1. Overview of Viral Haemorrhagic Fevers (VHFs)

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1.1 What are viral haemorrhagic fevers (VHFs)?

Viral haemorrhagic fever agents (VHFs) are zoonotic diseases that may cause a haemorrhagic syndrome in humans. These illnesses are caused by four distinct families of viruses: the arenaviruses, bunyaviruses, filoviruses, and flaviviruses (table 1). VHFs of particular concern however are those that have demonstrated the potential for person-to-person spread and they are the focus of this document. These are: Ebola, Marburg, Lassa fever and other Arenaviruses, and Crimean-Congo haemorrhagic fever.

Table 1. Haemorrhagic fever (HF) viruses and the diseases they cause

Family	Virus	Disease
Arenaviruses	**Lassa virus**	**Lassa fever**
	Junin virus	Argentine haemorrhagic fever
	Chapare virus	Bolivian haemorrhagic fever
	Machupo virus	Bolivian haemorrhagic fever
	Sabia virus	Brazilian haemorrhagic fever
	Guanarito virus	Venezuelan haemorrhagic fever
	Lujo virus	**Lujo haemorrhagic fever**
Filoviruses	**Ebola virus**	**Ebola haemorrhagic fever**
	Marburg virus	**Marburg haemorrhagic fever**
Bunyaviruses	**Crimean-Congo haemorrhagic fever virus**	**Crimean-Congo haemorrhagic fever (CCHF)**
	Rift Valley virus	Rift Valley Fever
Flavivirus	Yellow fever virus	Yellow fever
	Kyasanur forest disease virus	Kyasanur forest disease
	Omsk haemorrhagic fever virus	Omsk haemorrhagic fever
	Alkhurma haemorrhagic fever virus	Alkhurma haemorrhagic fever

BOLD indicates potential for person-to-person spread.

1.2 Features of VHFs

VHFs share a number of common features:

- they are all RNA viruses with a lipid envelope;
- they are zoonotic and/or vectorborne; their survival is dependent on an animal or insect host;
- the viruses are geographically restricted to the areas where their host species live;
- humans are not the natural reservoir for any of these viruses;
- human cases occur sporadically;
- they can cause severe life-threatening disease.

VHFs tend to be found in tropical and subtropical areas such as **Africa, South America and the Middle East. CCHF is endemic in Asia, south-eastern Europe, and Africa**. Many wild and domestic animals, ticks, and mosquitoes are known to carry some of the VHF agents, although the reservoirs have not been identified for all VHF agents. Environmental conditions in Ireland *do not support* the natural reservoirs or vectors of any of the viruses that cause haemorrhagic fevers.

While for most European countries, including Ireland, the risk of epidemic spread in the general population is negligible, cases of VHF are occasionally *imported* into Europe. Given the ever-increasing speed and volume of air travel in today's world, the risk that individuals incubating VHF may arrive in non-endemic regions is increasing.

The pathophysiological effects of VHFs result in their characteristic clinical features. The range of clinical features of VHFs is relatively uniform. All are characterised by fever and bleeding (for certain viruses this can be minor feature). At the more severe end of the spectrum these include vascular leakage, coagulation defects and multi-organ system failure. Fever is almost invariably the first symptom and commonly occurs with influenza-like symptoms including malaise, exhaustion, tender myalgia, sore throat, injected conjunctivae, headache, vomiting, and diarrhoea. The initial non-specific symptoms can be followed by facial and truncal flushing, a maculopapular rash, the formation of petechiae, ecchymoses, overt bleeding, dependent oedema and hypotension that can progress to shock from approximately day 5 of infection.

