case-control studies, and the mechanistic evidence is speculative. There is limited evidence that greater adult attained height, or the factors that lead to it, are a cause of endometrial cancer. The causal factor is unlikely to be tallness itself, but factors that promote linear growth in childhood.

7.12.5.7 Other exposures
Other exposures were evaluated. However, the data were either of too low quality, too inconsistent, or the number of studies too few to allow conclusions to be reached. These were as follows: cereals (grains) and their products; fruits; pulses (legumes); tofu and soya; poultry; fish; eggs; milk and dairy products; coffee; alcohol; carbohydrates; dietary fibre; total fat; animal fats; saturated fatty acids; cholesterol; protein; retinol; beta-carotene; vitamin C; vitamin E; energy intake; and lactation.

7.12.6 Comparison with previous report

7.12.6.1 General
See 7.1.6.1, and box 3.8 in chapter 3.

7.12.6.2 Specific
The finding here on physical activity is new. The evidence on body fatness and on abdominal fatness (not considered separately in the previous report’s matrices) has strengthened.

7.12.7 Conclusions

The Panel concludes:
The evidence that body fatness is a cause of cancer of the endometrium is convincing; abdominal fatness is probably also a cause.
Physical activity probably protects against this cancer.
There is limited evidence suggesting that non-starchy vegetables protect against endometrial cancer, and that red meat, and also the factors that lead to greater adult attained height, or its consequences, are causes of this cancer.

7.13 Cervix

Cervical cancer is the second most common cancer in women worldwide. Around half a million cases were recorded in 2002, accounting for around 10 per cent of all new cases of cancer in women (5 per cent overall). It is most common in Africa, some parts of Asia including India, and in Latin America. It is most common in relatively young women. Five-year survival rates are around 50 per cent. It is the third most common cause of cancer death in women.
Overall, the Panel notes that food and nutrition and associated factors are not significant factors in modification of the risk of cancer of the cervix, although general nutritional status may affect a woman’s vulnerability to infection.
Life events that protect against cervical cancer include having relatively few sexual partners. The reverse also applies. Infection with HPV is a necessary cause of this cancer, and smoking tobacco increases risk.

The Panel judges as follows:
There is limited evidence suggesting that carrots protect against cervical cancer.
In final summary, there is no strong evidence, corresponding to judgements of “convincing” and “probable”, to conclude that any aspect of food, nutrition, and physical activity modifies the risk of cervical cancer.

The cervix is the neck of the womb. The part of the cervix inside the cervical canal is called the endocervix. The part on the outside is the ectocervix. Most cervical cancers start where these two parts meet. There are two main types, squamous cell carcinoma and adenocarcinoma. Occasionally, mixed carcinomas, with features of both types, occur. Approximately 80 per cent of cervical cancers are squamous cell carcinomas, with most of the rest being adenocarcinomas. Both types of cervical cancer are covered in this Report.

7.13.1 Trends, incidence, and survival

Age-adjusted rates of cervical cancer are decreasing, particularly in high- and middle-income countries, although there are insufficient data to derive trends in low-income countries. In high-income countries, the incidence of adenocarcinomas has increased since the 1970s, both absolutely and relative to squamous cell carcinomas. The prevalence appears to be increasing disproportionately in young women.
Cervical cancer is predominantly a disease of low-income countries, with overall rates nearly twice as high in middle- to low- as in high-income countries. Around the world, age-adjusted incidence rates range from more than 40 per 100 000 women in parts of Africa, South America, and Melanesia, to less than 10 per 100 000 in North America and parts of Asia. However, rates are relatively high elsewhere.
in Asia, for example in India and Bangladesh. In the USA, rates are higher among both African-American and Hispanic-American women than in white women. The incidence of many cancers rises with age, but cervical cancer peaks in younger women, between the ages of 30 and 45.6 However, mortality does not follow the same pattern, and rises with age. Most women in high-income countries, and to varying degrees in other countries, have access to preventive screening programmes that are designed to detect precancerous lesions. If these are identified and removed, the incidence of this cancer is reduced. After a screening programme was implemented in the UK in 1988, cervical cancer incidence (age-standardised rate) has fallen by nearly 60 per cent.6 It is generally well accepted that better access to cervical screening programmes worldwide would decrease both the incidence and mortality rates for this cancer.267 More recently vaccination against HPV has become a preventive option.

