

Chapter 16

Impact of Eel Viruses on Recruitment of European Eel

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16.1 Introduction

Eels have an uncommon catadromic life cycle with exceptional migratory patterns to their spawning grounds several thousand kilometres away: the European eel (*Anguilla anguilla*) travels over 5,500 km to the Sargasso Sea (Schmidt 1923; McCleave and Kleckner 1987; Tesch 1982; Tesch and Wegner 1990); the American eel (*A. rostrata*) migrates over 4,000 km also to the Sargasso Sea (Castonguay and McCleave 1987; McCleave and Kleckner 1987; Tesch and Wegner 1990); the Australian eel (*A. australis*) travels over 5,000 km into the Pacific Ocean to spawn (Jellyman 1987); and the Japanese eel (*A. japonica*) travels over 4,000 km to an area near the Marianna Islands in the Philippines to spawn (Tsukamoto 1992). Evidently such long distance swimming will place those fishes under extra stress caused by the long starvation period, the high energy cost of the journey, and the many changes in the environment such as salt water, darkness, high pressure, and low temperatures, among other stress factors. Stress is often a basis for disease in eel, especially in intensive eel culture (Haenen and Engelsma, 2005 unpublished finding).

Nowadays, global transport of live fishes for aquaculture has facilitated the global spread of pathogens from diseased to healthy stocks. Within the last few decades, aquaculture has become an important production branch in our society. Its global production has more than doubled between 1986 and 1996 in tonnage and value, and over one quarter of human fish consumption at world scale is now produced in aquaculture (Naylor et al. 2000). The Netherlands is one of the leading eel producing & trading countries (Heinsbroek and Kamstra 1995). Blanc (1997) showed that nearly 100 pathogens have been introduced into European water bodies since the introduction of aquaculture. Worldwide many diseases are known in both wild and cultured eel. Parasites, for example trematodes, *Anguillicola*

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crassus (nematode), and *Myxidium giardi* (*myxosporean*) occur naturally in wild eel populations, mostly in low numbers, without causing mortality (Kjøie 1988; Van Banning and Haenen 1990; Borgsteede et al. 1999). However, under culture conditions, with eels kept in high densities, they may be harmful. Eel pathogenic bacteria like *Vibrio vulnificus*, *Vibrio anguillarum*, *Pseudomonas anguilliseptica* and *Edwardsiella tarda* may also cause disease, especially when a stress factor is involved or when the eel is injured (Veenstra et al. 1993; Austin and Austin 1999; Haenen and Davidse 2001). As far as we know, the clinical signs are often more severe under culture conditions compared to in the wild.

Several eel viruses are known to cause disease in eels. Three of them are found regularly in Europe: Eel Virus European (EVE), Eel Virus European X (EVEX), and Herpesvirus anguillae (HVA). They are treated, one by one, below. Most of the studies on eel viruses are related to cultured eel, and data on wild eel viral infections are

Table 16.1 Eel viruses isolated from preadult and adult eels (unless elvers are mentioned), several species, of different geographic locations with or without clinical signs. EVE: European Virus of Eel; EVEX: Eel-Virus European X, HVA: Herpesvirus anguillae, IPNV: Infectious Pancreatic Necrosis Virus; (references in the table)

Country	Location	Species	Clinical signs	Virus	Reference
Denmark	Wild eel	<i>A.anguilla</i> (elvers & eels)	-&+	EVEX	Jørgensen et al. 1994
	Wild eel	<i>A.anguilla</i> (elvers & eels)	-&+	IPNV(EVE)	Jørgensen et al. 1994
France	Wild eel	<i>A.anguilla</i> (elvers & eels)	-&+	EVEX	Jørgensen et al. 1994
	French W-coast	<i>A.anguilla</i> elvers	-	EVEX-like	Castric and Chastel 1980
	Eel farm	<i>A.anguilla</i>	+	herpes-like	Jørgensen et al. 1994
	Wild eel	<i>A.anguilla</i>	+	herpes-like	Jørgensen et al. 1994
	Wild eel	<i>A.anguilla</i> (elvers & eels)	-&+	IPNV(EVE)	Jørgensen et al. 1994
Germany	River Mosel	<i>A.anguilla</i>	+	HVA	Lehmann et al. 2005
	Eel farm	<i>A.anguilla</i>	+	HVA	Bergmann unpublished 2006
	Wild eel	<i>A.anguilla</i> elvers	-	EVEX	Shchelkunov et al. 1989
Italy	Eel farm	<i>A.anguilla</i>	-	EVEX & EVE	Van Ginneken et al. 2004
	Eel farm	<i>A.anguilla</i>	+	EVEX	Van Ginneken et al. 2004
Japan	Tokyo (source: France)	<i>A.anguilla</i> elvers	-	EVEX	Sano et al. 1977

