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Role of diet in primary prevention of colorectal cancer

Colorectal cancer and primary prophylaxis

In the group of cancerous diseases colorectal cancer was classified by the World Health Organization (WHO) on the third position from the aspect of the number of cases and deaths (Kubiak et al., 2014). In 2012, 1,360,602 people worldwide fell ill with colorectal cancer, and approximately 693,933 died due to this cause (Król, Kapka-Skrzypczak, 2011). Such a high position in the ranking by the WHO is the result of poorly effective actions in the area of primary and secondary prevention (Schuz et al., 2015), despite the fact that this is one of the best recognized cancerous diseases, and knowledge concerning the stimuli triggering the processes of its development is comprehensive. This is a diet-related disease which means that the risk of occurrence of the disease is modified by means of dietary habits.

Early detection of colorectal cancer improves treatment prognoses. However, the majority of patients undergo the therapy too late, which results in a low effectiveness of the treatment methods applied (Kubiak et al., 2014; Król, Kapka-Skrzypczak, 2011). Late detection of the disease results from problems associated with an effective system of diagnosing. At an early stage, the symptoms are difficult to observe, additionally, low awareness of both physicians and patients indicates the necessity for placing a greater emphasis on actions biased towards primary and secondary prevention (Kubiak et al., 2014; Schuz et al., 2015; Cancer Prevention WHO, 2016). Dietary and health education modifying factors related with life style should prove effective.

An argument in favour of primary prophylaxis are also costs of cancer treatment, which in the European Union have been estimated at 126 billion EUR (data of 2009). Oncologic treatment considerably burdens the national budgets and also has its social aspect. Despite the fact that cancerous diseases afflict mainly older population, a large number of new cases is observed in the population group at productivity age, which entails difficult the estimation of indirect costs related with the occurrence of the disease, i.e. costs of productivity and work absenteeism, as well as costs of patient care (Luengo-Fernandez et al., 2013). It is presumed that implementation

of a health promoting life style, i.e. an adequate diet, increased physical effort or maintenance of a proper body weight, would result in a decrease in the number of new cases of colorectal cancer even by 47% according to the World Cancer Research Fund (WCRF) (Anderson et al., 2015). Thus, the improvement of the level of health knowledge in a time of unfavourable demographic changes related with the ageing of societies, poses a challenge to the present health and educational systems.

Diet and its components in colorectal cancer

From the aspect of an individual and possibilities to avoid a cancerous disease, so-called modifiable factors are of great importance, such as physical activity and diet. Food and the methods of its production, processing, preservation and storage may, on the one hand, trigger negative processes in the colorectal region; however, on the other hand, they may play a considerable role in cancer prophylaxis. Life span is increasing; and consequently, a longer life is related with an increased exposure to carcinogens; therefore, it is worthwhile using a diet as a modifiable carcinogenic factor in order to reduce the risk of development of the disease. An appropriately balanced diet and its individual components may favour, delay or even prevent negative phenomenon taking place in this section of the gastrointestinal tract.

The International Agency for Research on Cancer (IARC) in association with the European Union (UE) have issued recommendations concerning prophylaxis of cancerous diseases entitled the "European Code Against Cancer". Into the factors conducive for the development of colorectal cancer have been classified: obesity, consumption of red meat and its products, and alcohol (Schuz et al., 2015; Thomson, 2015).

Scientific studies confirm the relationship between BMI (Body Mass Index) and WHR (Waist to Hip Ratio), and the risk of contracting colorectal cancer (Pischon et al., 2006; Renehan et al., 2012; Tandon et al., 2015). In individuals with BMI higher than 25 kg/m^2 the risk of occurrence of colorectal cancer increases by 30–50% (Kiciak et al., 2012). Per each subsequent 1 kg/m^2 exceeded over the normal value of BMI this risk increases by 2% (Gray et al., 2012). It was found that morbidity increases in males in whom body weight gain took place in adult life (over the age of 18), and decreases with body weight loss (Renehan et al., 2012; Gray et al., 2012; Rapp et al., 2008). A similar relationship was not observed among females (Chan et al., 2011; Migdał et al., 2014). This may result from the fact that higher tendencies towards the development of the disease were noted in abdominal (visceral) obesity typical of males, than in gluteal-femoral (gynoid) obesity typical of females (Doyle et al., 2012; Lee et al., 2016).

