Opinion Article

Can Ulcerative Dermal Necrosis (UDN) in Atlantic salmon be attributed to ultraviolet radiation and secondary Saprolegnia parasitica infections?

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\textbf{A R T I C L E  I N F O}

Article history:
Received 20 April 2021
Received in revised form 7 February 2022
Accepted 18 February 2022

Keywords:
Atlantic salmon
Saprolegnia parasitica
Skin damage
Stress factor
Ulcerative dermal necrosis
Ultraviolet radiation

\textbf{A B S T R A C T}

Ulcerative dermal necrosis (UDN), a chronic skin condition, affects primarily mature wild salmonids returning from the sea to freshwater for their spawning. The involvement of water moulds such as Saprolegnia parasitica as a secondary pathogen in this disease is clear but the identification of a primary cause or of primary pathogen(s) remains elusive. In this opinion article, we re-visit UDN regarding epidemiology, pathology and aetiology and speculate the potential involvement of UV radiation in the initiation of UDN in salmonid fish returning from the sea.

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1. Introduction

The first reported outbreak of ulcerative dermal necrosis (UDN) was in 1877, occurring in Scottish rivers of the Solway Firth. However, according to a Report of HM Inspector of Salmon Fisheries (\textit{England and Wales}, 1882), the disease had existed in rivers for a long time before initially being referred to as ‘Salmon Disease’. Evaluation of related literature has shown the existence of UDN at periodic intervals since the 1860s.
UDN as primarily a lesion of epidermal and dermal layers of the head area is believed to start during homeward migration and characteristically found in adult fish as they congregate prior to entering freshwater for upstream river migration. Confirmation of UDN however requires histological examination of early skin lesions which are considered to be the only specific signs of the disease. The likelihood of a correct diagnosis is reduced by the lack of an agreed UDN case definition and also by the infection of the skin lesions by water moulds, such as Saprolegnia parasitica that are a normal part of the river ecological system. Despite the similarity between saprolegniosis (the disease caused by Saprolegnia species) and UDN i.e. grey patches mostly on unscaled skin areas, saprolegniosis does not cause skin ulcers nor cell necrosis. It should be noted that S. parasitica should also be considered as a primary pathogen, but we do not discuss this further in this current opinion article (references include: Belmonte et al., 2014; Sandoval-Sierra et al., 2014; Trusch et al., 2018).

Due to dramatic losses and public concern regarding the 1960–70 outbreak several groups attempted to identify the aetiological agent/s. However, to date the primary cause/s of this complex condition remains unsolved. Historically, some researchers thought that the available evidence suggested that Saprolegnia is the primary parasite in UDN or that an extremely close relationship existed between an unknown primary parasite and Saprolegnia (Stuart Fuller, 1968), others defined putative bacteria or virus aetiology (Carbery and Strickland, 1968). Indeed, combinations of bacteria are occasionally detected in cultures of UDN lesions, although these are primarily fish pathogens and not related to the characteristics of skin lesions of UDN (Bissett, 1946; Roberts, 1993). Thus, although many studies have been led regarding the potential involvement of bacteria, oomycetes or virus, the specific factors leading to the onset of the characteristic lesions of UDN in wild salmonids remain to be elucidated. In this opinion article we will discuss the possible synergic association between oomycete and environmental factors, more precisely ultraviolet (UV) radiation, in the occurrence of UDN.

2. How prevalent is UDN?

Originally it was believed the disease was restricted to wild Atlantic salmon (Salmo salar L.), but later UDN like conditions were also observed and reported affecting, sea trout (Salmo trutta L.), brown trout (Salmo trutta L) as well as farmed rainbow trout (Oncorhynchus mykiss gairdneri) (Eiras et al., 1988; Lounatmaa and Janatunien, 1978; Roberts, 1993). To date, the disease has been reported on several occasions in the United Kingdom and Ireland (Stirling, 1879; Carbery and Strickland, 1968; Roberts et al., 1972; Roberts, 1993, Noguera and Marcos-Lopez, 2019), France (De Kinkelin and Le Turdu, 1972), Sweden (Johansson and Ljungberg, 1977), Switzerland (Meier et al., 1977), Finland (Lounatmaa and Janatunien, 1978), Portugal (Eiras et al., 1988), Norway (Poppe, 2016) and Poland (Ciepłński et al., 2018), with huge economic losses of wild salmon broodstock occurring during an epidemic (Roberts, 1993; Roberts et al., 1972).