(continued)

Table 16.1 (continued)

Country	Location	Species	Clinical signs	Virus	Reference
	Cage culture	<i>A.japonica</i>	+	EVEX-like	Kobayashi and Miyazaki 1996
	Eel farm	<i>A.anguilla</i> & <i>A.japonica</i>	+	HVA	Sano et al. 1990
	Eel farm	<i>A.japonica</i>	+	HVA	Kobayashi and Miyazaki 1997
	Eel farm	<i>A.japonica</i>	+	HVA	Lee et al. 1999
	Eel pond farm	<i>A.japonica</i>	+	EVE	Egusa 1970
	Eel pond farm	<i>A.anguilla</i>	+	EVE	Sano 1976
Morocco	Sebou	<i>A.anguilla</i>	-	EVEX	Van Ginneken et al. 2004
The Netherlands	Lake Grevelingen	<i>A.anguilla</i>	-	EVEX	Van Ginneken et al. 2004
	Lake Grevelingen	<i>A.anguilla</i>	+	EVEX	Van Ginneken et al. 2005
	Eel indoor farms	<i>A.anguilla</i>	+	EVEX	Haenen et al. unpublished 2001
	Lake Grevelingen	<i>A.anguilla</i>	+	HVA	Van Ginneken et al. 2004
	Lake Lauwers	<i>A.anguilla</i>	+	HVA	Van Ginneken et al. 2004
	River Rhine	<i>A.anguilla</i>	+& -	HVA	Haenen et al. unpublished 2008
	Eel indoor farms	<i>A.anguilla</i> elvers	+	HVA	Davidse et al. 1999; Van Ginneken et al. 2004
	Eel indoor farms	<i>A.anguilla</i>	+	HVA	Davidse et al. 1999
	River Merwede	<i>A.anguilla</i>	+	HVA	Haenen et al. unpublished 2008
	Eel indoor farms	<i>A.anguilla</i>	+	EVE	Haenen et al. 2001
	Eel indoor farms	<i>A.anguilla</i>	+	REOVirus	Haenen et al. unpublished 2001
	Lake Grevelingen	<i>A.anguilla</i>	-	picorna-like	Van Ginneken et al. 2004
	Eel indoor farms	<i>A.anguilla</i>	+	orthomyxo	Haenen et al. unpublished 2001
New Zealand	Tekawata	<i>A.dieffenbachi</i>	-	picorna-like	Van Ginneken et al. 2004
	Tekawata	<i>A.dieffenbachi</i>	-	EVEX	Van Ginneken et al. 2004
Sweden	Wild eel	<i>A.anguilla</i> (elvers & eels)	-&+	EVEX	Jørgensen et al., 1994
	Wild eel	<i>A.anguilla</i> (elvers & eels)	-&+	IPNV(EVE)	Jørgensen et al., 1994
United Kingdom	Wild eel	<i>A.anguilla</i> (elvers & eels)	-&+	EVEX	Jørgensen et al., 1994
	Wild eel	<i>A.anguilla</i> (elvers & eels)	-&+	IPNV(EVE)	Jørgensen et al., 1994

scarce. In Table 16.1, results from various authors on the occurrence of eel viruses in eel species from several geographical regions are presented.

16.2 Eel Virus European (EVE)

Eel Virus European (EVE) is a double stranded RNA (biRNA) virus, with an icosahedric form, a size of 68–77 nm, and serologically closely related to infectious pancreatic necrosis virus (IPNV) (Sano 1976; Sano et al. 1981; Okamoto et al. 1983). EVE was first described by Egusa (1970) in pond – cultured Japanese eel (*Anguilla japonica*). It caused branchionephritis, with mortalities up to 50%. In 1976, EVE was isolated for the first time from European eel (*A. anguilla*) in Japan with a renal pathology (Sano 1976). The virus was isolated from European elvers, which had been imported into Japan. The infection spread to Japanese eels and this resulted in mass mortalities, with swollen and congested gills and glomerulonephritis (Egusa 1976; Sano 1976). EVE was suspected to cause disease in natural eel populations by Wolf (1988), but this has not yet been confirmed.

