Hepatitis E in Namibia: A Historical Review

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Summary

BACKGROUND

Namibia has had three outbreaks of Hepatitis E Virus (HEV), in 1983, 1995 and 2017. HEV is particularly dangerous to pregnant women. The objective of this study was to present a thorough review of the history of HEV in Namibia; the genotypes which have appeared since 1983, and the possible reasons for the nationwide spread of HEV that has occurred since 2017.

MATERIALS AND METHODS

As this is a review article, no primary research data will be presented. However, an exhaustive literature study has been undertaken and there will be in-depth discussion of the findings of primary researchers in Namibia and elsewhere.

RESULTS

The first two episodes were confined to the Rundu area. The 1983 outbreak may have been genotype 1; that of 1995 contained genotypes 1 and 2. The genotype of 2017 episode has not been clearly established. Increased road traffic may have spread HEV during 2017-2020. Lack of clean water and washing facilities, and lack of awareness of what causes HEV, are the main factors in spreading it.

CONCLUSIONS AND RECOMMENDATIONS

There remain challenges to the containment of HEV. A recent government initiative to stop COVID-19 has helped slow its progress. Both infections are propagated by poor hygienic practice and lack of clean water.

Keywords: *Hepatitis E, Disease Control, Poor Hygiene, Namibia*

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Introduction

Namibia: The historical background and context

Namibia lies on the South-West coast of Africa, bordered by Angola, Zambia, Botswana and South Africa. It was a German colony from 1884 until 1920 when it became South-West Africa, a mandated territory of South Africa. German rule was characterised by repression of the native peoples and a genocidal campaign against the Herero and Nama tribes. This was followed by racial oppression, including Apartheid, imposed by the South African government from 1920 until Independence in 1990 [1-4]. Namibia is the

driest country in Africa and suffers frequent and prolonged droughts [5]. Its economy, traditionally based upon mineral exploitation, fishing and pastoral agriculture, has been augmented in recent years by increased tourism [6]. It is twice the size of Germany but sparsely populated, with only 2.6 million inhabitants [7]. It is divided into fourteen regions, and has eleven officially recognised indigenous languages [8]. Although economically ranked as a "Middle-Income" country, ranking it alongside countries such as Turkey, Botswana, China and Brazil for economic prosperity and quality of life, for many Namibians life is hard, a fact which is reflected in Namibia's relatively high Gini co-efficient [9-12].

Hepatitis E: A global phenomenon

The first documented occurrence of a form of hepatitis distinct from the previously-recognised types A and B hepatitis was in Tunisia in 1950 [13], while the first large-scale outbreak occurred in India in 1955 [14, 15]. It is characterised by, *inter alia*, acute jaundice (which, with a positive IgM/IgG [16], is a definitive characteristic of HEV [17]), dark urine, pale stools, yellowing of the whites of the eyes, lethargy and fatigue, and in extreme cases liver failure, hepatic coma and death [18]. This 'non-A, non-B' type hepatitis was first postulated to be an unique infective agent after an outbreak in Kashmir in 1978 [19], and HEV was finally recognised as a separate strain in 1983 [20], though speculative research indicates that it may have existed for many hundreds of years [21-25]. Since then, it has spread around the world, attaining epidemic status mainly in the poorer, low and middle income countries of Africa, the Middle East, Latin America and Asia [26]; in one estimate, as many as 30% of the world's population could be infected by it [27]. By 2005 more than 3.4 million cases of symptomatic HEV were being diagnosed per annum across the globe, with 70,000 of those resulting in death and a further 3000 resulting in stillbirths [28, 29]. Hepatitis E is now the leading cause of acute viral hepatitis globally [30], and is of serious concern, as other variants of hepatitis are rarely fatal to pregnant women [31]. It is estimated that 19-25% of all maternal deaths in the developing world occur in women with jaundice and that as much as 58% of maternal deaths in hospitalised women infected with hepatitis is due to HEV [32], while Patra *et al* give a similar figure of 15-20% Case Fatality Rate (CFR) in women infected with HEV [33]. According to Stanaway *et al*, the rate of deaths from viral hepatitis of all strains has increased by 63% over the 1990-2013 period [34]. With a CFR equal to that of Cholera and Measles [35], HEV stands as a major threat to health and well-being across the globe. Yet scant attention has been given to this global epidemic; it did not even rate a mention in the World Health Organisation (WHO) list of 'neglected infectious diseases' of 2012 [36].

