



Cancer prevention – review paper

Anna Maria Lewandowska^{1,A-B,D-E} , Tomasz Lewandowski^{1,A-B,E} , Marcin Rudzki^{2,E-F} ,
Sławomir Rudzki^{3,E-F} , Barbara Laskowska^{1,B,D,F} 

¹ State School of Technology and Economics, Jarosław, Poland

² Chair and Department of Jaw Orthopaedics, Medical University, Lublin, Poland

³ Chair and Department of General and Transplant Surgery and Nutrition, Medical University, Lublin, Poland

A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation,

D – Writing the article, E – Critical revision of the article, F – Final approval of article

Lewandowska AM, Lewandowski T, Rudzki M, Rudzki S, Laskowska B. Cancer prevention – review paper. *Ann Agric Environ Med.* 2021; 28(1): 11–19. doi: 10.26444/aaem/116906

Abstract

Introduction. Every year there is an increase in the number of cases and deaths due to the majority of cancers. Currently, these diseases constitute the second cause of death in Poland and the USA. The number of cases of malignant neoplasms in Poland has more than doubled over the last three decades. According to the National Cancer Registry, in Poland about 95.5 people per thousand die every year from malignant neoplasms. Current epidemiological data on cancer is worrying because the World Health Organization predicts a significant increase in the incidence of cancer in the general population. This problem, which is significant on the global scale, demands the search for more effective prevention methods so that more and more attention is paid to both primary and secondary prevention. Prevention and early detection of cancer have become a priority for the national health policy of many European countries. Numerous studies around the world prove that reducing the risk of cancer is most effective through adopting a healthy lifestyle, avoiding exposure to carcinogens, combined with regular screening.

Objective. The aim of the study was to review knowledge on cancer prevention, including the latest research results.

Conclusions. Due to the systematic increase in the incidence of cancer, a strong emphasis should be placed on prevention. Preventive actions bring benefits not only to the individual, but are an important aspect of health policy. The importance of primary prevention in relation to healthy people has been demonstrated, including secondary prevention aimed at controlling risk factors in relation to persons exposed to them. The combination of these activities becomes an important element in maintaining the health of the individual, as well as society.

Key words

cancer, Risk Factors, cancer prevention

INTRODUCTION

Every year there is an increase in the number of cases and deaths due to the majority of cancers. Currently, these diseases constitute the second cause of death in Poland and the USA [1]. The conducted analyses show that in Poland in 1990–2018 the main causes of deaths were cardiovascular diseases and cancer, causing about 70% of all deaths. The number of cases of malignant neoplasms in Poland has more than doubled over the last three decades. According to the National Cancer Registry, about 95.5 per thousand people in Poland die every year from malignant neoplasms. At the beginning of the 1990s, malignant tumors accounted for less than 25% of all deaths, at the beginning of the current century they accounted for about 23%, in 2015 26%, and in 2016 they accounted for 27.3% of all deaths [2, 3]. Analysis of the level and trends of Potential Years of Life Lost (PYLL) due to main causes of death in Poland in 2002–2011, taking into account place of residence based on the Central Statistical Office data, shows that PYLL due to cancer among men in rural areas is 19.4%, in urban areas – 20.9%, women in rural areas – 39.9%, and in urban areas – 41.7% [4].

Current epidemiological data on cancer is worrying because the World Health Organization predicts a significant increase in the incidence of cancer in the general population.

In 2025, the number of new cases per year will increase from 14 million to 19 million, in 2030 – to 21.7 million, and in 2035 – up to 24 million [5, 6]. This problem, which is significant on the global scale, demands the search for more effective prevention methods so that more and more attention is paid to both primary and secondary prevention. Prevention and early detection of cancer have become a priority for the national health policy of many European countries [7]. Numerous studies around the world prove that reducing the risk of cancer is most effective through adopting a healthy lifestyle, avoiding exposure to carcinogens, combined with regular screening [8]. On the one hand, it is possible by systematic physical examinations, mass screening tests, preferably in the environment of science, work, life, on the other hand by raising cancer awareness. All preventive measures only make sense if they are carried out continuously [9, 10, 11].

OBJECTIVE

The aim of the study is a review of knowledge on cancer prevention, including the latest research results.

MATERIALS AND METHOD

To review the evidence for the literature search, use was made of PubMed and included articles published between 2000 –

Address for correspondence: Anna Maria Lewandowska, State School of Technology and Economics in Jarosław, Poland
E-mail: am.lewandowska@poczta.fm

Received: 07.11.2019; accepted: 20.01.2020; first published: 12.03.2020

2019. Combinations of the following key-words were used: ‘cancer risk factors’, ‘cancer prevention’, ‘primary cancer prevention’, ‘secondary cancer prevention’ (key words: cancer, risk factors, cancer prevention). From this search, a total of 1,955 potentially relevant articles were identified. This number was reduced to 1,105 articles after screening titles and abstracts. The studies were in English and Polish. After reaching a list of potentially relevant articles, the full text of each paper was appraised, with particular emphasis on articles presenting environmental risk factors for cancer. This was in order to more extensively present study characteristics and results in order to present the information from each study, allowing the reader a more thorough assessment of the current literature to draw interpretations and final conclusions.

RESULTS

More than 80% of cancers are associated with lifestyle, which is why health-promoting behavior is most important for cancer prevention, influenced by – on the one hand – health knowledge and its use in everyday life, and on the other – effective motivation. There is a consensus that better access to information on cancer prevention can increase awareness in the field of prevention and influence the formation of pro-health activity. Knowledge of cancer risk factors allows taking preventive action. However, the final assessment of the effectiveness of such activities depends on the proper recognition of the subjects’ motivation to obtain information on health and its determinants [12, 13]. According to the concept of health by the World Bank, the health of the population depends primarily on the education of society. Acquiring appropriate education, and thus acquiring the relevant knowledge, depends not only on the quality of the education system, but also on the access to innovative methods of teaching and information acquisition. It has been documented that young people often use the Internet to search for health information, but only one-in-four declares that the information obtained this way has changed their own health behaviours [14, 15, 16]. Research results confirm that health-related information provided online helps to implement intervention programmes aimed at changing inappropriate behaviours, as well as increasing participation in preventive examinations [17, 18, 19]. It should be noted that the transferred knowledge should be adapted to the age, needs, attitudes and skills of the client or patient, while educational programmes should be conducted both in the traditional and online form. They should cover the entire society, should be planned for decades and consistently implemented throughout this period to bring measurable and intended effects. Without this determination in implementation, it will never be possible to realize the hopes placed in preventive actions [9, 20, 21, 22].

Over the past decade, epidemiological studies of various populations have confirmed the importance of proper diet, including specific nutritional factors, in preventing and controlling the incidence of non-communicable diseases; thus enabling nutritional intervention and changing their impact on the human body [23]. Epidemiological studies have shown that in populations whose diet contains high amounts of refined sugar, salt, animal fat, red and processed meat, an increased risk of cancer is observed. According to

the latest recommendations of the World Cancer Research Fund, consumption of red meat should be limited to 500 g per week, and a very small amount of this may be sausage. The EPIC study showed that people who ate about 80 g of red meat on a daily basis had a one-third increase in their risk of developing colorectal cancer. There is more and more evidence confirming a similar correlation for pancreatic and stomach cancer. Red meat and sausages contain particularly large amounts of heme, which can irritate and damage the mucosa of the large intestine, stimulate bacteria living in this part of the digestive system, and also stimulate bacteria living in this part of the digestive system to produce carcinogens [24, 25, 26]. According to the AICR Report of the American Institute for Cancer Research, consumption of sugar and sugar-sweetened beverages may not affect appetite in the same way as food. They provide excess calories, not only causing weight gain, overweight and obesity, but also show an indirect correlation to the occurrence of cancer [24].

