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Lifestyle Changes May Prevent Cancer

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1. Introduction

Cancer development is a result of interactions among environmental and hereditary factors (Kim, 2006). The majority of genetic abnormalities, which increase the risk of cancer are not hereditary, but a result of DNA damage occurring during lifetime. The causes of DNA damage include internal (nutrient metabolism, cell hormones) and/or external factors (diet, insufficient physical activity, tobacco use, exposure to chemical agents and radiation) (International Life Sciences Institute [ILSI], 2005; World Cancer Research Fund [WCRF], 2007; Kryston et al., 2011). Epidemiological studies have shown that diet and lifestyle are the most important external factors implicated in the development of malignant diseases (ILSI, 2005; WCRF & AICR, 2007; Go et al., 2003).

Why the same environmental factors cause different changes in human genome among individuals is a question for scientists searching unique combinations of factors leading to cancer (Brower, 2011). There is a growing body of evidence that many cancers are not caused by mutations in genes, but by chemical modifications that alter the way genes function. Chemical modifications of genes are called epigenetic changes. Epidemiological studies have shown that diet and lifestyle may cause such changes (Brower, 2011).

According to World Cancer Report (Boyle & Levin, 2008) 12.4 million new cases of cancer were reported in 2008. Malignant diseases accounted for 7.6% of all deaths during the same year and estimations are that in 2030, cancers will be responsible for 17.0% of deaths worldwide. The number of new cases is expected to rise, especially in developing countries (Boyle & Levin, 2008).

In total, malignancies account for 83 million disability-adjusted life years (DALYs). The estimated global economic loss from cancer is US$ 895.2 billion, measured by the economic value of DALYs (American Cancer Society & Livestrong, 2010).

The aforementioned data, coupled with rising health care expenditures for cancer patients, led to cancer being seen not only as a health problem, but also as a political issue (WHO, 2011a, 2011b). Identification of cancers as the leading non-communicable diseases [NCDs], together with cardiovascular diseases, type 2 diabetes and chronic obstructive pulmonary diseases, was followed by an initiative to include NCDs among the global development goals that will succeed the Millennium Development Goals in 2015 (The NCD Alliance, 2011).

Scientists around the world declared war on cancer (Waldorp, 2011). Preventive medicine has been recognized as the most promising field in reducing the risk of malignant diseases development. As diet and lifestyle are believed to be the most important external factors implicated in cancer development, nutrition care process [NCP] (American Dietetic Association [ADA], 2006a, 2008) and medical nutrition prevention [MNP] are considered the most important tools in cancer risk management (Key et al., 2004; Béliveau & Gingras, 2007).

NCP and MNP can be used to reduce the potential of nutrition misinformation to increase the cancer risks in populations and individuals who are inadequately educated about healthy food and lifestyle choices (ADA, 2006b).

Registered dietitians are well aware of dietary reference values and safe upper limits for nutrients intake in different population groups, but these recommendations neglect genetic differences in population subgroups. Therefore, special scientific disciplines - nutrigenetics and nutrigenomics - study the effects of different foods and food constituents on gene expression (Fenech et al., 2011). There is a distinct difference between nutrigenetics and nutrigenomics. Nutrigenetics studies effects of genetic variations on body response to diet and nutrition, while nutrigenomics studies health effects of gene alterations influenced by food constituents (Milner, 2006a). Both disciplines aim to determine the optimal nutrient intake and nutrient combinations (called nutriom) in order to sustain the genome and support the physiological processes of gene expression, metabolism and cell functioning. These findings are expected to be used in cancer risk reduction in dietetic practice (Milner, 2006a).

2. Risk factors related to lifestyle and the possibility of cancer prevention

Many lifestyle factors are related to cancer risks, but at the same time they are highly preventable or modifiable.

2.1 Body composition

Nutrition transitions, taking place in developing countries since the 20th century, toward more energy-dense foods, associated with insufficient physical activity, resulted in pandemic prevalence of overweight and obesity (Popkin, 1995). Overweight and obesity are metabolic disorders and the leading causes of NCDs. Worldwide, 4.8% of all deaths and 2.3% of DALYs are attributable to overweight and obesity (WHO, 2009a).

Overweight and obesity are associated with increased risk of some cancer localizations (Calle et al., 2003; Lagergren, 2011; Fontham & Su, 2008; Stoll, 2002; Ma et al., 2008). Visceral adiposity and central obesity are risk factors for some cancers (AICR & WCRF, 2009). Fat mapping (adipotopography) is an emerging biomedical field dealing with localization and amount of intra-abdominal adipose tissue [IAAT] in the human body. Subjects with higher amount of intra-abdominal adipose tissue are at greater risk for insulin resistance, diabetes and cancer (Thomas et al., 2011). The recommended body-mass index [BMI] for cancer risk reduction is 21.5 kg/m^2 on a population scale, and between 18.5 kg/m^2 and 25.0 kg/m^2 on individual level (AICR & WCRF, 2009; Food and Agriculture Organization of the United Nations [FAO], 2004). Individuals with BMI within the physiological range, but with high-risk waist circumference [WC], are classified as thin-on-the-outside, fat-on-the-inside [TOFI]

phenotype (Thomas et al., 2011) and they are at greater risk of certain cancer localizations. TOFI is a pathological phenomenon, synonymous with "metabolically obese", whereas thin-on-the-outside, thin-on-the-inside [TOTI] is an acronym for healthy adipose tissue distribution (Thomas et al., 2011).

TOFI phenotype may be used to explain the rising incidence of cancers in developing countries. Rapid economic growth and social transitions happening in these countries affect dietary and other lifestyle choices. Energy-dense foods become easily accessible to consumers, while the level of physical activity decreases, promoting overweight and obesity (Popkin, 2001).

