Alcohol Consumption and Cancer: A Literature Search and a Proposal

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Abstract

The correlation between alcohol and cancer is well-known. Recently, the International Agency for Research on Cancer (World Health Organization) included the consumption of alcoholic beverages, as well as the ethanol and acetaldehyde present in alcoholic beverages, in Group 1. A causal relationship has been established between alcohol and the occurrence of several types of cancer, particularly those with onset in the oral cavity, pharynx, larynx, esophagus, colon-rectum, liver, and breast. To date, a safe amount has not been established. Instead, a dose-dependent correlation has been demonstrated between cancer risk and alcohol intake in both males and females who consume alcohol on a regular basis. In subjects who consumed or consume alcohol in a risky / harmful way, or in patients with a history of alcohol dependence or alcohol problems, screening and oncological prevention programs should be customized with regard to the general population.

INTRODUCTION

The correlation between the consumption of alcoholic beverages and the onset of some types of tumor is well known, as is the fact that the ingestion of any type of alcoholic beverage is associated with the possible development of cancer. In the international literature there are numerous experimental and epidemiological studies and data that have indicated this for years. This causal relationship has been confirmed by the publication of volumes 96 (2010) and 100E (2012) by the International Agency for Research on Cancer (IARC - World Health Organization) [1-3].

In Table 1 the risk is shown in relation to grams / day. It is worth pointing out that the risk is cumulative. One Alcoholic Unit (AU) is represented by 10-12 grams of ethanol, which occurs on average in 125 ml of wine at 12%, in 330 ml of beer at 4.5%, in 80 ml of other alcoholic drinks or cocktails at 18 %, or 40 ml of spirits at 36%.

The National Institute for Health and Clinical Excellence suggests this classification by using a common means of identification such as the Alcohol Use Disorders Identification Test (AUDIT) [4]: If the score is greater than 8, this means the consumer is at risk; if the score is between 16 and 19, this means harmful use; if the score is greater than 20, this means severe alcohol dependence.

From the methodological point of view in this literature search, we selected studies that we considered most significant (PubMed). In the light of the selected data and personal experience a proposal is put forward for monitoring cancer in alcohol dependence (AD) patients and in those with risky / harmful consumption.

The mechanisms of carcinogenesis

The mechanisms of carcinogenesis induced by alcohol are numerous. The following are the most accredited [1,2,5-9]: 1) The production of toxins and carcinogens such as acetaldehyde, oxygen-free radicals and products arising from lipid peroxidation, 2) interference with the absorption of certain nutrients, 3) altered metabolism of some nutrients, 4) inhibition of some detoxification mechanisms, 5) activating enzyme (cytochrome P450-2E1; CYP2E1), 6) increased oxidative stress, 7) immune suppression, 8) changes in membrane fluidity, 9) alteration of cell proliferation / apoptosis, 10) stimulus to the processes of
Central control the mechanisms of anti-apoptosis. These last two factors sub-regulate the genes that systems. These systems are also inhibited by IL-6 and nuclear induction of nitric oxide synthase by ethanol inhibits DNA repair alcohol dehydrogenase and aldehyde dehydrogenase) [9,10].

Individual genetic patterns (especially genetic polymorphisms of neoplastic cell lines. The number of adducts varies in relation to and chromosomal alterations that favor the development of neoplastic cell lines. The number of adducts varies in relation to individual genetic patterns (especially genetic polymorphisms of alcohol dehydrogenase and aldehyde dehydrogenase) [9,10].

The acetaldehyde and nitric oxide produced through the induction of nitric oxide synthase by ethanol inhibits DNA repair systems. These systems are also inhibited by IL-6 and nuclear factor kB. These last two factors sub-regulate the genes that control the mechanisms of anti-apoptosis.