Regular consumption of fruit and vegetables significantly contributes to reducing the risk of developing stomach, colorectal, rectal and breast cancer. Europe-wide research by the European Prospective Investigation into Cancer and Nutrition (EPIC) has shown that eating very large amounts of vegetables and fruit – 400–800 g daily, can reduce the risk of developing cancer of the mouth, throat, larynx and esophagus by up to a third, and stomach and lung cancer – by a quarter [24]. Vegetables and fruits are a source of carotenoids, folic and ascorbic acid, as well as bioactive ingredients such as phenols, flavonoids, isothiocyanates and indoles, fibres that are anticarcinogenic [27, 28, 29]. Consumption of legumes, especially cooked beans, protects and reduces the risk of developing colorectal cancer due to the presence of saponin, oligosaccharides, phenolic compounds and isoflavonoids [30, 31]. Epidemiological studies show a clear relationship between a diet high in garlic and a low incidence of gastrointestinal neoplasms. Onion, leeks and chives also have anti-cancer effects [32, 33]. Acting at the stage of initiation and promotion, they inhibit tumour growth. They have antiproliferative and, by activating enzymes – catalase, superoxide dismutase or glutathione reductase – also antioxidant properties. Garlic cloves contain many chemical compounds, among them diallyl sulfide (DAS), diallyl disulfide (DADS), diallyl trisulfide (DATS), dipropyl sulfide (DPS), dipropyl disulfide (DPDS). Based on research conducted by the National Cancer Institute (NCI) in the United States, garlic was identified as one of the vegetables with the greatest potential anticancer effect, and the Council of Europe classified DADS as a substance that can be used as a food additive. However, it has not been specified in what form it should be consumed to achieve the maximum therapeutic effect [34, 35, 36].

Recommendations for primary prevention include in the first place chemoprevention, which, as it turns out, can have a positive effect on stopping or slowing down the early stages of the carcinogenesis process. Plant polyphenols, one of the antioxidant groups, play a significant role in prevention. Resveratrol is a polyphenol which belongs to flavonoids. Its largest amounts can be found in red grapes, berries, nuts and cocoa, and the effect on the body is anti-inflammatory, antioxidant, stimulates natural antioxidant cell protection systems, plays a protective role against uncontrolled cell division, inhibits angiogenesis, intensifies the process of apoptosis, and thus demonstrates preventive action at three

levels of cancer development: initiation, promotion and progression [37, 38].

Another antioxidant and carotenoid is lycopene found in tomatoes and their preparations, a substance with multidirectional, antioxidant, anti-inflammatory and immunomodulatory activity. It induces the process of apoptosis but inhibits angiogenesis and cell proliferation. A positive dependence has been demonstrated between lycopene intake and the development of prostate cancer. It is particularly beneficial in a concentration of 1–4 μM , reducing the risk of developing prostate, lung, breast cancer, gastrointestinal cancer and leukemia [35, 39, 40]. Quercetin is one of the most common plant flavonoids. Its sources are chokeberry, blueberry, peach, broccoli, Brussels sprouts, onion, oregano, tarragon and coriander. It is a substance that induces the process of apoptosis, stops or slows the progression of tumours, and is antiproliferative [41, 42]. Genistein is a natural plant compound, one of the main soy isoflavones, which has anti-cancer properties. The results of epidemiological studies suggest that consuming soybean seeds, which are the source of this isoflavone, may contribute to reducing the incidence of breast, colon, prostate, thyroid and head and neck cancers [43].

Undoubtedly, carotenoids that give plants a yellow to red colour, are of great importance in cancer prevention. Chemo-preventive effects have been demonstrated for vitamin A, β -carotene, fenretinide, a synthetically derived derivative of vitamin A. According to a Report issued by the World Cancer Research Fund (WCRF) and the American Institute for Cancer Research (AICR), food products containing carotenoids are protective against oral, throat, larynx and lung cancers. A positive correlation has also been demonstrated between β -carotene intake and reduced risk of developing esophageal carcinoma [26, 35, 44]. Randomized research conducted in China among approx. 30,000 people at risk of oesophageal and gastric cancer showed that the lowest mortality in the studied tumours occurred when β -carotene and selenium were taken simultaneously [45, 46, 47]. Research on vitamin E supplementation has shown that high doses of this vitamin can reduce the risk of bladder cancer. The benefits of long-term use of vitamin E have been demonstrated in a survey conducted in 2001 in the USA – a 10-year intake of vitamins C and E contributed to a reduction in the incidence of colon and rectal cancer [48, 49, 50, 51]. However, according to recent IARC reports on beta-carotene and vitamin E supplementation, β -carotene supplements increase the risk of lung cancer in smokers, while vitamin E supplements do not provide general anti-cancer protection [24].

Vitamin D also exhibits pro-apoptotic and antiproliferative properties in relation to cancer. Studies show that people living at higher latitudes are more likely to develop cancers such as Hodgkin's disease, pancreatic, colorectal, ovarian and prostate cancers. There are reports in the literature showing that people who develop breast, colorectal or prostate cancers in the summer-autumn season, in which skin synthesis of vitamin D is increased, have a greater survival rate than those diagnosed in the winter-spring season [52, 53, 54, 55]. Studies have also shown that a deficiency of folic acid in the diet may increase the risk of colorectal, uterine and breast cancer. Its preventive role is related to the function that this compound plays in DNA methylation, nucleic acid synthesis and S-adenosylmethionine. Low folate levels can

damage chromosomes and hypomethylate the genome. Folic acid deficiency also leads to excessive cellular proliferation [56, 57, 58].

A positive effect on the human body has also been shown for unsaturated omega-3 fatty acids, mainly long-chain polyunsaturated fatty acids, such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) [30]. In chemoprevention, at various levels of carcinogenesis, glucosinolates can also be used to reduce the activity of enzymes that activate carcinogens and induce enzymes involved in detoxification. They can also capture electrophilic metabolites and reactive forms of oxygen, and activate mechanisms that repair DNA. The highest amount of glucosinolates can be found in cruciferous plant seeds and fresh vegetables, whereas their level decreases over storage time [59, 60, 61].

Dietary fibre is one of the components of food that has a significant impact on health, and since the 1980s research results on the anticancer effects of fibre have been published [23, 62, 63, 64]. As a result of the research, Howe et al. found that an increase in fibre intake by 13 g/day reduces the risk of cancer by 31% [65, 66]. The European Prospective Investigation on Cancer and Nutrition (EPIC), coordinated by the International Agency for Research on Cancer (IARC), tested over half-a-million people to assess the relationship between diet and cancer. The research involved: Denmark, France, Germany, Greece, Italy, The Netherlands, Norway, Spain, Sweden and the United Kingdom. In a published report, the researchers stated that compared to people consuming 15 g of fibre daily, in those consuming 35 g of this ingredient the risk of cancer decreased by 40% [67].

In 2007, the World Cancer Research Fund (WCRF) published another comprehensive report on the impact of diet, physical activity and body weight on cancer risk. The report suggests that foods with a high fibre content reduce the risk of cancer [68, 69]. Zhang et al. published a paper comparing the impact of fibre intake on the risk of stomach cancers. It was found that an increase in dietary fibre intake by 10 g/day reduces the risk of developing gastrointestinal cancer by up to 44% [70]. Dong et al. compared the results of 10 scientific studies in which over 700,000 women participated, of whom 16,848 were diagnosed with breast cancer. The authors stated that among women consuming higher amounts of dietary fibre, the percentage of cases was lower by 11%, and an increase in fibre intake by 10g /day reduces the incidence rate by 7% [70].

Currently, a diet based mainly on plant products is recommended, including at least five portions of vegetables and fruits a day. The diet should contain whole grains, replacing purified sources of carbohydrates [24, 26, 27, 72, 73, 74]. A poorly balanced diet leads to overweight and obesity, which, as shown by the results of population studies, is becoming a serious epidemiological threat in developed countries.

A correlation was found between overweight, body mass index and increased risk of cancer, particularly of the large intestine and breast. Epidemiological studies have shown that overweight and obesity, which are becoming a growing epidemic in most countries, are associated with increased risk of cancers of various localizations [75, 76]. In a 2002 monograph, the International Agency for Research on Cancer (IARC) a thesis was given that there is sufficient evidence to recognize overweight and obesity as the cause of cancer of the oesophagus, endometrium, kidney, colon

and breast. In 2007, these studies were confirmed by the World Cancer Research Fund (WCRF), which also declared that there is convincing evidence of the impact of obesity on the development of rectal, pancreatic and gallbladder cancer. Recent WCRF reports from 2014 and 2015 have added ovaries and prostate cancers. The American Cancer Society (ACS) estimates that among 1.5 million new cases of cancer diagnosed every year, at least 20% are a result of obesity. Epidemiological studies also show that obese patients treated oncologically have worse prognoses and greater mortality than patients with normal BMI [30, 58].