Higher amount of IAAT or visceral adiposity is usually related to increased serum levels of insulin-like growth factor [IGF-1], insulin, leptin, sex hormones and adipocytokines (tumor necrosis factor-alpha [TNF-α], interleukin-6 [IL-6], C-reactive protein [CRP], adiponectin, resistin, visfatin, apelin), all known as cancer and type 2 diabetes risk factors (Donohoe et al., 2011; Bon, 2008; Redinger, 2008). Epidemiological studies show that type 2 diabetes patients are at higher risk for developing many types of cancer. There is an ongoing effort in research of shared risk factors for cancer and type 2 diabetes supported by the American Diabetes Association and the American Cancer Society. This research strives to find a biological link between cancer and type 2 diabetes, as well as to explain whether diabetes mellitus therapy affects the risk for cancer development (Giovannucci et al., 2010).

Taller individuals are more likely to get cancer, due to cells being stimulated by IGF-1 and growth hormone continuously, increasing the possibility of DNA replication error, some of which may lead to malignant alterations (Hernandez et al., 2009).

Overweight and obesity are usually the result of external factors, and are therefore, preventable and treatable (WHO, 2005, 2008a) risk factors for some cancers (Table 1).

Cancer site	USA [%]	UK [%]	Brazil [%]	China [%]
Endometrium	70	56	52	34
Esophagus	69	75	60	44
Mouth, pharynx & larynx	63	67	63	44
Stomach	47	45	41	33
Colon	45	43	37	17
Pancreas	39	41	34	14
Breast	38	42	28	20
Lung	36	33	36	38
Kidney	24	19	13	8
Gallbladder	21	16	10	6
Liver	15	17	6	6
Prostate	11	20	n/a	n/a
Total for these cancers combined	34	39	30	27
Total for all cancers	24	26	19	20

Table 1. Estimated percentage of cancers that could be prevented by healthy diet, regular physical activity and healthy weight in selected countries based on the conclusions of the 2007 WCRF/AICR Diet and Cancer Report (n/a – exposure data not available). Adapted from AICR & WCRF, 2009.

According to Fair & Montgomery, "nutritional energy intake is a modifiable factor in the energy balance-cancer linkage". Animal studies showed that reduction of energy intake by 10.0 to 40.0% decreases cell proliferation, by increasing apoptosis due to insufficient angiogenesis (Fair & Montgomery, 2009). Although known to have anticarcinogenic potential, decreased energy intake alone cannot reduce the risk of cancer, since energy expenditure depends highly on the physical activity level. Regular physical activity helps weight loss, reduction of IAAT, serum insulin, IGF-1 and adipocytokines levels, hence reducing the risk of cancer (WHO, 2008a).

Body mass reduction and maintenance of healthy body mass calls for accurate determination of daily energy requirements (Table 2), depending on age, gender and physical activity levels (U.S. Department of Agriculture [USDA] & U.S. Department of Health and Human Services, 2011).

		Physical Activity Level		
Gender	**Age [years]**	**Sedentary [kcal/day]**	**Moderately active [kcal/day]**	**Active [kcal/day]**
Child (female and male)	2-3	1000-1200[a]	1000-1400[a]	1000-1400[a]
Female[b]	4-8	1200-1400	1400-1600	1400-1600
	9-13	1400-1600	1600-2000	1800-2400
	14-18	1800	2000	2400
	19-30	1800-2000	2000-2200	2400
	31-50	1800	2000	2200
	51+	1600	1800	2000-2200
Male	4-8	1200-1400	1400-1600	1600-2000
	9-13	1600-1800	1800-2200	2000-2600
	14-18	2000-2400	2400-2800	2800-3200
	19-30	2400-2600	2600-2800	3000
	31-50	2200-2400	2400-2600	2800-3000
	51+	2000-2200	2200-2400	2400-2800

Table 2. Estimated calorie needs per day by age, gender and physical activity level (a. The calorie ranges accommodate needs of different ages within the group. Children and adolescents need more calories at older ages, and adults need less calories at older ages.; b. Not including pregnant and breastfeeding females.) Adapted from: U.S. Department of Agriculture [USDA] & U.S. Department of Health and Human Services, 2011.

High waist circumference (WC) values increase the cancer risks, and they are undoubtedly related to all-cause mortality in middle aged men and women (Bigaard et al., 2005). Health risks of high waist circumference values in adolescence and young adulthood are similar to those of middle aged individuals. Efforts are being made to standardize WC reference data in accordance with age, gender, body height and ethnicity. Standardization of WC by BMI, site of waist measurement, meal timing and phase of respiration are suggested (Wang, 2006). According to Wang the "unit for WC standardization that investigators will accept logically and mathematically and that would increase the use of WC measurement in the clinical field" should be similar to the percentile system used in children's growth rate nomograms, or T-score used in bone density evaluation (Wang, 2006). Cut-off values of WC by gender and ethnicity are shown in table 3.

Country/Ethnic group	Gender	Waist circumference
Europids[a] In the USA, the ATP III values (102 cm male, 88 cm female are likely to continue to be used for clinical purposes)	Male	> 94 cm
	Female	> 80 cm
South Asians Based on Chinese, Malay and Asian-Indian population	Male	> 90 cm
	Female	> 80 cm
Japanese	Use South Asian recommendations until more specific data are available	
Ethnic South and Central Americans	Use South Asian recommendations until more specific data are available	
Sub-Saharan Africans	Use European recommendations until more specific data are available	
Eastern Mediterranean and Middle East (Arab) populations	Use European recommendations until more specific data are available	

Table 3. Cut-off values of waist circumference by gender and ethnicity excluding children younger than 6 years (a. In future epidemiological studies of populations of Europid origin, prevalence should be given using both European and North American cut-points to allow better comparisons). Table adapted from IDF, 2006.

