Journal of
Alcoholism & Drug Dependence

Akhtar, J Alcoholism Drug Depend 2013, 1:1
DOI: 10.4172/2329-6488.1000e104

Editorial Open Access

Alcohol, Gut Microbiota and Epithelial Barrier: An Interdependent Relationship

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Alcohol remains the most abused substance which affects every organ system within the body and causes enormous loss of economic and public health resources. According to an estimate, alcoholism affects 18 million adults in the U.S. and costs over 185 billion dollars annually. An association between alcohol and traumatic injuries of all types has long been recognized. Nearly one million burn injuries are reported every year within the United States, half of which occur under the influence of alcohol intoxication. Studies have established that alcohol exposure is not simply a risk factor for traumatic injuries but that it negatively complicates post-injury outcomes. In burn patients, alcohol intoxication at the time of injury leads to delayed wound healing, increased susceptibility to infection and longer hospital stays. In addition, intoxicated patients die of smaller burns. Regardless of alcohol exposure, severe burn injury results in organ dysfunction and disability, often leading to premature death. Yet, the mechanism(s) by which alcohol enhances post burn pathogenesis remains largely unclear.

The adult human intestine houses more than 100 trillion bacteria composed of an estimated 500-1000 different species of aerobic, facultative and anaerobic bacteria. Under healthy conditions the intestine maintains a barrier which prevents intestinal bacteria and their products from reaching systemic organs. However, this barrier is compromised following insult, including alcohol exposure, burn and other traumatic injuries. In such instances, intestine-derived bacteria and their products present a major clinical problem to burn/trauma victims, patients with alcohol exposure as well as ICU patients and patients developing multiple organ failure.

Recent studies suggest that gut microbiota plays critical roles in maintaining an effective gut mucosal barrier and driving immune homeostasis. There exist interdependent relationships between gut microbes, mucosal immunity and the gut epithelium. Under homeostatic conditions, these interrelationships not only prevent disease, but also generate appropriate immune responses. Disturbances in gut microbiota underlie a number of human diseases including alcoholic liver disease, obesity as well as severe systemic inflammation response syndrome following burn injury. Changes in gut microbiota may alter bacterial-epithelial cell interactions and contribute to gut tissue damage and leakiness following alcohol and burn injury.

The detection of bacteria and their products by the epithelial cells is achieved via a family of specialized receptors commonly known as pattern recognition receptors (PRR). The well characterized PRR include members of the Toll-like receptor (TLR) and nucleotide oligomerization domain (Nod)-like receptor (NLR) families. Among the receptors identified, Nod-1 is one of the major determinants in sensing and mediating the effects of Gram-negative bacteria. Nod-1 is expressed ubiquitously in both intestinal epithelial and immune cells. The ligation of TLR and Nod receptors by their respective agonists results in the activation of downstream inflammatory pathways, leading to activation of NFkB, a master transcription factor involved in the release of multiple inflammatory mediators. It is likely that both TLR and Nod act in synergy to drive the production of inflammatory cytokines following ethanol and burn injury. In summary, the dynamics by which a shift in gut microbiota, after ethanol and burn injury, drives gut tissue damage involve an array of interdependent pathways.

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Received December 21, 2012; **Accepted** December 26, 2012; **Published** December 28, 2012

Citation: Akhtar S (2013) Alcohol, Gut Microbiota and Epithelial Barrier: An Interdependent Relationship. J Alcoholism Drug Depend 1: e104. doi:10.4172/2329-6488.1000e104