Macroscopically UDN develops from focal to small, superficial grey to white areas of roughened skin, to deep ulcers involving much of the head and covered with a grey-white fungal mycelium (Fig. 1). Lesions appear primarily in unscaled areas of the body, initially mostly the fish head (snout, operculum) or near the tail, such as the adipose fin (Munro, 1970; Roberts et al., 1970; Roberts and Hill, 1976). It is believed that this skin damage favours further secondary infections caused predominantly by Saprolegniales (Roberts, 1993). Oomycete infections can gradually spread all over the entire body of the fish leading to serious damage to the epidermis and dermis under the mycelial excrecence (Johansson et al., 1982; Munro, 1970; Roberts and Hill, 1976).

Gardner (1974) reported a case of UDN disease affecting osmoregulation in Atlantic salmon. Their research showed a decrease in plasma osmotic pressure along with the sodium concentration, and suggested a negative effect of changes in salt level on disease severity. More recent studies (Kurhalyuk et al., 2009, 2010; Tkachenko et al., 2014) claimed that UDN affects antioxidative defence mechanisms in trout, causing a decrease in the antioxidant enzyme activities and total antioxidant activity in the muscle, heart and liver tissues, and an increase in oxidative stress markers.

Recently, a new finding has pointed to fungi belonging to the genus Fusarium potentially playing a role in UDN (Pe˛kala-Safiańska et al., 2020). Fusarium infections are reported in aquatic animals and notably cause black gill disease in crustaceans (Sarmiento-Ramírez et al., 2010). Species from the genus Fusarium are known to produce mycotoxins (e.g. nivalenol, zearelenone and moniliformin), which affect both animals and humans (Sugiura, 2012; Asam et al., 2017). Interestingly, zearelenone was detected in the organs of brown trout (Salmo trutta morpha trutta) that were affected by UDN. These brown trout were collected from the Stupia River in Poland and toxicological analysis of organs (i.e. gastrointestinal tract, liver, kidney) revealed the presence of zearealenone which supports the hypothesis of a possible involvement of Fusarium sp. in UDN (Pe˛kala-Safiańska et al., 2020). Further studies on this will be informative, although many detailed studies have been carried out on the aetiology of UDN, none so far have been able to conclusively demonstrate the role of a specific factor or organism.

Why has the aetiology of UDN remained elusive for so long? Are there synergistic cryptic factors involved? Could one factor be UV radiation?

3. Ultraviolet radiation and fish

Ultraviolet radiation (UVR) is divided into 3 spectral wavelengths bands: UVA (320–400 nm), less harmful; UVB (280–320 nm), moderately harmful and UVC (200–280 nm), highly harmful but mostly absorbed by stratospheric ozone and oxygen (McKenzie et al., 2007). In nature, an increasing number of stations and networks have shown that there has been an increase in solar UVR at the surface of and within aquatic systems which corresponds with stratospheric ozone depletion (Wassmann et al., 2010).
Stratospheric ozone dynamics and climate change interact strongly, increasing the potential exposure of fish to UVR under water. Fish are then exposed to new and complex interactions between UVR and environmental stressors (biotic and abiotic), which potentially affect fish growth and survival (Alves and Agusti, 2020).

Aquatic ecosystems play an important role in nature and they are being threatened by the increased UV radiation (Hader et al., 2011). Nevertheless, aquatic environments vary tremendously in their UV attenuation. Depending on the water transparency, organic/inorganic material, among others, different optical properties are achieved. Freshwater habitats have a higher UV absorption (Bullock, 1982; Hader et al., 2007) than seawater habitats.

Despite reports in the literature as early as 1820 suggesting a correlation between dorsal skin damage in fish and ultraviolet light, it was not until the 1960s, when the pioneering work of Jerlov (1968) and Steeman-Nielsen (1964) on UV penetration in oceanic waters was published, that the potential hazard of solar UV within the aquatic ecosystem was recognised (Bullock, 1988).

Some of the UVR effects described in fish include: a reduction in growth, an impaired development, behaviour changes, development of skin and eye lesions, a reduction in mucus producing goblet cells in the skin, an impaired immune system, increased diseases susceptibility, DNA damage and a series of metabolic and physiological stress changes (Browman et al., 2003; Hunter et al., 1982; Salo et al., 2000a; Sandrini et al., 2009; Sharma and Katiyar, 2005; Kaweewat and Hofer, 1997). Stress factors have been implicated in the onset of UDN. Stress in fish may lower the immune system subsequently making fish more susceptible to disease (O’Brien, 1974; Beckmann et al., 2020). Such stress could be encountered naturally notably during adaptation of salmon returning from the sea to freshwater. This also may be compounded by UVR should the levels of the rivers be particularly low.