Key recommendations for long term maintenance of healthy body mass are as follows (Grace, 2011):

1. Being physically active (at least 60 minutes of moderate physical activity daily),
2. Choosing low energy and low fat foods,
3. Eating breakfast regularly,
4. Monitoring body mass,
5. Catching body mass gain before it turns into overweight or obesity,
6. Establishing a consistent healthy diet and proper lifestyle choices.

2.2 Cancer protective diet versus diet-related cancer risk

Nutrition is the source of life. Healthy diet with optimal nutrients intake prevents nutrient deficiency and helps to maintain or improve health. Types of nutrients and their amounts sufficient for sustaining life and maintaining good health are well known and recommendations for nutrients intake are given according to age, gender, pregnancy or lactation (Smolin & Grosvenor, 2010). A number of factors (e.g. genetic background, physical activity level) affect individual's optimal nutrients intake, so it is still not possible for dietitians to practice personalized nutrition.

The inter-individual variability of responses to different food constituents is being scrutinized (Simopoulos & Milner, 2010). The scientific position that genetic predisposition to complex diseases is a result of small variations of a large number of genes and their ability to interact with specific ecological factors is strongly supported in available literature. Therefore, results of nutrigenomic and nutrigenetic research could, through practical and clinical use of new knowledge, reduce the risk for malignant alterations and help control the burden of cancer as the second most common cause of death in the world (Kinsella & He, 2009; WHO, 2011c). It is expected that nutrigenetics and nutrigenomics will shed some light

on the complex relations between nutritional molecules, genetic variations and the biological system, thus facilitating the concept of personalized nutrition. Development of nutrition guides intended for individual use depends on agricultural production and food availability (Simopoulos & Milner, 2010).

At this moment, only population-based nutrition guides exist. They first appeared in early 20th century (USDA, 2002). The first significant guide was the Exchange List for Meal Planning (American Diabetic Association & ADA, 2003) and it was followed by globally accepted MyPyramid (USDA, 2011a). In June 2011, USDA published the enhanced version of nutrition guide called ChooseMyPlate (USDA, 2011b), based on Dietary Guidelines for Americans, published earlier in 2011 (USDA & U.S. Department of Health and Human Services, 2011). The use of nutritional guides is still encouraged in dietetics practice, because they can help manage cancer risks by changing diet and lifestyle.

No specific food can be labelled as anticarcinogenic, but certain food constituents have the potential to reduce cancer risks (WCRF & AICR, 2007; WHO, 2003). Diet rich in fresh vegetables, fruits and low in red and processed meat has been referred to as chemoprotective, while foods with high glycemic indices and sweetened beverages have been linked to hyperinsulinemia, overweight, obesity and increased cancer risks (WCRF & AICR, 2007; Kushi et al., 2006).

2.2.1 Grains and grain products

The amount of nutrients in cereals and cereal products depends on the degree of refinement and processing. Dietary fibers, antioxidants, phenols, lignans and phytoestrogens, present in whole grain products may reduce the risk for some types of cancer (Schatzkin et al., 2007). Dietary fibers are defined as partially or completely indigestible carbohydrates consisting of 3 or more monosaccharide units (Gray, 2006). "Isolated indigestible carbohydrates shown to have beneficial physiological effects in humans" are called functional fibers (Institute of Medicine, 2005). The fiber content of carbohydrates, determines the food's glycemic index [GI] (Smolin & Grosvenor, 2010; Gray, 2006; Barclay, 2008). Foods labelled with "low GI" on nutrition claims (good sources of dietary fibers) are recommended for risk reduction of certain cancers (Barclay, 2008; Buttriss & Stokes, 2008). Nutrition (and health) claims have enormous potential for chronic diseases risk reduction and health promotion and improvement, but better education of consumers on this issue is necessary (Buttriss & Stokes, 2008; Jovičić et al., 2010; Bonsmann et al., 2010). Key recommendation concerning grains and grain products category is to pay attention to the GI and dietary fiber content per 100 g or per serving of foods.

2.2.2 Fruits and vegetables

During the last decade of the 20th century, scientists started emphasizing that high intake of fruits and vegetables may reduce cancer risks. That is why the "5-A-Day" program was developed by the American National Cancer Institute (Havas et al., 1994), but later studies failed to back up the optimistic findings from the 90s (Boffeta et al., 2010; Willet, 2010; Cancer Council Australia, 2007). There is an ongoing effort to find specific fruits and vegetables and their constituents (or their interactions) that are responsible for cancer risk reduction (Table 4).

Organisation Review	**Highest Evidence Convincing**	**Moderate Evidence Probable**	**Lower Evidence Possible / Limited**
WCRF/AICR (2007)		Mouth (f&v) Pharynx (f&v) Larynx (f&v) Oesophagus (f&v) Stomach (f&v) Lung (f)	Nasopharynx (f&v) Colon & rectum (f&v) Pancreas (f) Liver (f) Lung (v) Ovary (v) Endometrium (v)
IARC (2003)		Oesophagus (f&v) Stomach (f) Colon & rectum (v)	Mouth (f&v) Pharynx (f&v) Larynx (f&v) Kidney (f&v) Colon & rectum (f) Bladder (f) Stomach (v) Lung (v) Ovary (v)
WHO/FAO (2003)		Oral Cavity (f&v) Oesophagus (f&v) Stomach (f&v) Colon & rectum (f&v)	
COMA (1998)	Oesophagus (f&v)	Stomach (f&v) Colon & rectum (v)	Breast (f&v)
WCRF/AICR (1997)	Mouth (f&v) Pharynx (f&v) Oesophagus (f&v) Stomach (f&v) Colon & rectum (v) Lung (f&v)	Larynx (f&v) Pancreas (f&v) Breast (f&v) Bladder (f&v)	Ovaries (f&v) Cervix (f&v) Endometrium (f&v) Thyroid (f&v) Liver (v) Prostate (v) Kidney (v)

Table 4. Conclusions from the major cancer prevention reports regarding the cancer protective effect of fruit (f) and vegetables (v). Used with permission of Cancer Council Australia (www.cancer.org.au) (Cancer Council Australia, 2007).