Contaminated water is universally implicated in all outbreaks [37-39], being conspicuously associated with the use of river water used for drinking, bathing and excretion, and appearing prominently in populations displaced by war to areas without any previous clean-water sources [40]; it is estimated that 20-40% of urban water supplies in the developing world are prone to disinfectant failure [41].

HEV has 4 genotypes known to affect humans [42, 39], of which 1 and 2 are isolated entirely from infected humans; it is passed via the faeco-oral route, is acquired from contaminated water, and accounts for most HEV infections in low and middle income countries [43]. Types 3 and 4 occur in Europe and North America, the Far East, China and Japan, are zoonotic in origin (often from pigs), and only pass to humans from infected meat; they are mainly a disease of developed world, high income countries [44,

45], although evidence shows that type 4 has for some time been emerging as a main source of HEV in China [46]. Genotypes 1 and 2 appear to affect mainly young adult males in the age range 15-40, and pregnant women; types 3 and 4 seem to affect the elderly more. The reason for the age- and sex-specific groupings of those affected by types 1 and 2 is not yet fully understood, but since types 3 and 4 are most often diagnosed among the elderly in the developed world, and transmission of those types is specific to contaminated meat, there is probably a causative link for types 3 and 4 to be found in the kind of food supplied to the elderly in the developed world [47-50]. The proliferation of HEV may also be explained by its great genetic adaptability and ability to mutate [51].

HEV, along with the other strains of Hepatitis, is a largely self-limiting disease, with a fatality rate in the general population of up to 2% (more usually <1%); but, for reasons not yet fully understood, pregnant women exposed to genotypes 1 and 2, particularly those in the last trimester, are at much greater risk of fatality (as much as 40%); types 3 and 4 are not associated with maternal mortality [51]. HEV types 1 and 2 are particularly prevalent in Africa, with epidemics reported in all parts of the continent [52]. Namibia has experienced three large-scale epidemics of HEV since the strain was first isolated, in 1983, 1995 and 2017 [53].

Hepatitis E in Namibia: The 1983 outbreak

The first large outbreak of HEV in Namibia was in the latter half of 1983 (when Namibia was still South West Africa) [54, 55]. It occurred in the Kavango region, in the North of the country, bordering on Angola and separated from it by the Okavango river. A large number of Angolans, refugees from the civil war in Angola, were in Kavango at the time, swelling the local population [56] and living mainly in camps and informal townships [57] around the town of Rundu, the region's main centre. While Rundu (population at the time approximately 5000) and a further two nearby formal settlements had treated water and a piped sewerage system, the five major informal settlements around the town, with many Angolan refugees, had no such facilities; three of them had standpipes with both treated river-water and untreated borehole water, one had water piped directly from shallow (untreated) wells, and the fifth had water taken directly from the river. All the water sources were inadequate, forcing people from all five informal settlements to use the river-water. Toilet facilities were almost non-existent - those that did exist were sited near or next to water supplies, food-stalls and beer breweries [58]. The situation had been exacerbated by drought in the first half of the year, forcing people inland to migrate to the Rundu area and thus further increasing pressure on available water supplies [59].

Few medical and public health records of the 1983 outbreak appear to have been kept at the time, and the author has been unable to find any good supporting historical data which describe it. The lack of good health records is likely to be multifactorial, including perhaps the possibility of the ongoing Angolan war spilling over into Namibia, difficulties faced by the local health authorities in coping with the influx of Namibians and Angolans into the settlements and, indeed, the probability that the Apartheid regime of the time (consistent with the history of White colonialism there since the nineteenth century) did not consider the welfare of the black population a high priority. Consequently, the full extent of this outbreak is unknown.

