The airwaves have been abuzz over a recent publication concerning the negative impact of alcohol on cancer-related deaths. The results of the article in the American Journal of Public Health confirm results of other studies. The WineDoctors would like to review the relationship between alcohol and cancer and focus on the impact of alcohol on breast and head and neck cancers.

Ethanol has been classified as the most important carcinogen in alcoholic beverages. The epidemiological evidence of the carcinogenicity of alcoholic beverage consumption shows little indication that alcohol's carcinogenic effects depend on the type of alcoholic beverage. A linear association has been noted for average daily alcohol consumption and cancer risk. There is a clear dose response meaning that the more alcohol one consumes, the higher that individual's cancer risk. Some other compounds found in wine (lead, arsenic, ethyl carbamate, acetaldehyde) may pose risks below thresholds normally tolerated for food contaminants.

Worldwide in 2002, about 389 100 cases of cancer were attributable to alcohol drinking—3·6% of all cancers, and 232 900 deaths from cancer deaths were attributable to alcohol consumption—3·5% of all cancer deaths. A causal link has been established between alcohol consumption and cancer of the upper aerodigestive tract (ie, of the oral cavity, pharynx, or esophagus); liver; colon; rectum; and breast. An association has been observed between alcohol consumption and cancer of the pancreas. There is evidence suggesting lack of carcinogenicity for cancer of the kidney and non-Hodgkin lymphoma.

Breast CA
Large cohorts of postmenopausal women have been evaluated, indicating a link between estrogen receptor positive cancers, but not triple negative cancers with regard to alcohol consumption. Data suggests that genetic/familial cancers testing positive for BRCA1 and 2 markers show no association with alcohol use. Precancer or DCIS has no association with alcohol use. However, women who consume alcohol and have hormonally sensitive estrogen receptor positive or progesterone receptor positive cancers might be prone to an increased risk of recurrence. This has to be weighed with the information that moderate alcohol consumption may have a cardio-protective effect in these patients.

The above associations do suggest a different physiologic basis for occurrence. Proposed mechanisms of action might include increased rates of circulating estrogen in women who consume alcohol in the menopause and possible additive effect with higher levels of circulating estrogen even in menopause in obese women.

Head and Neck CA
A clinical link between the chronic consumption of alcohol and head and neck cancer has been observed for decades. Alcohol was described initially as a risk enhancer only in smokers. A number of epidemiological studies have now provided sufficient evidence that chronic alcohol consumption increases the risk of head and neck cancer independent of exposure to tobacco smoke. The systemic effects of alcohol interact with local changes in the morphology and function of the salivary glands. In addition, alcohol leads to accumulation of pathologic microbes

within the mucosa, leading to chronic infection.

Acetaldehyde is a metabolite of alcohol oxidation is derived either from ethanol or tobacco and appears to act in the upper digestive tract as a local carcinogen in a dose-dependent and synergistic way. There us strong epidemiological, biochemical and genetic evidence supporting the role of the first metabolite of alcohol oxidation--acetaldehyde--as a common denominator. Acetaldehyde has direct carcinogenic and mutagenic effects by modifying DNA

ALDH2-deficiency and high active ADH1C result in two- to threefold salivary acetaldehyde concentrations after a dose of alcohol and this prevails for as long as ethanol is present in the blood and saliva. Alcohol is metabolized to acetaldehyde locally in the oral cavity by microbes representing normal oral flora. Poor oral hygiene, heavy drinking and chronic smoking modify oral flora to produce more acetaldehyde from ingested alcohol. Also, tobacco smoke contains acetaldehyde, which during smoking becomes dissolved in saliva. Via swallowing, salivary acetaldehyde of either origin is distributed from oral cavity to pharynx, esophagus and stomach.