TIME-RESTRICTED EATING FOR IMPROVED METABOLIC HEALTH: ALIGNING MEALS WITH CIRCADIAN RHYTHMS

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Background. Ancient man relied on their circadian clock, driven by natural light to regulate feeding behaviors. Modern eating habits and artificial light have disrupted our circadian rhythm which could lead to metabolic diseases. Certain appetite and satiation hormones have varying levels based on the circadian rhythm. **Objective of the study.** To Investigate the optimal daily eating window based on circadian rhythms for improved metabolic health and the influence of the suprachiasmatic nucleus (SCN) on hunger hormones. **Material and methods.** This article is based on information gathered from many publications and literature published since 2017 that are accessible on PubMed, PMC, Google Scholar and NCBI. **Result.** Eating habits like time restricted eating (TRE) and intermittent fasting with a feeding window of 6-10 hours (8am - 6pm) and a fasting period of 14-18 hours aligns with the circadian rhythm and has been shown to benefit weight loss and improve insulin sensitivity due to synchronization of peripheral hormone secretion with the SCN. TRE has also been shown to lower triglyceride and LDL cholesterol levels. It is recommended that food intake begins at 8am after the cortisol peak, signaling the beginning of the activity phase, and end at the ghrelin and insulin peak around 6pm. Ghrelin levels increase across the day while leptin has higher levels at night. Circadian misalignment alters these hormones and therefore eating behaviors which may lead to impaired glucose tolerance and increased fat accumulation. **Conclusion.** This study highlights the importance of circadian rhythm in regulating appetite hormones and metabolic health. By synchronizing mealtimes with hormonal fluctuations within an eating period, TRE promotes optimal metabolic function and prevents chronic metabolic disorders. **Keywords:** Circadian rhythm, Intermittent fasting, Suprachiasmatic nucleus.

INVESTIGATING THE LINK BETWEEN ALCOHOL USE DISORDER AND THE SEVERITY OF ACUTE RESPIRATORY DISTRESS SYNDROME

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Background. Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are acute respiratory failure syndromes with a high mortality rate. Byproducts of alcohol metabolism like acetaldehyde and reactive oxygen species lead to oxidative stress and inflammation which could worsen recovery outcomes, due to increased risks for conditions like systemic inflammation, sepsis and immune dysfunction. **Objective of study**. Investigate the mechanisms by which alcohol use helps the development of ALI and ARDS. Explore Wnt/β-catenin pathway as a therapeutic target to counteract these effects. **Material and methods**. This article is based on information gathered from publications and literature published on PubMed, Google Scholar and NCBI. **Results.** Patients with alcohol use disorder (AUD) have a 2-4 times higher risk of developing ARDS. People with a history of alcohol abuse have twice the risk of developing sepsis and patients with sepsis are twice as likely to develop ARDS. Alcohol is a major risk factor for the development of ALI/ARD as it increases the risk of aspiration and pulmonary infection and disrupts the immune system and non-immunologic host defense mechanisms leading to immune dysregulation of alveolar macrophages and dysfunction of the alveolar epithelial barrier. Alcohol Use Disorder (AUD) heightens ALI/ARDS risk by decreasing immune defenses and lung barrier functions via affecting membrane permeability, glutathione depletion and impairment of macrophage function. Wnt/β-catenin pathway offers therapeutic potential by suppressing epithelial mesenchymal transition for reducing lung injury. **Conclusion**. Alcohol consumption greatly increases the risk of ALI and ARDS by disrupting immune defenses and weakening lung barriers, causing enhanced inflammation and oxidative stress. Targeting mechanisms like the Wnt/β-catenin pathway may offer therapeutic benefits to counteract these effects. **Keywords:** ALI, ARDS, alcohol, immune dysfunction, oxidative stress.