There are, however, records for sixty-four infected patients recovered by a field team during October 1983 which show that the outbreak appears to have started in July of that year (at which time the average

presentation in Rundu Hospital of a jaundiced patient, with suspected hepatitis, was one person per week) and peaked during September, with a maximum twenty-six cases reported in one week, before tailing off sharply in October. Of these sixty-four, 46 (72 %) were males, all affected were in the 15-39 age group, and of the seven fatalities, six (86%) were pregnant women [60]. The responses, if any, of the authorities to this outbreak, are as yet unknown but, as Isaacson remarks [61], the field team that visited Rundu in October of 1983 found no systematic record-keeping had been undertaken, so presumably the response to the outbreak was, and remained, at local level [62].

The 1995 outbreak

This outbreak also occurred in and around Rundu and, once again, was thought to be the result of a contaminated water supply, which on this occasion was compromised by drought, and by maintenance work done on the supply pipes six months before the identification of the new outbreak [63]. A more thorough audit of the 1995 outbreak (compared with that of 1983) showed that around 600 patients were infected. This time the affected population was much less homogenous, occurring in both males and females in the age range 5 days-80 years. The median age of subjects was 25 years. All the patients included in the study had overt symptoms of viral hepatitis (jaundice and hypochondriacal pain), and at least three were fulminant [64], including one pregnant woman. HEV antibodies were detected in 75% of icteral [65] patients, with an incidence of 13% in patients aged <10 and 81% in those aged > 50 (though none were detected in children aged <5). Approval for the study was given by the Ministry of Health and Social Services, and consent for the use of specimens for the study was obtained from all patients.

During this outbreak there seemed to be a variation in the genotypes involved. In 1983 the genotypes were, insofar as can be ascertained, predominantly type 1. In the 1995 outbreak around 86% of the isolates shared a nucleotide-identity with a genotype 2 "Mexican" isolate, which also appeared in an outbreak in Nigeria in 1997 [66, 67]. Figures for cases and age/sex identity for this outbreak are sparse, but Teo [68] reports that no pregnant women died in this outbreak, despite the diagnosed presence of fulminant hepatic failure among them.

The 2017 outbreak - An anomaly, or a sign of things to come?

The 1983 and 1995 outbreaks of HEV in Kavango region [69] had been localised to settlements around Rundu. In 2017, the outbreak led to an epidemic that spread from Khomas region (housing the capital, Windhoek) across all of Namibia's 14 regions [70]. The first confirmed case of HEV, in what became an epidemic, occurred in Windhoek district. A patient was admitted to hospital (from the informal settlement of Havana, on the outskirts of Windhoek) with the signs and symptoms of hepatitis on 13 October 2017. On 14 December 2017, with increasing cases reported, the government declared outbreaks of HEV in the informal settlements of both Havana and Goreangab (also on the outskirts of Windhoek). The first fatality, a 26-year-old woman in her third trimester of pregnancy, occurred on 19 December 2017 (four days post-partum). By 7 June 2018, 1507 cases had been reported both in and beyond Khomas, including sixteen deaths, six of which (37%) were in pregnant women [71]. By November 2018 the reported-case figure had more than doubled to 3,800 across six of the country's regions, with thirty-three deaths recorded (fourteen of whom - 42% - were pregnant women) [72]. In January 2019 reported cases stood at 4,277 over seven regions, with forty deaths, of which seventeen (40%) were maternal [73], forcing the Namibian government to admit that "So far none of the

regions with Hepatitis E outbreaks have been contained" [74]. By 11 August 2019 the total was at 6,151 [75], leading the government, in September 2019, to both declare an epidemic of HEV and to admit that HEV was now the leading cause of maternal deaths in the country [76].