Fruits and vegetables contain dietary fibers, vitamins, minerals and other bioactive molecules, as well as a large percentage of water which makes them low-energy foods. Replacing high-energy dense foods with fruits and vegetables may lead to body mass reduction and, consequently, to cancer risk reduction (Willet, 2010; Cancer Council Australia, 2007; Slimani & Margetts, 2009; Novaković et al., 2010).

Nutritional and health benefits of indigestible oligosaccharides contained in fruits and vegetables are being pointed out in scientific journals. Oligosaccharides are dietary fibers which are generally highly fermentable. Some oligosaccharides, namely fructooligosaccharides, inulin and lactulose are valued for their prebiotic properties (Gray, 2006; Roberfroid et al., 2010; Mujal et al., 2009). High-energy dense nutrition is low in indigestible oligosaccharides and other dietary fibers. Chronic high fat intake and low dietary fiber intake may lead to gut microflora changes, specifically to Bifidobacterium spp. number decrease (Cani & Delzenne, 2009). Altered microflora may play an important role in obesity initiation and homeostasis perturbation (Cani et al., 2007a; Cani et al., 2009;

Heilbronn & Campbell, 2008). Lipopolysaccharides [LPS] from Gram negative bacteria (in altered gut microflora) promote secretion of inflammatory cytokines. LPS pass through intestinal wall, travel by chylomicrons to target organs where they promote inflammation-induced metabolic disorders (obesity, insulin resistance, macrophage infiltration into adipose tissue, liver steatosis). Bifidobacterium spp. as an important actor in healthy gut microflora, decreases the levels of intestinal endotoxins and enhances mucosal barrier function (Tuohy et al., 2005). Increasing the intake of prebiotics may be a beneficial strategy in keeping the number of Bifidobacterium spp. optimal and gut microflora healthy and functional, reducing low-grade inflammation (Tohuy et al., 2005; Cani et al., 2007b). According to Goodlad et al. "prebiotic dietary fibers may also modulate other targets prone to influence metabolic disorders associated with obesity, such as gut peptides" (Goodlad et al., 1987). Decreasing low-grade inflammation and beneficially influencing the body mass, prebiotics (together with probiotics) may be a factor in cancer risk reduction (Grey, 2006; Tuohy et al., 2005; Cani et al., 2007a; Goodlad et al., 1987; World Gastroenterology Organisation, 2008; Donaldson, 2004; Yan & Polk, 2010). Jain et al. indicated that certain synbiotics (combinations of prebiotics and probiotics) are more efficient in vivo than in either treatment alone, but that more research is needed to identify the most potent synbiotics (Jain et al., 2010).

2.2.3 Milk and dairy products

Milk and dairy products are sources of many essential nutrients, e.g. amino acids, fatty acids, lactose, vitamin D, vitamin A, vitamin B_{12}, calcium, potassium and they are prerequisites for growth and development, as well as for optimal bone health (USDA & U.S. Department of Health and Human Services, 2011; Smolin & Grosvenor, 2010). The strength of evidence suggesting that intake of milk and dairy products is associated with reduced cardiovascular and type 2 diabetes risks and blood pressure lowering is moderate (Smolin & Grosvenor, 2010; Huth & al., 2006). A large number of epidemiological studies were conducted in order to explore the relations between milk and dairy products consumption and cancer risks, but the results were largely equivocal (Huth & al., 2006; Quigley, 2011; Järvinen, 2011; Van der Pols et al., 2007). Chronic excessive intake of dairy fat can promote development of malignancies, but certain fatty acids from milk, like conjugated linoleic acid [CLA] can inhibit cancer growth. Vitamin D_3 may reduce the risk of prostate cancer. Calcium from milk and dairy products may have protective properties against colon cancer, while bovine lactoferrin from whey may inhibit colon carcinogenesis (Tsuda et al., 2000; Aune et al., 2011).

The aforementioned contradictory results do not undermine the importance of milk and dairy products in daily diet. Dietary Guidelines for Americans, 2010 recommendations for dairy intake are in accordance with cancer risk reduction (USDA & U.S. Department of Health and Human Services, 2011; Smolin & Grosvenor, 2010).

2.2.4 Protein foods

ChooseMyPlate nutrition guide classifies meat, fish, eggs and their products, legumes, nuts and seeds as protein foods (USDA, 2011b). Proteins have not been linked with increase in obesity, diabetes or cancer risks (WHO, 2007a; Joslin Diabetes Center & Joslin Clinic, 2007),

but foods from this group are rich sources of fats, oils and other substances that might affect cancer risks (Smolin & Grosvenor, 2010; USDA, 2011b).

The majority of case-control studies suggested a positive link between meat consumption and colorectal cancer risk (WCRF & AICR, 2007; Gonzalez, 2006; Chan et al., 2011).

Globally, meat and meat products account for 8.0% of the daily energy intake (FAO, 2011). On account of essential amino acids, fatty acids, vitamin B_{12}, folates, iron, copper and zinc content, meat should be consumed on a daily basis. The amount and the percentage of fat in consumed meat, as well as the type of processing correlate with the incidence of colorectal malignancies (Gonzalez, 2006; Chan et al., 2011; Cross & Sinha, 2004). Risk factors associated with consumption of meat and meat products include nitrates, nitrites, N-nitroso compounds, heterocyclic amines and polycyclic aromatic carbohydrates. Iron is also considered to be a risk factor, because of its prooxidative properties (WCRF & AICR, 2007; Cross & Sinha, 2004; Eichholzer & Gutzwiller, 1998; Lanou & Svenson, 2010; Butler et al., 2003; Zheng et al., 2009). The overall evidence supports limiting red and processed meat intake in order to reduce the risk of colorectal cancer (WCRF & AICR, 2007; Key et al., 2004; Béliveau & Gingras, 2007; Kushi, 2006).