It should be remembered that increased CYP2E1 activity leads to the activation of pro-carcinogenic substances found in environmental tobacco smoke and certain foods such as polycyclic hydrocarbons, hydrazine and nitrous mine [9,11]. CYP2E1 reduces the tissue levels of retinol and retinoic acid, which have particular relevance in the regulation of cell growth and differentiation. The decisive factor is the influence of ethanol and acetaldehyde on the transfer mechanisms of the methyl groups. In case of hypomethylation there is an activation of the oncogenes, whereas in case of hypermethylation a deactivation of the tumor suppressor genes occurs.

To maintain a sufficient methylation capacity, it is also appropriate to maintain a fair number of lipotropic substances including nutrients such as choline, betaine and methionine, which are essential for the formation, transport and transfer of methyl groups to the “target molecules.” Ethanol promotes tumor progression via the induction of angiogenesis. In fact, an increase in endothelial growth factors and tumor angiogenesis has been found [12,13]. Polymorphisms and genetic mutations greatly affect the ratio of alcohol to cancer.

Genetic variations related to the metabolism of ethanol and acetaldehyde, oxidative stress, lipid storage mechanisms, the metabolism endotoxins and the mechanisms of fibro genesis have all been identified. The carriers of these polymorphisms are predisposed to cancer through social consumption [2]. More incisive genetic modifications are related to aldehyde dehydrogenase (ALDH) and alcohol dehydrogenase (ADH). An altered function of ALDH results in a buildup of acetaldehyde, which is highly toxic and carcinogenic. The normal allele is represented by ALDH2 * 1, while the inactive variant is ALDH2 * 2. The presence of the variant involves the accumulation of a higher concentration of acetaldehyde. This favors the mechanisms of carcinogenesis.

Single nucleotide polymorphism (SNP) may also be present in the structure of ADH. The presence of ADH1B * 2, for example, ensures an activity 40 times higher. In these individuals the metabolism of ethanol will be more effective and faster. This provides protection against cancer [9].

From these examples it can be deduced that a genetic pattern, at an equal frequency of consumption and dosing, leads to unpredictable individual variations.

The correlation of alcohol consumption and cancer

In the past twenty years, the meta-analyses linking the consumption of alcohol with cancer have been particularly numerous. The cancers related to consumption are: oral cavity, pharynx, larynx, esophagus, colon-rectum, liver and breast. There is a possible correlation with stomach, pancreas, lung, prostate and bladder cancers.

Boffetta et al. (2006) [14] estimated that about 3.6% of cancers in the world are related to the consumption of alcoholic beverages (5.3% in males and 1.7% in females). Considering that in different areas of the world there is no consumption of alcohol, mainly for religious reasons, this percentage is particularly significant in the Western world and in Eastern Europe.

### Table 1: Chronic consumption of alcoholic beverages: Proposal of a scheme for the identification of risk in a healthy subject.

<table>
<thead>
<tr>
<th>Risk Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Very low risk:</strong></td>
<td>Woman 3g / day between 45 and 65 years</td>
</tr>
<tr>
<td></td>
<td>Man 9 g / day between 45 and 65 years</td>
</tr>
<tr>
<td><strong>Low Risk:</strong></td>
<td>Woman &lt;10 g / day</td>
</tr>
<tr>
<td></td>
<td>Man &lt;20 g / day</td>
</tr>
<tr>
<td><strong>Risky Consumption:</strong></td>
<td>Woman 10-40 g / day</td>
</tr>
<tr>
<td></td>
<td>Man 20 and 60 g / day</td>
</tr>
<tr>
<td><strong>Harmful Consumption:</strong></td>
<td>Doses greater than 12 grams over 65 years</td>
</tr>
<tr>
<td></td>
<td>Man over 60 g / day</td>
</tr>
</tbody>
</table>