By 7th January 2020, Ministry of Health figures showed that nearly 7000 cases of HEV had been reported since the outbreak began, with fifty-nine deaths reported to date [77]. By February 2nd 2020 the case-total had risen to 7,247, and the epidemic had reached all fourteen regions of Namibia [78]. At the end of April 2020 it had reached 7,587, with a total of sixty-five deaths reported. Of this sixty-five, thirty-six (55%) were female, of which twenty-three (63%) were maternal (pregnant twelve, post-partum ten and miscarriage one). The epidemic, after a brief respite during which the rate and number of infections dipped temporarily during a two-week period in February-March 2020 [79], peaked at 7,853 cases up to July 2020, but with no further deaths from April [80].

It was noted, in most government comments on the epidemic released to the press, that the highest concentration of cases were found in the informal townships of Khomas (around Windhoek) and Erongo (a coastal region, with the informal settlements around Swakopmund, the capital, the focus of HEV infection) [81] and that residents of the informal settlements of these regions were largely unable to access clean water. Thus, of the 7,247 cases reported up to February 2020, 6,068 (84%) came from Khomas and Erongo, both of which regions have high levels of informal settlements. Of this 6,068, 2,677 (37%) came from three of Namibia's largest informal settlements (Havana and Goreangab in Khomas, and the Democratic Resettlement Community in Erongo) [82].

The genotype of the 2017 outbreak in Namibia has been claimed to be type 2, though clear diagnostic identification of this strain is absent in the literature [83, 84]. There were outbreaks of genotype 1 in Chad in 2016 [85] and Nigeria in 2017 [86] but, given the endemic and varied nature of HEV strains in Africa, it is not sensible to try and extrapolate the Namibian variety from these or similar examples. Different genotypes can co-exist in neighbouring countries with no apparent reason for the presence of either one being manifest instead of the other. For instance, sixteen studies showed that while genotype 1 predominated in the Central African Republic, Sudan, Chad, Egypt and Namibia, genotype 2 co-existed with it in Central African Republic, Chad and Namibia [87]. Another study showed that when genotype 1 was more common in Sudan, genotype 2 was more common in neighbouring Chad [88]. As in 1995, it is eminently possible that types 1 and 2 co-exist in Namibia.

Unlike the previous two outbreaks of HEV, the episode in 2017 was not isolated, but rather sustained over time and widespread throughout the whole country. The specific reasons for this change in behaviour of HEV in Namibia are unconfirmed, but various factors may play a part. Inferential statistics show that motor transport, and thus the potential for spreading the virus nationwide, appears to have increased greatly since the 1983 and 1995 outbreaks (see below, in 'Discussion'). The shift from rural to urban living, due to economic depression and drought, has been pronounced in recent years. Over the last decade or so, Namibia's population growth has been around 1.4% per annum, but the rate of growth of informal settlements resulting from the migration from countryside to towns has been 8-15% per annum, so the government estimates that now around 40% of urban households are located in informal settlements with minimal infrastructure, little or no clean water or functioning toilets, and poor hygiene [89]. While these and other, as-yet unknown,

factors may explain the nationwide spread of HEV from 2017, the causes of that spread must be better understood in order to help the authorities control the epidemic.

Discussion

Each of the three Namibian outbreaks exhibits characteristics unique to itself. The 1983 outbreak was alone in that it adhered to all the characteristics now recognised as typical of HEV, and featured no new manifestations of the disease. It occurred near a river, in settlements inhabited by displaced people with no reliable source of clean drinking water, affected the 15-39 age group, the majority of who were male, and displayed a mortality rate coherent with percentages shown to be a feature of HEV-infected, pregnant females. And, though records do not appear to indicate the strains of the 1983 genotype, there is no evidence that it was anything other than genotype 1, which would be expected at this time and place. The 1995 outbreak was typical in that it shared the same location as the 1983 episode and had the same aetiology (contaminated water). However, this time it did not appear to cause any fatalities, at least not among the most vulnerable group (pregnant women), and it affected the entire age range and both sexes, more or less equally. The 1995 genotype was characterised by the appearance of a new strain, the Mexico variant, with which the virus exhibited a closer nucleotide identity than it did with pre-existing, genotype 1 strains, and which thus placed it within the genotype 2 category.