Several cases of sunburn due to overexposure to high natural solar radiation resulted in numerous losses in aquaculture fish farms during the 1980–1990s, particularly in those where fish were grown in outdoor tanks (Bullock and Coutts, 1985; Bullock, 1982, 1988). As mentioned above for UVR effects described in fish, abnormal behavioural changes have also been recorded associated with UDN and it was proposed that UDN causes disruption of the Central Nervous System of the fish which could explain these behavioural changes (Roberts et al., 1970). Under particular weather conditions and river levels, UVR could initiate or exacerbate early pre mycotic lesions, usually limited to epidermal acantholysis and pemphigoid-like degeneration of the lower layers, alongside swelling and degeneration of the melanophores immediately below the basal layer (Bruno et al., 2013). Since the cases of sunburn reported in the 1980s - 1990s, more recently, Sweet et al. (2012) described extensive melanosis and melanoma (skin cancer) in wild populations of the coral trout Plectropomus leopardus due to UVR exposure. UV radiation can also act as an indirect virulence factor by promoting the loss of epidermal integrity facilitating the entry of pathogens and causing osmotic disturbances (Zagarese and Williamson, 2001). Lesions are rapidly colonised by pathogens leaving the original damage difficult to identify. Alterations related to the fish physiological stress response, increased plasma cortisol (stress hormone) concentration followed by blood lymphopenia and granulocytosis were detected in roach (Rutilus rutilus) exposed to moderate levels of UVB doses but no visible signs of oedema, sunburns or signs of infection in the skin of fish were induced (Jokinen et al., 2001; Salo et al., 1998, 2000a, 2000b).

Skin lesions were also found in natural fish populations inhabiting environments with high UV radiation levels, such as residual waters on the tidal flats (Berghahn et al., 1993). Several researchers have also observed the deficiency of mucus on the surface of the skin in areas affected by UDN (Roberts et al., 1970; Johansson et al., 1982). Moreover, Kaweewat and Hofer (1997) found that the number of goblet cells (mucus secreting cells) in the dorsal epidermis of two salmonids species (Oncorhynchus mykiss and Salvelinus alpinus) was significantly reduced following UV-B radiation. A lower number of goblet cells would likely result in less mucus production and thus a reduced protective barrier against pathogens.

Interestingly, UV in combination with Saprolegnia also seems to play a significant role in the decline of amphibians world-wide (Kiesecker and Blaustein, 1995). It was found that a synergistic effect between UV-B radiation and Saprolegnia increased the mortality of amphibian embryos compared with either factor alone.
4. Conclusion

Evidently Saprolegnia species play an important role in UDN outbreaks since sporulating mycelia infected tissue is the main visual sign in UDN-affected fish. We know that warmer, drier springs - weather conditions with higher UV intensity - can be correlated with increased S. parasitica infections. If the river depth decreases during hot and dry weather, salmonids returning from the sea will have less chances of escaping from UV radiation. This could lead to longer UV radiation exposure, which in turn gives rise to changes in behaviour, more skin wounds, fewer goblet cells, reduced mucus levels and ultimately increased stress levels. Stressed and immuno-compromised fish are more prone to an increased incidence of S. parasitica infections (Beckmann et al., 2020; de Bruijn et al., 2012; Johansson et al., 1982; Kaweewat and Hofer, 1997; Roberts et al., 1970; Salo et al., 2000a; Zagares and Williamson, 2001). It is interesting to note that deforestation has exposed rivers and therefore reforestation would increase shading, reduce river temperatures and also reduce UV exposure (Dugdale et al., 2018), which could in turn reduce the incidences of UV radiation inflicted skin damage of the salmon.

In summary, we would like to hypothesise that ulcerative dermal necrosis is a complex type of skin condition where increased UV-radiation exposure of salmonid fish plays a relevant role as a stress factor initiating or exacerbating skin damage with the consequent decrease of mucus, that allows secondary infections to take place (Saprolegnia species and possibly other microbes such as Fusarium and bacteria), which ultimately results in what is commonly known as UDN.

Declaration of competing interest

None declared.

Acknowledgements

This work was partially funded from the European Union’s Horizon 2020 research and innovation program under the Marie Skłodowska–Curie grant agreement No 766048 (PROTECTA).

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