The severity of symptoms will vary depending on the causative virus. Occasionally, a "VHF syndrome" will develop, which is a severe, and potentially life-threatening multisystem syndrome, typified by a combination of capillary leak syndrome and bleeding diathesis, leading to the characteristic swelling and bleeding seen in more severe diseases such as EVD and Marburg virus, CCHF and the South American arenavirus infections. The internal bleeding can result in hepatic damage, myocarditis, encephalitis and consumptive coagulopathy. Patients with fatal infections typically die during the second week of illness. Convalescence may be prolonged in those who survive. In the case of diseases such as Lassa, Dengue Rift Valley Fever and Yellow fever, bleeding is considerably less marked, and can frequently be absent.

Lassa fever is the most commonly imported VHF, with 9 cases imported to the UK between 1980 and 2022.[1] The most recent case was imported to the UK in early 2022 from Mali.[1,2] In May 2015 a case of Lassa fever was imported to the United States from Liberia, bringing the total number of imported cases in the US to eight. Two cases of Marburg haemorrhagic fever were imported from Uganda in 2008, one to the Netherlands and the other to the United States.[3] Three cases of Crimean-Congo haemorrhagic fever (CCHF) have been imported into Western Europe.

Only one VHF is endemic in Europe; CCHF has been endemic in Bulgaria since the 1950s when a large outbreak occurred from 1954 to 1955 during which 487 cases were notified. Cases have also been notified in neighbouring countries including Albania, Kosovo, Turkey and Ukraine, as well as in south western regions of the Russian Federation. The first case of CCHF in Greece was notified in 2008. The first local case of CCHF in Western Europe was reported in Spain in August 2016, with secondary transmission reported in a healthcare worker.[4] Changes in climate may contribute to the further spread of the *Ixodid* ticks, the vector for CCHF, and consequently to the geographic spread of CCHF in southern Europe.[5]

While many VHFs were initially considered to be highly transmissible between humans, this hypothesis has not been substantiated. Although nosocomial transmission has occurred in areas with endemic disease, accumulated evidence shows that transmission of these viruses does not commonly occur through casual contact.[6-8] Several importations to non-endemic countries have

occurred without subsequent disease outbreaks. While secondary cases of Marburg have been documented, a limited number of secondary cases of Lassa fever have been identified following an importation episode. One involved the physician who was treating the patient, who seroconverted but remained asymptomatic.[9] Lassa fever in a traveller returning from Mali in February 2022 was associated with secondary transmission to two family members in the UK, one of whom died. [1,2] This represents the second known episode of secondary transmission of Lassa fever in Europe. [2]

Persons at highest risk of secondary infection are those who are in closest contact with an infected person or his/her body fluids. Such persons include those with prolonged or close contact with patients, those providing direct medical and nursing care, and laboratory workers handling blood, tissue or other specimens.[10]

The key characteristics of the VHFs of public health importance are outlined in Table 2, including their distribution, vectors and reservoir hosts, incubation period and case fatality rate. Further details of the disease and viruses are given in Sections 1.3 to 1.9.

Information on current outbreaks can be found on the HPSC website.

1.3 Ebola haemorrhagic fever

Ebola was first recognised in 1976 in the Democratic Republic of Congo. It is a severe, often fatal disease in humans and other primates, though the mortality can be reduced through appropriate supportive care as demonstrated during the 2014-2016 outbreak in West Africa. [11] Ebola typically appears in sporadic outbreaks, usually within a health-care setting. Outbreaks have been occurring with increasing frequency since the mid 1990s, with 11 outbreaks reported from 1990-1996 compared with 6 for the period 1976-1989.[12] Five varieties of Ebola virus are known to exist, four of which are known to cause VHF in humans: Ebola Zaire, Ebola Sudan, Ebola Ivory Coast and Bundibugyo Ebola. The fifth subtype, Ebola Reston, has caused VHF in non-human primates but not in humans. [13, 14]

