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Hepatitis D is rare or non-existent in hepatitis B virus-infected Indigenous Australians in the Northern Territory

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Hepatitis D virus (HDV), the causative agent of delta hepatitis or hepatitis D, infects 18 million people worldwide with significant regional variation in prevalence.[1] As it requires an individual to be co-infected with hepatitis B virus (HBV) to maintain its replication, its distribution tends to mirror that of chronic HBV infection globally. Although there has been a decline in HDV infection rates over the past few decades in Europe, it remains a significant problem in parts of sub-Saharan Africa, South America and the Asia-Pacific region, as well as in high risk populations such as injecting drug users (IDU) and men who have sex with men (MSM).

In Australia, Hepatitis D is a notifiable disease. There were 34 cases reported nationally in 2010 and 36 in 2011. There has only been one case notified from the Northern Territory (NT) in the past 10 years (this was in a non-Indigenous IDU).[2] Data from the 1980s looking at hepatitis B positive IDUs in Melbourne and MSM in Sydney reported HDV infection rates of 19-20% and 4.4% respectively.[3,4] Current Gastroenterological Society of Australia

Table 1: Patient demographics and HDV status (n=55).

Median age	36.4 years (IQR 28-46)
Male	28 (51%)
Indigenous status	55 (100%) Indigenous Australian
Home region[a]	East Arnhem Land 16 (29.1%) West Arnhem Land 9 (16.4%) Tiwi Islands 6 (10.9%) Darwin urban 1 (1.8%) Katherine region 11 (20.0%) Daly River region 12 (21.8%)
eAg status	Positive 30 (55%) Negative 25 (45%)
eAb status	Positive 25 (45%) Negative 30 (55%)
HDV antigen and antibody	Negative 55 (100%)

a Main residence during first five years of life.

Chronic Hepatitis B guidelines[5] recommend baseline testing for HDV in HBV-infected individuals from higher risk countries – specifically the *Pacific Islands, Mediterranean, parts of South America and Africa*.

Hepatitis B is endemic in NT Indigenous communities and there is uncertainty as to whether they constitute a high-risk group who would warrant HDV testing at the time of initial hepatitis B diagnosis. We sought to address this question by prospectively recruiting a cohort of hepatitis B positive Indigenous individuals.

Between June 2010 and June 2012, following approval from the local human research ethics committee, and written informed consent, we tested 55 hepatitis B positive Indigenous individuals from across the Top End of the NT for HDV antibody and antigen. We found no positive results (Table 1). A subset of 13 samples was further tested for HDV RNA and all were not detected. Hence we estimate the prevalence of HDV in HBV-infected Indigenous people of the NT to be 0% (one-sided 95% confidence interval 0%-6.5%).

Based on these prospectively collected data, HDV infection is either rare or does not exist in NT Indigenous communities and we can therefore recommend that screening is not routinely required.

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