Preparing meals by steam cooking or braising in accordance with EPIC and IARC recommendations is a form of cancer prevention. In 2002, acrylamide (AA) was discovered as a by-product of preparation of heat-treated foods with high carbohydrate content, such as snacks, potato chips, bread, cereal products and coffee. AA is metabolized in the body to form glycidamide epoxide (GA), genotoxin. AA and GA form adducts with DNA and amino acids in haemoglobin, increasing the risk of developing endometrial, ovarian, pancreatic, breast and esophagus cancer [24, 30].

Among the factors contributing to the formation of tumours are also deficiencies in or complete lack of physical activity. Recent reports show that physical activity can influence the risk of cancer through a variety of mechanisms. Deficiencies or lack of psychical activity lead to overweight which, in turn, increases the levels of circulating estrogens, androgens, insulin and insulin-like growth factors. These factors are related to the growth of cells as well as tumours. Reduced psychical activity also leads to increased exposure of breast tissue to circulating ovarian hormones as well as to food retention in the large intestine, thus increasing the duration of the potential mutagenic effects on the intestinal lining [59]. The relation between physical activity and the risk of breast, colorectal and endometrial cancer has been demonstrated. Most likely, it also helps reduce the risk of prostate and lung cancer. Multicentre epidemiological studies conducted nowadays, following the principles of evidence-based medicine, have specified, the role of physical activity undertaken systematically to prevent cancer. The impact of physical activity on the body is multidirectional: it affects the immune system, releasing catecholamines and cortisol, which increase the amount of natural killer. Regular physical activity causes the loss of adipose tissue. This increases the adiponectin concentration, lowering the concentration of TNF- α and IL-6 that can damage DNA, inhibit apoptosis and facilitate tumour invasion [77, 78, 79, 80]. Physical activity also lowers the levels of sex hormones, and increases sex hormone-binding globulin levels which, in turn, reduces the risk of breast cancer. Physical activity improves intestinal peristalsis and accelerates intestinal passage, thus reducing exposure to carcinogens contained in foods [58, 59].

Undoubtedly, physical activity is an important preventive factor in cancer. According to the recommendations of the American Institute for Cancer Research, it helps reduce the risk of cancer and prevents weight gain, overweight and obesity, which increase the risk of cancer. Researchers recommend aiming at a minimum of 150 minutes of moderate or 75 minutes of intense physical activity per week. In cancer prevention and weight control, a higher level of activity provides even more benefits. 45- 60 minutes of moderate physical activity should be achieved daily. Over 60 minutes a day provides additional health benefits [61, 81, 82].

A very important issue in the primary prevention of cancer is the reduction of active and passive exposure to tobacco smoke that is the single and main cause of cancer. According to the WHO FCTC, all tobacco products wholly or partly made of tobacco leaves used for smoking, chewing or sniffing, are sources of various carcinogens and other toxic factors. Some carcinogens are ingredients of the tobacco plant itself, including nitrosamines [TSNA] – including N-nitrosornicotine [NNN], 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone [NNK]), most of which are formed when tobacco is burned (i.e. polycyclic aromatic hydrocarbons [PAHs], specifically benzo[a]pyrene [83, 84, 85, 86]. According to the International Agency for Research on Cancer (IARC), more than 70 components of tobacco smoke are considered to be carcinogens to laboratory animals, and 16 of them are recognized as carcinogenic to humans. The strongest activity is demonstrated by: benzene, dimethylnitrosamine, ethylmethylnitrosamine, diethylnitrosamine, nitrosopyrrolidine, hydrazine and vinyl chloride [59, 83]. A report from the United States National Cancer Institute and the Centers for Disease Control and Prevention shows that over 300 million people in the world are smokeless users of tobacco products, stressing that most of them live in Southeast Asia, where the oral cancer rate is the highest in the world [87]. Research conducted by the American Cancer Society shows that tobacco smoking is causally related to at least 16 types of cancer. IARC classified smoking as the cause of haematopoietic tumours, cancer of the larynx, squamous cell carcinoma of the mouth, cancer of the throat, bronchitis, esophagus, pancreas, kidney, bladder, lung cancer, large intestine, nasal cavity and paranasal sinuses, esophagus, stomach, pancreas, liver, bladder, cervix cancer. There is also evidence showing that smoking can cause breast cancer and leukemia in children [59, 83, 88].

Passive smoking is also carcinogenic, the smoke from the glowing tip of a cigarette is four times more harmful than that inhaled by the smoker. Passive smoking increases the risk of lung cancer by a quarter, increases the risk of laryngeal and oesophageal cancer, as well as of childhood leukemia and cancer of the larynx, throat, brain, bladder rectum and stomach (American Cancer Society Report) [83, 89, 90].

Epidemiological studies have indicated that there is a causal connection between alcohol consumption and an increased risk of cancer. Alcohol consumption increases the risk of cancer of the mouth, throat, larynx, oesophagus, liver and breast [91, 92, 93]. The magnitude of the risk varies and depends on the amount of alcohol consumed, its type, and other factors. Even small amounts may increase the risk of cancer. Some studies have shown that having one alcoholic drink a day (6–8 g of pure ethanol) raises the risk of breast cancer by 11%, whereas two drinks a day increase the risk of colorectal cancer by 8%. The International Agency for Research on Cancer (IARC) has identified alcohol as a Class I carcinogen for liver cancer [92, 93]. Both high and low consumption of alcohol (10–12%), is a risk factor for cancer. One portion of pure alcohol (10 g) per day for women – one glass of wine, a glass of beer or a small glass of strong alcohol, is considered acceptable and relatively safe. A portion is 30 ml of vodka (40 per cent/vol), 100 ml of wine (12 per cent/vol.), 285 ml of strong beer (4.9 per cent/vol.) or 375 ml of light beer. (3.5 per cent/vol.) For men, the acceptable daily intake is twice as high [91, 93].

Alcohol enhances neoplasia by acting ‘directly on the mucous membrane, eliminating the lipid component of the barrier that surrounds the granularity of the epithelial layer, or indirectly, by impairing detoxifying function of the liver’. Recent research has shown that the systematic and long-lasting use of alcohol-based mouthwashes is a contributing factor in the development of head and neck cancers, regardless of habitual smoking and consumption of alcoholic beverages. Taking into account only cancers of the oral cavity, pharynx and larynx, using a mouthwash with alcohol twice a day increases the risk of cancer more than tenfold in smokers, over five times for drinkers and nearly fivefold for non-drinkers [93].

Currently, there are many strategies and programmes that can prevent young people from starting to use tobacco, including limited advertising and promotion, increasing excise duties, measures aimed at limiting access of tobacco to minors, education and counter-advertising. Epidemiological data indicate the effectiveness of primary prevention in tobacco control, which motivates further actions in this direction and the creation of effective control programmes [58, 59].

It has been shown that contamination of the natural and working environment is the cause of cancer, which is difficult to avoid during individual life, but preventive measures can be used to a small extent. Activities in this area are widely known and include reduction of smoke and dust emissions from factories, vehicles, natural fertilization on farms, proper drinking water treatment, or application of health and safety standards protecting pregnant women and nursing mothers by limiting exposure to chemicals in a professional environment. An important preventive element is limiting the exposure of children and pregnant and breastfeeding women to household products, pesticides, incense smoke and hair dyes [59].

Nowadays, a lot of attention is paid to the role of electromagnetic fields in the cancer process and to reducing the exposure of its impact on the human body. Regardless of the magnitude of the permissible exposure to the electromagnetic field, it is recommended to apply the principle of prudent avoidance and limit exposure at home, the workplace and in the environment. This can be easily achieved by limiting the presence of electrical wiring, household electrical appliances, office equipment, television sets or computers, and by not placing these devices near places of rest and sleep. Reducing the impact of waves produced by cell phones and microwave ovens is also considered, especially in children [60, 94].

The prevention of skin cancers includes reduced exposure to ultraviolet radiation. The best method to reduce exposure is to avoid solar radiation during its peak activity hours between 10.00 – 15.00 and to wear appropriate clothing (long sleeves, a cover for the head), and at the time of exposure to use creams with a UV absorbing filter greater than 15 (SPF>15) [59, 60, 95]. In modern sunscreen products, several chemical filters and a physical filter are usually used to increase the protection spectrum. Chemical filters penetrate the surface layers of the epidermis that on isomerizing absorb the radiation energy and turn it into thermal energy. Modern chemical filters included in sunscreen products with proven results and high safety profile are camphor derivatives (Mexoryl), avobenzone and benzotriazoles (tinosorb). Physical filters reflect and scatter A and B radiation. The physical protectors used are titanium dioxide, zinc oxide and iron oxide. In the preventive action, attention should also be paid to the manner

of application of the preparations used, which significantly affects their effectiveness. This applies to the amount of the product used and its durability after application. It is assumed that ‘it is appropriate to apply about 2 mg of preparation per 1 cm² of the body surface, which is about 2–3 g on the face of an adult human and about 10 g on the body surface of a child’.