The exact location, origin and natural reservoir of Ebola remains unknown, but researchers believe that the virus is zoonotic, native to the African continent. Current evidence suggests the fruit bat, as a potential reservoir for Ebola virus. Ebola-specific antibodies have been detected in three species of fruit bat in Africa (*Hypsignathus monstrosus, Epomops franqueti* and *Myoncyteris torquata*) and in *R. amplexicaudatus,* a common species of fruit bat in the Philippines.[15-17] The exact mode of transmission to humans is unknown. Once infected, the virus is then transmitted to others via direct contact with blood, secretions, organs or other bodily fluids of infected persons. Traditional burial practices, including washing and dressing the body of the deceased, have been identified as a risk factor. These practices were identified as the sole significant risk factor associated with being a probable or confirmed case in an outbreak in Uganda from December 2007 to January 2008.[18]

Confirmed cases and outbreaks of Ebola have been reported in various African countries since 1995, including Gabon, Democratic Republic of Congo, Uganda, Ivory Coast, Republic of Congo, South Africa and Sudan. The mortality rates have ranged from 50-89%.[13] In 1976 a laboratory worker in the UK became infected as a result of a needle stick injury while working on specimens from a case

of Ebola HF but he recovered.[6] In 2004 there were two further cases due to laboratory accidents, one in the US and the other in Russia. Both were due to needle stick injury. One case recovered (US) but the other died as a result of the infection (Russia).[19]

Ebola Reston virus was first reported in 1989 from several quarantine facilities in Reston, Virginia, USA, where monkeys from the Philippines became ill and died. There were similar reports from other facilities in the USA and Italy which also housed monkeys from the same monkey facility in the Philippines.[12, 20] No further cases of Ebola Reston were reported after the closure of the affected facility in the Philippines in 1997. However, in October 2008, Ebola Reston infection was confirmed in pigs in the Philippines for the first time. A threat assessment by ECDC in January 2009 stated that European swine and monkey handlers should be considered potentially at low risk for exposure when handling animals from the Philippines. It also stated that further assessment was needed to determine the risk of transmission to humans through the eating of uncooked contaminated meat.[21]

The largest event was the 2014-2016 Ebola outbreak in West Africa (Guinea, Liberia and Sierra Leone), where there was intense and sustained transmission for the first time. Associated with this extensive outbreak, Ebola cases were imported into Italy, Nigeria, Mali, Senegal, Spain, the UK and the USA. Other recent significant events include the 2018-2020 outbreak in the Democratic Republic of the Congo, the 2020 outbreak in the Democratic Republic of the Congo and the 2022 outbreak in Uganda.

In December 2019, the United States Food and Drug Administration approved Ervebo, the first vaccine for the prevention of Ebola virus disease caused by Zaire ebolavirus.[22] The vaccine is licensed for individuals aged 18 and older. It was administered to over 350,000 people in Guinea between 2014 and 2015, where it was shown to be safe and effective. It was used in the Democratic Republic of the Congo during the 2018-2020 Ebola virus disease outbreak under the "*compassionate use*" protocol.[23]

1.4 Lassa fever

Lassa fever, an Old-World Arenavirus, is an acute viral illness that occurs in West Africa. The illness was first reported in 1969 when two missionary nurses died in Nigeria. Lassa fever is endemic in parts of West Africa including Guinea, Liberia, Sierra Leone and Nigeria. The reservoir of Lassa virus is the multimammate rat (*Mastomys* genus).

Humans can be infected in several ways. Rats shed the virus in urine and droppings and therefore primary transmission is likely to be through direct contact with these materials. Infection can also occur following inhalation of particles containing virus. Secondary transmission can also occur through person-to-person contact. In Ireland, such secondary transmission is most likely to occur in a healthcare setting either by coming into contact with the virus in blood, tissue or secretions of a case, or by breathing in airborne particles which the patient can produce by coughing. In endemic areas the majority of cases (80%) are asymptomatic, but of those hospitalised approximately 15-20% of patients die. The overall case fatality rate is 1%. The death rates are particularly high for women in the third trimester of pregnancy and for foetuses, about 95% of which die in the uterus of infected

expectant mothers. Following recovery, the most common complication across all groups is deafness, which occurs in approximately 33% of cases. [24]