Dietary fatty acid composition plays a role in the process of carcinogenesis and tumor proliferation (WCRF & AICR, 2007; Fenech et al., 2011; WHO, 2008a; FAO, 2004; USDA & U.S. Department of Health and Human Services, 2011; Chan & Giovannucci, 2010; Hu et al., 2011a). The single most prominent issue responsible for global increase of total dietary fat and saturated fatty acids intake is the process of hydrogenation of oils (USDA & U.S. Department of Health and Human Services, 2011; Van Stuyvenberg, 1969). Total hydrogenation transforms unsaturated fatty acids from fish oil and plant oils to saturated fatty acids, while partial hydrogenation produces unsaturated trans-fatty acids. When consumed, trans-fatty acids act as saturated (USDA & U.S. Department of Health and Human Services, 2011; Van Stuyvenberg, 1969). WCRF (WCRF & AICR, 2007) and North Carolina Colon Cancer Study (Vinikoor et al., 2009) claim that specific effects of trans-fatty acids on cancer risk is not known, since positive correlation linking trans-fatty acids intake and overall cancer prevalence was detected using mailed food questionnaires (Hu et al., 2011b).

Data on health effects of fatty fish intake, the correlation between fatty fish intake and gastric cancer (Wu et al., 2011), breast cancer (Terry et al., 2003; Murff et al., 2011), prostate cancer (Terry et al., 2003; Allen et al., 2004) and colorectal cancer (Aune et al., 2009a; Vinikoor et al., 2009; Daniel et al., 2009) are inconsistent (Daniel et al., 2009). Long-chain polyunsaturated fatty acids from fish are seen as promising nutrients in cancer prevention, but currently there is not enough supportive scientific evidence. On the other hand, intake of α-linolenic acid might be a risk factor for cancer (Daniel et al., 2009; Colquhoun et al., 2009). New studies, both experimental and epidemiological, are necessary to shed more light on these findings.

The link between eggs consumption and cancer has not been researched as extensively. Currently, the evidence suggest that eggs intake may be linked to increase in colon, rectal, bladder and ovarian cancer risks, putting into consideration the use of dietary alternatives to eggs (Zhang et al., 2003; Radosavljević et al., 2005; Aune et al., 2009).

Legumes are known to be beneficial in cardiovascular and type 2 diabetes risk reduction, but there is limited evidence that legumes consumption may reduce the risk of stomach, colorectal, and kidney cancer. Further investigations of these complex relations are still waited upon (Kolonel et al., 2000; Aune et al., 2009b).

Effects of nuts and seeds intake on cancer risks are limited and inconclusive (WCRF & AICR, 2007).

Aflatoxin contamination of fungus-contaminated crops and legumes remains a serious food safety problem, as aflatoxin is known to be a risk for liver cancer (Goldman & Shields, 2003; Wogan et al., 2004).

2.2.5 Table salt

Chronic intake of excessive amounts of table salt is a possible gastric cancer risk factor (WCRF & AICR, 2007; ADA, 2006a; Key et al., 2004; Béliveau et al., 2007; WHO, 2003, 2005; AICR & WCRF, 2009; Donaldson, 2004). As positive correlation between *Helicobacter pylori* infection and high salt intake has also been noted, it is possible that there is a synergy between the two factors in gastric cancer promotion. Another mechanism of stomach cancer promotion proposed is that high salt intake damages the gastric mucose, increasing the possibility of endogenous mutations, which leads to hypergastrinemia and decrease in number of gastric parietal cells, and thus to cancer promotion (Wang et al., 2009).

Processed foods are the main source of salt in the diet, and in order to reduce cancer risks in adult population, population-level strategy should start by decreasing salt intake among children, adolescents and young adults (Trajković-Pavlović et al., 2010a, 2010b).

2.2.6 Vitamins, minerals and other bioactive molecules

Micronutrients, such as vitamins and minerals, are essential nutrients in maintaining good health, while other bioactive molecules, such as phytochemicals (substances of plant origin) and zoochemicals (substances of animal origin) are non-essential, but may improve human health (Kaput, 2006). Recommendations for vitamin and mineral intake exist for different population groups (Institute of Medicine, 1998, 2000, 2001), but it is not possible to quantify the need for phytochemicals (Smolin & Grosvenor, 2010).

Many vitamins (e.g vitamin E, vitamin C), minerals (e.g selenium) and phytochemicals (e.g flavonoids) are parts of the antioxidant defense system. Substances with antioxidant properties were seen as promising in lowering risks from chronic diseases and, among them, cancer. In vitro experiments have confirmed that these molecules have antioxidant properties, but the initial hypothesis that antioxidant substances can prevent cancer (or other chronic diseases) in humans has yet to be confirmed (WCRF & AICR, 2007; Mamede et al., 2011).

The impact of calcium and vitamin D on breast and colorectal cancer is being intensively researched during the last ten years, but, up to this point, no definite answers can be given about these relations (Lin et al., 2007; Lappe et al., 2007; Manson et al., 2011).

In the complex pathways of carcinogenesis, there are numerous processes that could be targeted by phytochemicals in order to lower the risk of disease development. The interest

in phytochemicals has grown substantially over the years and it has not lessen, although screening for potential chemopreventive molecules requires a systematic and wide-range approach (Tan et al., 2011; Milner, 2004). In order to reduce the risk of cancer in human population, many experiments studying herbs and spices and their effects on carcinogenesis were conducted on animals. The possibility of using herbs and spices as substitutes for unhealthy food constituents (e.g added sugars, added fat, table salt) can contribute to chemopreventive potential of herbs and spices (Tapsell et al., 2006).