The effect of obesity on carcinogenic processes in the colorectal region is explained in various ways. Metabolic changes accompanying an excessive increase in adipose tissue lead to an increase in the resistance to insulin, intensification of inflammatory states in the body, modification in the level of sex hormones, changes in the production of adipokines, cause oxidative stress and changes in immune

response. All these processes favour carcinogenesis (Schuz et al., 2015). In addition, nutritional behaviours leading to obesity, such as: an excessive consumption of processed products with low nutritional value, i.e. low content of fibre, micro- and macroelements, and a high contribution of simple carbohydrates and hydrogenated vegetable fats, affect the composition of the microflora of the colon and its peristalsis. In turn, the motility of the colon exerts an effect on the duration of the contact of gastric contents with the colon wall. The retention of undigested products triggers carcinogenic processes, while disorders in the bacterial flora of the colon weaken its structure and protective functions, leading to dysbiosis and intensified endotoxemia, and cause a chronic inflammatory state (Nowak, Libudzisz, 2008).

Also, the relationship between the occurrence of colorectal cancer and the consumption of products which have a high glycaemic index (GI) and glycaemic load (GL) is significant. Among Canadians surveyed from the aspect of daily diet during the period 1994–1997, those whose diet was characterized by high GI and GL more frequently contracted colorectal cancer (Hu et al., 2013).

Other observations made among the American population, the diet of which is based on high GI and GL, revealed a positive correlation between the above--mentioned indices and the prevalence of colorectal cancer (Gil et al., 2010), especially among individuals whose physical activity was on a low level. A high GI diet increased the risk of colorectal cancer by 69% in females and 87% in males (Ciok, Dolna, 2005).

The classification of food based on the above-mentioned indices specifies the effect of diet on the level of glucose in blood. Products with a high GI and GL cause the states of glycaemia, i.e. an elevated concentration of free glucose in blood, above the values adopted as normal. Glucose accompanies growth and proliferation of cancer cells (Dudziak, Regulska-Ilow, 2013). In addition, chronically persisting hyperglycaemia typical of type 2 diabetes is most often related with hyperinsulinaemia accompanying overweight and obesity, and enhances the processes of initiation and progression of cancer. Epidemiological studies showed that in patients with type 2 diabetes the risk of colorectal cancer increases even 1.5 times (Dudziak, Regulska-Ilow, 2013; Dąbrowski, 2010).

In response to the above-quoted reports, for the purpose of cancer prevention, the American Institute for Cancer Research (AICR) recommends the maintenance of normal body weight, irrespective of age, and a proper weight circumference, through physical activity and an adequate diet in order to avoid visceral obesity (Schuz et al., 2015). Elimination from the diet products containing easily absorbed carbohydrates, i.e. with a high GI and GL, should also, to a large extent, protect the intestine against carcinogenic processes (Gil et al., 2010; Dudziak, Regulska-Ilow, 2013).

The International Agency for Research on Cancer (IARC) considered red meat (pork, beef, and mutton) as a factor possibly causing colorectal cancer (Group 2A), whereas its products (cold cuts, sausages, pates etc.) as carcinogenic factors in the discussed section of the gastrointestinal tract (Group 1). This classification was based

on scientific reports which showed that the risk of contracting colorectal cancer increased by 14% per each 100 g of daily red meat and meat products consumed (Chan et al., 2011). The risk related with red meat concerns the colon rather than the rectum, whereas the consumption of its products affects the development of cancer concerning these both sections of the gastrointestinal tract (Chan et al., 2011; Fatima et al., 2009).

