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BEYOND THE BREW: COFFEE'S ROLE IN LIVER IMPAIRMENT

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Chronic liver disease (CLD) represents a significant global health burden, progressively advancing through stages of inflammation and fibrosis to cirrhosis, irrespective of its underlying etiology be it viral hepatitis, alcohol overconsumption, or metabolic dysfunction (Muriel, 2017; Tapper and Parikh, 2023).While the liver does display the remarkable ability to regenerate (replace damaged or lost tissue with fresh/healthy cells), persistent chronic damage can result in systemic liver inflammation, which if left unchecked can progress to fibrosis (the formation of scar tissue), cirrhosis (advanced, irreversible scarring), and ultimately, hepatocellular carcinoma (HCC).

Coffee has become as a promising transformable factor in liver health, with a growing body of research investigating its anti-fibrotic properties. Worldwide evidence suggests that coffee, one of the world's most consumed beverages, may offer hepatoprotective effects (Saab et al., 2014). However, the dose-response relationship between coffee intake and the risk of advanced liver fibrosis remains to be fully researched. Regular consumption of black coffee (2 to 4 cups) significantly benefits reduce fatty liver by lowering elevated enzymes, reduce inflammation, reduce risk of fibrosis and cirrhosis and also acting as an effective antioxidant against liver damage.

The beneficial effects of coffee and caffeine extract against liver fibrosis have been demonstrated by several studies using standard rodent models of experimental liver fibrosis induced by intoxication with dimethylnitrosamine (DMN), carbon tetrachloride (CCl4), or thioacetamide (TAA). In almost every study, ingestion of coffee blocked toxin-induced liver fibrosis and cirrhosis. Of note, conventional filtered coffee is the form generally used in most of the published studies supporting its protective role.

The anti-fibrotic effects of coffee are attributed to a complex interplay of its bioactive compounds, including caffeine, chlorogenic acids, and diterpenes (Vargas-Pozada et al., 2025). Caffeine acts as an antagonist of adenosine receptor subtype 2A (A2AR) on hepatic stellate cells (HSCs), the primary fibrogenic cells of the liver. This antagonism disturbs a profibrotic signalling cascade involving Cyclic Adenosine Monophosphate (cAMP), Protein Kinase A, and Extracellular signal-Regulated Kinases1 and 2 (ERK 1/2), thereby inhibiting HSC activation, proliferation, and the synthesis of extracellular matrix components like procollagen type I. And also a separate pathway involving p38 mitogen-activated protein kinase (p38 MAPK) influences procollagen type III production (Wang et al., 2014). Coffee components suppress the activation of the NLRP3 inflammasome (an oligomeric protein complex involved in innate immunity) and the Nuclear Factor Kappa B (NF-κB) signalling pathway. This leads to a reduction in the release of pro-inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNF-α), interleukin-1β, and interleukin-6, effectively decrease the chronic inflammatory microenvironment that drives fibrogenesis (Vargas-Pozada et al., 2022). Coffee activates of the transcription factor Nrf2, which express of endogenous antioxidant enzymes like superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). This action reduces oxidative stress, a key mediator of hepatocellular injury and HSC activation (Vicente et al., 2014).

Progression of liver injury to fibrosis to cirrhosis is a slow, but deadly, process. The number of North American and European patients with CLD is increasing, Thus identification of simple measures that can slow fibrosis and prevent cirrhosis in at-risk patients is critical. Because coffee consumption appears to have salutary effects on human health overall, coffee is an attractive lifestyle measure that patients can take.