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Impact of sleep on gastrointestinal cancer

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Abstract

Sleep problems have become a significant public health concern, affecting a large portion of the global population and have been linked to increased morbidity and mortality. The incidence of gastrointestinal (GI) cancers continues to rise, posing a substantial burden on healthcare systems worldwide. This editorial aims to delve into the impact of sleep on GI cancers, including esophageal, gastric, colorectal, hepatobiliary, and pancreatic cancer. Recent literature investigating the potential connections between GI cancers and sleep was reviewed. We considered aspects such as sleep duration, sleep disorders, and circadian rhythmicity, in order to explore the underlying mechanisms that can contribute to the development of GI cancers and propose avenues for future research.

Key Words: Sleep; Cancer; Gastrointestinal cancer; Esophagus; Stomach; Colon; Liver

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Core Tip: Sleep problems are a growing global health concern, affecting a large population, while the rise in gastrointestinal (GI) cancers poses a significant burden on healthcare systems. This editorial explores the impact of sleep on GI cancers, reviewing up-to-date literature.

INTRODUCTION

Sleep, the state the body requires as a process of recovery, is a vital life function that takes approximately one-third of every day. We often encounter the discussion of which is more important, quality *vs* quantity, in sleep. The simple answer is they are both important. However, it is crucial to understand that quantity and quality of sleep are closely associated. Quality of sleep means several things, including subjective feeling of rest, sleep efficiency, and normal sleep architecture (normal ratio of each sleep stage). The subjective feeling of rest is especially heavily associated with its duration. Typical sleep duration for adults ranges from six to nine hours, which is impacted by daily activities and environmental factors.

Sleep disorders encompass a wide range of conditions that disrupt normal sleep quality. The common sleep disorders, including insomnia, obstructive sleep apnea (OSA), and circadian rhythm disorders, are well known to play a role in chronic medical conditions and mental health disorders but have been understudied in cancer[1]. Commonly proposed mechanisms of cancer development, such as intermittent hypoxia, dysfunction to anti-tumor immunity, gut microbiome, and metabolic homeostasis, are also regularly discussed in the mechanism of sleep impact on medical conditions, suggesting a potential link between sleep disruption and cancers. The relationship between sleep disorders and types of cancer is still being investigated with possible bidirectional association[2,3].

There has been an increase in data supporting the relationship between sleep and various types of cancer, including breast[4], prostate[5,6], thyroid[7], lung[8], and gastrointestinal (GI) cancer[9-11]. Globally, GI cancers, such as esophageal, gastric, and hepatobiliary cancers, represent a major cause of morbidity and mortality [12]. Given the limitations in data, we were particularly intrigued by the recent study that explored the association between sleep and GI cancer[13]. While the primary mechanism of the pathophysiology between cancer and sleep disorders is not fully understood, early findings from this study indicated that sleep disorders and GI cancers, in particular, had an association around the time when the cancer diagnosis is initially made, suggesting that sleep disorders may be an early sign of cancer. In this editorial, our specific focus lies in the association between various aspects of sleep and the risk for specific GI cancers, including esophageal, gastric, hepatobiliary, and pancreatic cancer.

CURRENT INSIGHTS INTO SLEEP AND GI CANCER

The connection between inadequate sleep quality and cancer is gradually being elucidated as the multifactorial etiology of cancer is being better understood, involving cellular responses, hormonal alteration, immune function, and dietary factors.

Inflammatory, immune and cellular responses

Sleep disturbance is linked to the activation of the sympathetic nervous system, triggering inflammatory activity in human studies that can become chronic[14]. This chronic inflammation, in turn, fosters the initiation, progression, and metastasis of cancer. A landmark study in humans by Irwin *et al*[15] revealed how exposure to short sleep can quickly impact immunity against cancer, with a single night of four hours of sleep decreasing 70% of natural killer (NK) cells circulating in the immune system compared to eight hours of sleep. C-reactive protein and interleukin (IL)-6, common inflammatory markers, were associated with sleep disturbance and long sleep duration (> 8 h) whereas these markers were not associated with short sleep duration (< 7 h)[14]. In university students, the increase in IL-1 has a positive correlation with the Athens Insomnia Scale and Pittsburgh Sleep Quality Index (PSQI)[16]. What's more, chronic sleep deprivation in an animal study has also been reported to compromise the anti-tumor immune response, impacting NK cells and cytotoxic T cells[17]. In another animal model study that assessed the association between tumor growth and sleep disruption, sleep-deprived mice showed a 200% increase in the progression of tumor growth compared to the control group. Furthermore, *in vitro* study has demonstrated that chronic sleep deprivation affects the activation of macrophages, particularly M2 macrophages, that promote cancer growth[18].