If phytochemicals are added to foods, such foods become functional. Functional foods are foods that provide healthy benefits beyond basic nutrition, when consumed as part of a varied diet on a regular basis, at effective levels (ADA, 2009; Howlett, 2008). According to some authors, health effects of consuming functional foods containing bioactive substances or pharmaceuticals may be as beneficial as consumption of those substances from their natural sources (Howlett, 2008). Other authors claim that chemopreventive properties of fruits and vegetables are a result of synergistic and additive effects of phytochemicals acting together in their natural environment, and therefore, cannot be imitated by functional foods (Tapsell et al., 2006; Liu, 2003; Milner, 2006b).

Global use of dietary supplements containing vitamins, minerals and other bioactive compounds, although already enormous, is still on the rise. Dietary supplements can reduce the risk of deficiencies and promote optimal health, but should not be considered substitutes for a well-balanced, healthy diet (Mason, 2007). Evidence supporting the use of dietary supplements in cancer risk reduction are scarce, so population-based recommendation is to increase the percentage of people who are achieving optimal nutrition without the use of dietary supplements (WCRF & AICR, 2007; Myung et al., 2009). Determination of oxidative stress-based biomarkers should be regarded as "indication" for using antioxidant supplements (Ziech et al., 2010), although this is not financially viable yet.

Mediterranean diet is considered to be protective against cancers, opposite to USA and Northern Europe diet patterns. Adoption of Mediterranean eating pattern in USA and Northern Europe may help cancer risk reduction (Simopoulos, 2001; Verberne et al., 2010).

2.3 Physical activity

Regular physical activity, besides leading to fitness, provides many health benefits (Warburton et al., 2006; Miles, 2007). Physical activity increases overall well-being, and if regular, improves quality of life, helps body mass maintenance and therefore, reduces cancer risks (WCRF & AICR, 2007; WHO, 2003, 2005, 2008a, 2009b; AICR & WCRF, 2009). Cancer risks in regularly physically active are up to 40.0% lower than among physically inactive (Newton & Galvão, 2008).

Insufficient physical activity is considered to be the fourth leading risk factor in overall mortality. WHO holds insufficient physical activity responsible for 6.0% of global deaths (WHO, 2009a). Dropping levels of physical activity worldwide are in part responsible for rising prevalence of NCDs, including cardiovascular diseases, diabetes and cancer (WCRF & AICR, 2007; WHO, 2005, 2009a; Fair & Montgomery, 2009; AICR & WCRF, 2009; Newton & Galvão, 2008; Tucker et al., 2011; Hardman et al., 2011; Wannamethee et al., 2001). Insufficient physical activity accounts for 21.0-25.0% of breast and colon cancer burden (WHO, 2009a, 2010a)

Physical inactivity is a modifiable lifestyle choice and a cancer risk factor, and it is therefore of great public health significance (WCRF & AICR, 2007; WHO, 2005; AICR & WCRF, 2009; Warburton et al., 2006; Miles, 2007; Newton & Galvão, 2008; Tucker et al., 2011; Hardman, 2001; Wolin et al., 2009), but meeting physical activity recommendations has proven to be as big of a challenge on an individual level, as on the society one.

The complexity of cancer - physical activity interactions should be assessed on gene level, too. Thune & Furberg (Thune & Furberg, 2001) believe that "genetic predisposition to be physically active, combined with the knowledge that cancer is a genetic localized disease, warrants studies in general population and high-risk groups alike".

Physical activity is divided into inactivity, insufficient activity and sufficient activity. Sufficient activity is subdivided into "meeting current recommendations" (moderate physical activity) and "highly active" (WHO, 2007b, 2009a, 2009b, 2010a).

"Meeting current recommendations" is possible by 2.5 hours of moderate physical activity, or 1 hour of vigorous physical activity per week. Both are equivalent with 600 metabolic equivalents [MET] per week. Highly active individuals' energy expenditure is equivalent to 1600 MET per week. Metabolic equivalent is the ratio of energy consumption during a specific physical activity to energy consumption while sitting and resting. One MET is defined as the resting metabolic rate obtained during quiet sitting and is set by convention to 3.5 ml O_2/kg/min or 1 kcal/kg/h (WHO, 2010a). Physical activities are classified according to energy needed for their performance, using MET as a reference value. On a population level, moderate physical activity should be set as a goal for health benefits and cancer risk reduction. Indivividuals and population groups that are already moderately active, should be encouraged to become highly active.

In the "Global Recommendations on Physical Activity and Health" (WHO, 2010a), WHO aims to establish dose-response relationship between physical activity and the consequent health benefits, as well as to identify the frequency, duration, intensity, type and total amount of physical activity needed for health benefits, such as cancer or other NCDs risk reduction (WHO, 2007b, 2009b, 2010a). Currently, it is believed that diabetes, heart diseases and cancer risks, including breast and colon cancer risks, can be reduced among people who are 18 or older by 150 minutes of moderate intensity aerobic activity the least, or 60 minutes of vigorous activity weekly. At least 60 minutes of moderate to vigorous physical activity can reduce NCDs risks in 5-17 year-olds (WHO, 2009a, 2009b). For added health benefits, introduction of physical activity, with the amount, frequency, duration and intensity being gradually increased, is recommended for inactive adults, older adults and those limited in activity by their disease (WHO, 2010a).

Public health goals set by new physical activity recommendations include achieving and maintaining optimal health (WCRF & AICR, 2007; WHO, 2003, 2005, 2007, 2008a, 2009a, 2009b, 2010a; Wolin et al., 2009).

2.4 Tobacco, alcohol use and everyday drinks

Although alcoholic beverages and everyday drinks are considered foods, together with tobacco use, their consumption may pose a health risk. On the other hand, safe drinking water is not a health risk, but a prerequisite for optimal health.

2.4.1 Tobacco use

Tobacco is a plant that contains nicotine, various carcinogens and toxins, and it is considered addictive (WHO, 2011d). Around 4000 chemicals have been detected in tobacco smoke, with more than 50 of them identified as carcinogenic (WHO, 2006). Tobacco dependence is classified as a disease under the International Classification of Diseases [ICD-10] (WHO, 1999).