In Europe, Jørgensen et al. (1994) tested 2,092 pools of healthy wild elvers and cultured juvenile European eels, from Denmark, UK, France and Sweden for the presence of virus. Of the 91 virus isolates they typed 38 viral strains from all four countries to IPNV. In The Netherlands, EVE was isolated several times from diseased juvenile cultured *A. anguilla* with haemorrhages in the skin, skin lesions and a congested and red head, with mortalities up to 50% (Haenen et al. 2000 unpublished findings). Van Ginneken et al. (2004) isolated EVE from clinically healthy adult farmed European eel from Italy, which had a double virus infection of EVE and EVEX (Eel Virus European X, a rhabdovirus, see below). At present there are no publications available on EVE isolated from wild silver eels.

16.3 Eel Virus European X (EVEX)

The first rhabdovirus from eel was isolated by Sano et al. (1976) from European elvers imported into Japan from Cuba with a haemorrhagic disease. The virus was named Eel Virus American (EVA). Sano et al. (1977) described the isolation of a second rhabdovirus, from European eels imported from France, and named it Eel Virus European X (EVEX). EVEX is a RNA-virus, and has a bullet-like form and a size of 170–175 × 90–95 nm (Sano et al. 1977). Hill et al. (1980) showed that EVA and EVEX were closely related. Infected elvers in Japan showed vascular congestion of the abdominal surfaces and pectoral and anal fins, and histology revealed extensive haemorrhaging and necrosis of kidney, muscle, pancreas and liver (Sano 1976). One of the rhabdoviruses isolated by Castric and Chastel (1980) from apparently healthy elvers from French coastal waters showed similarities to EVA and EVEX. In infection experiments, this virus isolate was shown to be a-virulent for elvers (Castric and Chastel 1980; Nishimura et al. 1981). EVEX was also isolated from healthy elvers

imported to the USSR from Western Germany (Shchelkunov et al. 1989), and it was shown to be pathogenic to 4-year old eel after i.p. injection. Jørgensen et al. (1994) tested 2,092 pools of healthy wild elvers and cultured eels *A. anguilla*, from Denmark, UK, France and Sweden. From 91 virus isolates, 47 were eel typed as rhabdovirus (EVEX/EVA), isolated from all four countries (Castric et al. 1984).

Kobayashi and Miyazaki (1996) found EVEX/EVA-like virus in Japanese eels from cage culture showing cutaneous erosion and ulceration at 15°C. The disease has sometimes occurred since then and caused mass mortalities in Japanese eel during stocking before shipping. In experimentally induced infections cutaneous lesions developed in eel, accompanied with necrosis of the dermal fibrocytes, haemorrhage and inflammatory cellular infiltration, and diffuse necrosis of the haematopoietic tissue, renal tubules, splenic pulps and hepatic parenchyma (Kobayashi and Miyazaki 1996). They proposed to name the disease *rhabdoviral dermatitis* in Japanese eel. Haenen et al. (unpublished findings) found clinical signs of EVEX in cultured European eel: lethargy, haemorrhages, haemorrhagic skin with tiger-like patterns, skin lesions, a red head, and severe anorexia, with mortalities below 20% at eel farms, or higher, depending on stress factors. They only rarely isolated EVEX. More recently, clinical signs of viral infection have been found in European eel from Lake Grevelingen, The Netherlands, used for a swim tunnel experiment (Van Ginneken et al. 2005). The eels showed some haemorrhages and anaemia, and died after 1,000–1,500 km swimming in the tunnel (Van Ginneken et al. 2005). EVEX was isolated from these eels. The stress factor of the swim tunnels might have induced virus replication, and disease. Van Ginneken et al. (2004) isolated EVEX from Dutch adult wild European eel, farmed Italian European eel, wild adult European eel from Morocco, and wild adult *Anguilla dieffenbachi* from New Zealand, but apart from the European eel from Italy, which showed haemorrhages and red patches on the skin, they showed no clinical signs of disease. It was suggested, that eel virus infections could be a contributing factor in the worldwide decline of the eel stocks (Van Ginneken et al. 2005). However, due to the fact that no EVEX was found in a disease screening of 80 wild silver eel originating from the River Rhine (The Netherlands; Haenen et al. 2008, unpublished findings), there seems to be a very low prevalence of EVEX in at least Dutch silver eels.