Melatonin effects

Short sleep, sleep disturbance, and evening chronotype are associated with low melatonin. Melatonin is believed to have an anti-carcinogenic profile through various mechanisms, including eliminating reactive oxygen species (ROS), promoting a DNA repair system, activating NK cell function, hindering the initiation phase of tumorigenesis, and impeding the growth of cancer cell lines in humans[19]. The effects of melatonin in many types of GI cancer have been reported including esophageal cancer suppression, gastric cancer cell growth inhibition, enhancement of the efficacy of chemotherapy in hepatocellular carcinoma (HCC), colorectal cancer, and pancreatic cancer[20].

Sleep quality and quantity

For the majority of studies, the reference category for sleep duration was seven to eight hours per night. Short sleep

duration was typically defined as less than seven hours each night, while long sleep was characterized as exceeding eight hours per night[14]. Sleep fragmentation and insufficient sleep duration have been shown to amplify cortisol secretion, heighten insulin resistance, and contribute to weight gain, obesity, and diabetes each representing an independent risk factor for cancer[21,22]. Long sleep may indicate disrupted sleep continuity, impacting immune function and leading to an elevated release of pro-inflammatory cytokines as mentioned earlier. Long sleep has also been related to sedentary behavior and a less healthy lifestyle[23]. In patients with OSA, the intermittent hypoxia and disrupted sleep are believed to be the pathophysiological mechanisms that lead to upregulation of oxidative stress, hypoxia-inducible factors-1 transcription factor, ROS, vascular endothelial growth factor and DNA mutation, ultimately promoting tumor progression[24,25]. Duration of sleep has been shown in subsequent studies to impact GI cancers.

Circadian rhythm and its impact

Shift workers, especially night shift workers, are exposed to unnatural light at night, which can reduce melatonin secretion and may increase inflammatory cytokines production, circadian gene dysfunction, and impairment of DNA repair, leading to tumorigenesis. Lastly, chronotype impact on GI cancers can be linked to lifestyle, dietary, gut microbiota, and dietary factors. Individuals with a morning chronotype are more likely to have a healthier lifestyle. For example, smoking less and adhering to the Mediterranean diet in morning chronotype individuals have been associated with reduced risk of cancer[26]. On the other hand, the evening chronotype usually has poorer eating habits, potentially leading to heightened cancer risk[27]. Considering dietary factors is essential when estimating the risk of GI cancer. For gut microbiota that is associated with GI cancer, a decrease of *Alistipes* and an increased *Lachnospira* in individuals with a morning chronotype were found. The evening chronotype also exhibits higher insulin resistance and low-density lipoprotein levels, which are linked to cancer development[10].

ESOPHAGEAL CANCER

Limited research exists linking sleep factors such as sleep duration, sleep disorders, chronotypes, and their potential effects. This section explores available evidence and highlights the investigation into this specific GI malignancy.

Esophageal cancer is one of the most common types of cancer worldwide known risk factors for two types: esophageal squamous cell carcinoma (ESCC) and esophageal adenocarcinoma (EAC). Esophageal cancer has not been reported to have a significant correlation with sleep duration[28]. However, a notable association of sleep duration has been observed when classified by type of esophageal cancer. Few studies have examined the association between sleep disorders and these two types of esophageal cancer. In a prospective study that investigated sleep duration on EAC and ESCC risk in the United Kingdom Biobank, poor sleep behavior (defined as less than six hours or greater than nine hours of sleep a day and occasional daytime napping) was associated with a high risk of EAC, independent of genetic risk, but was not seen for ESCC[29].

Previous sleep disorders, including sleep apnea, insomnia, hypersomnia, circadian rhythm disorders, narcolepsy, parasomnia, sleep-related movement disorders, and unspecified sleep disorders that were found within one year of cancer diagnosis of these GI cancers, revealed a positive association with GI cancers overall, suggesting that sleep disorders may be a harbinger of GI cancers[13]. However, esophageal cancer alone failed to show a significant association with sleep disorders in subgroup analysis, which supported the findings from the propensity score matching study for participants with and without sleep disorders by Wu Zheng *et al*[30]. Moreover, there was no association between sleep symptoms such as daytime sleepiness, and snoring, with esophageal cancer both overall or within each subtype[29,31].

Concerning shift work, rotating shift male workers, known to have disruptions to circadian patterns, were found to have higher risks of esophageal cancer[32]. Surprisingly, fixed night shift work with persistent light exposure at night was not associated with esophageal cancer[32,33]. What is more, the investigation of chronotype and esophageal cancer revealed that the morning chronotype did not exhibit any apparent connection with esophageal cancer. In contrast, individuals with evening chronotype exhibited an elevated risk, specifically for ESCC, following a two-year enrollment period[13,29].