The carcinogenic effect of red meat has not been ultimately explained and requires further studies. One of the causes is the presence of heme iron which generates free radicals activating a number of unfavourable processes leading to changes in the colorectal region (Fatima et al., 2009; Samraja et al., 2015). In addition, during processing, especially an exposure of red meat to a high temperature, results in the formation of mutagenic substances, such as heterocyclic amines (HCA), or polycyclic amines, polycyclic aromatic hydrocarbons (PAHs) (Majcherczyk, Surówka, 2015; Klusek et al., 2011). Although the effect of consumption of red meat requires recognition of the mechanisms of effect on carcinogenic processes in the colon, there is no doubt concerning the carcinogenic effect of industrial meat products. The IARC placed these products in the group of factors with a high carcinogenic effect on humans (Group 1) (Can et al., 2011). Especially the substances used in meat processing, such as dyes, preservatives, acidity regulators, antioxidants, stabilisers and emulsifiers, flavour enhancers and others, are not indifferent to health (Majcherczyk, Surówka, 2015). Cold cuts are a rich source of PAHs produced during the processes of the smoking of meats. They may impair the genome and favour carcinogenic processes. The PAHs include several hundred compounds, 15 of which have a potentially carcinogenic effect. Nitrogen compounds added to cold cuts products for preservation purposes, during digestion are transformed into nitrosamines – strongly carcinogenic compounds (Migdał et al., 2014).

Concerning scientific publications, the WHO recommends the limitation of consumption of red meat to 500 g per week and avoidance of consumption of processed cured meat products (Schuz et al., 2015).

The risk of development of colorectal cancer is 1.4 times higher in individuals who regularly consume more than 50 g of ethyl alcohol per day, compared to abstainers. The relationship between colon and rectal cancer and the consumption of alcohol has been observed, irrespective of the type of alcohol consumed, because the carcinogenic factor in this case is ethanol and its metabolite – acetaldehyde (Schuz et al., 2015; Kasicka-Jonderko, 2012).

The effect of alcohol on the development of colorectal cancer results from disorders in digestion and absorption caused by ethanol metabolites, i.e. acetaldehyde and acetic acid. They exert an effect on motor and secretory functions of the gastrointestinal system, leading to flatulence and inefficiency of digestion, as well as disorders in the absorption of micro- and macro-components. This results in the deficiency of vitamins which play the role of antioxidants in the human body. In individuals who abuse alcohol, a slowing down of intestinal motility has

been observed, which leads to an excessive development of intestinal flora and its translocation. Ethanol increases the production of nitric oxide, superoxide free radicals and peroxynitrite anions, which enhance the permeability of the intestines by the weakening of tightness of the mucous (Kasicka-Jonderko, 2012).

The WHO classified alcohol beverages, ethanol and acetaldehyde, into important carcinogenic factors (Group 1) and recommended the limitation of alcohol consumption to two drinks in the case of males and one drink for females per day, i.e. 20–30 g of ethanol daily (Schuz et al., 2015; Cancer Prevetion WHO, 2016).

Food components and nutritional habits decreasing the risk of occurrence of colorectal cancer include the maintenance of normal body weight and appropriate consumption of fruits, vegetables and cereal products (Schuz et al., 2015). In the context of prophylaxis of colon cancer, many scientific studies emphasize the role of milk and milk products (Kuczyńska et al., 2013; Cichosz, Czeczot, 2012).

Non-starchy food of plant origin is indicated in the European Code Against Cancer as a basis of diet (Schuz et al., 2015). However, evidence for the presence of the relationship between a higher consumption of vegetables and fruits and the risk of contracting colorectal cancer is not unequivocal. The relation between a reduced risk of occurrence of colorectal cancer and consumption of fruits and vegetables was observed based on scientific reports indicating that this disease is less common among vegetarians than individuals who consume meat (Orlich et al., 2015; Key et al., 2014). The effectiveness of anti-cancer effect in this section of the gastrointestinal tract has been scientifically confirmed only for some groups of vegetables, such as cruciferous plants, to which belong cabbage, brussel sprouts, cauliflower, broccoli, turnip, kale, wasabi (Japanese horseradish), or *Alliacae*, e.g. garlic, onions, leeks or chives (Wu et al., 2013; Wroński, 2013). Cruciferous plants contain glucosinolates, substances which are precursors of compounds which play functions in the protection of cells against DNA damage by carcinogenic factors and reactive oxygen species, and do not allow the development of cancer. Glucosinolates exert an effect on many processes taking place in a cell, such as: regulation of the level of transcription factors, signalling paths, regulation of cell cycle and apoptosis. While exerting an effect on cell cycle, they enable the degradation of cancerously changed cells at early stages of tumour development. Glucosinolates show anti--angiogenic, anti-inflammatory, anti-bacterial and anti-viral effects (Śmiechowska et al., 2008; Zalega, Szostak-Węgierek, 2013).