Attention should also be paid to factors potentially reducing the product’s persistence on the skin surface and weakening its effectiveness, that is mechanical removal during physical activity, by rubbing clothing, towels and contact with water. Features of a suitable photoprotective product are a wide spectrum of photoprotection, photostability, long-term protection, water-resistance and good cosmetic quality [96, 97, 98].

Due to the increased role of infectious agents in cancer formation, it is important to reduce exposure to biological agents such as viruses, bacteria, fungi, protozoa. Preventive actions consist of taking care of proper housing conditions (dry and not mouldy), limiting the possibility of infection and avoiding early infections in infants under three months old. People with a family history of liver cancer or liver disease should be tested for HBV and HCV, children of mothers who are carriers of HBV or HCV should receive immunoglobulins after birth, and women who are carriers of HTLV-1 should not breastfeed their children. It is also recommended to test for *Helicobacter pylori* in people with a family history of stomach cancer. Primary prevention also includes protective vaccinations, which should not be limited to compulsory vaccinations, but above all, supplementary vaccinations against hepatitis A and B, Haemophilus Influenzae type B, chickenpox, mumps, influenza and pneumococcal infections [99]. Protective vaccinations, as preventive measures, have led to a lower incidence rate of hepatitis B in Poland. As the epidemiological data show, the introduction of vaccination in all newborns in 1994–1996 contributed to the improvement of the health situation among the youngest children. The ‘incidence of hepatitis B in 1997 for children aged 0–3 years was on average 2.5/100,000’ [100].

In the primary prevention of benign and neoplastic lesions caused by genital types of human papillomavirus (HPV), according to WHO guidelines, preventive vaccines are a breakthrough [101]. In 2006, the European Medicines Evaluation Agency approved two vaccines for use in Europe, as they proved effective by reducing the risk of developing cervical dysplasia. Mass vaccination should be used in girls between 12 – 15 years of age, which creates a real opportunity to prevent cervical cancer [102–114]. Secondary prevention of cancer is mass screening, aimed at early detection of cancer in people without symptoms of the disease and reduction of related mortality. Secondary prevention screening has a beneficial effect on reducing cancer mortality. Examples of early detection through screening are cytological, gynecological, microbiological and virological tests in the prevention of cervical cancer [99, 110]. Cytological tests should be considered an obligatory form of screening for endothelial neoplasia. The American Society of Cervical Colposcopy and Pathology (ASCCP), together with the American Cancer Society (ACS), recommend cytological tests from the age of 21, or 3 years after the onset of sexual activity. Currently, the Population Program for the Prevention and Early Diagnosis of Cervical Cancer and the Population Programme for the Early Detection of Breast Cancer are being implemented as a part of the National Programme

for Combating Neoplastic Diseases. Current information on places where free tests are performed in the breast and cervical cancer prevention programme can be found on the National Health Fund websites. Besides, as a part of the National Programme for Combating Neoplastic Diseases, a programme of care for families with a high hereditary risk of developing malignant neoplasms, is planned, including early detection of breast and ovarian malignancies in such families [5, 111, 112, 113].

Secondary prevention of ovarian cancers includes early diagnosis of gonadal dysgenesis and androgen insensitivity syndrome, sex chromatin evaluation in all female neonates, and ultrasound screening to assess the presence of alveolar apparatus in the gonads. In the process of early breast cancer detection, an important element in secondary prevention is breast self-examination, ultrasound and radiological examination. The effectiveness of breast self-examination in conjunction with a radiological examination in detecting breast cancer at an early stage reaches nearly 90%. Radiological breast examinations are aimed at detecting mammary gland cancer at an early stage of development, where the disease does not yet show symptoms. Ultrasound examination is particularly valuable in women with a dense radiological structure of the breast, with a high content of glandular tissue. An additional advantage of this test is its non-invasiveness and harmlessness, which is why it can be performed safely in pregnant women [114–122].

Breast Imaging Reporting and Data System (BIRADS) have developed an assessment/classification system that should be the standard for MMG descriptions. According to the recommendations of the diagnostic and therapeutic procedure in malignant neoplasms of 2013, screening consisting of mammography for women without clinical symptoms is the best method for the early detection of breast cancer. Assessment of the effectiveness of breast cancer prevention by mammographic screening showed a significant reduction in mortality from breast cancer (20–35%) among women aged 50–69 [122, 123, 124, 125]. Mammography is most often used in the examination of breasts with a higher structural content of adipose tissue than glandular tissue, i.e. in older women. It is worth mentioning that MMG is not recommended in the 20–39 age group due to the high density of breast tissue, typical of young women [123, 126].

Other examples of secondary prevention screening include determination of urinary catecholamine secretion for neuroblastoma detection, computed tomography screening for lung cancer, frequent physical examination and abdominal ultrasonography in individuals with birth defects to detect Wilms' tumour [109, 110].

Undoubtedly, the future in secondary prevention will be genetic tests, which are currently the main method of determining the risk of developing the hereditary forms of cancer. An example is the BRCA1 and BRCA2 gene analysis, which aims to determine the hereditary predisposition to ovarian, breast and other cancers. Research on the BRCA2 gene shows that mutation in this gene is responsible for 35–45% of inherited breast cancer in women, increase the risk of breast cancer to 85%, of ovarian cancer – 15–20%, and the risk of developing the breast cancer in men to 5–10% [114]. Also, knowledge of the early signs of cancer in society, thorough self-observation and self-examination, also allows the observation of disturbing symptoms and reporting for preventive check-ups, which will enable early detection of the disease.

CONCLUSIONS

Due to the systematic increase in the incidence of cancer, a strong emphasis should be placed on prevention. Preventive actions bring benefits not only to the individual, but are an important aspect of health policy. As a part of prevention, apart from improving the health situation of society, there is a consequent reduction in health care expenditure. The importance of primary prevention in relation to healthy people has been demonstrated, including secondary prevention aimed at controlling risk factors in relation to persons exposed to these factors. The combination of these activities becomes an important element in maintaining the health of the individual, as well as society. A basic condition for the high effectiveness of screening programmes, in addition to the broad coverage of populations at high risk of cancer, however, is to raise awareness. Education is an inseparable element of preventive measures, it not only increases health awareness, but above all has a measurable impact on health behaviour, including regular testing at set intervals [115, 116].

REFERENCES

1. American Cancer Society. Cancer Facts and Figures, Atlanta 2015. <http://www.cancer.org/acs/groups/content/@editorial/documents/document/acspc044552.pdf> (27.12.2015).
2. Gajda M, Kowalska M. Internet in interventional studies on cancer prevention. *Hygeia Public Health*. 2016; 51(2): 115–123.
3. Swora-Cwynar E, et al. Impact of selected food ingredients on the prevention of cancer. *Cosmos. Probl Biol Sci*. 2018; 67, 2 (319): 391–396.
4. Krzyżak M, Maślach D, Szpak A, Piotrowska K, Florczyk K, Skrodzka M, Owoc A, Bojar I. Tendencje dotyczące potencjalnych utraconych lat życia z powodu głównych przyczyn zgonów w populacji miejskiej i wiejskiej w Polsce, 2002–2011. *Ann Agric Environ Med*. 2015; 22 (3): 564–571. 10.5604/12 321 966.1168657
5. Wiszniewska M, et al. The prophylactic operational model integrated with occupational healthcare – Prophylactic of some types of cancers among women. *Med Pr*. 2018; 69(4): 439–455, <https://doi: 10.13075/mp.5893.00665>
6. Fitzmaurice C, et al. The global burden of cancer 2013. *JAMA Oncol*. 2015; 1(4): 505–527, <https://doi: 10.1001/jamaoncol.2015.0735>
7. Fern LA, et al. How frequently do young people with potential cancer symptoms present in primary care? *Brit J Gen Pract*. 2011; 5: 223–230.
8. Adamowicz K, Zaucha JM, Majkovic M. Assessment of the state of knowledge regarding cancer prevention among patients of the Breast Cancer Prevention Clinic Nowotwory. *J Oncol*. 2011; 61(5): 449–456.
9. Chybicka A. Nutrition, movement and cancer in children. *Medical News*. 2003; 72(4): 327–329.
10. Ferlay J, Shin HR, Bray F, Forman D, Mathers C, Parkin DM. GLOBOCAN 2008 v2.0, Cancer Incidence and Mortality Worldwide: IARC Cancer Base No. 10 [Internet]. Lyon, France: International Agency for Research on Cancer; 2010. Available from: <http://globocan.iarc.fr>, accessed on 9.11.2012. 2.
11. Independent UK Panel on Breast Cancer Screening, The benefits and harms of breast cancer screening: an independent review. *Lancet*. 2012; 380: 1778–1786.
12. Perek D. Problems in diagnosing cancer in children. *Pediatric Rev*. 2008; 38(3): 209–213.
13. Miyawaki R, Shibata A, Ishii K, Oka K. Obtaining information about cancer: prevalence and preferences among Japanese adults. *BMC Public Health*. 2015; 15(1): 145, <https://doi: 10.1186/s12889-015-1510-2>.
14. Ettl G, Nathanson I, Ettl D, et al. How do adolescents access health information? And do they ask their physicians? *Perm J*. 2012; 16(1): 35–38, <https://doi: 10.7812/tpp/11-125>.
15. Hendershot CS, Otto JM, Collins SE, et al. Evaluation of a Brief Web-Based Genetic Feedback Intervention for Reducing Alcohol-Related Health Risks Associated with ALDH2. *Ann Behav Med*. 2010; 40(1): 77–88, <https://doi: 10.1007/s12160-010-9207-3>.