The overall 5-year survival rate is approximately 50 per cent: 61 per cent in high-income countries compared with 41 per cent in middle- to low-income countries.124 This cancer accounts for somewhat over 4 per cent of all cancer incidence (around 10 per cent in women) but only around 4 per cent of all cancer deaths (just over 9 per cent in women). Also see box 7.1.1.

### 7.13.2 Pathogenesis

Virtually all cervical cancers are associated with HPV infection (see box 7.13.1), and a woman’s nutrition status may influence her susceptibility to this infection.268 However, the majority of women with HPV do not develop cervical cancer. Therefore, HPV infection is a necessary but not a sufficient cause of cervical cancer. Women become susceptible to developing cervical cancer following HPV infection, but other environmental factors are required for the cancer to develop.

These factors may include toxins such as polycyclic aromatic hydrocarbons (see box 4.3.4) from tobacco smoke, food, or other environmental sources, which have been found in the mucus lining the cervix.269
7.13.3 Other established causes

(Also see chapter 2.4 and 7.1.3.1.)

Life events. Early sexual experience and a relatively high number of sexual partners increase the risk and severity of HPV infection, and may be seen as indirect causes of cervical cancer.220 222

Tobacco use. Smoking tobacco makes a woman twice as likely to develop cervical cancer.10 Tobacco by-products have been found in the cervical mucus of women who smoke. The effect of smoking is independent of that of viral infection.10 273

Infectious agents. HPV infection (see box 7.13.1) is a necessary but not sufficient cause of cervical cancer.273 274

Medication. Dethylstilboestrol (a synthetic oestrogen, now withdrawn) used by women during pregnancy is a cause of vaginal and cervical clear-cell adenocarcinoma in their daughters.275

7.13.4 Interpretation of the evidence

7.13.4.1 General
For general considerations that may affect interpretation of the evidence, see chapters 3.3 and 3.5, and boxes 3.1, 3.2, 3.6 and 3.7.

‘Relative risk’ is used in this Report to denote ratio measures of effect, including ‘risk ratios’, ‘rate ratios’, ‘hazard ratios’, and ‘odds ratios’.

7.13.4.2 Specific
Considerations specific to cancer of the cervix include:

Confounding. High-quality studies adjust for HPV infection. Early studies that failed to adjust for HPV status have reduced validity.

7.13.5 Evidence and judgements

In total, 154 publications were included in the SLR for cervical cancer. Fuller summaries of the epidemiological, experimental, and mechanistic evidence are to be found in Chapters 4–6.

The full SLR is contained on the CD included with this Report.

7.13.5.1 Carrots
(Also see chapter 4.2.5.1.3.)
Five case-control studies and one ecological study investigated carrots. All of the case-control studies showed decreased risk for the highest levels of intake compared with the lowest, statistically significant in three. The case-control studies all used hospital-based controls and none adjusted for HPV status. The single ecological study showed non-significant increased risk with high intake of carrots.

Some carotenoids, including beta-carotene and alphacarotene, which are found at high levels in carrots, are precursors of vitamin A. They also have properties independent of their pro-vitamin A activity. Carotenoids are recognised antioxidants, and low blood levels of dietary antioxidants are associated with HPV persistence.276

The evidence, from case-control studies only, is sparse but consistent. There is limited evidence suggesting that carrots protect against cervical cancer.

7.13.5.2 Other exposures
Other exposures were evaluated. However, the data were either of too low quality, too inconsistent, or the number of studies too few to allow conclusions to be reached. These were as follows: non-starchy vegetables; fruits; milk; retinol; vitamin E; alcoholism; body fatness; and adult attained height.

Although data suggest that alcoholism is related to increased risk, the Panel concludes that this is likely to be due to factors other than alcohol intake itself.

7.13.5.3 Exposures as related to non-invasive cancer outcomes
The following exposures were evaluated. However, the data were either too sparse, too inconsistent, or the number of studies too few to allow conclusions to be reached: vitamin A (as beta-carotene, alpha-carotene, or retinol); folate; vitamin C; vitamin E; and lycopene.

7.13.6 Comparison with previous report

7.13.6.1 General
See 7.1.6.1, and box 3.8 in chapter 3.

7.13.6.2 Specific
The previous report found that vegetables and fruits, and carotenoids (not carrots specifically), and also vitamins C and E possibly protect against cervical cancer.

7.13.7 Conclusions

The Panel concludes:

There is limited evidence suggesting that carrots protect against cervical cancer. The evidence is too limited to conclude that any aspect of food, nutrition, and physical activity directly modifies the risk of this cancer.