Lassa fever is the most common of the VHFs. Some studies indicate that 300,000 to 500,000 cases of Lassa fever and 5,000 deaths occur annually across West Africa. With such a large number of cases, there is a greater possibility of Lassa fever being imported into Europe than any of the other VHFs. [24]

1.5 Marburg haemorrhagic fever

Marburg virus was first recognised in 1967 when outbreaks of haemorrhagic fever occurred simultaneously in Marburg and Frankfurt in Germany, and in Belgrade in the former Yugoslavia. A total of 32 people were infected as a result of those first affected having been exposed to blood, organs and cell cultures from African green monkeys imported from Uganda.[25] Thirty one cases were hospitalised while the other case was ill but did not require hospitalisation. Marburg infection was retrospectively serologically diagnosed in this patient. There were 6 secondary cases which were a result of: needle stick injuries (n=3); sexual intercourse (n=1); knife cut at post mortem (n=1); as well as one case of nosocomial transmission. The wife of the Belgrade index case was a physician and had drawn blood at home for testing and so this was considered nosocomial transmission. There were 7 deaths among the reported cases (case fatality rate 22%).[26]

Marburg virus is indigenous to Africa and while the geographic areas in which it is endemic are unknown, they appear to include at least parts of Uganda, Western Kenya, Angola and perhaps Zimbabwe. Recent studies implicate the African fruit bat as the reservoir host of the Marburg virus but further study is required to determine if there are other host species. The fruit bat is widely distributed across Africa, extending the area at risk of outbreak for Marburg haemorrhagic fever beyond that previously suspected. [27]

When the 1967 outbreak occurred in Europe, the virus had arrived with imported monkeys from Uganda. The next imported case did not occur until 1975 in Johannesburg and the patient had most likely been exposed while travelling in Zimbabwe. A travelling companion and a nurse were subsequently infected. In 1982, a case was identified in an 18 year old from the same rural part of Zimbabwe in which the case from 1975 had stayed. This patient recovered and there were no secondary cases. [28] Three cases (two deaths) have been associated with western Kenya. In 1980, a French engineer who travelled in western Kenya was infected and died. A physician who tried to resuscitate the case in a Nairobi hospital was infected but recovered. In 1987 a boy who visited a park near to where the engineer was infected was also infected and died. There were no secondary cases.[29]

Outbreaks have been reported in the Democratic Republic of Congo (1998-2000), Angola (2004) and Uganda (2007, 2012), mostly among mine workers. [30] In 2008, two cases (one death) were reported in tourists, one Dutch and the other American, returning from Uganda. Both travellers had visited a well-known cave inhabited by fruit bats in a national park. [3, 31] How the virus is transmitted from animals to humans remains unknown.

While the case fatality rate was initially thought to be significantly lower than that of Ebola, analysis of recent outbreaks in the Democratic Republic of Congo has shown that this is also greater than 70%. A case fatality rate of 90% was documented in an outbreak in Angola in 2004-2005. [32] Recovery from Marburg can be slow and known sequelae include orchitis, recurrent hepatitis, transverse myelitis and uveitis.

1.7 Crimean-Congo haemorrhagic fever

Crimean-Congo haemorrhagic fever (CCHF) was first described in the Crimea in 1944. In 1969 it was recognised that the virus causing Crimean haemorrhagic fever was the same as that responsible for an illness identified in 1956 in the Congo, hence the linkage of the two names.

