We are grateful to the reviewer and editor for their positive comments and insightful suggestions. We have studied them carefully and added a summary table indicating the actions, roles and effects of each micronutrient in the etiology of ALD. We upload a response letter that includes a point-to point response to the issues raised in the peer-review report. Moreover, we have also gone through the paper thoroughly, corrected all the typos, and double-checked each of the reference to ensure all necessary information is provided, which we hope meet with approval.

#Reviewer #1:

Scientific Quality: Grade A (Excellent)
Language Quality: Grade A (Priority publishing)
Conclusion: Accept (General priority)
Specific Comments to Authors: The manuscript describes the relationship between pathological condition of chronic alcoholic liver disease and micronutrients such as zinc, iron, cupper, selenium, magnesium, vitamin D, and vitamin E. The contents deal with a current topic and cover related literatures widely. Thus, the manuscript deserves publication in WJG. For readers' convenience, I suggest authors to add a summary table indicating the actions, roles and effects of each micronutrient in the etiology of the disease.

We are pleased to see a high level of enthusiasm for our paper. We thank the reviewers for the valuable comments. Following the reviewer's suggestion, we have added a summary table indicating the actions, roles and effects of each micronutrient in the etiology of ALD (**Table 1**).

Table 1 Micronutrients imbalance in patients with ALD

Micronutrient	Metabolic status	Effects	Potential mechanisms	Representative references
Zinc	Deficiency	Intracellular signaling transduction, inflammatory response, ROS production, immunoregulation	Decrease the tight-junction proteins, increase the risk of intestinal barrier dysfunction; Inhibition of oxidative stress; Disturb dendritic cells' ability to respond to LPS; Activate apoptosis	[15, 17, 24]
Iron	Overload	Control the transportation of oxygen; DNA biosynthesis; ATP synthesis	Activate HSC, promoting liver fibrosis; Induce ferroptosis and mitochondrial dysfunction; Provoke oxidative damage through Fenton reaction; influence myelination and neurotransmitters	[34-37, 43] [45]
Copper	Deficiency / overload	The precise function of bone marrow and central nervous system; A cofactor of many antioxidases	Interacts with other trace elements, and function as a cofactor of antioxidase that is responsible for antioxidant defense	[48-51]

Selenium	Deficiency	Antioxidant property	Increase the enzyme activity of glutathione peroxidase and protect against oxidative injury; participant in autophagy, caspase-involved apoptosis, and NF-kB-implicated inflammation regulation	[52, 55-58]
Magnesium	Deficiency	Participate in enzymatic reactions, neurotransmission, glycolysis, and mitochondrial function	Perturb the extrusion of cellular magnesium through Na^+-dependent and Na^+-independent manner	[66-68]
Vitamin D	Deficiency	Anti-fibrosis, anti-tumor, and anti-inflammation; Immunomodulatory	Not yet fully understood	[73, 74]
Vitamin E	Deficiency	Antioxidative properties; protected against hepatocyte necrosis and maintained mitochondrial integrity	Diminish alcohol-induced oxidative damage, and improve antioxidant defense; Regulate the EGFR-AKT and EGFR-STAT3 pathways.	[84, 88-90]