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Comparative Susceptibility of Brown Trout and Rainbow Trout to *Discocotyle sagittata* (Monogenea)

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ABSTRACT: The susceptibility of brown trout (*Salmo trutta*) and rainbow trout (*Oncorhynchus mykiss*) to the monogenean *Discocotyle sagittata* in the United Kingdom was assessed by experimental infection of naive fish. One month postinfection with 100 oncomiracidia/host, brown trout harbored significantly lower burdens (27.7 worms/host ± 4.13 SE) than rainbow trout (47.8 worms/host ± 3.90; $P = 0.002$). This indicates that the consistently lower prevalence and intensity of *D. sagittata* recorded in naturally infected farmed fishes reflects differences in susceptibility to the parasite. The outcome may be related to the comparatively short-term association of this parasite with rainbow trout (introduced to Britain in the 1880s) compared with the established native host–parasite association.

Discocotyle sagittata is a monogenean occurring on freshwater salmonid fishes in the Northern Hemisphere (Williams and Jones, 1994). In Europe, 2 salmonid species are cultivated widely, the native brown trout (*Salmo trutta*) and rainbow trout (*Oncorhynchus mykiss*), which was introduced in the late 19th Century. Farmed trout on the Isle of Man, located in the Irish Sea, harbor high burdens of *D. sagittata* and exhibit high mortality rates attributable to parasite-induced anemia (Gannicott, 1997). Field data covering several years show that brown trout exhibit lower parasite prevalence and intensity than rainbow trout in the same farm (Gannicott, 1997), suggesting that the 2 fish species may differ in their susceptibility. Farmed brown trout have been shown to possess higher anti–*D. sagittata* immunoglobulin titers than rainbow trout (Rubio-Godoy et al., 2003b). Antibodies may therefore be involved in mediating acquired immunity to the parasite. Nonspecific immunity to *D. sagittata* may also be distinct in *O. mykiss* and *S. trutta*. Sera from these 2 salmonid species differ in their ability to kill *D. sagittata* oncomiracidia in vitro, the lethal effect being mediated by the alternative activation pathway of complement (Rubio-Godoy et al., 2004). This study was undertaken to assess the susceptibility of brown trout and rainbow trout to a controlled infection.

Farmed fishes that had become naturally infected on the Isle of Man were transported to Bristol, maintained in 500-L tanks at 13 C, and used as a source of parasite eggs. Eggs were collected by filtering tank water through a 125-μm nylon mesh, washed in dechlorinated water, and incubated at 13 C in 12:12 photoperiod. Under these conditions, the eggs of *D. sagittata* hatch after ca. 28 days at the start of each dark period (Gannicott and Tinsley, 1997), and oncomiracidia can be collected under a dissecting microscope. Brown trout and rainbow trout fingerlings (ca. 20 cm in length) were purchased from a parasite-free farm. (Trout from this farm were known to be naive because successive samples consistently showed absence of *D. sagittata* infection; the farm uses its own spring water, and there is no catchment that could produce contamination.) Fifteen fish of each species were infected individually by exposure to 100 oncomiracidia in tanks in which they were kept for 24 hr at 13 C (Rubio-Godoy and Tinsley, 2002). Fish were then maintained in 250-L tanks and killed 1 mo later; 2 rainbow trout died for unknown reasons before the end of the experiment. Gills were immediately dissected into individual arches, placed in water in separate petri dishes, and screened under the dissecting microscope. Parasite intensity per gill arch was recorded for the 2 host species. The developmental stage of each worm was determined by counting the number of clamps present on the haptor; a mean developmental index (DI) was calculated for worms from individual fish. DI values range from 1 for freshly attached worms with 1 pair of clamps, to 4.5 for sexually mature worms with 4 pairs of clamps (Rubio-Godoy and Tinsley, 2002). Data were analyzed using SPSS for Windows 10.0 (univariate analysis of variance; general linear model analysis). The use of parasitological terms is according to the recommendations of Bush et al. (1997).

All experimentally exposed fish were infected at the time of dissection, 1 mo postinfection. Brown trout had a mean intensity ± SE of 27.7 ± 4.13 worms/host (range 14–70) and rainbow trout 47.8 ± 3.90 worms/host (range 24–73) (Fig. 1). Worms recovered from *O. mykiss* presented a mean DI ± SE of 1.67 ± 0.08 and those from *S. trutta* of 1.69 ± 0.04. Intensity significantly differed between the 2 host species ($F_1 = 12.274$; $P = 0.002$), but DI indices did not differ. Parasite burdens were more overdispersed in brown trout than in rainbow trout, with variance to mean (V/M) ratios of 9.27 and 4.14, respectively.