CCHF is a severe illness in humans with a high mortality, but fortunately human illness occurs infrequently. Animal infection is more common. Animals become infected with CCHF from the bite of infected *Ixodid* ticks (*Hyalomma* genus). Humans who become infected usually do so from direct contact with blood or other tissues from infected animals or directly from a tick bite. The majority of cases have occurred in those involved with the livestock industry such as agricultural workers, slaughterhouse workers and vets. However, CCHF has repeatedly caused nosocomial outbreaks with high mortality rates, which puts healthcare workers, including those working in laboratories, at serious risk of infection.[33] In Bulgaria from 1950 to 1974, 42 health care workers were infected with CCHF with a case fatality rate of 40%. The case fatality rate among 14 healthcare workers in Turkey from 2003 to 2009 was 28%. [33] Percutaneous exposure presents the highest risk of transmission. [33]

The geographical distribution of the virus is widespread. The disease is endemic in parts of Africa, Asia, the Middle East and Eastern Europe. In Africa, outbreaks have been reported from South Africa, Congo, Mauritania, Burkina Faso, Tanzania and Senegal. An outbreak in China in 1965 had a case fatality rate of 80%. A large number of cases have also been reported from Middle Eastern countries such as Iraq, United Arab Emirates, Saudi Arabia and Oman. Since 2000, outbreaks have been reported in Albania, Kosovo, Turkey, Pakistan, Iran, Mauritania and Kenya. [33] Most recent data from South Africa reported 3 cases in 2009 (case fatality rate 33%), compared with 11 cases in 2008 and just one case in 2007. [34]

In Europe, CCHF is currently endemic only in Bulgaria where a total of 1,568 CCHF cases were notified from 1953 to 2008, with an overall case fatality rate of 17%. However, there has been an increase in cases and outbreaks of CCHF recorded in other countries in the region such as Albania, Kosovo, Turkey and Ukraine as well as south-western regions of the Russian Federation. This increase has been attributed to climate and anthropogenic factors such as changes in land use, agricultural practices and movement of livestock, all of which may influence tick-host dynamics. The first case of CCHF in Greece was recorded in June 2008 in an agricultural worker with a tick bite in an area just a few kilometres from a previously documented Bulgarian outbreak. [35] The first local case of CCHF in Spain was reported in August 2016, in a man who had a tick bite in the Ávila province, 300km away from an area where evidence of CCHFV circulation was found in 2010. [36] A secondary case occurred in a nurse who had assisted with endotracheal intubation of the index case. [4]

An inactivated suckling mouse brain-derived CCHF vaccine is currently used in Bulgaria to protect military and medical personnel, farmers and people living or working in endemic areas. The initial doses are given at days 0 and 30, the third dose one year later. Booster doses are then given every five years. [37]

In September 2008 ECDC held a meeting to review the current epidemiological situation of CCHF in Europe and to identify gaps in prevention and control. It was concluded that integrated control measures are essential and should include vector control, vaccination programmes, improved therapy strategies, diagnostic tools, surveillance (human and animal) and public awareness. Given the high case fatality rate and outbreak potential, CCHF poses a serious risk in Europe, as demonstrated by the increased geographic spread of cases. [35, 38]

CCHF cases have only rarely been imported into Western Europe, including a case imported from Zimbabwe to the UK in 1998; a case imported from Bulgaria to Germany in 2001; and a case imported from Senegal to France in 2004.[38]

1.8 Other Old-World and New-World Arenaviruses

New-World Arenaviruses are a group of rodent-associated Arenaviruses in South America similar to the Lassa group in Africa (Old-World), but antigenically unrelated to the African viruses.

Junín virus, which causes Argentinean Haemorrhagic Fever, was the first of these to be recognized (isolated in 1958). It occurs in a limited agricultural area of the pampas in Argentina. Machupo virus, which causes Bolivian Haemorrhagic Fever, is also geographically restricted. Isolated in 1963, it is found in the remote savannas of the Beni province of Bolivia. In December 2003 a newly discovered Arenavirus, Chapare virus, was identified as the cause of haemorrhagic fever in rural Bolivia in an area outside the known Machupo endemic region. [39] The Guanarito (Venezuelan Haemorrhagic Fever) and Sabia (Brazilian Haemorrhagic Fever) viruses are the most recent additions to this family. [40, 41] The Junín and Machupo viruses are associated with serious disease and both have peak incidence during May and June.[41]

