did not show significant associations.

Bladder

For five evaluable cohort studies of fruit consumption, the mean relative risk for high versus low consumption was 0.87 and the range 0.63–1.12. For four evaluable case–control studies, the mean relative risk for high versus low consumption was 0.74 and the range 0.53–0.95.

For three evaluable cohort studies of vegetable consumption, the mean relative risk for high versus low con-

sumption was 0.94 and the range 0.72–1.16. For the three evaluable case–control studies, the mean relative risk for high versus low consumption was 0.89 and the range 0.66–1.04.

Most studies appropriately adjusted for potential confounding by age, gender, energy intake and smoking. In one cohort study, the estimates were stratified by smoking habits and an inverse association was found, mainly in current heavy smokers.

Kidney

One of the two cohort studies did not show an association with total fruit or vegetable intake. The other, although indicating an inverse association with total fruit, had too few cases to be informative.

For seven evaluable case–control studies of fruit consumption, the mean relative risk for high versus low consumption was 0.76 and the range 0.20–1.20.

For four evaluable case–control studies of vegetable consumption, the mean relative risk for high versus low consumption was 0.86 and the range 0.30–1.60.

The case–control studies were conducted in Australia, China, Europe and the USA and all cases were histologically confirmed. Most studies used population controls and response rates were relatively high. Potential confounding by body mass index and smoking was addressed in all analyses. However, recall bias cannot be excluded as an explanation of the results.

Brain

Three case–control studies of adult and five of childhood brain cancers have considered fruit and vegetable consumption, usually as a part of studies with other primary dietary hypotheses. All studies in adults and three studies in children showed inverse associations with fruit and/or vegetable consumption.

Thyroid

There were no cohort studies of thyroid cancer, and none of the three available case–control studies found a significant association with total fruit and vegetable consumption.

Non-Hodgkin lymphoma

In both of two cohort studies of fruit consumption, a non-significant inverse association was found. There was only one casecontrol study, which showed no evidence of an inverse association.

Among three cohort studies of vegetable consumption, a significant inverse association was seen in one. There was only one case–control study, which showed no evidence of an inverse association.

Leukaemia

Only one cohort study that considered green-yellow vegetables but not fruit consumption was available. No inverse association with risk was found.

Preventable fraction

The Working Group estimated that the preventable fraction for low fruit and vegetable intake would fall into the range of 5–12%. This is only a crude range of estimates and the proportion of cancers that might be preventable by increasing fruit and vegetable intake may vary beyond this range for specific cancer sites and across different regions of the world.

Intermediate markers of cancer

In experimental dietary studies in humans relying on intermediate endpoints related to disease risk, individual fruits and vegetables have been shown to modulate biological processes relevant to cancer, including biotransformation enzymes, antioxidant enzymes, oxidative damage to macromolecules, DNA adducts. Results are sometimes inconsistent, depending on the fruit or vegetable consumed, and the type of intervention which may differ greatly in duration, sample size and study design.

Experimental studies

Cancer and pre-malignant lesions

A study in rats with complete pathological examination showed that mixed fruits and vegetables did not significantly affect the spontaneous rates of total cancer or of cancer in any organ. A few well controlled rodent studies have provided evidence for preventive effects on carcinogen-induced colon cancer or adenomas of mixed fruits and vegetables at levels relevant to human dietary intake. In an additional study using tumour-prone transgenic mice, mixed fruits and vegetables also decreased the multiplicity of intestinal polyps in males fed a low-fat diet.

Other animal experiments have evaluated the efficacy of individual fruits or vegetables in decreasing cancer risk. These experiments have generally been performed with doses of fruits or vegetables that were high compared with human dietary intakes. Most of the 30 studies conducted in four different animal species and in different organs provided good evidence that high doses of individual fruit and vegetables can decrease tumour yield after a challenge by chemical carcinogens. The majority of the tumour-preventive effects have been observed in the colon, mammary gland or oesophagus. Some evidence also points to the potential of individual fruits and vegetables at high doses to decrease incidence of cancers of the bladder, liver, oral cavity and skin.