Tobacco use has grown into a global epidemic. The number of tobacco users is on the rise in middle and low income countries, while in decline in developed countries (Mackay et al., 2006; IARC, 2004; WHO, 2008b). Smoking is the second principal cause of global mortality participating with 8.7% (3.7% DALYs) (WHO, 2008b, 2009a; IARC, 2004). At the same time, smoking is the most modifiable single risk factor for malignant diseases and other NCDs (WHO, 2009a; Danaei et al., 2009).

Smoking causes 71.0% of lung cancer deaths (WHO, 2009a), but it is also a risk for other cancer localizations, like throat, mouth, esophagus, stomach, pancreas, kidney, bladder and cervix (WHO, 1999, 2011d). Not only smokers die of smoking-related lung cancer – 4300 secondhand smokers die from lung cancer every year in USA (IARC, 2004; WHO, 2008b, 2011e).

Tobacco use, in any form, is unhealthy. Attempts were made to create less toxic versions of tobacco products, but such products were unacceptable by consumers and consequently failed. Although not widely used, in some parts of the world smokeless tobacco represents a significant form of tobacco use (WHO, 2006; IARC, 2004, 2006, 2008).

Definitions of smoking and related terms – secondhand tobacco smoke [SHS], environmental tobacco smoke [ETS] or other people's smoke and smoking free area – are given in WHO Framework Convention on Tobacco Control [WHO FCTC] as recommended terms for smoking and secondhand smoke (WHO, 2006, 2011e).

WHO FCTC aims to protect public health policy makers from commercial interests, to provide guidance for protection against tobacco smoke, to regulate the contents of tobacco products, to implement rules for labelling, advertising and promotion of tobacco products, to educate people and communicate information about tobacco addiction. Ultimately, the goal is to decrease the number of tobacco users and increase the number of former smokers (WHO, 2011e; IARC, 2008).

There are several aspects of harmful effects of smoking. Dietary nutrient intake and plasma folate level can also be affected by smoking status. Depletion of plasma folate, an antioxidant, together with depletion of other dietary substances, might be a factor in early onset of tobacco-related morbidity and mortality in smokers. Beneficial effects of Mediterranean diet on smokers' health have been documented and they are presumably related to optimal ratio of omega-6 to omega-3 polyunsaturated fatty acids and significant amounts of bioactive molecules (Vardavas et al., 2008, 2011).

In conclusion, all tobacco products should be considered harmful and addictive, and strict regulations should be implemented in order to control the tobacco epidemic (WHO, 2011e; IARC, 2008). Public health researchers of the Oxford Vision 2020 Program, underlined not only the societal, but the individual responsibility towards health and healthy lifestyle

choices. Being aware of the health risks related to tobacco use is not merely enough – motivational campaigns should be designed in order to cut down smoking prevalence by 8.0 to 10.0% per year, and achieve the resulting prevalence of less than 10.0% in all social groups (Yach et al., 2005).

2.4.2 Alcohol use

WHO has estimated that alcohol consumption causes 3.5% of global deaths (6.2% among males, and 1.1% among females) (WHO, 2011f), and it is responsible for 4.5% of DALYs (WHO, 2011f).

The link between alcohol intake and cancer is well documented. There is a positive correlation between oral, pharyngeal, laryngeal, esophageal, liver, colorectal (in men) (WCRF & AICR, 2007; WHO, 2009a, 2011f; Testino & Borro, 2010; Baan et al., 2007) and breast cancer and intake of more than 30 g of alcohol/day (3 standard drinks/day) (Allen et al., 2009; Boyle & Boffetta, 2009). Heavy drinking may correlate with higher risk of lung and pancreatic cancer, but the epidemiological evidence supporting this hypothesis is weak (WCRF & AICR, 2007; WHO, 2011f; Testino & Borro, 2010).

Heavy drinking is defined as intake of more than 80 g of alcohol per day, or more than 5 to 6 standard drinks per day (Pöschl & Seitz, 2004).

More than 30 codes of ICD-10 include the term "alcohol" in their name or definition, documenting the importance of alcohol-related health impairment (WHO, 1999).

Health effects of alcohol vary depending on the age, gender and other characteristics of consumers, but they also depend on the setting and context of drinking (Pöschl & Seitz, 2004).

International Agency for Cancer Research has stated that "acetaldehyde associated with alcoholic beverages is carcinogenic to humans (Group 1)" and confirmed the Group 1 classification of alcohol consumption and of ethanol in alcoholic beverages (Secretan et al., 2009).

Acetaldehyde is the first and the most toxic metabolite of alcohol metabolism. Alcohol dehydrogenase [ADH] oxidizes alcohol to acetaldehyde, which is then converted to acetate by aldehyde dehydrogenase [ALDH]. Ethanol also inhibits DNA methylation and interacts with retinoid metabolism. Tissue-specific levels of ethanol are in correlation with the amount of alcohol ingested, but they also depend on the genotype coding for ethanol-metabolizing enzymes, predominantly ALDH (Seitz & Stickel, 2007; Boffeta & Hashibe, 2006; Seitz & Becker, 2007).

Effects of acetaldehyde, elevated estrogen levels, production of oxygen radicals and changes in folate and vitamin B_6 metabolism, may be mechanisms responsible for the genotoxicity of alcohol (Seitz & Stickel, 2007; Boffeta & Hashibe, 2006; Seitz & Becker, 2007).

Persons with achlorhydric gastritis who consume alcohol are at greater risk of stomach cancer. Absence of hydrochloric acid creates an environment favorable for thriving of ADH-containing bacteria that can metabolize carbohydrates to ethanol and acetaldehyde (Seitz & Becker, 2007).