16. Jander A, Crutzen R, Mercken L, et al. A Web-based computer-tailored game to reduce binge drinking among 16 to 18 year old Dutch adolescents: development and study protocol. *BMC Public Health*. 2014; 9(14): 1054, [https://doi: 10.1186/1471-2458-14-1054](https://doi.org/10.1186/1471-2458-14-1054)
17. Dedert EA, et al. Public health clinical demonstration project for smoking cessation in veterans with posttraumatic stress disorder. *Addict Behav*. 2010; 35(1): 19–22, [https://doi: 10.1016/j.addbeh.2009.08.007](https://doi.org/10.1016/j.addbeh.2009.08.007)
18. Momin B, et al. Traditional and innovative promotional strategies of tobacco cessation services: a review of the literature. *J Community Health*. 2014; 39(4): 800–809, [https://doi: 10.1007/s10900-014-9825-y](https://doi.org/10.1007/s10900-014-9825-y)
19. Myung SK, McDonnell DD, Kazinets G, et al. Effects of Web- and computer-based smoking cessation programs: meta-analysis of randomized controlled trials. *Arch Intern Med*. 2009; 169(10): 929–937, [https://doi: 10.1001/archinternmed.2009.109](https://doi.org/10.1001/archinternmed.2009.109)
20. Puckett M, Neri A, Thompson T, et al. Tobacco cessation among users of telephone and web-based interventions-four states, 2011–2012. *MMWR Weekly*. 2015. 63(51): 1217–1221.
21. Sarna L, Bialous SA, Zou XN, et al. Evaluation of a web-based educational programme on changes in frequency of nurses' interventions to help smokers quit and reduce second-hand smoke exposure in China. *J Adv Nurs*. 2016; 72(1): 118–126, [https://doi: 10.1111/jan.12816](https://doi.org/10.1111/jan.12816)
22. Strecher VJ, McClure JB, Alexander GL, et al. Web-Based Smoking-Cessation Programs. *Am J Prev Med*. 2008; 34(5): 373–381, [https://doi: 10.1016/j.amepre.2007.12.024](https://doi.org/10.1016/j.amepre.2007.12.024)
23. Bienkiewicz M, Bator E, Bronkowska M. Dietary fiber and its importance in health promotion. *Probl Hig Epidemiol*. 2015; 96(1): 57–63.
24. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. AICR, Washington 2018.
25. Kirsh VA, et al. Supplemental and Dietary Vitamin E, β -Carotene, and Vitamin C Intakes and Prostate Cancer Risk. *J Natl Cancer Inst*. 2006; 15; 98(4): 245–54, [https://doi: 10.1093/jnci/djj050](https://doi.org/10.1093/jnci/djj050)
26. Lee IM, et al. Vitamin E in the Prevention of Prostate Cancer: Where Are We Today. *J National Cancer Inst*. 2006; 98, 4, 15: 225–226.
27. Klein EA, et al. Vitamin E and the Risk of Prostate Cancer: Updated Results of The Selenium and Vitamin E Cancer Prevention Trial. *JAMA*. 2011; 12, 306(14): 1549–1556, [https://doi:10.1001/jama.2011.1437](https://doi.org/10.1001/jama.2011.1437)
28. Sesso HD, Buring JE, Christen WG, et al. Vitamins E and C in the prevention of cardiovascular disease in men: the Physicians' Health Study II randomized controlled trial. *JAMA*. 2008; 300(18): 2123–33, [https://doi:10.1001/jama.2008.600](https://doi.org/10.1001/jama.2008.600)
29. Greenwald P, Anderson D, Nelson SA, Taylor PR. Clinical trials of vitamin and mineral supplements for cancer prevention. *Am J Clin Nutr*. 2007; 85(1): 314–317, [https://doi: 10.1093/ajcn/85.1.314S](https://doi.org/10.1093/ajcn/85.1.314S)
30. International Agency for Research on Cancer. World Health Organization. EPIC Study. <https://epic.iarc.fr/highlights/highlights.php> (11.01.2020).
31. Kruk J. Fruits and vegetables consumption and the risk of breast cancer. *Contemporary Oncology*. 2006; 10(5): 224–230.
32. Petrovic Voin, Nepal Anala, Olaisen Camilla, et al. Anti-Cancer Potential of Homemade Fresh Garlic Extract Is Related to Increased Endoplasmic Reticulum Stress. *Nutrients*. 2018; 10(4): 450, [https://doi: 10.3390/nu10040450](https://doi.org/10.3390/nu10040450)
33. Marciniak K, Włodarczyk-Marciniak B. Anticancer properties of garlic. *Post Fitoter*. 2008; 2: 90–95.
34. Lanou AJ, Severson B. Reduced cancer risk in vegetarians: an analysis of recent reports. *Cancer Manag Res*. 2011; 3: 1–8, [https://doi: 10.2147/CMR.S6910](https://doi.org/10.2147/CMR.S6910)
35. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. AICR, Washington 2007.
36. Kromołowska R, Wołosiak R, Sadowska A. Natural anticancer substances in food. *Post. Techn. Przetw. Spoż.* 2011; 1: 87–91.
37. Athar M, Back JH, Tang X, Kim KH, Kopelovich L, Bickers DR, Kim AL. Resveratrol: a review of preclinical studies for human cancer prevention. *Toxicol Appl Pharmacol*. 2007; 1, 224(3): 274–283, [https://doi: 10.1016/j.taap.2006.12.025](https://doi.org/10.1016/j.taap.2006.12.025)
38. Aluyen JK, Ton QN, Tran T, Yang AE, Gottlieb HB, Bellanger RA. Resveratrol: potential as anticancer agent. *J Diet Suppl*. 2012; 9(1): 45–56, [https://doi: 10.3109/19390211.2011.650842](https://doi.org/10.3109/19390211.2011.650842)
39. Salman H, Bergman M, Djaldetti M, Bessler H. Lycopene affects proliferation and apoptosis of four malignant cell lines. *Biomed Pharmacother*. 2007; 61(6): 366–369, [https://doi: 10.1016/j.biopha.2007.02.015](https://doi.org/10.1016/j.biopha.2007.02.015)
40. Zalega J, Szostak-Węgierek D. Nutrition in cancer prevention. Part III. Diets of anticancer properties. *Probl Hig Epidemiol*. 2013; 94(1): 59–70.
41. Ostrowska L. What should be directed in the choice of diet for the patients with metabolic syndrome? *Wybrane Problemy Kliniczne* 2011; 2: 11–18.
42. Kobylńska A, Janas KM. Health – promoting effect of quercetin in human diet. *Post Hig Med Dosw*. 2015; 69: 51–62.
43. Radzikowski C, Wietrzyk J, Grynkiewicz G, Opolski A. Genisteina – izoflawnonoid soi o zróżnicowanym mechanizmie działania – implikacje kliniczne w leczeniu i prewencji chorób nowotworowych. *Postępy Hig Med Dosw* (online). 2004; 58: 128–139.
44. Gryszczyńska A, Gryszczyńska B, Opala B. Carotenoids. natural sources, biosynthesis, influence on human body. *Postępy Fitoter*. 2011; 2: 127–143.
45. Jacobs C, Hutton B, Ng T, Shorr 2, Clemons M. Is there a role for oral or intravenous ascorbate (vitamin C) in treating patients with cancer? A systematic review. *Oncologist* 2015; 20(2): 210–223, [https://doi: 10.1634/theoncologist.2014-0381](https://doi.org/10.1634/theoncologist.2014-0381).
46. Zabłocka K, Biernat J. The influence of selected dietary components on lung cancer risk – vitamins and provitamins. *Bromatol Chem Toksykol*. 2010; 43(2):145–151, [https://doi: 66b60a43-fec6-46f9-b075-e48b6cb66c71](https://doi.org/10.1016/j.bcp.2013.07.018)
47. Kong P, Cai Q, Geng Q, et al. Vitamin intake reduce the risk of gastric cancer: meta-analysis and systematic review of randomized and observational studies. *PLoS One*. 2014; 9(12): e116060, [https://doi: 10.1371/journal.pone.0116060](https://doi.org/10.1371/journal.pone.0116060)
48. Joško J, Ratman R, Ratman K. Angiopreventive role of vitamins. *Współcz Onkol*. 2008; 12(4): 168–172.
49. Slatore CG, Littman AJ, White E. Long-Term Use of Supplemental Multivitamins, Vitamin C, Vitamin E, and Folate Does Not Reduce the Risk of Lung Cancer. *Am J Respir Crit Care Med*. 2008; 177(5): 524–530, [https://doi: 10.1164/rccm.200709-1398OC](https://doi.org/10.1164/rccm.200709-1398OC)
50. Cardenas E, Ghosh R. Vitamin E: a dark horse at the crossroad of cancer management. *Biochem Pharmacol*. 2013; 86(7): 845–852, [https://doi: 10.1016/j.bcp.2013.07.018](https://doi.org/10.1016/j.bcp.2013.07.018)
51. Wang YY, Wang XL, Yu ZJ. Vitamin C and E intake and risk of bladder cancer: a meta-analysis of observational studies. *Int J Clin Exp Med*. 2014; 7(11): 4154–4164.
52. Pericleous M, Mandair D, Caplin ME. Diet and supplements and their impact on colorectal cancer. *J Gastrointest Oncol*. 2013; 4(4): 409–423, [https://doi: 10.3978/j.issn.2078-6891.2013.003](https://doi.org/10.3978/j.issn.2078-6891.2013.003)
53. Garland CF, Garland FC, Gorham ED, et al. The role of vitamin D in cancer prevention. *Am J Public Health*. 2006; 96(2): 252–61, [https://doi: 10.2105/AJPH.2004.045260](https://doi.org/10.2105/AJPH.2004.045260)
54. Mohr SB, Gorham ED, Kim J, et al. Could Vitamin D sufficiency improve the survival of colorectal cancer patients? *J Steroid Biochem Mol Biol*. 2015; 148: 239–44, [https://doi: 10.1016/j.jsbmb.2014.12.010](https://doi.org/10.1016/j.jsbmb.2014.12.010)
55. Bergman P, Sperner S, Höjjer J, Bergqvist J, Björkhem-Bergman L. Low vitamin D levels are associated with higher opioid dose in palliative cancer patients – results from an observational study in Sweden. *Plos One*. 2015; 10(5): e0128223, [https://doi: 10.1371/journal.pone.0128223](https://doi.org/10.1371/journal.pone.0128223)
56. Czyżewska-Majchrzak Ł, Paradowska P. The effects of folate deficiency and its supplementation. *Nowiny Lek*. 2010; 79(6): 457–463.
57. Czeczot H. Folic acid in physiology and pathology. *Postępy Hig Med Dosw*. 2008; 62: 405–419.
58. Eyre H, et al. Preventing Cancer, Cardiovascular Disease, and Diabetes: a Common Agenda for the American Cancer Society, the American Diabetes Association, and the American Heart Association. *Diabetes Care*. 2004; 27(7): 1812–24, [https://doi: 10.2337/diacare.27.7.1812](https://doi.org/10.2337/diacare.27.7.1812)
59. Vance RB. Common interests and common goals: achieving greater progress in preventive health through strategic collaborations. *CA Cancer J Clin*. 2004 J; 54(4): 188–9, [https://doi: 10.3322/canjclin.54.4.188](https://doi.org/10.3322/canjclin.54.4.188)
60. Ghawi S.K, Methven L, Niranjan K. The potential to intensify sulforaphane formation in cooked broccoli (*Brassica oleracea* var. *italica*) using mustard seeds (*Sinapis alba*). *Food Chem*. 2012; 138(2–3): 1734–1741, [https://doi: 10.1016/j.foodchem.2012.10.119](https://doi.org/10.1016/j.foodchem.2012.10.119)
61. <https://www.wcrf.org>. World Cancer Research Fund. American Institute for Cancer Research. Analysing research on cancer prevention and survival. The cancer proces. 2018
62. Park Y, Hunter JD, Spiegelman D, et al. Dietary fiber intake and risk of colorectal cancer. A pooled analysis of prospective cohort studies. *JAMA*. 2005; 294(22): 2849–2857, [https://doi: 10.1001/jama.294.22.2849](https://doi.org/10.1001/jama.294.22.2849)
63. Kimokoki WR, Newby PK. Dietary Patterns, Smoking, and Cardiovascular Diseases: A Complex Association. *Curr Nutr Rep*. 2013, 2(2): 113–125.
64. Tapsell LC, Neale EP, Probst Y. Dietary Patterns and Cardiovascular Disease: Insights and Challenges for Considering Food Groups and

