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ESPS Peer-review Report
Name of Journal: World Journal of Gastroenterology
ESPS Manuscript NO: 5132
Title: The Role of H2S in Portal Hypertension and Esophagogastric Junction Vascular Disease
Reviewer code: 01407353
Science editor: Qi, Yuan
Date sent for review: 2013-08-18 19:37
Date reviewed: 2013-09-04 17:28

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[ ] Grade A: Priority Publishing	Google Search:	[ ] Accept
[ ] Grade B (Very good)	[Y] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[Y] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	
[ ] Grade D (Fair)		BPG Search:	[ ]Rejection
[ ] Grade E (Poor)	[ ] Grade D: rejected	[ ] Existed	[ ] Minor revision
		[ ] No records	[Y] Major revision

COMMENTS TO AUTHORS

Major concerns 1) Can Authors explain the enormous difference in numbers of patients and controls when comparing the "Material and Methods" (200 patients with cirrhosis-induced portal hypertension and 100 healthy controls, respectively) and the "Results" sections –Table 1- (23 patients and 25 controls, respectively). 2) Rather than including only cirrhotic patients with portal hypertension and compare them with healthy subjects, it would be better to include also a group of cirrhotics without portal hypertension. In this way, it could be possible to compare directly cirrhotics with and without portal hypertension, and even to test if H2S is associated with portal hypertension independently from liver dysfunction. 3) In Results, Authors speak about an inverse correlation between H2S plasma levels and portal diameter. However, they refer to Table 1), where no correlation is shown and only the difference in portal diameter between patients and controls is presented. 4) In the "Results" section, page 11th, Authors state that "low H2S concentrations directly affected the proliferation and apoptosis of vascular smooth muscle cells, which in turn leads to pathological changes in the blood vessels of the esophago-gastric junction region". However, since no data from upper endoscopy is presented, this statement lacks support. Rather, if gastroscopies were performed and data are available, it would be very interesting to compare H2S plasma levels in patients with/without esophago-gastric varices. 5) Determination of ERK1/2 in the whole liver tissue is completely aspecific, and the statement that, "because pERK1/2 expression directly regulates cell proliferation, …in the SPH model liver cell proliferation is stimulated" (pag 12th, first line) is speculative. ERK1/2 determination could acquire some significance if determined in portal vein smooth muscle cells. Minor concerns 1) In Figure 3), panel C, data concerning Bcl-2 and Bcl-XL levels in primary portal vein smooth muscle cells are presented. However, in the the "Material and

Methods" section, these cell line is not "introduced". Please give details. 2) In Figure 4), data from rabbit omentum vascular smooth muscle cells are presented. However, again, in the the "Material and Methods" section, this cell line is not "introduced". Please give details. 3) In the "Western blot Method" section, it is written that Bcl-XL and Bcl-2 were determined in liver tissues. However, in the "Results" section and in Figure 3) it appears that they were determined only in lysates from smooth muscle cells. Please specify. 4) Authors should not mention hepatocyte apoptosis in the "Discussion" section (page 13th, line 8th from the bottom) since they don't provide data about it. 5) The phrase "long term cirrhotic liver tissues showed symptoms of reduced apoptosis rates" is generic and the word "symptoms" is completely wrong in this context. 6) English should be better revised with the aid of a native English speaker.

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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[ ] Grade A: Priority Publishing	Google Search:	[ ] Accept
[ ] Grade B (Very good)	[ ] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[ ] Grade C (Good)	[Y] Grade C: a great deal of language polishing	[ ] No records	
[Y] Grade D (Fair)		BPG Search:	[ ]Rejection
[ ] Grade E (Poor)	[ ] Grade D: rejected	[ ] Existed	[ ] Minor revision
		[ ] No records	[Y] Major revision

COMMENTS TO AUTHORS

H2S, NO, CO all have biological effects, some of them rather being rather similar. Any claim of an H2S impact has to control the other gases as well. Method for measuring apoptosis should be explained more in detail. FACS is mentioned but not in the results? The number of measurments should be indicated Presentation of data are presented as mean ± SD only is justified if normal distributed. $p<0.05$ should be considered statistically significant only if not multiple comparisons are done. The study started with 200 patients, but in table 1 there are left only 23 with portal hypertension? What is the concentration of H2S? Why using so high concentrations in the cell culture? Magnification of histological images? Electron microscopy showed cell damage in the controls, but less with low concentration of H2S? What is low concentration, how many repetitions, what time of culturing? Is there any optimum concentration of H2S? Correlation betwwen liver damage Child-score and H2S measurements? any other measurments of liver enzymes as parametr of liver cell damage?