The rodent hosts of Arenaviruses are chronically infected and virus is shed into the environment in the urine and droppings. Human infection occurs by direct contact of broken skin with rodent excrement, or through the inhalation of particles contaminated with rodent urine or saliva.[41] Laboratory–acquired infection of Junín and Machupo viruses have been reported; 21 cases (1 death) and 6 cases (1 death), respectively, up to 1980.[42, 43]

In September-October 2008 a novel Old-World Arenavirus, Lujo virus, was identified as the cause of a nosocomial outbreak in South Africa. There were five patients involved in the outbreak, four of whom died. Three of the cases were secondary infections and one tertiary infection occurred. The patient who recovered was treated with Ribavirin and the institution of barrier nursing procedures prevented further spread. Lujo virus was the first highly pathogenic Arenavirus to be identified in Africa in 40 years and highlights the possibility that pathogenic Arenaviruses could be more widespread in Africa than previously thought.[44]

1.9 Flaviviruses

The flaviviruses cause a range of illness from self-limiting febrile illness to severe hepatitis and haemorrhagic fever. Of these, the viruses that cause Kyasanur forest disease, Omsk and Alkhurma haemorrhagic fevers are of greatest public health concern due to their potential as bioterrorism agents rather than their potential for person-to-person transmission.

Kyasanur forest disease, caused by Kyasanur forest disease virus, is a haemorrhagic disease transmitted to humans principally from the bite of infected ticks. While larger animals such as goats, cows or sheep may be infected with Kyasanur forest disease virus, there is no evidence that they have a role in its transmission, including transmission via unpasteurised milk. [45] There is no evidence of direct person-to-person transmission. Up to 1979, 87 cases of laboratory-acquired infections were reported. [45, 46] The virus is limited to Karnataka State, India. The case fatality rate is 2% - 10%.[45, 46]

Omsk haemorrhagic fever is also geographically limited and is found in the western Siberian regions of Omsk, Novosibirsk, Kurgan and Tjumen, with seasonal occurrence in each area coinciding with vector activity. Transmission to humans is from the bite of an infective tick but data also suggests direct transmission to humans from muskrat and from water contaminated with the virus. There is no evidence of direct transmission from person-to-person, and no outbreaks within a hospital or family have been reported, with the exception of family outbreaks where multiple family members were involved in the hunting of muskrats and the removal and treatment of their skins. The disease is thought to be under-reported as mild cases are frequently misdiagnosed or not reported.[47] Between 1946 and 1958 there were 972 cases but the incidence decreased dramatically from 1960 and in 1988 only 3 cases were reported. In 1989 22 cases were reported and outbreaks followed in 1990 (29 cases) and in 1991 (38 cases). In 1998 7 cases were reported, with 3 classified as severe and 1 death. [47] Laboratory-acquired infections have been documented including two cases due to aerosols generated from a broken vial in a centrifuge.[48] A total of 23 laboratory-acquired cases were reported.[47] The case fatality rate is 0.4% -2.5%.[47]

Alkhurma haemorrhagic fever virus is genetically very closely related to Kyasanur forest disease. It is found in the Makkah and Najran provinces on the west coast of Saudi Arabia. It is also tick-borne but the reservoir host has not been documented but probably includes sheep, camels and goats. Humans who become infected usually do so directly from a tick bite, by contact with infected blood on a skin wound, or from consumption of unpasturised milk of infected animals. In the first three months of 2009, 4 cases were reported in the Najran province. The case fatality rate is 25% - 30%.[49]