- Nutrient Sources. *Curr Atheroscler Rep.* 2019; 21(3): 9, [https://doi: 10.1007/s11883-019-0770-1](https://doi.org/10.1007/s11883-019-0770-1).
65. Huxley RR, Woodward M, Clifton P. The Epidemiologic Evidence and Potential Biological Mechanisms for a Protective Effect of Dietary Fiber on the Risk of Colorectal Cancer. *Curr Nutr Rep.* 2013; 2: 63–70.
 66. Schulze MB, Martínez-González MA, Fung TT, Lichtenstein AH, Forouhi NG. Food based dietary patterns and chronic disease prevention. *BMJ.* 2018; 13(361): 2396, [https://doi: 10.1136/bmj.k2396](https://doi.org/10.1136/bmj.k2396)
 67. Bingham SA, Day NE, Luben R, et al. Dietary fibre in food and protection against colorectal cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC): an observational study. *Lancet.* 2003; 3(361): 1496–1501, [https://doi: 10.1016/s0140-6736\(03\)13174-1](https://doi.org/10.1016/s0140-6736(03)13174-1).
 68. Wiseman M. The second World Cancer Research Fund/American Institute for Cancer Research expert report. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. *Proc Nutr Soc.* 2008; 67(3): 253–6, [https://doi: 10.1017/S002966510800712X](https://doi.org/10.1017/S002966510800712X)
 69. Aune D, Chan DS, Lau R, et al. Dietary fibre, whole grains, and risk of colorectal cancer: systematic review and dose-response meta-analysis of prospective studies. *BMJ.* 2011; 343: d6617, [https://doi: 10.1136/bmj.d6617](https://doi.org/10.1136/bmj.d6617)
 70. Zhang Z, Xu G, Ma M, Yang J, Liu S. Dietary fiber intake reduces risk for gastric cancer: a meta-analysis. *Gastroenterol.* 2013; 145(1): 113–120, [https://doi: 10.1053/j.gastro.2013.04.001](https://doi.org/10.1053/j.gastro.2013.04.001)
 71. Dong JY, He K, Wang P, Qin LQ. Dietary fiber intake and risk of breast cancer: a meta-analysis of prospective cohort studies. *Am J Clin Nutr.* 2011; 94(3): 900–905, [https://doi: 10.3945/ajcn.111.015578](https://doi.org/10.3945/ajcn.111.015578)
 72. Rudkowska I. Functional food for health: Focus on diabetes. *Maturitas.* 2009; 62(3): 263–9, [https://doi: 10.1016/j.maturitas.2009.01.011](https://doi.org/10.1016/j.maturitas.2009.01.011)
 73. US Department of Health and Human Services, US Department of Agriculture. 2015–2020 Dietary Guidelines for Americans. In: US Department of Health and Human Services, US Department of Agriculture, editors. 8th Edition ed. Washington DC, United States of America 2015.
 74. Bingham SA, Day NE, Luben R, Ferrari P, et al. Dietary fibre in food and protection against colorectal cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC): an observational study. *Lancet.* 2003; 361(9368): 1496–501, [https://doi: 10.1016/s0140-6736\(03\)13174-1](https://doi.org/10.1016/s0140-6736(03)13174-1)
 75. Farvid MS, Cho E, Chen WY, Eliassen AH, Willett WC. Adolescent meat intake and breast cancer risk. *Int J Cancer.* 2015; 136(8): 1909–1920, [https://doi: 10.1002/ijc.29218](https://doi.org/10.1002/ijc.29218)
 76. Fung TT, Hu FB, Hankinson SE, Willett WC, Holmes MD. Low-carbohydrate diets, dietary approaches to stop hypertension-style diets, and the risk of postmenopausal breast cancer. *Am J Epidemiol.* 2011; 174(6): 652–660, [https://doi: 10.1093/aje/kwr148](https://doi.org/10.1093/aje/kwr148)
 77. Irwin ML, Wilder-Smith A, McTiernan A, et al. Influence of pre- and postdiagnosis physical activity on mortality in breast cancer survivors: The Health, Eating, Activity, and Lifestyle Study. *J Clin Oncol.* 2008; 26(24): 3958–64, [https://doi: 10.1200/JCO.2007.15.9822](https://doi.org/10.1200/JCO.2007.15.9822)
 78. Emaus A, Thune I. Physical activity and lung cancer prevention. *Recent Results Cancer Res.* 2011; 186: 101–133, [https://doi: 10.1007/978-3-642-04231-7_5](https://doi.org/10.1007/978-3-642-04231-7_5).
 79. Hojman P, Gehl J, Christensen JF, Pedersen BK. Molecular Mechanisms Linking Exercise to Cancer Prevention and Treatment. *Cell Metab.* 2018; 9, 27(1): 10–21, [https://doi: 10.1016/j.cmet.2017.09.015](https://doi.org/10.1016/j.cmet.2017.09.015)
 80. Grossmann ME, Nkhata KJ, Mizuno NK, Ray A, Cleary MP. Effects of adiponectin on breast cancer cell growth and signaling. *Metab. Br J Cancer.* 2008; 29, 98(2): 370–379, [https://doi: 10.1038/sj.bjc.6604166](https://doi.org/10.1038/sj.bjc.6604166)
 81. Neilson HK, Friedenreich CHM, Brockton NT, Millikan RC. Physical activity and postmenopausal breast cancer: Proposed biologic mechanisms and areas for future research. *Cancer Epidemiol. Biomarkers Prev.* 2009; 18(1): 11–27, [https://doi: 10.1158/1055-9965.EPI-08-0756](https://doi.org/10.1158/1055-9965.EPI-08-0756)
 82. Friedenreich CM, Woolcott CG, McTiernan A, et al. Alberta physical activity and breast cancer prevention trial: Sex hormone changes in a year-long exercise intervention among postmenopausal women. *J Clin Oncol.* 2010; 28(9): 1458–1466, [https://doi: 10.1200/JCO.2009.24.9557](https://doi.org/10.1200/JCO.2009.24.9557)
 83. Leon ME, Peruga A, McNeill A, Kralikova E, Guha N, Minozzi S, Espina C, Schüz J. European Code against Cancer, 4th Edition: Tobacco and cancer. *Cancer Epidemiol.* 2015; 39(1): 20–33, [https://doi: 10.1016/j.canep.2015.06.001](https://doi.org/10.1016/j.canep.2015.06.001)
 84. Carroll Chapman SL, Wu LT. E-cigarette prevalence and correlates of use among adolescents versus adults: a review and comparison. *J Psychiatr Res.* 2014; 54: 43–54, [https://doi: 10.1016/j.jpsychires.2014.03.005](https://doi.org/10.1016/j.jpsychires.2014.03.005)