The 2017 outbreak started in Windhoek and spread rapidly through most of the rest of the country; however, there is a documented history of infected people from Windhoek having travelled from there to Swakopmund and other towns and back again, which is likely to have caused the spread beyond what might otherwise have occurred [90]. There is no evidence that the 1983 and 1995 outbreaks spread countrywide in the same way as that of 2017; travel in Namibia in 1983 was very restricted for the black population, and there is no evidence that this was significantly different in 1995.

However, the 2017 outbreak has not resolved but has continued up to the present, has infected every region of the country and has broadly followed the pattern of other global outbreaks of HEV. The affected age-groups are largely consistent with pre-existing patterns, and the female CFR follows the same pattern and proportion. There are anomalies in the 1995 and 2017 outbreaks that were not apparent in the 1983 episode, which conformed to the profile of a genotype 1, HEV epidemic. The 1995 outbreak showed evidence of a new strain of HEV being in play. The 'Mexico' strain is the only classified member of genotype 2 [91]; the markedly lower level of severe illness and absence of detected mortality might indicate that genotype 2 strains, or at least this particular variety, may provide some kind of safeguard against lethality.

Reasons for the demographics of HEV – Is alcohol a factor?

A likely reason that men in the 15-40 age-range contract HEV more than any other age and gender group is that this is the cohort most likely to be found in public drinking places. A common feature of Namibian society, particularly in informal settlements, is the sharing of drinking vessels in beer-bars and shebeens. Men of this age range might be expected to be the more common frequenter of such places, and children, older adults and women less so, possibly explaining the higher incidence of infection noted in men of the 15-40 age group. Is the fact of the greatest percentage of most infections being men in this age-range merely a reflection of the greater likelihood of such people being exposed to more infection-vectors, such as shared beer-jars in drinking places that serve alcoholic drinks made from contaminated

water? Admittedly, studies have shown anomalies in the demographic pattern traditionally associated with HEV. For instance, a study from an outbreak in Uganda published in 2010 showed that the CFR for children under 2 was 12%, while that for pregnant women, usually the most vulnerable group, was 6.9% [92]. There does not as yet appear to be any explanation for such an anomaly - however, this particular one might be the exception proving the rule.

In other parts of the world studies have been done of normative behaviour, and of the age and gender, of habitual drinkers, and they all seem to show that most drinking is done by males in the 15-40 year age group. For instance, a study from Sri Lanka showed that, of a cohort of 85 alcohol-drinking subjects, less than 5% (4 out of 85) were women, meaning the remaining 95% were men; of these, 78% were under the age of 50, with only 9% under 30 and 22% in the 51-65 age group [93]. A survey in Samoa demonstrated that, out of 4,803 subjects (all in the 15-49 age range), only 393 women (8.4%) drank [94]. A Chinese study showed that, out of a surveyed 1270 people who drank, of whom 83 had alcoholic liver disease, only one woman was found to ever drink at all [95]. A 2006 survey in Brazil showed that the proportion of men who drank was 3-5 times greater than that of women [96], and that of the men, 6% of 14-18 year-olds, over 40% of 18-29 year-olds, 20% of 30-50 year-olds and about 7% of 50+ year-olds drank [97]. And, as with the Chinese example above, a study from India, dealing with risk-factors (chewing tobacco and drinking) for oral cancer, showed that so few women of the cohort drank that they were left out of the statistical analysis [98]. In a study of 48 low-and middle-income countries, 11.8% of men examined drank as against 3.42% of women, while in low income countries 7.76% of men drank while only 3.28 of women did [99]. Combined with the fact that most drinkers around the world are men in the 15-40 age-group is the fact that, even in those who might not be deemed "problem drinkers", a degree of hepatic impairment is likely to build up over time. A combination of drinking in insanitary conditions and drinking steadily over time could alone, and quite simply, explain why men in this age range develop more symptoms of HEV than any other [100].