### Table 2: Oncological screening in alcohol.

<table>
<thead>
<tr>
<th>System</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral Cavity -</td>
<td>Neurological examination</td>
</tr>
<tr>
<td>Pharynx</td>
<td>ENT Visit</td>
</tr>
<tr>
<td>Larynx</td>
<td>Testing for Hp infection and/or endoscopy with biopsies</td>
</tr>
<tr>
<td>Esophagus /</td>
<td>Fecal occult blood testing and/or colonoscopy</td>
</tr>
<tr>
<td>Stomach</td>
<td>If cirrhosis; US every 6 months</td>
</tr>
<tr>
<td>Colon-Rectum</td>
<td>If chronic alcoholic liver disease; US every 6 months</td>
</tr>
<tr>
<td>Hepato-biliary</td>
<td>If chronic pancreatitis alcoholic liver disease; US every 6 months</td>
</tr>
<tr>
<td>System</td>
<td>Testing for HBV, HCV, HIV infection</td>
</tr>
<tr>
<td>Lung</td>
<td>Chest X-ray</td>
</tr>
<tr>
<td>Breast</td>
<td>Mammography and US &gt; 40 years</td>
</tr>
</tbody>
</table>

angiogenesis, 11) weight gain, 12) genetic polymorphisms of alcohol dehydrogenase (ADH) and acetaldehyde dehydrogenase (ALDH).

The metabolism of ethanol by ADH entails the production of acetaldehyde and NADH, while from CYP2E1 acetaldehyde and oxygen-free radicals (ROS) are formed. The NADPH is reoxidized to NADH in the mitochondria with the further formation of ROS. ROS and reactive species of nitrogen (RSN) subsequently induce lipid peroxidation. Acetaldehyde, ROS and products of lipid peroxidation determine direct genotoxic activity through the formation of “adducts,” which involves mutations and chromosomal alterations that favor the development of neoplastic cell lines. The number of adducts varies in relation to individual genetic patterns (especially genetic polymorphisms of alcohol dehydrogenase and aldehyde dehydrogenase) [9,10].

The acetaldehyde and nitric oxide produced through the induction of nitric oxide synthase by ethanol inhibits DNA repair systems. These systems are also inhibited by IL-6 and nuclear factor kB. These last two factors sub-regulate the genes that control the mechanisms of anti-apoptosis.

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The most affected areas of the world among males are: European area C (including the Russian Federation) with a correlation of about 9%; European area A (covering Western Europe); African area E; and Western Pacific B, with a correlation of approximately 6.5%. Regional differences in terms of risk can be attributed to several factors: regional variations in the prevalence of consumption, recruitment procedures, other carcinogenic substances present in alcoholic beverages, the local genetic structure of different populations and other associated risk factors.

The cancers most causally related to alcohol worldwide are: 30.4% of cancers of the oral cavity and pharynx, 18.5% of cancers of the esophagus, 3.2% of cancers of the colon-rectum, 9.4% of cancers of the liver, 23% of cancers of the larynx and 4.5% of breast cancers in women. In addition, 0.5% of all cancer deaths globally are alcohol-related: 25.9% of deaths from cancers of the oral cavity and pharynx, 18.1% from cancers of the esophagus, 3.1% from cancers of the colon and rectum, 9.4% from cancers of the liver, 21.4% from cancers of the larynx and 4.1% from cancers of the breast [14].

It has been shown that in Europe (Denmark, Germany, Greece, Italy, Spain and Great Britain) 10% of cases in males and 3% in females can be attributed to alcohol consumption [15]. In both sexes, the percentage is higher for cancers of the upper digestive tract-plane (44% males, 25% females), followed by cancers of the liver (33% males, 18% females), then colorectal cancer (17% in males, 4% in females) and finally breast cancer (5%).

The percentage increases if the daily dose exceeds two units / day for men and 1 unit / day for women: 10% of colorectal cancers, 27% of liver cancers and 30% of cancers of the upper digestive tract.

Recently, Nelson et al. [16] found that in the USA, 3.5% of all cancer deaths are attributable to alcohol. In particular, 26-35% of all alcohol-related cancers are caused by a consumption of less than 20 grams per day.

Meta-analyses by Bagnardi et al. (2001) [17], Corrao et al. (2004) [18], Boffetta et al. (2006) [14] and Nelson et al. [16] show, in addition, that an increased risk is dose dependent.