Based on the current evidence, both insufficient and excessive sleep durations may be contributing factors to EAC. The specific types of esophageal cancer should be considered when evaluating these associations. Further studies are needed to explore the impact of sleep disorders on esophageal cancer. Different types and settings of shift work as well as the types of cancer may have varying impacts on cancer risk. It is important to consider circadian preferences and disruptions, as there is potential relevance to specific types of esophageal cancer.

GASTRIC CANCER

Gastric cancer previously had a high incidence rate, but has shown a declining trend over the past several decades. Many changes to known risk factors for gastric cancers from reduction in tobacco smoking to improved sanitation, and diagnosing and treating *Helicobacter pylori*, have played an integral role in this trend. The association between sleep and gastric cancer has not been well understood, but there have been some studies to assess potential associations.

Gastric cancers are classified anatomically by location as cardia or noncardia, and histologically by type as diffuse or intestinal type, this separates types of gastric cancer into different risk profiles. Gastric cancer was investigated across five case-control studies from the Stomach Cancer Pooling Project Consortium, using stratified analysis by key factors of sex,

smoking status, socioeconomic status, anatomical site, histological type, and self-reported sleep duration[9].

Short sleep duration has been reported to be associated with gastric cancer in prior studies, this relationship was not found in this study[9,13,23,34]. However, a long sleep duration of at least nine hours was noted to be associated with gastric cancer, similar to previous studies. This relationship remained consistent whether categorized by anatomical site or histological type. Similar to esophageal cancer, gastric cancer did not exhibit a significant relationship with overall sleep disorders[13,30]. Interestingly, female adults with narcolepsy had a higher risk for gastric cancer[35]. Human leukocyte antigen haplotypes and immunological derangement in narcolepsy may be associated with cancer susceptibility. Frequent long naps were associated with an increased risk of gastric cancer and were exacerbated by night shift work[23]. However, there was no association between night shift work alone and gastric cancer[32,33]. In terms of chronotype, there was an inverse correlation between morning chronotype and gastric cancer[10] which supported the findings seen in gastroenteropancreatic neuroendocrine tumors, with lower progression and metastatic risks in the morning chronotype and higher risk in the evening chronotype[36].

While the connection between short sleep duration and gastric cancer remains unclear, a notable link can be seen with long sleep durations. Disruptions to circadian rhythm suggest increased gastric cancer risk, but the data remains controversial. Among sleep disorders, only narcolepsy exhibits a significant relationship with gastric cancer. Further studies are needed to explore the mechanisms for this association. The morning chronotype may be a protective factor for gastric cancer, aligning with patterns observed in other related tumors.

COLORECTAL CANCER

Colorectal cancer is a common and lethal cancer worldwide with the World Health Organization estimating it as the third most common diagnosed cancer and second leading cause of cancer-related death. Thus, early detection and treatment are vital. Furthermore, the relationship between sleep and colorectal cancer has been implicated relationship between sleep and colorectal cancer, further emphasizing the importance of understanding their relationship.

The evidence from a meta-analysis by Gong *et al*[19] demonstrated that long sleep duration (greater than nine or ten hours) increased the risk of colorectal cancer. A short sleep duration (less than six hours) has been linked to an increased risk of colonic adenomas development[37]. Nevertheless, neither short nor long sleep durations were associated with an elevated risk of colorectal cancer-specific mortality[38]. A sleep duration of seven to eight hours was associated with a lower risk of colorectal cancer[19]. Frequent daytime napping, occurring six to seven times per week, and extended durations exceeding 30 min, were associated with increased odds of colorectal cancer[23]. A study with the National Health Insurance Research Database of Taiwan to estimate the risk of colorectal cancer in those with sleep disorders found a significant positive association[39]. However, recent findings did not reveal a significant correlation with the overall history of sleep disorders, except if having more than five years of any sleep disorder diagnosis[13,30]. When factoring in psychiatric disorders, patients with sleep disorders and concomitant depression were found to have five times the incidence rate of colorectal cancer compared to the control group[39]. Further prospective studies are needed to prove causality. There is conflicting evidence on OSA, the most common sleep-related breathing disorder, there are conflicting evidence of OSA in the development of colorectal cancer[5,40]. Despite a non-significant relationship between OSA and colorectal cancer in a meta-analysis; subgroup analysis on studies with a follow-up period longer than five years indicated a significantly higher risk for colorectal cancer[4,41]. Adjustments for co-morbidities and variations in follow-up duration may influence the slow progression nature of the disease. Exposure to light at night may be associated with an increased risk of colorectal cancer in some studies[42]. However, similar to gastric cancer, night shift work did not show an association with colorectal cancer in meta-analysis[33]. The result for colorectal cancer outcomes in this meta-analysis displayed moderate heterogeneity, which is supported by this finding of a recent large study[32,33]. As with gastric cancer, a morning chronotype was linked to a reduced risk of colorectal cancer[10].