In turn, plants of the *Alliacae* species contain sulfur compounds, the anti--cancer effect of which is based on the induction of apoptosis in colon cancer. Their bactericidal effect was also confirmed; therefore, the WCRF recommends the daily consumption of fresh garlic (Wroński, 2013).

Although there is no unequivocal evidence for a direct relationship between the risk of contracting colorectal cancer and general consumption of vegetables and fruits, their indirect effect may be proved, i.e. that they eliminate factors which are conducive for the development of this disease. Due to the content of dietary

fibre they have a low energy density, even with a higher volume, thus resulting in the maintenance of normal body weight. Also, dietary fibre favourably affects the motility of the colon and prevents the stagnation of gastric contents in this section of the gastrointestinal tract. The stagnation of food results in a long-term contact of intestinal walls with secondary bile acids, which exert a negative effect on colon cells (colonocytes) (Doyle et al., 2012). It has been confirmed that especially deoxycholic acid affects necrosis, hyperplasia, and induction of DNA damage of cells (Renehan et al., 2012). Fibre is also a natural medium for favourable bacterial flora (Szczepańska et al., 2010).

Some vegetables and fruits contain insulin. This is a natural prebiotic occurring in, e.g. chicory, leek, garlic, onion, Jerusalem artichoke, or bananas, which stimulates growth and metabolic activity of some species of intestinal bacteria, which favourably affects the functioning of the gastrointestinal tract. In the large intestine, insulin is subject to fermentation and contributes to an increase in lactic acid and acetic acid bacilli, substances which inhibit the growth of putrefaction bacteria. As a result of the insulin fermentation processes, short chain fatty acids are produced in the colon, including butter acid, causing the destruction of cancer cells (Schuz et al., 2015; Zalega, Szostak-Węgierek, 2013). Similarly, fermented plant products, such as soya, cabbage, or cucumbers, due to the content of lactic acid create the environment favourable for the development of intestinal microflora, capable of synthesising the enzymes which decompose potentially carcinogenic compounds in the lumen of the colon (Zalega, Szostak-Węgierek, 2013).

In vegetables and fruits there are many biologically active substances of confirmed anti-cancer effect, such as: vitamins C, E, and A, and β-carotene, folic acid, selenium, polyphenols, anthocyanins, among others. They may constitute an active instrument in the chemoprevention of colorectal cancer (Schuz et al., 2015).

The benefits resulting from the consumption of vegetables and fruits are considerable. However, this group of products is not excluded from risk factors, which are related with human activity. The problem concerns the preparations such as: pesticides and herbicides used in the processes of growth and storage of plants, as well as aflatoxins. These preparations have been preventively classified as carcinogenic, because they may induce cancerous processes if in these products a proper level of inhibitory factors, such as vitamin C and E, flavonoids and polyphenols is not simultaneously found (Hasik, 2001). However, as a result of human interference, the content of vitamins and minerals in cultivated plants decreases.

Despite the lack of unequivocal evidence for the relationship between higher overall consumption of vegetables and fruits, and a lower risk of development of colorectal cancer, the potential of this group of products in the prevention of the disease is tremendous.

Whole grain cereal products are a rich source of dietary fibre. A negative correlation was found between the consumption of fibre and occurrence of colorectal cancer. In individuals who consume 35 g of fibre per day the risk of development of

gastrointestinal cancer decreases by as much as 40%, compared to those who consume only 15 g of fibre daily (Bienkiewicz et al., 2015).