It has also been reported that 68% of cancers of the upper aero-digestive tract are related to the contemporary consumption of alcohol and smoking tobacco [19]. Selected studies have demonstrated that the dose-response effect is also present in non-smokers.

For medium dosages of 21.5 units / day in males and 16.4 units / day in females (population of alcoholics), Thyngesen et al. (2009) [20] found an increased risk at other sites: the gallbladder, the digestive organs and the peritoneum, pleura, lung, kidney, prostate, cervix, ovary and skin. This study included 15258 males and 3552 females, who were followed up for a period of about 40 years.

Saieva et al. [21] calculated that alcohol dependency in a population increased the mortality rate by just under 5-fold compared to the general population. Alcohol-dependent women were the most affected; this was confirmed in a study by Thyngesen et al. [20], which found that the possibility of the occurrence of cancer can affect different areas of the body.

**CANCER SITES**

**The Oral Cavity and Pharynx**

In a previous review [13] we evidenced how alcohol is the most recognized risk factor in the cancer of the oral cavity and the pharynx, and together with tobacco it is the cause of most cases in developed countries (75% of cases). The relative risk (RR) is also significant in non-smokers [22].

In the oral cavity the detrimental effects of alcohol are well known on both an extracellular and intercellular level. Via these mechanisms, carcinogens can act on the proliferative activity of stem cells on the basal layer. In patients with oropharyngeal cancer a significant increase in the concentration of salivary acetaldehyde has been detected. This is also related to poor oral hygiene and to cigarette smoking. These behaviors increase the production of acetaldehyde by means of bacterial flora [5]. Seitz and Meier [5] evidenced how smoking changes the flora from being predominantly Gram −, progressively evolving to being predominantly Gram + with a concomitant increase in the concentration of acetaldehyde by 50-60%, as compared to non-smokers. It is worth pointing out that smokers who are exposed to even moderate amounts of alcohol can significantly increase the concentrations of acetaldehyde, whereby the most affected oral sites are the ventral tongue and the floor of the mouth [8,9,13].

Altieri et al. [22] detected a dose-dependent risk, with an odds ratios (OR) of 2.1 for multivariate 3-4 units / day, 5 for 5-7 units / day, 12.2 for 8-11 units / day and 21.1 for more than 12 units / day [13,19]. A meta-analysis by Tramacere et al. [23] recently showed an increase in risk for a consumption of less than 1 unit / day, with a progressively dose-dependent increase. More recently, Roswall and Weiderpass [24] reported the conclusions of the World Cancer Research Fund (WCRF)/American Institute of Cancer Research (AIRC) [25]. The WCRF identified five cohort studies and the AIRC identified nine studies. They showed a RR per drink/weekly of 1.24 (95% confidence interval, 1.18 to 1.30) [24,25].

**The Larynx**

Numerous epidemiological studies have shown that alcohol is an independent risk factor for laryngeal cancer [1,2].

Islami et al. [26] found in a recent meta-analysis that the risk significantly increases for doses of little more than one unit / day. This increase in risk is also present in non-smokers [27].

The risk of laryngeal cancer is influenced by genetic variations: in particular, the polymorphisms of genes that control DNA repair capability (“nucleotide excision repair”) [28].

**The Esophagus**

Between 50% and 75% of all cases of esophageal cancer are attributable, in both males and females, to alcohol. Ethanol promotes, through alterations to the motility and tone of the lower esophageal sphincter, gastro-oesophageal reflux and...
consequent esophagitis [13]. This disease is a condition of risk for the development of Barrett’s esophagus (intestinal metaplasia), dysplasia and adenocarcinoma. The chronic inflammation makes the esophageal mucosa more susceptible to nitrosamines and polycyclic aromatic carbohydrates [13]. These substances may be present in alcoholic beverages or in pre-cancerous substances converted in the liver. In addition, we remember the direct carcinogenic action of acetaldehyde that is formed in the oral cavity from the metabolism of ethanol by bacterial flora [13].