 85. World Health Organization International Agency for Research on Cancer, Smokeless tobacco and some tobacco-specific N-nitrosamines. *IARC Monogr Eval Carcinog. Risks Hum.* 2007; 89: 1–592.
 86. Diethelm PA, Farley TM. Refuting tobacco-industry funded research: empirical data shows decline in smoking prevalence following introduction of plain packaging in Australia. *Tob Prev Cessation* 2015; 1(6): 2459–3087, [https://doi: 10.18332/tpc/60650](https://doi.org/10.18332/tpc/60650)
 87. Stark MJ, Rohde K, Maher JE, et al. The impact of clean indoor air exemptions and preemption policies on the prevalence of a tobacco-specific lung carcinogen among nonsmoking bar and restaurant workers. *Am J Public Health.* 2007 August; 97(8): 1457–1463, [https://doi: 10.2105/AJPH.2006.094086](https://doi.org/10.2105/AJPH.2006.094086)
 88. Becher H, Belau M, Winkler V, Aigner A. Estimating lung cancer mortality attributable to second hand smoke exposure in Germany. *Int J Public Health.* 2018 Apr; 63(3): 367–375, [https://doi: 10.1007/s00038-017-1022-1](https://doi.org/10.1007/s00038-017-1022-1)
 89. Thomas JL, et al. Metabolites of a tobacco-specific lung carcinogen in children exposed to secondhand and thirdhand tobacco smoke in their homes. *Cancer Epidemiol Biomarkers Prev.* 2011 Jun; 20(6): 1213–21. [https://doi: 10.1158/1055-9965.EPI-10-1027](https://doi.org/10.1158/1055-9965.EPI-10-1027)
 90. Cogliano VJ, Baan R, Straif K, et al. Preventable exposures associated with human cancers. *J Natl Cancer Inst.* 2011; 103(24): 1827–39. [https://doi: 10.1093/jnci/djr483](https://doi.org/10.1093/jnci/djr483)
 91. Chang JS, Straif K, Guha N. The role of alcohol dehydrogenase genes in head and neck cancers: a systematic review and meta-analysis of ADH1B and ADH1C. *Mutagenesis* 2012; 27(3): 275–86. [https://doi: 10.1093/mutage/ger073](https://doi.org/10.1093/mutage/ger073)
 92. Franceschi S, Bidoli E, Herrero R, Muñoz N. Comparison of cancers of the oral cavity and pharynx worldwide: etiological clues. *Oral Oncol.* 2000 Jan; 36(1): 106–15, [https://doi: 10.1016/s1368-8375\(99\)00070-6](https://doi.org/10.1016/s1368-8375(99)00070-6)
 93. World Health Organization International Agency for Research on Cancer. Personal habits and indoor combustions. A review of human carcinogens. *IARC Monogr. Eval. Carcinog. Risks Hum.* 2012; 100: 1–538.
 94. Herzog B, Wehrle M, Quass K. Photostability of UV absorber systems in sunscreens. *Photochem Photobiol.* 2009; 85(4): 869–78, [https://doi: 10.1111/j.1751-1097.2009.00544.x](https://doi.org/10.1111/j.1751-1097.2009.00544.x)
 95. Guan VX, Probst YC, Neale EP, Batterham MJ, Tapsell LC. Identifying usual food choices at meals in overweight and obese study volunteers: implications for dietary advice. *Br J Nutr.* 2018; 120(4): 472–480, [https://doi: 10.1017/S0007114518001587](https://doi.org/10.1017/S0007114518001587)
 96. Holick MF. Sunlight. Sunlight, ultraviolet radiation, vitamin D and skin cancer: how much sunlight do we need? *Adv Exp Med Biol.* 2014; 810: 1–16.
 97. Rice M. For the European Medicines Agency, A Decade of Challenges. *J Natl Cancer Inst.* 2005; 97, 19(5): 1403–1405, [https://doi: 10.1093/jnci/dji355](https://doi.org/10.1093/jnci/dji355)
 98. Zieliński S. Diversity in cancer rates, control programs matches diversity of European Union. *J Natl Cancer Inst.* 2005; 5; 97(19): 1398–9, [https://doi: 10.1093/jnci/dji352](https://doi.org/10.1093/jnci/dji352)
 99. Stefanowicz A, Kulik T, Pacian A, Dyduch A, Żołnierczuk-Kieliszek D. Importance of training family physicians in oncology in the prophylaxis of malignant cancer in phc. *Med Og.* 2007; 13(4): 293–302.
 100. Małecka I, Wysocki J. Hepatitis B – prevention. *Guide for GPs.* 2001; 4, 5(68): 108–111.
 101. Majewski S, Sikorski M. A breakthrough in the primary prevention of cervical cancer and other changes associated with HPV infections. *Guide for GPs.* 2007; 2(10): 108–113.
 102. Griesser H, Sander H, Walczak C, Hilfrich RA. HPV vaccine protein L1 predicts disease outcome of high-risk HPV+ early squamous dysplastic lesions. *Am J Clin Pathol.* 2009; 132(6): 840–845, [https://doi: 10.1309/AJPCU0HBFFFGDTV](https://doi.org/10.1309/AJPCU0HBFFFGDTV)
 103. Carlos RC, Dempsey AF, Patel DA, Dalton VK. Cervical Cancer Prevention Through Human Papillomavirus Vaccination. Using the “Teachable Moment” for Educational Interventions. *Obstet Gynecol.* 2010; 115(4): 834–838, [https://doi: 10.1097/AOG.0b013e3181d502d7](https://doi.org/10.1097/AOG.0b013e3181d502d7)
 104. Khan K, Curtis CR, Ekwueme DU, Stokley S, et al. Preventing Cervical Cancer. *Cancer* 2008; 113(10 suppl): 3004–12.
 105. Dempsey AF, Gebremariam A, Koutsky L, Manhart L. Behavior in early adolescence and risk of human papillomavirus infection as a young adult: results from a population-based study. *Pediatrics.* 2008; 122(1): 1–7, [https://doi: 10.1542/peds.2007-2515](https://doi.org/10.1542/peds.2007-2515)
 106. Rand CM, Shone LP, Albertin C, Auinger P, Klein JD, Szilagyi PG. National health care visit patterns of adolescents: implications for delivery of new adolescent vaccines. *Arch Pediatr Adolesc Med.* 2007; 161(3): 252–259, [https://doi: 10.1001/archpedi.161.3.252](https://doi.org/10.1001/archpedi.161.3.252)
 107. Insinga RP, Dasbach EJ, Elbasha EH. Assessing the annual economic burden of preventing and treating anogenital human papillomavirus-related disease in the US: analytic framework and review of the literature. *Pharmacoeconomics.* 2005; 23(11): 1107–1122, [https://doi: 10.2165/00019053-200523110-00004](https://doi.org/10.2165/00019053-200523110-00004)

