MATHEWS Open Access Journals

ISSN: 2474-6797

Short Communication | Mathews Journal of Cancer Science

Suggested Model to Induce Esophageal Cancer and Its Precursor Lesions in the Esophagus of Rats

Dennis Stamp[1] *

[1]23 Fairmar Ave. Toronto, On. Canada. M8Y2C7.

Corresponding Author: Dennis Stamp, 23 Fairmar Ave. Toronto, On. Canada. M8Y2C7, **Tel:** + 416- 239- 8708; **Email:** Dennis.stamp@gmail.com

Received Date: 02 Feb 2018

Accepted Date: 23 Feb 2018

Published Date: 26 Feb 2018

Citation: Stamp D. (2018). Suggested Model to Induce Esophageal Cancer and Its Precursor Lesions in the Esophagus of Rats. M J Canc. 3(1): 014.

INTRODUCTION

In a previous communication [1] it was suggested that liposomes loaded with UDCA (ursodeoxycholic acid) could be used to introduce anti -carcinogens into the esophagus of patients. Here we explore the possibility of using rats instead of patients. The reasons for shifting to rats are obvious, they will always be available and can produce valuable information without inconvenience to humans.

In this study rats would be fed a diet containing Sodium Cholate Hydrate originating from either Ox or Sheep bile and which is over 99% pure. It is manufactured and sold by Sigma Chemical Company and consists of 4 bile acids. These BAs are the sodium salts of deoxycholic acid (DCA), taurocholic acid (TCA), Glycocholic acid (GCA) and Cholic acid (CA). TCA and GCA are conjugated bile acids and are linked to taurine or glycine. All these bile acids, except the taurine conjugates, are insoluble at pH values less than pH4. TCA, being a sulphonic acid, is soluble at all pH values in the GI tract. According to the Hofmann and Mysels Principle [2] "Free and Glycine conjugated bile acids are insoluble at acid pH but as the pH is increased, their solubilities would increase exponentially until the Critical Mycellar Contration (CMC) is reached at pH > 7 when they would become almost completely soluble. " Sodium Cholate hydrate is mixed with the Standard AIN 76 Diet at approximately 10mg per Kg of diet. At the CMC, the bile acids would easily enter the esophageal cells and start carcinogenesis.

Sodium cholate hydrate has some unusual properties among which are

1) It has a pH range of pH 5 to pH 8, is used in membrane technology and in culture media. As well, it is a non- denaturing detergent.

Of the 4 bile acids mentioned above

1) It was recently discovered by Bernstein et al [3] that DCA is a colon carcinogen in mice. Why the esophagi of these animals were never checked for their ability to produce esophageal cancers will never be understood. DCA is the most hydrophobic and carcinogenic BA made in the animal body and as well, it is functional in other areas of research such as removal of submental fat in humans.

2) Sho Sato et al [4] have developed a model to produce EAC and ESCC in rats and has cited many references showing that Taurocholic acid exposure promotes esophageal squamous cell carcinoma (ESCC) and adenocarcinoma (EAC) in rats.

EXPERIMENTAL

Rats are fed ad lib with a mixture of sodium cholate combined with the Standard AIN-76 Diet (Standard American Institute Of Nutrition Diet) and at a concentration of ~10 mg/ Kg of diet. After about one month of feeding, 2 rats would be sacrificed and their esophagi would be removed and prepared for histological examination to detect signs of cancer. This feeding regimen would be continued until signs of esophageal cancer, or one of its precursor lesions was seen. The remaining rats would then be given the liposome-UDCA treatment to see if curative properties are detected. Once you have a working model, you can now find ways to treat the disease with a UDCA- liposome preparation. There are many ways of improving the efficiency of liposomes, one is by treating them with polyethylene glycol.

REFERENCES

1. Dennis Stamp. (2016). Proposed use of deoxycholic acid and urso deoxycholic acid in a treatment regimen for Barrett's esophagus. Mathews Journals of Cancer Science. 1 (1): 1-2.

2. Hofmann A and Mysels K. (1992). Bile solubility and precipitation in vitro and in vivo: The role of conjugation, pH, and Ca ions. Lipid Res. 33(5): 617-626.

3. Bernstein C, Holubec H, Bhattacharyya A, Nguyen H, et al, (2011). Carcinogenicity of Deoxycholic Acid, a Secondary Bile acid. Arch of Toxicol. 85(8): 863-871.

4. Sho Sato, Hiroto Yamamoto, Ken-ichi Mukaisho, Hattori T, et al, (2014). Continuous Taurocholic Acid Exposure Promotes Esophageal Squamus cell Carcinoma Progression due to Reduced Cell Loss resulting from Enhanced Vascular Development. PLOS|one. 9(2): e88831.