Several studies (prospective and case control) have shown a significant association between esophageal cancer (particularly squamous cells) and alcohol. The risk is dose dependent [1,2,13]. The relative risk (RR) increases significantly when there is an association with smoking. If you consume more than 20 cigarettes / day with 1-4 units / day, the RR increases to 5.1 times; if you consume between 4 to 8 units / day, the RR increases to 12.3; and if consumption is greater than 8 units / day, the RR increases by 44.4-fold [29]. The synergistic effect of this combination is important, because the majority of subjects who use alcohol are also chronic smokers (about 75%).

Jarl and Gerdtham [30] affirm that the risk is reversible following drinking cessation. They conclude that it is most likely that about 16 years are required until all elevated risk has disappeared.

The Stomach

The general population has shown a modest increase in risk [2]. A high risk has been reported in the alcohol-dependent population, especially in males [2]. The risk of gastric cancer has also increased in social drinkers suffering from infection by Helicobacter pylori (Hp). It is known, in fact, that this bacterium possesses ADH and, therefore, there is an increase in the gastric mucosa of acetaldehyde [6].

The Colon-Rectum

Several meta-analyses have evidenced a positive dose-response relationship between alcohol consumption and colorectal cancer [9,13]. These studies have shown an increase in relative risk of about 10-20% with a chronic consumption of about 50 g / day. Likewise, there is an association for both colon and rectal cancer [1,2,13].

Concerning cancer of the colon-rectum, an RR of 7.4 with a dosage of 20 g / day [9] was indicated. An important role is played by the production of acetaldehyde by bacterial flora.

For this reason ALDH polymorphisms are particularly relevant. It has been suggested that, in this area, alcohol may promote cancer by disturbing the equilibrium characterized by a balanced transfer of methyl groups in combination with an appropriate concentration of folates. It has been proposed that the role of alcohol may be particularly relevant in the presence of precancerous conditions such as, for example, aberrant crypt foci [29,31].

There is no consistent evidence of any influence from smoking [1,2].

The Liver

The association of alcohol consumption and hepatocellular carcinoma (HCC) is well known: different assessments made by both American and Italian groups have indicated that such a correlation is present in 32-45% of cases. The occurrence of HCC in the context of early-stage chronic liver disease is possible (especially in combination with other risk factors). However, the chain of events is as follows: steatosis, steatohepatitis, steatofibrosis, cirrhosis and HCC. Cirrhosis itself is a risk factor for the onset of HCC, even in subjects who have achieved abstinence. The possibility of the evolution, and especially the speed of the above sequence is certainly influenced by genetic patterns [32] and by risk factors or associated comorbidities.

Hassan et al. [33] found this to be true for the intake of large amounts of alcohol (80 g / day of ethanol) in association with HCV or HBV-related hepatitis, with an OR of 53.9 (in cases of isolated hepatitis an OR of 19.1, and in cases of isolated alcohol consumption an OR of 2.4). In cases with a correlation between alcohol (> 80 g / day) and diabetes mellitus (both insulin dependent and non-insulin dependent), there was an OR of 9.9 (for isolated diabetes an OR of 2.4).

In many experiments a model of alcohol-induced hepatocarcinogenesis has been proposed [8]. This model consists of a first phase (initiation, direct genotoxicity) and a second phase characterized by promotion / progression (angiogenesis induction). In the first phase we witness a mechanism of direct genotoxicity caused by oxidative stress and the formation of adducts, in association with a reduction in anti-oxidant mechanisms [7,34]. Moreover, an aberrant methylation has been evidenced, Toll-like-receptor alterations and shorter telomere. In particular, the telomere length decreases in relation to an increase in drink-units per day [35].

The close correlation between the consumption of alcoholic beverages and DNA fragmentation suggests that oxidative stress, which is a result of ethanol metabolism, may be responsible for direct genotoxic action [36]. A discontinuation of alcohol consumption was estimated to reduce the annual risk by 6-7% [36].

