Inflammation, sleep and detoxification
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The biochemical mechanisms responsible for sleep regulation are very complex. Interleukin-6 (IL-6) and tumor necrosis factor (TNF) are fatigue-inducing cytokines, and the daytime secretion of IL-6 is negatively influenced by the quantity and quality of the previous night's sleep. Recent studies suggest that IL-6 is a putative 'sleep factor' and its circadian secretion correlates with sleep and sleepiness. IL-6 is elevated in disorders of excessive daytime sleepiness such as narcolepsy and obstructive sleep apnea. It correlates positively with body mass index and may be a mediator of sleepiness in obesity. The secretion of this cytokine is stimulated by total acute or partial short-term sleep loss reflecting the increased sleepiness experienced by sleep-deprived individuals. (Vgontzas AN Z. M.-B., 2002)

Studies that evaluated the 24-hour secretory pattern of IL-6 in healthy young adults suggest that IL-6 is secreted in a biphasic circadian pattern at about 08.00 and 21.00, and there are two zeniths at about 19.00 and 05.00 h. In contrast, following sleep deprivation or in disorders of sleep disturbance, (e.g. insomnia), IL-6 peaks during the day and, based on the level of stress (cortisol) system activity, contributes to either sleepiness and deep sleep (low cortisol) or feelings of tiredness and fatigue and poor sleep (high cortisol).

This study concluded that IL-6 is a mediator of sleepiness and its circadian pattern reflects the homeostatic drive for sleep. (Vgontzas AN B. E., 2005)

Other studies have shown that excessive levels of IL-6 may cause insomnia and conversely chronic sleep deprivation has been shown to further elevate IL-6 levels. (Vgontzas AN Z. M.-B., 2002) The poor quality of insomniacs' sleep may be associated with a hypersecretion of IL-6 and TNF during the daytime, which, in turn, correlates with the fatigue experienced by these patients. (Krueger, 2008)
A novel treatment protocol may be to use anti-inflammatory supplementation during the day to reduce the level of these two cytokines and treating insomnia as an inflammatory disorder.

It goes without saying that if sleep is disturbed then the detoxification process will also be negatively affected.

**Inflammation and detoxification**

One of the liver’s most important functions is to convert metabolic products and toxins into safe, soluble substances which can be eliminated. A number of biochemical pathways are involved in liver biotransformation. Phase II liver detoxification reactions involve the conversion of the products generated from Phase I into hydrophilic substances to allow for rapid elimination. If the liver is inflamed these processes may underfunction leading to toxicity and free radical accumulation. This then leads to a vicious cycle of further liver irritation and inflammation.

**Toxicity, Inflammation and acidity**

IL-1 beta, IL-6 and TNF have all been reported to affect hepatic metabolism in a variety of ways. The best known and most studied effects being the synthesis and secretion of a variety of glycoproteins during the acute phase response. Other effects of cytokines include changes in hepatic glucose, glycogen and triglyceride, lipoprotein, mineral and xenobiotic/steroid metabolism. (Lacour S, 2005)

However, significant hepatotoxicity has been noted in several well-described clinical states and experimental models whose pathobiology is thought to be mediated by particular cytokines.

Studies of clinical and experimental sepsis and endotoxemia strongly suggest that cytokines can in fact play a role in hepatic damage.

Whether the hepatotoxicity represents an exaggeration or alteration of normal beneficial homeostic response or whether hepatotoxicity is caused by high cytokine levels is unknown. Further suggesting possible detrimental effects of elevated cytokines is that the clinical manifestations of several acute and chronic hepatic diseases might be cytokine mediated.

Pro-inflammatory cytokines such as TNF, IL-1 beta and IL-6 are released into the bloodstream both from the liver and from distal sites during hepatic toxic injury (Lacour S, 2005)
Body pH has an impact on almost every aspect of health. Antibiotics, surgery, drugs, sugar and many of the foods we habitually eat imbalance pH by contributing to acidity. Fortunately there are simple steps we can take to eliminate acidity and improve the body’s pH balance, boosting immunity and eliminating pain and chronic symptoms of disease. Use of Alkalysing diets and buffer the acidity with potassium are effective ways to correct unbalanced pH.

**Support the relevant organs throughout the detoxification process**

Support of the GIT, liver and kidneys is essential to reduce exposure to the potential free radical and toxic storm produced during detoxification. Glutamine, zinc and vitamin A support mucosal membrane integrity, GAG barrier function and immune function.

High level toxin exposure depletes glutathione faster than it can be produced or absorbed leading to increased susceptibility to diseases associated with elevated toxins such as cancer, chronic fatigue syndrome and auto-immune diseases. Cysteine, glycine, glutamine, and vitamin C are the precursor nutrients for glutathione production. Supplementation with these nutrients and also alpha lipoic acid may help to replenish glutathione levels.

Quercetin, turmeric, glycine and vitamin C reduce inflammation systemically. Anti inflammatory support is required due to the increase of free radicals via Phase I and phase II liver detox. Turmeric may also help stimulate the flow of bile and inhibit Helicobacter pylori growth (Mahady GB, 2002)

Glycine, glutamine and potassium help support kidney health & function (Ming Yin, 2002) Quercetin (SINGH Devinder, 2004) and glycine (TANG, XIE, & A., 2006) may protect kidneys from free radical damage and reduce renal inflammation

**Conclusion**

A comprehensive nutritional and herbal formula that supports GIT, liver and kidney function and providing additional anti-inflammatory support may prove a valuable foundation for detox, sleep and inflammation.

The original article can be found at:

Bibliography


Mahady GB, P. S. (2002). Turmeric (Curcuma longa) and curcumin inhibit the growth of Helicobacter pylori, a group 1 carcinogen. Anticancer Res., 22(6C):4179-81.


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