



**COMMENTARY ON A ROLE OF VITAMIN B6 IN COLORECTAL CANCER: FURTHER  
INVESTIGATION IS REQUIRED**

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**ABSTRACT**

Colorectal cancer is the third most common cause of death worldwide and the second most common cause of death in United States. Dietary factors are the most modifiable important cause and consumption of vitamin B6 over a long period of time has been associated with reduced risk of colorectal cancer. So a large cohort or interventional study is needed to further confirm this study.

Vitamin B6 is a water soluble vitamin and takes part in more than 100 co-enzyme reactions. It may influence colorectal cancer risk in multiple ways, including its role in one carbon metabolism related DNA synthesis and methylation. The active form of vitamin B6 is Pyridoxal 5 phosphate (PLP) and is most commonly used to measure Vitamin B6 status. It also reduces inflammation, cell proliferation and oxidative stress. But observatory studies of dietary or dietary plus supplemental intake of vitamin B6 and colorectal cancer risk have been inconsistent. A PLP level of more than 20mmol/L is an indicator of adequate vitamin B6 status in adults.

The main sources of vitamin B6 in the United States include fortified cereals, starchy vegetables, beef and poultry and the recommended daily allowance (RDA) for vitamin B6 intake is 1.7 mg/d for men and 1.5 mg/d for women aged 51 years or older, although some groups like smokers, blacks, seniors and current and previous oral contraceptive users require higher intake.

Vitamin B6 may influence carcinogenesis through its role in methylation and DNA synthesis while animal models have shown that supplemental Vit B6 suppressed cell proliferation and reduced the number of tumors in the colon by inhibiting angiogenesis, suppressing nitric oxide and reducing oxidative stress. Also low vitamin B6 status has been linked to chronic inflammation, a potential risk factor for colorectal cancer.

While there have been several studies conducted, results largely remain inconclusive. A cohort study conducted by the **Karolinska institute**, Stockholm Sweden and

**Harvard School of Public health** showed that alcohol consumption increases the risk of colorectal cancer significantly in women having low vitamin B6 intake as compared to those with a higher intake. They observed that vitamin B6 reduced the colorectal cancer risk significantly.

Only two double blinded randomized, placebo controlled trials, the Norwegian Vitamin trial and the Western Norway Vitamin B Intervention Trial included 6837 participants were followed. The pooled analysis of data from these two trials showed no benefit of Vitamin B6 supplementation on incidental or fatal colorectal cancer.

There have been a number of confounding factors associated with the PLP levels and cancer risk. Individuals with high vitamin B intake tend to have healthy behaviors such as higher physical activity, less smoking and higher intakes of folate, calcium and vitamin D that may also reduce the risk of colorectal cancer.

Interaction with other factors might also result in inconsistent results. Alcohol consumption may decrease vitamin B6 levels, so the level of Vit. B6 levels might have a stronger association with colorectal cancer risk. A stronger inverse association with plasma PLP concentrations has been reported among alcohol drinkers compared to non drinkers. However current studies are limited and found no evident pattern.

Conclusion: Vitamin B6 intake and colorectal cancer in relatively nourished populations have been inconsistent

and a better understanding is needed of the determinants of plasma PLP concentrations. Further studies should focus on confounding factors.

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