The evidence for antitumorigenic effects during the initiation phase is strong, whereas the evidence for late effects in carcinogenesis by fruit and vegetables is weaker, with mostly negative results from animal studies.

Intermediate markers of cancer

Mixed fruits and vegetables at levels relevant to human dietary intakes increased the activity of both phase I and phase II xenobiotic-metabolizing enzymes in rat liver. High doses of individual fruits or vegetables, including broccoli, Brussel sprouts and garlic, mainly induced phase II enzymes. An increase in phase II enzyme activities and a decrease in DNA damage were observed to parallel decreased tumour yields in a dose-dependent manner in a few studies. Some effects have been observed on other potential early risk factors for cancer, including carcinogen–DNA binding, lipid oxidation, DNA damage and mutation.

Mechanisms of cancer prevention

Extensive study of fruit and vegetables in human intervention studies and in animal models has provided a wealth of information on the variety of mechanisms by which a diet high in fruit and vegetables may contribute to reduced cancer risk.

Fruit and vegetables, at moderate intake levels, can modulate phase I and phase II enzymes in both animals and humans. Statistically significant phase II enzyme induction has been observed in human volunteers consuming single vegetables (most experiments were performed with *Brassica* vegetables). It is therefore likely that modulation of xenobiotic-metabolizing enzymes, in particular phase II enzymes, could contribute to prevention of human cancer. Enzyme induction is dose-dependently linked in animal studies with a decrease in genetic damage and tumorigenesis.

While the evidence is inconsistent that fruit and vegetables decrease direct oxidative DNA damage, evidence is more consistent for a decrease in lipid oxidation, a source of indirect oxidative damage to DNA. Nonetheless, the evidence linking direct or indirect oxidative DNA damage with risk of cancer is weak. The evidence for other mechanisms, including inhibition of endogenous formation of carcinogens, carcinogen–DNA binding, cytogenetic damage and post-initiation effects, by fruits and vegetables is weak.

In conclusion, the best, but still tentative, evidence for a mechanism of cancer prevention by fruit and vegetables is related to xenobiotic-metabolizing enzyme modulation, while antioxidant mechanisms are less well substantiated.

Associations with diseases other than cancer

Following a number of earlier ecological studies, analytical observational investigations, in particular several cohort studies, have shown inverse associations between consumption of fruit and vegetables and risk of coronary heart disease or stroke. The results of these studies are not entirely consistent; however the inverse associations found in the large cohorts, better controlled for confounding factors, provide evidence supporting a protective effect. Results from randomized clinical trials of diets rich in fruit and vegetables indicate the efficacy of such diets in lowering systolic and diastolic blood pressure over periods of weeks and months.

Two recent randomized trials show that lifestyle and diet changes, including the substitution of energy-dense dietary fats with fruit and vegetables, improve glucose tolerance and prevent occurrence of type 2 diabetes. In two large prospective studies, frequent intake of fruit and vegetables has been associated with decreased risk of senile cataract. Less data suggesting associations with fruit and vegetables are available for other chronic conditions such as osteoporosis, senile macular degeneration, Alzheimer disease and Parkinson disease.

Carcinogenic effects

There is no evidence from human studies of carcinogenicity of consumption of fruit and vegetables as a class. In one study in rats, a fruit and vegetable mixture fed at dietary levels relevant to humans did not affect spontaneous cancer incidence in any organ, after complete pathological examination. As a part of western-type diets, mixed fruit and vegetables at dose levels relevant to human exposures had the ability to increase intestinal tumours in one rat experiment and in one transgenic mouse experiment. There is no published evidence for a net increase in tumours after dosing with any individual fruit or vegetable at high doses in rodents.

Toxic effects

The relatively few adverse effects reported for individual fruits and vegetables were caused by specific components in a few kinds of fruit and vegetables and cannot be regarded as a general adverse effect of these classes of food.