Dietary fibre from cereals shows a greater importance in the prevention of colorectal cancer than fibre from vegetables and fruits (Schuz et al., 2015).

Dietary fibre is not digested and absorbed in the small intestine. It passes to the colon in an unaltered form, and it is in this section of the gastrointestinal tract that its partial or total fermentation takes place. As mentioned above, fermentation processes stimulate the growth or metabolic activity of some strains of intestinal bacteria. By hydrolysing fibre, the bacteria obtain energy necessary for their own development. Favourable bacterial flora regulates the functioning of the immune system, development of the intestine, production of vitamins (biotin and vitamin K), as well as enzymes. One of the products of fermentation processes are short chain fatty acids: acetic acid, propionic acid and butyric acid. It has been confirmed that these acids affect an increase in the proliferation of colonocytes and apoptosis of cancer cells (Bienkiewicz et al., 2015; Schuz et al., 2015).

A diet rich in whole grain cereal products supplies the body with a higher amount of dietary fibre, which exerts a beneficial effect on the motility of the large intestine. Due to this, gastric content is not retained in the colon, and is passed at a proper speed to the subsequent sections of the gastrointestinal tract, therefore shortening the duration of contact of digested food with the intestinal wall. This prevents constipations which are considered as factors conducive to the occurrence of colon and rectal cancer.

Despite many advantages resulting from the consumption of fibre, the supply of fibre for the maintenance of health should not be excessively high, because it may hinder the absorption of nutrients and the use of mineral components (Bienkiewicz et al., 2015).

Milk and its products are considered as factors of probable protective effect on the large intestine against cancer. Many clinical and epidemiological studies suggest that this group of products is important in the reduction of risk of development of colon and rectal cancer, indicating, at the same time, the need for carrying out further observations (Kuczyńska et al., 2013).

Preventive properties of milk are considered in association with its composition. Milk and dairy products are the source of easily absorbed vitamins and mineral components, as well as short chain fatty acids. Classified into anti-oxidants present in milk are all vitamins soluble in its fats, i.e. vitamins A, D, E and K, which protect cells against reactive oxygen species, and vitamins soluble in water – vitamin C and vitamins of group B. Other strong antioxidants are contained in milk fat (Cichosz, Czeczot, 2012; Kuczyńska et al., 2013).

Calcium present in milk may bind bile acids forming with them insoluble calcium salts, thus deactivating the carcinogenic factor. It also shows a capability for exerting a direct effect on the intestinal epithelial cells, regulating their cell cycle; also limits the growth and differentiation of altered cells (Gałaś et al., 2013).

The high digestibility of calcium from milk is not only due to its high content in the product, but also due to the presence of other biocomponents, including a beneficial proportion of calcium to phosphorus and the presence of vitamins from group D (Cichosz, Czeczot, 2012). Vitamin D also contributes to the inhibition of cancerous processes in the large intestine. An insufficient supply of vitamin D may disrupt the growth and functioning of colonocytes (Olejnik et al., 2010).

In recent studies on the anti-cancer properties of milk, attention is paid to milk fat and contained in it natural isomers *trans*: conjugated linoleic acid (CLA) and vaccenic acid. CLA is considered as the most active anti-oxidant in milk fat which shows an inhibitory effect on the development of cancer cells in the colon (Cichosz, Czeczot, 2012). The anti-cancer effect of this acid in humans was confirmed at a dose of 3 per day. In milk products, its content range between 2.9–30 mg/g of fat, while with such products as yellow cheese or butter, it reaches 385 mg/100 g of fat (Kuczyńska et al., 2013).

Ester lipids, 13-methyltetradecanoic acid, α-tocopherol, coenzyme Q_{10}, and short chain saturated fatty acids also show anti-oxidative and immunosuppressive properties. A great advantage of milk is that the oxidants present in it are very effective, although their number is small. This results from an active co-action of individual compounds, which create a synergistic system where the regeneration of one component may take place at the cost of the other, e.g. coenzyme Q_{10} reconstructs α-tocopherol from the tocopherol radical, whereas α-tocopherol may renew β-carotene. In addition, anti-oxidants of the lipophilic environment supplement the effect of the antioxidant of the hydrophilic environment.