16.4 Herpesvirus anguillae (HVA)

Herpesvirus anguillae (HVA) was isolated in 1985 from diseased Japanese eels and European eel in Japan (Sano et al. 1990). It is an enveloped double-stranded DNA-virus, with an icosahedric form, and a size of the nucleocapsid of 110 nm (Davidse et al. 1999). Infected eels showed reddening of the ventral skin, especially below the mouth (Fig. 16.1), and a swollen and haemorrhagic kidney and spleen. Furthermore, necrosis was found in skin, gills, liver and spleen. Kobayashi and Miyazaki (1997) isolated HVA from Japanese eels with skin lesions, and tested the pathogenicity of HVA from cutaneous lesions of Japanese eel. Lee et al. (1999) described gill filament necrosis in farmed Japanese eels infected with HVA.



Fig. 16.1 Diseased cultured European eel *Anguilla anguilla* with a HVA-infection. The eel shows a patchy pattern of haemorrhages in the skin, and severe haemorrhagic fins (Haenen et al. 2002, with permission)

In Europe, Békési et al. (1986) found herpes-like particles in skin lesions of European eel. From 1977–1992 Jørgensen et al. (1994) found herpes-like virus in three pools of farmed and two pools of wild adult healthy European eels from France, by electron microscopy and syncytia-induction in cell culture. By use of EK-1 (eel kidney cells, Chen et al. 1982) Davidse et al. (1999) isolated, replicated, and typed the virus for the first time in Europe from diseased cultured European eel. Optimal growth temperature was 20–25°C (Smail and Munro 2001) and 26°C (Davidse et al. 1999). Clinical signs of HVA outbreaks in farmed European eel were variable: predominantly lethargy, anemia, haemorrhages in skin and fins with a tiger-like pattern, ulcerative skin and fin lesions, haemorrhages in the gills and fins, a red head, a slightly congested head, anorexia, an enlarged spleen, a pale spleen, kidney, and liver with petechial haemorrhages in the liver, pinkfat by small diffuse haemorrhages, haemorrhages in the mesenteria and muscle, and ascites with blood, and congested gill epithelium (Davidse et al. 1999; Haenen et al. 2002).

Van Nieuwstadt et al. (2001) infected clinically healthy farmed juvenile European eel with HVA. The eels showed a response in specific antibodies against the virus, but no disease. It was concluded, that they had probably prior contact with HVA, and were protected by their specific antibodies. HVA was isolated from the eels, and treatment with the stress hormone dexamethasone provoked virus release. The recrudescence of HVA by a stress hormone suggested, that HVA can establish a latent infection in eel (Van Nieuwstadt et al. 2001). Infection of eel with HVA resulted in mortalities from 1% to 6.8% in Japan (Sano et al. 1990), and 10% or higher in cultured European eel in Europe, depending on stress factors like a suboptimal water quality (Haenen et al. 2002).

Van Ginneken et al. (2004) isolated HVA from diseased adult wild and farmed European eels in the Netherlands. The wild eels originated from Lake Grevelingen (one of ten fish positive) and Lake Lauwers (ten of ten fish positive). In both cases the eel showed typical symptoms like reddening with petechial haemorrhages (Van Ginneken et al. 2004). The virus has also been detected in diseased silver eels of the River Merwede in autumn 2003 (The Netherlands; Haenen et al. 2008, unpublished results). In autumn 2004, one out of 6 adult wild European eel origi-

nating from Kornwerderzand (The Netherlands) was found HVA positive without clinical signs. In 2005, 80 silver eels from the River Rhine were investigated for the presence of pathogens, including eel viruses (Haenen et al. 2008, unpublished findings). HVA was detected in 2005 in 40% of the eels, of which most eels showed some fin haemorrhages but no internal signs of viral disease. However, the clinical observation of fin haemorrhages was a-specific, as they were also seen in virus negative eels. From these observations we suggest that HVA is widespread in wild European eel, supporting the hypothesis of Van Nieuwstadt et al. (2001) and Haenen et al. (2002).