The WHO Global Strategy to Reduce the Harmful Use of Alcohol (WHO, 2010b) aims to raise awareness of health, social and economic aspects of alcohol abuse, and the relationship between alcohol and disease development. It points to the importance of effective stakeholders involvement in preventing harmful effects of alcohol. It also aims to provide support for national efforts to reduce the overall effects of alcohol abuse.

USDA recommendations for alcohol intake not linked to increase of cancer risks are 28 g of alcohol per day for healthy adult men, and half of that amount for healthy women (USDA & U.S. Department of Health and Human Services, 2011). The European Code Against Cancer recommends up to 20 or 30 g/day of alcohol for healthy men and, again, half of that amount for healthy women (Boyle et al., 2003). AICR made recommendations simple by advising 2 standard drinks for healthy men and 1 standard drink for healthy women (WCRF & AICR, 2007).

Tobacco and alcohol use are major risk factors for malignancies of different localizations, but mainly of the gastrointestinal tract (Testino & Borro, 2010; Pelucchi et al., 2008; Seitz & Cho, 2009) and the two also act in synergy increasing the cancer risk. Tobacco smoking combined with alcohol use increase the tissue levels of acetaldehyde, while alcohol helps in activation of different procarcinogens in tobacco smoke by induction of cytochrome-P450-2E1-dependent microsomal biotransformation system in mucose cells of the upper digestive tract and liver (IARC, 2004; Testino and Borro, 2010; Seitz & Cho, 2009). Pancreatic cancer risk is 4.3-fold higher in people who smoke more than 20 cigarettes per day and drink more than 21 standard drinks per week, than in non-smoking people who drink less than 7 standard drinks per week (Talamini et al., 2010).

2.4.3 Coffee and tea use

Coffee and tea drinking is a widespread habit. Both coffee and tea contain many bioactive substances (antioxidants, phenols) with in vitro anticarcinogenic characteristics (Ferruzzi, 2010). Consumption of 3 cups of coffee per day (equivalent to 300 mg of caffeine) is considered to be moderate (Tverdal et al., 2011).

It is still not known whether coffee consumption increases the risk of any type of cancer. Evidence concerning the link between coffee and esophageal and pancreatic cancer are inconsistent and difficult to interpret, due to the confounding effects of tobacco and alcohol use (WCRF & AICR, 2007; Ferruzzi, 2010).

Laboratory experiments and animal testing showed chemoprotective activity of tea polyphenols, but there are not enough evidence to confirm the same in human population (WCRF & AICR, 2007; Lambert & Yang, 2003).

2.4.4 Soft drinks

Soft drinks consumption shows an increase of 5.0% per year, that is an increase from 467 to 552 billion litres from 2004 to 2007 (Zenith International, 2005, 2008). Cola drinks and carbonated soft drinks reportedly accounted for 42.0% of 467 billion litres of soft drinks consumed within one year, in contrast to fruit juices, nectars and fruit drinks which accounted for 8.0% in 2004 (Zenith International, 2005).

Currently, findings on the effects of soft drinks on cancer risks are limited and inconsistent (WCRF & AICR, 2007).

Soft drinks are significant contributors to added sugar intake which leads to body mass increase, overweigh, obesity and consequently to NCDs. Eminent international organizations do not consider soft drinks to be a part of healthy and active lifestyle (WCRF & AICR, 2007; WHO, 2005; USDA & U.S. Department of Health and Human Services, 2011).

2.4.5 Water as a fluid of choice

Only air is more crucial for life than water. Access to safe drinking water is a basic human right, as well as a condition for achieving and sustaining optimal health (WHO, 2011e).

However, contaminated water is a great health risk. Inorganic arsenic from drinking water is a proven risk for lung cancer and there is limited evidence that it is also a risk factor for kidney and bladder cancer (WCRF & AICR, 2007).

Safe drinking water is a nutrient *per se*. Because it plays many important roles in the human body, water is the fluid of choice as a part of healthy lifestyle. There has been a steady growth in the sales of bottled water (6.1% in 2007) (Zenith International, 2008) which is an encouraging fact in accordance with today's active and healthy lifestyle.

Daily requirements for water depend on age, gender, pregnancy, lactation, physical activity level, diet and external conditions (Smolin & Grosvenor, 2010; ILSI, 2004).

3. Conclusion

The impact of nutrition and lifestyle on cancer development is evident. Still, there are many unanswered questions concerning the interactions among nutrition, environment and human genome. The existing and future knowledge should be used by public health professionals to promote cancer risk reduction on a population scale, especially in high risk subpopulations, as well as on the individual level. Further research and public health activities should be supported by health and government authorities.

"To eat is a necessity, to eat intelligently is an art." (François de La Rochefoucauld, French writer, 1613-1680)

"Walking is man's best medicine." (Hippocrates, ancient Greek physician, 460 BC – 377 BC)

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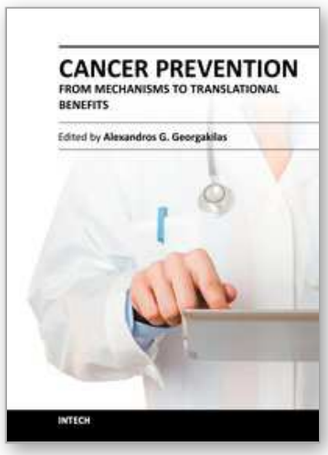
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This unique synthesis of chapters from top experts in their fields targets the unique and significant area of cancer prevention for different types of cancers. Perspective readers are invited to go through novel ideas and current developments in the field of molecular mechanisms for cancer prevention, epidemiological studies, antioxidant therapies and diets, as well as clinical aspects and new advances in prognosis and avoidance of cancer. The primary target audience for the book includes PhD students, researchers, biologists, medical doctors and professionals who are interested in mechanistic studies on cancer prevention and translational benefits for optimized cancer treatment.

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