The Pancreas

With regard to pancreatic cancer, smoking, in combination with certain mutations or polymorphisms, is certainly the main risk factor. The relative risk (RR) is affected by alcohol at 21 units or more of alcohol per week [37]. Recently, Bagnardi et al. [38] confirmed, through a meta-analysis (572 epidemiological studies) this correlation only in the presence of huge alcohol intake. An increase of 19% compared to non-drinkers was found. This correlation was confirmed even after the removal of the most important confounding factors: smoking, obesity and diabetes mellitus.

The Lung

A significant increase in risk in the general population has been reported in 16 cohort studies [2]. Populations with risky / harmful alcohol consumption and alcohol-dependent populations were found to be at high risk in 7 case studies. Korte
et al. [39] showed the relative risk as being between 1.53 and 1.86 in case-control studies for doses of higher than 5 drinks / day. Freudenheim et al. [40] reported an increased risk for doses of higher than 30 g / day. An increased risk for doses of higher than 15 g / day in the absence of cigarette smoke has also been demonstrated.

**The Prostate**

A correlation between alcohol and prostate cancer was reported in 2 out of 9 case. This Danish study by Piu et al reported by IARC [2] revealed a significant increase in prostate cancer as compared to the general population.

The relation between alcohol consumption and prostate cancer was recently confirmed by some authors [41,42]. Bagnardi et al. [38] reported a significant increase in risk in heavy drinkers. This correlation was not found in the Asian population [43]. To date there is no unanimous opinion on this correlation: more studies are needed before reaching a final conclusion.

**The Bladder**

To date, the correlation between alcohol consumption and bladder cancer has not been identified [38]. On the other hand, the consumption of alcohol promotes bladder cancer in smokers and probably also in moderate doses [44].

**The Breast**

4-5% of all breast cancers are alcohol related, and breast cancer accounts for 60% of alcohol-related tumors in women. Numerous epidemiological studies have shown a positive correlation between alcohol consumption and the incidence of cancer. A risk has also been defined for moderate consumption. It has been shown that for a 10 g / day increase in ethanol consumption, there is a 10% increase in the RR [45,46].

In fact, Chen et al. [47] found that the RR increased (though slightly) at doses of lower than 10 g / day, as well as in a subgroup characterized by the absence of estrogen receptors, whereas in a subgroup with a higher prevalence of progesterone receptors there was an augmented RR at doses of less than 5 g / day. This experience also confirmed there is no safe dose.

Kwan et al. [48] showed that in overweight women (especially in those who are post-menopausal) who had already developed breast cancer, the risk of suffering a relapse significantly increased with a consumption of 3-4 units / week. Recently, Romieu et al [49] confirmed the increased risk of alcohol on breast cancer development. In particular this risk has been evidenced in women who started drinking before their first full-term pregnancy. Ethanol consumption was positively associated with the risk of both estrogen receptor (ER) positive and ER negative breast cancer risk [13,50,51].

Pathogenic mechanisms are certainly to be found in the oxidative stress caused by the actions of acetaldehyde and by changes in nutrition (folate, Vit.B6 and B12), but the main cause is due to interactions with estrogen. It is known that estrogens are metabolized by ADH, therefore competing with ethanol. Particularly high concentrations of acetaldehyde were observed in association with high levels of estrogen during the menstrual cycle. The consumption of alcohol is probably involved in the early stages of carcinogenesis caused by an increase in insulin-like growth factor 1, which is linked to an increase in the binding protein 3 of this growth factor.

Alcohol is one of the few risk factors that can be eliminated in the prevention of cancer and in particular of breast cancer [13,52].