16.5 Other Viruses and Double Infections

Other viruses have been isolated from eels in lower frequencies. For example, in The Netherlands, between 1986–2001 a reovirus was isolated four times from diseased cultured European eel, showing a mortality of less than 5%, (Haenen et al. unpublished findings). Furthermore, two orthomyxo virus strains with a mortality of less than 25%, and one non-EVE biRNA virus with a mortality of 100% were isolated from diseased cultured sub-adult European eel in The Netherlands (Haenen et al. unpublished findings). As these viruses occur only rarely, the impact on the eel population in total will most probably be limited. In cultured eel double infections with two viruses can be observed: herpesvirus and reovirus, EVEX and reovirus, HVA and EVE, and EVEX and EVE are known combinations related to diseased cultured European eel (Haenen et al. 2003, unpublished findings). Thus far, no double infections were found in wild eels.

16.6 Discussion

16.6.1 *Viral Infections in Wild Eel*

As described above EVE, and EVEX both have been isolated from elvers. This implicates either a vertical transmission of the disease, from parent to offspring, or acquisition of the infection at an early stage in life. For IPNV, which is strongly related to EVE, vertical transmission has been shown in salmonids (Bullock et al. 1976; Mulcahy and Pascho 1984), via eggs and sperm of fish, but this is not yet proven for eels. In juvenile eels, all three viruses EVE, EVEX, and HVA have been found. In wild silver eels, only EVEX and HVA were found so far. In general the viruses seem to be wide spread in the wild European eel population with limited impact. This suggests EVE, EVEX and HVA to be endemic in Western Europe and the wild eel to have some resistance against these viruses.

16.6.2 *Stress and Immunosuppression during Migration*

Stress can be an important factor in inducing disease in eel, in its severity determining the seriousness of the disease. The stress source may either be physical (high water temperature), chemical (bad water quality, pollution), or biological (lack of feeding, predators, etc.). Eels prepare their long-distance migration by increasing energy reserves as stored fat, which can reach levels up to 30% of their body weight (Bruggeman 1983). Stored lipid reserves in wild eels are often contaminated with PCBs, which have an immuno-suppressive effect (see Chapter 15). For other pathogens like parasites and bacteria the immunosuppressive action of PCBs has been demonstrated in response to exposure to different PCBs. Sures and Knopf (2004) showed that PCB 126 suppressed humoral immune response in European eel: The exposure to PCB 126 increased the chance of infection of eel for the nematode *Anguillicola crassus*.

Also the migration and spawning itself could stress the migrating eel as a biological and physiological stress factor, and thereby suppress the resistance to pathogens. In general physiological stress, when associated with migration is a well known risk factor resulting in increased susceptibility of animals for infectious diseases. For bird migration it is known that migration stress often leads to reactivation of otherwise latent virus infections (Gylfe et al. 2000).

16.6.3 *Eel Viruses and Migration*

In a recent paper it was shown that a group of eels swimming continuously in swim tunnels at a speed of 0.5 BL s^{-1} became seriously ill (Van Ginneken et al. 2005). Those eels were found to be infected with the rhabdovirus EVEX (Eel Virus European X)-virus, and the other eels from the same batch did not develop this disease. The clinical signs were haemorrhages and extreme anemia in actively swimming eels. Possibly, the anemia was the result of internal blood loss, since the symptoms included blood in the abdominal fluid and haemorrhages all over the body. The observed changes in plasma lactate dehydrogenase (LDH), total protein and aspartate aminotransferase (AAT) were indicative of a serious viral infection. All experimental eels stopped swimming between 1,000 and 1,500 km during simulated migration, and died soon after that (Fig. 16.2). In contrast, non-infected animals from another batch swam 5,500 km, the estimated distance to the spawning ground of the European eel in the Sargasso Sea. Virus-positive eels showed a decline in hematocrit, which was related to the swim distance, while the healthy eels showed even a slightly increased hematocrit. Based on these observations, we concluded, that eel viral infections may adversely affect the spawning migration of eels, which might be a contributing factor to the worldwide decline of eel.