Anti-cancer effect of milk fat within the large intestine also consists in the stimulation of the functioning of the epithelium through short and medium chain saturated fatty acids. These compounds from the diet, as well as those produced by intestinal flora, affect an increase in the growth of colonocytes and apoptosis of cancer cells, and exert a stimulatory effect on motility. In addition, prostaglandins present in milk fat possess a capability for binding bacterial toxins and rotaviruses, thus preventing the development of inflammatory processes in the intestine (Cichosz, Czeczot, 2012).

Milk and its products are also a valuable source of proteins showing a strong anti-cancer effect, to which belong whey proteins and casein (Kuczyńska et al., 2013).

Conclusions

Colorectal cancer belongs to civilisation diseases. This is a diet-related disease, i.e. the risk of the development of the disease is modifiable by means of nutritional habits. The most important dietary factors favouring this disease include: obesity, consumption of red meat, meat products, alcohol, and consumption of products of high GI and GL. The risk of contracting the disease is decreased by: appropriate

consumption of whole grain cereal products, rich in fibre, proper supply of milk, as well as vegetables and fruits in the daily diet.

Colorectal cancer is most frequently diagnosed at older age, in an already advanced stage of the disease. Late diagnosis provides little chance for the effectiveness of the therapies undertaken. The human life span is constantly increasing, which is associated with a higher exposure to carcinogenic factors. Therefore, it is worthwhile using diet as a modifiable carcinogenic factor in order to reduce the risk of development of the disease.

References

Anderson A.S., Caswell S., Macleod M., Craigie A.M., Stead M., Steele R.J.C., 2015, *The BeWEL Team, Awareness of Lifestyle and Colorectal Cancer Risk: Findings from the BeWEL Study*, BioMed Research International, 1–5.

Bienkiewicz M., Bator E., Bronkowska M., 2015, *Dietary fiber and its importance in health promotion*, Problemy Higieny i Epidemiologii, 96(1), 57–63.

Cancer prevention WHO, http://www.who.int/cancer/prevention/en/, accessed 20.02.2016.

Chan D.S.M., Lau R., Aune D., Vieira R., Greenwood D.C., Kampman E., Norat T., 2011, *Red and Processed Meat and Colorectal Cancer Incidence: Meta-Analysis of Prospective Studies*, PLOS ONE, 6(6): e20456.

Cichosz G., Czeczot H., 2012, *Milk fat in prophylaxis of cancer diseases*, Polski Merkuriusz Lekarski, 33(195), 168–172.

Ciok J., Dolna A., 2005, *Glycemic index and cancer*, Contemporary Oncology, 4(9), 183–188.

Dąbrowski M., 2010, *Diabetes and cancer*, Diabetologia Praktyczna, 2(11), 54–63.

Doyle S.L., Donohoe C.L., Lysaght J., Reynolds J.V., 2012, *Visceral obesity, metabolic syndrome, insulin resistance and cancer*, Proceedings of the Nutrition Society, 71(1), 181–189.

Dudziak K., Regulska-Ilow B., 2013, *The importance of glycemic load of the diet in the development of cancer*, Postępy Higieny i Medycyny Doświadczalnej, 67, 449–462.

Fatima A., Haggar M., Boushey R.P., 2009, *Colorectal Cancer Epidemiology: Incidence, Mortality, Survival, and Risk Factors*, Clinics in Colon and Rectal Surgery, 22(4), 191–197.

Gałaś A., Sochacka-Tatara E., Augustyniak M., Kulig J., Jędrychowski W., 2013, *Intake of calcium and phosphorus in the etiology of colorectal cancer – the Krakow study*, Problemy Higieny i Epidemiologii, 94(1), 134–139.

Gil J., Stembalska A., Łaczmańska I., Sąsiadek M., 2010, *Sporadic colorectal cancer – factors modulating individual susceptibility to cancer*, Współczesna Onkologia, 14(3), 211–216.