108. American Academy of Family Physicians. Joint principles of the Patient-Centered Medical Home. *Del Med J*. 2008; 80(1): 21–22.
109. Knight SB, Crosbie PA, Balata H, Chudziak J, Hussell T, Dive C. Progress and prospects of early detection in lung cancer *Open Biol*. 2017; 7(9): 170070, [https://doi: 10.1098/rsob.170070](https://doi.org/10.1098/rsob.170070)
110. Smith RA, et al. Cancer screening in the United States, 2017: A review of current American Cancer Society guidelines and current issues in cancer screening. *CA Cancer J Clin*. 2017; 67(2): 100–121. [https://doi: 10.3322/caac.21392](https://doi.org/10.3322/caac.21392)
111. Oken MM, et al. Screening by chest radiograph and lung cancer mortality: the Prostate, Lung, Colorectal, and Ovarian (PLCO) randomized trial. *JAMA*. 2011; 306(17): 1865–1873, [https://doi: 10.1001/jama.2011.1591](https://doi.org/10.1001/jama.2011.1591)
112. Franc EL, et al. Human Papillomavirus and Cancer Prevention: Gaps in Knowledge and Prospects for Research, Policy, and Advocacy. *Vaccine*. 2012; 30(5): F175–F182, [https://doi: 10.1016/j.vaccine.2012.06.092](https://doi.org/10.1016/j.vaccine.2012.06.092)
113. Wentzensen N, Arbyn M. HPV-based cervical cancer screening- facts, fiction, and misperceptions. *Prev Med*. 2017; 98: 33–35, [https://doi: 10.1016/j.ypmed.2016.12.040](https://doi.org/10.1016/j.ypmed.2016.12.040)
114. Demarco M, et al. Validation of a Human Papillomavirus (HPV) DNA Cervical Screening Test That Provides Expanded HPV Typing. *J Clin Microbiol*. 2018; 56(5): e01910–17, [https://doi: 10.1128/JCM.01910-17](https://doi.org/10.1128/JCM.01910-17)
115. Schüz J, Espina C, Villain P, Herrero R, et al. European code against cancer 4th edition: 12 ways to reduce your cancer risk. *Cancer Epidemiol*. 2015; 39(1): 1–10, [https://doi: 10.1016/j.canep.2015.05.009](https://doi.org/10.1016/j.canep.2015.05.009)
116. Anderson AS, Key TJ, Norat T, et al. European code against cancer 4th edition: Obesity, body fatness and cancer. *Cancer Epidemiol*. 2015; 39(1): 34–45, [https://doi: 10.1016/j.canep.2015.01.017](https://doi.org/10.1016/j.canep.2015.01.017)
117. Scoccianti C, Cecchini M, Anderson AS, et al. European code against cancer 4th edition: Alcohol drinking and cancer. *Cancer Epidemiol*. 2015; 39(1): 67–74, [https://doi: 10.1016/j.canep.2015.01.007](https://doi.org/10.1016/j.canep.2015.01.007)
118. Brenner DR, Brockton NT, Kotsopoulos J, et al. Breast cancer survival among young women: A review of the role of modifiable lifestyle factors. *Cancer Causes Control*. 2016; 27(4):459–472, [https://doi: 10.1007/s10552-016-0726-5](https://doi.org/10.1007/s10552-016-0726-5)
119. Harris HR, Bergkvist L, Wolk A. Adherence to the World Cancer Research Fund American Institute for Cancer Research recommendations and breast cancer risk. *Int. J. Cancer*. 2016; 138(11): 2657–2664, [https://doi: 10.1002/ijc.30015](https://doi.org/10.1002/ijc.30015)
120. Fourtanier A, Moyal D, Seité S. Sunscreens containing the broad-spectrum UVA absorber, Mexoryl SX, prevent the cutaneous detrimental effects of UV exposure: a review of clinical study results. *Photodermatol Photoimmunol Photomed*. 2008; 24(4): 164–174, [https://doi: 10.1111/j.1600-0781.2008.00365.x](https://doi.org/10.1111/j.1600-0781.2008.00365.x)
121. Nomura SJ, Inoue-Choi M, Lazovich D, Robien K. WCRF/AICR recommendation adherence and breast cancer incidence among postmenopausal women with and without non-modifiable risk factors. *Int J Cancer*. 2016; 138(11): 2602–2615, [https://doi: 10.1002/ijc.29994](https://doi.org/10.1002/ijc.29994)
122. Oeffinger KC, Fontham ET, Etzioni R, et al. Breast cancer screening for women at average risk: 2015 guideline update from the American Cancer Society. *JAMA*. 2015; 314(15): 1599–15614, [https://doi: 10.1001/jama.2015.12783](https://doi.org/10.1001/jama.2015.12783)
123. Fletcher SW, Elmore JG. Mammographic screening for breast cancer. *N Engl J Med*. 2003; 348: 1672–1680, [https://doi: 10.1056/NEJMcp021804](https://doi.org/10.1056/NEJMcp021804)
124. Armaroli P, Villain P, Suonio E, et al. European code against cancer 4th edition: Cancer screening. *Cancer Epidemiol*. 2015; 39(1): 139–152, [https://doi: 10.1016/j.canep.2015.10.021](https://doi.org/10.1016/j.canep.2015.10.021)
125. Alemar B, Gregório C, Herzog J, et al. BRCA1 and BRCA2 mutational profile and prevalence in hereditary breast and ovarian cancer (HBOC) probands from Southern Brazil: Are international testing criteria appropriate for this specific population? *PLoS One*. 2017; 12(11): e0187630, [https://doi: 10.1371/journal.pone.0187630](https://doi.org/10.1371/journal.pone.0187630)
126. Santen RJ, Boyd NF, Chlebowski RT, et al. Critical assessment of new risk factors for breast cancer: Considerations for development of an improved risk prediction model. *Endocrine-Related Cancer*. 2007; 14(2):169–187, [https://doi: 10.1677/ERC-06-0045](https://doi.org/10.1677/ERC-06-0045)