The appearance of a new, possibly less lethal, strain in 1995 might explain the lack, or lower level, of mortality in HEV cases (in terms of virus propagation, it makes sense that a less lethal strain is more likely to spread wider and faster than one more lethal, since its victims will live longer, and thus be more likely to continue spreading the virus, than those who die early from a more lethal variant). It may also explain why a wider age-range was affected, and why women were affected equally to men. This latter phenomenon might also be explained by people having less resistance to the newer strain; but it does not explain why, if that was the case, it did not spread as much as, or faster than, the 1983 outbreak, given the postulated assumption of similar levels of mobility shared between the affected populations of 1983 and 1995. It also does not explain why a new strain would be less likely to be spread among the 15-50 male beer-drinking cohort than an old one. A provisional explanation may be that genotype 2 strains are in some way counteracted by drinking in beer-shops with shared drinking-vessels. On the other hand, it may not - the high CFR among pregnant women in a nationwide epidemic that may be wholly or in part genotype 2 (i.e., that of 2017) would indicate that this genotype has no protective characteristics at all, at least for women. The study of an outbreak of HEV genotype 2 in Nigeria in 2017 gives a tantalising glimpse of how genotype 2 might, or might not, have some protective

characteristics - of 146 confirmed cases of HEV, only two (1.37%) were fatal [101]. How would one extrapolate such a finding? On the one hand, the CFR was significantly lower than that of the average for HEV deaths – on the other, the two fatalities were both pregnant women. The extreme genetic adaptability of HEV may explain many of these anomalies [102], but as yet this remains moot.

The 2017 outbreak may have spread because of the amount of travel undertaken by those infected, if that incidence was in fact greater than that of populations during the previous epidemics. There is no direct evidence available to indicate that road travel has increased in Namibia since 1983 and 1995, but that it has can be inferred from existing data. Numerous studies show that an increase in GDP leads to an increase, not only in the numbers of vehicles on the roads of any given country, but also to an increase in road traffic accidents (RTAs), as the road safety and accident/injury prevention measures that commonly appear in developed countries to stabilise and reduce such events do not appear in time to prevent an increase in road traffic accidents in developing countries [103, 104]. In Namibia, the distance travelled by road vehicles increased from 4,722,048,700 kilometres in 2002 to 7,414, 761,800 in 2009; the number of RTAs rose from 10,915 in 2002 to 15,537 in 2009, and the crash rate (per 1000 vehicles) from 60.5 in 2002 to 67.6 in 2009 [105-107] - all figures indicating a general rise in the number of vehicles on the road and the distances travelled by them. It may reasonably be assumed that these figures increased again between 2009 and 2017. Thus, the numbers of those travelling from heavily-infected Windhoek townships to every other part of Namibia may explain the rapidity and reach of the 2017 outbreak.

Those seeking refuge from conflicts or drought, such as those fleeing drought-stricken areas of Namibia or war-torn Angola, might arguably be less able or willing to travel from their informal settlements to other areas in which they would not likely have any business. This alone might explain why the 1983 and 1995 outbreaks did not travel beyond Rundu. But in a case where those most afflicted by HEV were least likely to travel, why would the increase in traffic by 2017 facilitate the spread of the disease? An increase of traffic on the scale indicated by the figures quoted above might be an indicator of peace and prosperity - those travelling regularly, in a country at peace, for business, recreational or family reasons might be assumed to be relatively well-off, more likely to live in formal settlements with properly-treated water supplies, and thus be less likely to serve as vectors of infection. However, this idea is predicated on the notion that those living prosperous lives do not share the habits and standards of those in informal settlements. Unfortunately for this idea, it appears common for the members of all socio-economic levels of society in Namibia to travel back to their original homes (often farms in the countryside) from wherever they live for work, in order to partake of traditional social practices such as sharing food and drink in family or public settings, and without regard to hygiene issues which are implicated in the spread of HEV [108]; in such circumstances the levels of prosperity of any given traveller would be irrelevant. In their study of patients and staff affected by the 2017 outbreak in Omusati, Freeman *et al* showed that this was likely to be the case - of seventeen informal settlement residents interviewed in the Northern district of Omusati, eleven had travelled to Windhoek in the previous year (often more than once), two had travelled back and forth frequently to local towns up to 40 km away, and one had travelled regularly to and from Angola [109]. If the inhabitants of informal settlements could travel that frequently and that far by 2017, and those more well-off (but with similar hygiene-

free behaviours to the inhabitants of informal settlements) could also travel as much as they liked, the nationwide spread of HEV from 2017 is easily explained.