Gray L., Lee I.-M., Sesso H.D., Batty G.D., 2012, *Association of body mass index in early adulthood and middle age with future site-specific cancer mortality: the Harvard Alumni Health Study*, Annals of Oncology, 23, 754–759.

Hasik J., 2001, *The dietetic facilities of metabolic process. What the fitamins are?*, Postępy Fitoterapii, 2(3), 9–11.

Hu J., La Vecchia C., Augustin L.S., Negri E., de Groh M., Morrison H., Mery L., 2013, *The Canadian Cancer Registries Epidemiology Research Group, glycemic index, glycemic load and cancer risk*, Annals of Oncology, 24, 245–251.

Kasicka-Jonderko A., 2012, *Alcohol and the digestive system – should it always be blamed?*, Przegląd Gastroenterologiczny, 7(5), 264–275.

Key K.J., Appleby P.N., Crowe F.L., Bradbury K.E., Schmidt J.A., Travis R.C., 2014, *Cancer in British vegetarians: updated analyses of 4998 incident cancersina cohort of 32,491 meateaters, 8,612 fisheaters, 18,298 vegetarians, and 2,246 vegans*, American Journal of Clinical Nutrition, 100, Suppl. 1, 378–385.

Kiciak A., Całyniuk B., Grochowska-Niedworok E., Szczepańska E., 2012, *Selected eating habits of persons with neoplastic diseases*, HYGEIA Public Health, 47(3), 354–359.

Klusek J., Głuszek S., Kozieł D., 2011, *What is new in gastrointestinal cancer prevention – a review of the literature 2009–2010*, Przegląd Gastroenterologiczny, 6(2), 78–84.

Król S.K., Kapka-Skrzypczak L., 2011, *Human colon cancers as a major problem in Poland and in the world – medical and environmental issues*, Medycyna Środowiskowa, 14(4), 75–80.

Kubiak A., Kycler W., Trojanowski M., 2014, *Epidemiology and prevention of colorectal cancer in Poland*, Problemy Higieny i Epidemiologii, 95(3), 636–642.

Kuczyńska B., Nałęcz-Tarwacka T., Puppel K., 2013, *Bioactive components as an indicator of the health-beneficial quality of the milk*, Medycyna Rodzinna, 1, 11–18.

Lee S.E., Bum Jo.H., Kwack W.G., Jeong Y.J., Yoon Y.J., Kang H.W., 2016, *Characteristics of and risk factors for colorectal neoplasms in young adults in a screening population*, World Journal of Gastroenterology, 22(10), 2981–2992.

Luengo-Fernandez R., Leal J., Gray A., Sullivan R., 2013, *Economic burden of cancer across the European Union: a population-based cost analysis*, Lancet Oncology, 14(12), 1165–1174.

Majcherczyk J., Surówka K., 2015, *Heterocyclic aromatic amines as chemical hazard in thermally processed meat products,* Żywność. Nauka. Technologia. Jakość, 1(98), 16–34.

Migdał W., Dudek R., Kapinos F., Kluska W., 2014, *Wędliny Wędzone Tradycyjnie – Zawartość Wielopierścieniowych Węglowodorów Aromatycznych (WWA)*, [in:] *Właściwości Produktów i Surowców Żywnościowych. Wybrane Zagadnienia*, Polskie Towarzystwo Technologów Żywności, Kraków, 75–87.

Nowak A., Libudzisz Z., 2008, *Carcinogenic activity of intestinal microbiota*, Jakość, 61(6), 25–39.

Olejnik A., Tomczyk J., Kowalska K., Grajek K., 2010, *The role of natural dietary compounds in colorectal cancer chemoprevention*, Postępy Higieny i Medycyny Doświadczalnej, 64, 175–187.

Orlich M.J., Singh P.N., Sabaté J., Fan J., Sveen L., Bennett H., Knutsen S.F., Beeson W.L., Jaceldo-Siegl K., Butler T.L., Herring P., Fraser G.E., 2015, *Vegetarian Dietary Patterns and the Risk of Colorectal Cancers*, JAMA Internal Medicine, 175(5), 767–776.

