Proposed link between liver cancer and adeno-associated virus challenged in human gene therapy

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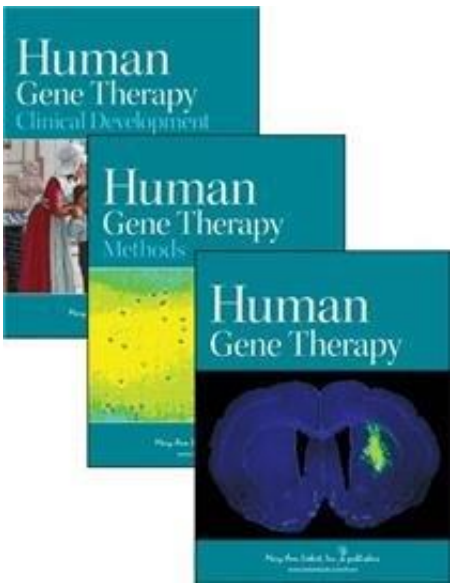

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The conclusion drawn from a recent study that insertion of adeno-associated virus 2 (AAV2) into human DNA causes mutations leading to the development of hepatocellular carcinoma (HCC) was resoundingly rejected by leading researchers in the fields of gene therapy and molecular genetics. Calling the conclusions of the study authors "an enormous leap from their data," the team of researchers challenge details of the experimental methods, interpretation of the findings, and

limitations of the study design in an Editorial published in *Human Gene Therapy*.

"Adeno-Associated Virus Type 2 and Hepatocellular Carcinoma?" is coauthored by Kenneth Berns, Barry Byrne, Terence Flotte, Guangping Gao, William Hauswirth, Roland Herzog, Nicholas Muzyczka, Thierry VandenDriessche, Xiao Xiao, Sergei Zolotukhin, and Arun Srivastava.

Challenging a recent Letter in Nature Genetics by Nault et al., the authors of the HGT Editorial instead suggest that "AAV infection might indeed be a key factor in preventing HCC in humans." Up to 90% of humans have AAV2 in their blood, yet HCC affects only about 10/100,000 people in the U.S.

In the Editorial, the authors assess the study design and data. They conclude that in most cases, AAV2 present in the DNA of liver cells from patients with HCC either slowed or had no effect on tumor growth, as it was detected in only 7% of HCC tumors and in 21% of adjacent normal liver tissue samples.

More information: The Editorial is available free to download on the *Human Gene Therapy* website until February 6, 2016.

Provided by Mary Ann Liebert, Inc