Of course, there is no direct evidence that the patterns and frequency of travel of those infected in 1983 and 1995 was the same as, or different from, those of the 2017 episode, so the possibility of more travel for the 2017 cohort being a causative factor for the spread of HEV must remain moot. However, the rapid epidemic rise of the 2017 outbreak, in combination with the evidentiary rise in motor transport during 1983-2017, seems too great an association of factors to ignore.

Conclusion

Namibia is not alone in Africa in having outbreaks of HEV - regular episodes have been reported in Chad, Ethiopia, Kenya, Nigeria, Sudan, South Sudan and Uganda, mainly in camps for displaced persons which, with their lack of clean water supplies, are notorious hotbeds of infection [110]. That HEV spreads because of people passing the virus to each other by the agency of contaminated water is beyond dispute; the rapid spread of HEV through Namibia from 2017 appears to be connected to the greatly increased mobility of the population, combined with universally-accepted social habits that propagate the spread of the disease. In such case, the key to controlling or eradicating HEV may lie not in treatment, but in preventing the emergence of the disease in the first place. In response to the advent of Covid-19 in Namibia, the Minister for Health, Dr. Kalumbi Shangula, announced in April 2020 the establishment of a Covid-19 Communications centre, which would address the control of the epidemic by using the WHO's 8 Pillars of Response Management (co-ordinated management and logistics; case management; infection prevention and control; surveillance; laboratory; points of entry; risk communication and community engagement, and mental health and psycho-social support) [111]. In May 2020 Dr. Shangula reported that these measures had not only been successfully deployed against Covid-19, but that it has also helped slow the spread of HEV, as the incidence of new cases had slowed and that "… as we continue the fight against Covid-19, we are killing two birds with one stone" [112]. The same measures employed to fight the spread of HEV are effective in combatting Covid-19, i.e. washing hands in clean water, so measures taken to encourage and facilitate this for the one are bound to be effective in reducing the other [113]. Of course, such measures depend on the availability of clean water, itself contingent upon the installation of effective sanitation arrangements, and until these become generally available the likelihood of further outbreaks remains high.

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Conflict of Interest

No conflict of interest has been identified.

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60. Ibid: 620.
61. Ibid: 620
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63. **Maila** *et al op. cit.* (note 53): 91-92, Nucleotide-identity with other African isolates, particularly the genotype 1 strain identified in the 1983 outbreak, was only in the 77-78% range.
64. Fulminant - coming on suddenly and with great severity; Merriam-Webster dictionary.
65. **Icteral** - of, or relating to, jaundice; Merriam-Webster dictionary
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69. Kavango district was split into Kavango East and Kavango West in 2013. However, all documents referring to HEV and used for this article only mention Kavango (presumably Kavango West, which holds the town of Rundu, the focus of most informal settlements in Kavango and the site of the 1983 and 1995 outbreaks), which complicates the tallying of districts.
70. The fourteen districts (North to South, more or less) are Kunene, Omusati, Oshana, Ohangwena, Oshikoto, Kavango East, Kavango West, Zambesi, Erongo, Otjozondjupa, Omaheke, Khomas, Hardap and Karas.
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81. **China**.org.cn. 26th April 2020.
82. **Bustamente** *et al*. *op.cit*. (note 78): 35.
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109. **Davies** *et al op.cit* (note 90).

110. **Spina** *et al op.cit* (note 85).

111. Namibia launches Covid-19 communications centre. WHO 17th April 2020.

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113. Tippy-taps used to fight infection. The Namibian 3rd March 2020.