Suboptimal sleep durations are linked to a higher risk of colorectal cancer. Both prolonged and frequent napping carry a heightened risk for colorectal cancer. The literature shows a potential higher risk of sleep disorders in colorectal cancer with sleep disorders, but additional research with long follow-up periods is needed to establish causality in these relationships. Chronotype significantly influences colorectal cancer and could play a crucial role in preventive strategies.

HEPATOBIILIARY-PANCREATIC CANCER

Hepatobiliary cancers are often common in individuals with chronic liver disease. There is very little evidence assessing the association between hepatobiliary-pancreatic cancer and sleep. This section delves into relevant research on the impact of sleep on the liver, biliary duct, and pancreatic cancer.

A prospective study of patients with advanced hepatobiliary-pancreatic cancer found that short and long durations were associated with increased mortality in the U-shape pattern[43].

This U-shape correlation pattern was found in sleep duration and incidence of HCC, hepatobiliary cancer and the mortality of HCC[44]. However, the correlation between sleep duration and individual cancer sites remains controversial. For instance, another study found a significant inverse correlation between sleep duration and HCC and hepatobiliary cancer[45]. For obese and postmenopausal patients in the United States, long sleep duration was found to be positively associated with an increased risk of liver cancer, but short sleep was not[46]. However, long duration of sleep may imply the compensation mechanism of preexisting medical conditions, including sleep disorders. Thus, it may not be the root cause of cancer genesis. Overall, there is no strong evidence suggesting a significant relationship between sleep duration

and liver or pancreatic cancer[28]. There was a positive correlation between the duration of daytime napping and both HCC and biliary tract cancer[44,45]. In a meta-analysis conducted by Wu *et al*[4], OSA was found to be associated with a higher risk for liver and pancreatic cancer. Nonetheless, recent studies have shown conflicting correlations between sleep disorders and pancreatic cancer, and no correlation has been observed in liver cancer[13,30]. Insomnia may be a risk factor for hepatobiliary cancer[45]. There was an association between a higher risk of liver cancer and the exposure to outdoor light at night or night shift[47]. Rotating shift work is associated with a decreased risk of liver cancer in Japanese men. In this study, there is no association between rotating shift work in the female gender and night shift work in both genders[32]. In contrast, rotating shift work is associated with a higher risk of biliary duct cancer[48]. In terms of chronotype, genetic liability to morningness was not associated with liver, biliary duct or pancreatic cancer in the combined analysis.

The association of sleep durations, sleep disorders, shift work and the risk of hepatobiliary-pancreatic cancer remains an area of interest, although conclusive evidence is currently lacking. The protective profile of morning chronotype against hepatobiliary-pancreatic cancer has not yet been fully elucidated, emphasizing the need for further studies to provide a more comprehensive assessment.

CLINICAL IMPLICATIONS

Lifestyle management, such as adhering to appropriate sleep durations based on age, can foster optimal development and health. For individuals with insufficient sleep duration, implementing interventions such as sleep hygiene to extend sleep duration may enhance sleep quality and potentially reduce the risk of GI cancer. Similarly, screening for underlying conditions that may lead to prolonged sleep duration, followed by appropriate treatment, might normalize sleep duration. Individuals with sleep disorders are likely to be encouraged to undergo early screening for tumor detection and treatment, adhering to established protocols, given the potential impact on GI cancer. Embracing the habits of a morning chronotype may confer health benefits, resulting in a lower risk of GI cancer. Interventions that may impact circadian rhythm and sleep quality, such as melatonin and light exposure adjustment, have been studied and may offer assistance in GI cancer treatment[20,48]. However, there is insufficient evidence to establish a clear recommendation for the interventions. More research will be required to elucidate the impact of sleep adjustments on cancer, as they may influence cancer outcomes.

CONCLUSION

Unfavorable sleep duration may increase the risk for GI cancers. Many studies indicated that an extended duration of nocturnal sleep, frequent napping, and insufficient sleep may have deleterious effects on health, increasing the risk for specific GI cancers. Future large-scale prospective studies, with additional stratification of individuals with sleep disorders and more detailed measurement of sleep duration, shift work status and chronotype, are needed to clarify the cause-effect relationship between sleep disturbances and GI cancers.

FOOTNOTES

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