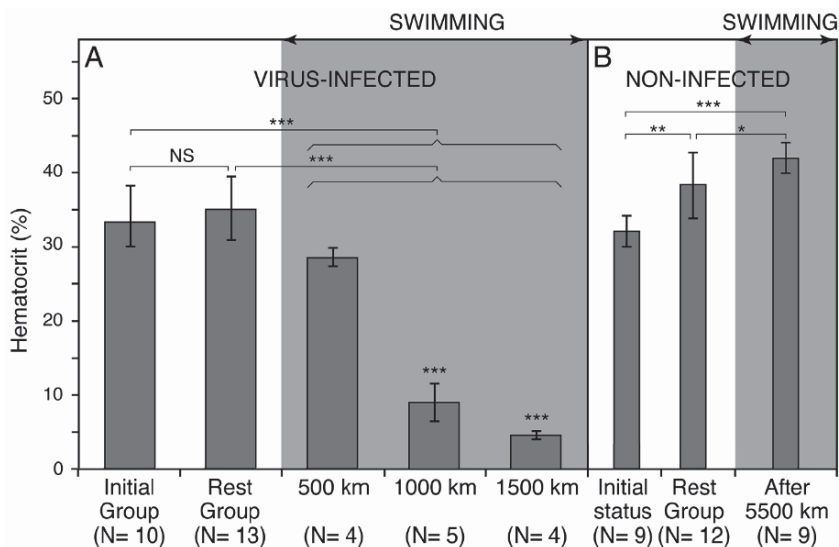


Fig. 16.2 Development of anaemia in swimming eels which were tested and were shown to be infected with EVEX. **A:** Haematocrit of European eels infected with EVEX-virus. Eels swam for up to 1 month during September 2000 in large 127-l Blazka swim tunnels. The decrease of red blood cells is negatively correlated with the distance covered. The rest group did not develop clinical signs of the disease. **B:** Haematocrit of virus-negative eels which swam 5,500-km during the period March-September at a continuous speed of 0.5 Body-length (BL) per second in the same set up. * $P \leq 0.05$, ** $P \leq 0.01$, *** $P \leq 0.001$ (Student t-test) (Van Ginneken et al. 2005. With permission)

16.6.4 Eel Migration and Water Temperature

The temperature range in which wild eels migrate has been studied. Silver eels migrate downstream in late summer and autumn, at temperatures around 15°C, while at lower temperatures (<10°C) they stop their migration behavior (Tesch 2003). In the autumn the North Sea remains at 12–15°C, the same applies for the upper 200 m of a large part of the Atlantic Ocean. From telemetry studies it was observed that silver eels, which were tracked when they left the continental slope off the Bay of Biscay and west of Spain, swam to depths of at least 400 m in the daytime, while they selected shallower water (50 to 215 m) at night (Tesch 1978). Studies on tracking eels in the western Mediterranean Sea provided information on thermal preference. Eels tended to swim in the 13°C hypolimnion, preferred 344 m, at day, but at night regularly crossed the thermocline during vertical migrations to 196 m depth into surface waters as warm as 18°C, according to a diurnal rhythm (Tesch 1989).

The distance to the continental shelf starting in The Netherlands is 1,000–1,500 km, and till then eels have to travel in this upper layer, which certainly remains above 10°C. As eels migrate further into the Atlantic, they are assumed to swim at depths

below the 500m zone. Below 500m the temperature drops rather fast from around 10°C at 500m to 4°C at 2,000m depth. This means, that it is likely that eels swim for most of their journey (4,000km) at temperatures between 10°C and 4°C. The temperature profiles of the water of the Atlantic ocean below 750m are rather stable at around 8°C (20–40° N), with the isotherm of 5°C between 1,000 and 1,500m. Steep temperature profiles can be found in the upper water layers.