Table 2. Key Characteristics of Viral Haemorrhagic Fevers

Family Disease (Virus)	Distribution	First Described	Person-to-person Transmission	Vectors	Reservoir Hosts	Incubation Period Usual (range)	Case Fatality Rate
Arenaviridae							
Lassa fever	West Africa, incl. Liberia, Guinea, Sierra Leone, Nigeria	1969	Y		Rodents of genus *Mastomys* shed virus in urine & faeces	6 - 21 days	1% overall, up to 20% of hospitalised cases
Argentine HF (Junin)	Argentina	1958	N		Rodents	7 -16 days	5-30%
Bolivian HF (Machupo)	Benin province Bolivia	1963	N		Rodents	7 -16 days	5-30%
Bolivian HF (Chapre)	Cochabamba province, Bolivia	2003	N		Unknown	Unknown	Unknown
Brazilian HF (Sabia)	Brazil	1990	N		Rodents	7 -16 days	
Venezuelan HF (Guanarito)	Venezuela	1989	N		Rodents	7 -16 days	
Lujo	Southern Africa	2008	Y		Possibly rodents	9-18 days (2 cases)	80% (4/5 cases)
Bunyaviridae							
Crimean-Congo HF	Africa, Eastern Europe, Middle East, Asia	1944	Y	*Ixodid* ticks (*Hyalomma* genus)	Small mammals, livestock, wide range of domestic & wild animals	1 -3 days (1 - 13 days)	10-50%
Rift Valley Fever	Africa		N	Mosquitoes	Sheep and cattle	3 - 12 days (few days - few months)	1%
Filoviridae							
Ebola	Central & Eastern Africa, incl. DR Congo, Gabon, Sudan, Ivory Coast, Uganda, Rep. of the Congo	1976	Y	-	African fruit bat	2 - 21 days	50-90%
Marburg	Eastern & Southern Africa, incl. Angola, DR Congo, Kenya, Zimbabwe & Uganda	1967	Y	-	African fruit bat	3 - 10 days	up to 90%
Flavivirdiae							
Yellow Fever	West Africa & South America	1927	N	Mosquitoes	Humans (monkeys)	3 - 6 days	
Kyasanur Forest Fever	India	1957	N	Ticks	Goats, cows, sheep	3 - 8 days	2-10%
Omsk HF	Siberia	1946	N	Ticks	Rodents, muskrat	3 - 8 days	0.4 – 2.5%
Alkhurma HF	Saudi Arabia (Makkah & Najran provinces)	1957	N	Ticks	Probably sheep, camels, goats	Probably 3-8 days	25-30%

1.10 Requirement to notify VHF to the World Health Organization under the International Health Regulations, 2005

The aim of the International Health Regulations (IHR) is to help the international community prevent and respond to acute public health risks that pose a serious risk to health worldwide and have the potential to cross borders. The IHR require countries to notify WHO of events that may constitute a Public Health Emergency of International Concern (PHEIC). This is done by the WHO IHR National Focal Point at HPSC. An event of VHF (Ebola, Lassa, or Marburg) "*shall always lead to utilisation of the algorithm in Annex 2 (page 52)* (a decision instrument for the assessment and notification of events that may constitute a PHEIC)*, because this disease has demonstrated the ability to cause serious public health impact and to spread rapidly internationally*" and would be notified to WHO.

The IHR 2005 came into force in 2007. There is a nationally agreed mechanism for responding to Public Health Emergencies of International Concern (PHEIC) and this is the National Public Health Outbreak Response Plan and Team (NPHORT). This would be activated in the event of a case of VHF arising in Ireland.

1.11 Establishment and role of the National Isolation Centre

The National Isolation Unit (NIU) for adult patients, located at the Mater Misericordiae University Hospital, Dublin (St. Bernard's Ward), is the national referral centre for ***High Risk*** suspected and confirmed cases of VHF.

Officially opened in December 2008, the self-contained unit has 12 beds including six lobbied, en-suite single rooms with negative pressure ventilation. Two of the isolation rooms are of high specification and are separate from the rest of the unit with different air-handling systems. It is designed to admit, isolate and treat patients suspected or diagnosed with highly infectious diseases.

There is currently no designated national paediatric referral centre for VHF. Contingency arrangements in the event of a paediatric case arising are currently in development.

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