Pischon T., Lahmann P.H., Boeing H., 2006, *Body size and risk of colon and rectal cancer in the European Prospective Investigation Into Cancer and Nutrition (EPIC)*, Journal of the National Cancer Institute, 98(5), 920–931.

Rapp K., Klenk J., Ulmer H., Concin H., Diem G., Oberaigner W., Schroeder J., 2008, *Weight change and cancer risk in a cohort of morethan 65 000 adults in Austria*, Annals of Oncology, 19, 641–648.

Renehan A.G., Flood A., Adams K.F., Olden M., Hollenbeck A.R., Cross A.J., Leitzmann M.F., 2012, *Body Mass Index at Different Adult Ages, Weight Change, and Colorectal Cancer Risk in the National Institutes of Health-AARP Cohort*, American Journal of Epidemiology, 176(12), 1130–1140.

Samraja A.N., Pearcea O.M.T., Läublia H., Crittenden A.N., Bergfelde A.K., Bandar K., Gregga C.J., Bingmana A.E., Secresta P., Diaza S.L., Varkia N.M., Varkia A., 2015, *A red meat-derived glycan promotes inflammation and cancer progression*, Proceedings of the National Academy of Sciences, 2(112), 542–547.

Schuz J., Espina C., Villain P., Rolando Herrero R., Leon M.E., Minozzi S., Romieu I., Segnan N., Wardle J., Wiseman M., Belardelli F., Bettcher D., Cavalli F., Galea G., Lenoir G., Martin-Moreno J.M., Nicula F.A., Olsen J.H., Patnick J., Primic-Zakelj M., Puska P., van Leeuwen F.E., Wiestler O., Zatonski W., 2015, *Working Groups of Scientific Experts, European Code against Cancer 4th Edition: 12 ways to reduce your cancer risk*, Cancer Epidemiology, 39, 1–10.

Szczepańska J., Wądołowska L., Słowińska M.A., Niedźwiedzka E., Biegańska J., 2010, *Frequency of dietary fibre intake and its relationship with the body mass of students*, Bromatologia i Chemia Toksykologiczna, 43(3), 382–390.

Śmiechowska A., Bartoszek A., Namieśnik J., 2008, *Cancer chemopreventive agents: Glucosinolates and their decomposition products in white cabbage (Brassica oleracea var. capitata)*, Postępy Higieny i Medycyny Doświadczalnej, 62, 125–140.

Tandon K., Imam M., Senousy Ismail B.E., Castro F., 2015, *Body mass index and colon cancer screening: The road ahead*, World Journal of Gastroenterology, 21(5), 1371–1376.

Thomson R., 2016, *Red meat and bowel cancer risk – how strong is the evidence?, Cancer Prevention, Cancer Research*, http://wcrf.org/int/blog/articles/2015/10/red-meat-and-bowel-cancer-risk-how-strong-evidence, accessed 05.04.2016.

Wroński K., 2013, *Consumption of garlic in patients with diagnosed colorectal carcinoma*, Nowa Medycyna, 2, 49–53.

Wu Q.J., Yang Y., Vogtmann E., Wang J., Han L.H., Lil H.L., Xiang Y.B., 2013, *Cruciferous vegetables intake and the risk of colorectal cancer: a meta-analysis of observational studies*, Annals of Oncology, 24, 1079–1087.

Zalega J., Szostak-Węgierek D., 2013, *Nutrition in cancer prevention. Part I. Plant polyphenols, carotenoids, dietary fiber*, Problemy Higieny i Epidemiologii, 94(1), 41–49.

Role of diet in primary prevention of colorectal cancer

Abstract

Colorectal cancer is a diet-related disease classified by the World Health Organization (WHO) on the third position from the aspect of the number of cases and deaths. The main factors promoting the development of colorectal cancer have been classified as obesity, consumption of red meat and alcohol. It is presumed that implementation of a health education, promoting life style, i.e. an adequate diet, increased physical effort or maintenance of a proper body weight, would result in a significant decrease in the number of new cases of colorectal cancer.

Key words: colorectal cancer, prevention, diet

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