In the Sargasso Sea and along the Gulf Stream warm water reaches rather deep, mainly due to the higher salinity. The Sargasso Sea is a massive sinking gyre with a diameter of some 2,000-km. The circulating water mass sinks due to the evaporation at the surface, which increases the salinity and therefore its density. At the surface the temperatures are above 30°C, falling rather slow with depth to about 16°C at 500m. A very stable water layer of virtually the same salinity and temperature (18–19°C) occurs between 100 and 400m over an enormous surface area (Worthington 1959). At deeper levels the temperature drops again quickly: at 500m 16°C and at 750m already 11°C. So, for migrating silver eels – assuming they are swimming at depths below 750m – it will be a rather sudden change in temperature when they arrive at the Sargasso Sea; the temperature will rise from 5–8°C to 11–12°C. This rise with 6°C will take place over a distance of a few hundred kilometres, which they cover easily in a few days. As silver eels cannot be stimulated to maturation at 12°C (see Chapter 13), it is likely that they will go to shallower waters after arriving at the Sargasso Sea. The vertical migration of the leptocephali occurs actually in the upper layers between 140m at night and at 350m daytime (Kleckner and McCleave 1982). These water layers have a year round constant temperature of 18–19°C (Worthington 1959). As the eggs float, going up by 100–200m over 2 days before hatching, spawning likely takes place at a greater depth. However, as maturation is temperature dependent this will not be much more than 400–500m. In a laboratory experiment the final preferred temperatures (FPT, the temperature an animal ultimately selects) of pre-migratory and migratory American eels were determined using chronic tests in a horizontal thermal gradient. Results indicated that both silver and yellow *Anguilla rostrata* in saltwater had mean FPTs of 17.5°C, which indicates that American silver eels have a preference for relatively high temperatures (Haro 1991).

16.6.5 Eel Viral Disease Dependent on Water Temperature

Temperature is an important factor involved in the development of viral disease in fish: Each virus has its appropriate optimal temperature for virus replication. For EVE this is between 15°C and 23°C (Haenen and Davidse 2001 unpublished findings), for EVEX between 10°C and 15°C (Shchelkunov et al. 1989), and for HVA between 20°C and 26°C (Smail and Munro 2001; Davidse et al. 1999). Outside these temperature ranges the viruses may persist but disease does not develop in the host. In this light, (latent) viral diseases of eel depend strongly on the environmental temperature. Given the fact, that, like discussed above, the eels are thought to swim

for a long time at temperatures between 4°C and 10°C, and in the Sargasso Sea they are supposed to swim in water of 10–20°C, the eels experience temperatures, which, dependent on the optimum per eel virus, may contribute to the reactivation of latent virus in the eel, and replication of eel virus, causing the development of viral disease in eel.

As presented in Table 16.1, many virus infections are found in farmed and wild eel populations in The Netherlands, which could be a threat for the global eel population. HVA and EVEX have been found in eels from several countries worldwide: EVEX now and then in the European eel, *Anguilla anguilla*, in the Netherlands, Italy, and Morocco, but also in another eel species: The New Zealand eel, *Anguilla dieffenbachi*, in this latter without clinical signs though. Moreover, Herpesvirus anguillae (HVA) has been isolated and identified in eel populations in different parts of the world: In cultured eels in Taiwan (Ueno et al. 1992, 1996; Chang et al. 2002), in cultured and wild eels in The Netherlands (Van Ginneken et al. 2004; Van Nieuwstadt et al. 2001; Davidse et al. 1999; O. Haenen et al. 2008, unpublished) and in cultured eels (S. Bergmann 2006, unpublished) and wild silver eels in Germany (Lehmann et al. 2005) EVE was never found in silver eel yet, so the disease risk of this virus in migrating eel is supposed to be small.

16.7 Conclusions

This study shows that EVEX, EVE, and HVA are found at several geographic places in wild and farmed European eel. Transport of clinically healthy but virus-infected elvers and eels may cause introduction of eel viruses into the virus uninfected waters with eels, both fisheries and aquaculture. No serious mortalities are known in wild eel populations due to eel viruses, but wild diseased eels are difficult to trace, particularly migrating silver eels.

Related to the fact that migrating silver eels are stressed during their spawning migration, and as a result immunosuppressed, with the assumption, that they swim for some weeks in ambient water temperatures for virus infections, the eels might get viral disease in these water bodies. To what extent this disease would occur and threaten the wild eel population and recruitment depends on the immune status of the eels, the water temperature, and the pathogenicity of the particular virus strain. Maturing eels have a depressed immune system, so, they do run a risk of developing the disease after arriving in the Sargasso Sea, the extend of it is still difficult to estimate.

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