**The evaluation of the international agency for cancer research (IARC)**

The IARC (World Health Organization) issued the final data on the relationship between alcohol / cancer in 2007 in Lyon, which were published in Monograph 96, 2010 [1] and confirmed in Monograph 100 E 2012 [2]. The IARC classification provides four target groups: Group 1: Substances carcinogenic for humans; Group 2A: Substances probably carcinogenic for humans; Group 2B: Substances with the possibility of being carcinogenic for humans; Group 3: Substances not classifiable as being carcinogenic for humans (still under study); Group 4: Substances probably not carcinogenic for humans.

In particular: 1) alcoholic beverages are carcinogenic for humans (Group 1). The consumption of alcohol has a causal relationship with cancers of the oral cavity, pharynx, larynx, colon-rectum, liver and breast, 2) the free acetaldehyde in alcohol promotes tumors of the esophagus and of the head and neck (Group 1). In relation to “point 1” it is interesting to note that the Agency has clearly used the word “consumption,” thus correlating alcohol and cancer not only in alcohol-dependent populations. Several authors have studied the role of free acetaldehyde. The most authoritative studies on this are by Lachenmeier et al. [53,54]:

The authors detected free acetaldehyde in all types of alcoholic beverage. They also found products where the free acetaldehyde was equal to 0, as distillation reduces the levels of acetaldehyde. Free acetaldehyde is able to act directly through contact [51]: this justifies the position in Group 1 of cancers of the head, neck and esophagus. The Agency stresses that aldehyde dehydrogenase genetic polymorphisms are particularly important.

The Lachenmeier study [55] identified other substances in alcoholic beverages included in Group 1: arsenic, formaldehyde, acrylamide, cadmium, benzene and others. In addition, an evaluation of the MOE (Margin of Exposition: this parameter relates the toxicological threshold to the exposure) revealed an impressive datum: a certain amount of ethanol, or its equivalent per day in units, is just as potent a carcinogen as other substances more likely to cause alarm in the general public.

**CONCLUSIONS AND PROPOSALS**

The conclusions to be drawn from the experimental and epidemiological evidence fully confirm the correlation between alcohol and cancer. In view of the relatively high frequency of certain types of cancer (esophagus, colon, liver, breast) and the
persistent chronic use of alcohol in our society, the link between alcohol and cancer should be taken into great consideration in both prevention programs, and for reaching an early diagnosis. The aim of prevention and health promotion programs should be to minimize the use of alcohol in the general population, reaffirming that the consumption of alcoholic beverages is risky, as alcohol is a toxic, carcinogenic substance, which involves not only alcoholics, but also social drinkers.

Some international institutions agree that there is no “safe” daily amount; however, “low risk” may be considered as follows: 20 g / day for males and 10 g / day for females [56].

It should be stressed, however, regarding the relationship between alcohol and cancer, several authors and international organizations have expressed the view, which we fully share, that the most responsible attitude of health professionals should be to not to specify “safe” dosages of alcohol. To date, in fact, the scientific evidence suggests a ratio of alcohol / dose-dependent cancer with no defined safe threshold [57-59].

The European Code Against Cancer affirms: “If you drink alcohol of any type, limit your intake. Not drinking alcohol is better for cancer prevention” [60]. In view of the high incidence of cancer in people with risky / harmful alcohol consumption and alcohol dependence, common screening and oncological prevention programs for the general population should be discussed again and put in place [61]. In addition, it should be noted that for a high percentage of cases (over 80% in our experience), there is a correlation with smoking.

All asymptomatic subjects with past or present risky / harmful alcohol consumption, and all subjects with a history of alcohol dependence or alcohol problems should undergo (at a frequency yet to be determined by the proper guidelines and compatibility with available economic resources): ENT check-ups, hepatobiliary-pancreatic ultrasonography, chest X-rays, breast ultrasonography, mammography, fecal occult blood testing and / or colonoscopy, testing for Helicobacter pylori infection and / or esophago-gastro-endoscopy (Table 2).

It should be emphasized that subjects should always undergo an appropriate internal medical assessment, taking into account the fact that alcohol can cause the development of around 60 different diseases and 11 different cancers [3, 62-64].

REFERENCES


42. Testino et al. (2016)