Naive individuals of both trout species exposed to experimental infection were found to be susceptible to *D. sagittata*. However, in response to the same exposure dose, brown trout appeared to be capable of regulating their worm burdens more effectively than rainbow trout maintained in identical conditions; this is supported by the higher V/M ratio recorded for *S. trutta*. Under conditions of natural transmission, fish would normally experience repeated invasion events. In experimental infections of the clawed toad *Xenopus laevis* with its natural monogenean parasite *Protopolystoma xenopodis*, successful primary infection (94% prevalence determined at patency) was followed, after the lapse of this infection, by a high degree of acquired immunity to secondary exposure (15% prevalence) (Jackson and Tinsley, 2001). If similar immune capabilities occur in this fish host–*D. sagittata* system, it would be predicted that *S. trutta* develops more powerful resistance to reinfection by its native parasite than *O. mykiss*. This would be in accordance with field data documenting different infestation levels (Gannicott, 1997; Rubio-Godoy et al., 2003b).

The apparent differential susceptibility may be related to the ancestral distribution of the 2 salmonid species and their possible coevolution with regional parasite strains. *Salmo trutta* is indigenous to Europe, northern Africa, and western Asia, whereas *O. mykiss* evolved in a restricted geographical area of the northern Pacific (Burgner et al., 1992). Rainbow trout was introduced from its native area throughout the world in the late 19th century, as eggs and fry to eastern North America and as eggs elsewhere. Wherever it was introduced as eggs, rainbow trout was free of monogenean parasites (Kennedy and Bush, 1994). *Discocotyle sagittata* is a specialist parasite (Marcogliese and Cone, 1991; Kennedy and Bush, 1994), occurring in low levels in several wild salmonid species throughout the Northern Hemisphere, e.g.,

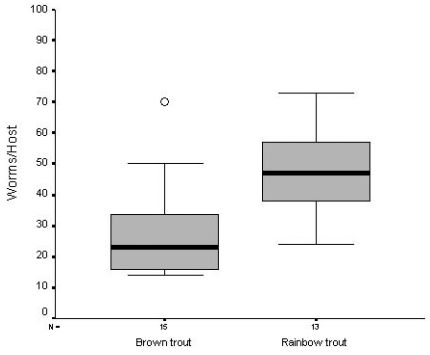

FIGURE 1. *Discocotyle sagittata* burdens in brown trout (*Salmo trutta*) and rainbow trout (*Oncorhynchus mykiss*) 1 mo postinfection with 100 oncomiracidia/fish. The figure illustrates the median (thick line), the interquartile range containing 50% of the values (box), and the range (whiskers) for each fish species. Note: the letter O represents an outlying data point (70 worms/host) well outside the 95% confidence interval calculated for the mean parasite intensity in brown trout.

in brown and sea trout (Chubb, 1977; Kennedy et al., 1991), rainbow trout (Kennedy et al., 1991), brook trout, *Salvelinus fontinalis* (Cone and Ryan, 1984); Atlantic salmon, *Salmo salar* (Cone and Ryan, 1984); and whitefish, *Coregonus* spp. (Chubb, 1977). In general, *D. sagittata* infection levels in all wild fish species studied have been found to be low, with the majority of salmonids harboring <6 worms/host. This could be an indication of long-term coexistence of hosts and parasites, resulting in low infection intensities that cause little or no harm to hosts (Mo, 1994). Thus, fish may evolve defensive mechanisms to local parasite strains or species, as exemplified by the varying success of gyrodactylids infecting salmonids from different geographical regions (Bakke et al., 1990; Buchmann and Uldal, 1997). Exposure of hosts to novel parasites can result in severe, unchecked infections leading to major mortality, as found for Norwegian salmon exposed to Baltic *Gyrodactylus salaris* (Mo, 1994).

Innate and adaptive immune mechanisms in fishes have been shown to mediate rejection of some monogenean parasites (Buchmann and Lindenstrøm, 2002). In particular, complement from different fish species kills gyrodactylids with efficiencies related to host susceptibility to the parasites (Buchmann, 1998; Harris et al., 1998). Brown trout serum is more effective at killing *D. sagittata* oncomiracidia than rainbow trout serum (Rubio-Godoy et al., 2004), which may in part explain the different susceptibility illustrated in this report. Nonetheless, rainbow trout are capable of developing partial immunity to *D. sagittata* after vaccination (Rubio-Godoy et al., 2003a). A significant negative correlation was found between antibody titers in vaccinated fish and *D. sagittata* intensity, suggesting that immunoglobulin may be involved in mediating protection (Rubio-Godoy et al., 2003a). Naturally infected, farmed fishes likewise have significantly elevated anti–*D. sagittata* antibody titers; on average, brown trout had higher immunoglobulin titers and lower parasite prevalence and intensity than rainbow trout (Rubio-Godoy et al., 2003b). Thus, immunity against *D. sagittata* apparently involves both innate and acquired components. The contrasting susceptibility of these 2 salmonid species to *D. sagittata* may illustrate different levels of coevolution of hosts with the parasites encountered in their environment.

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