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Aquaculture 246 (2005) 139–149

Aquaculture

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Genetic (co)variation in resistance to White Spot Syndrome Virus (WSSV) and harvest weight in *Penaeus* (*Litopenaeus*) *vannamei*

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Received 15 July 2004; received in revised form 1 February 2005; accepted 7 February 2005

Abstract

A total of 339 full-sib families (representing 143 paternal half-sib families) and 337 full-sib families (representing 145 paternal half-sib families) were respectively challenged with White Spot Syndrome Virus (WSSV) in a controlled environment and tested for growth performance under commercial growing conditions. The families were derived from two selected lines. The estimates of heritability ($h^2 \pm$ SE) for the two lines were 0.03 ± 0.02 and 0.07 ± 0.02 for WSSV resistance and 0.21 ± 0.04 and 0.20 ± 0.04 for harvest weight. Moreover, there was an unfavorable genetic correlation of -0.55 and -0.64 in the two lines between resistance to WSSV measured in controlled challenge tests at an average weight of 3 g and growth performance to harvest size in ponds. Thus, for simultaneous improvement of both traits breeding values for WSSV resistance must be obtained from data recorded on sibs of the breeding candidates.

Keywords: Shrimps; *Penaeus (Litopenaeus) vannamei*; Heritability; Genetic correlations; WSSV resistance; Harvest weight

1. Introduction

Over the past decade, infectious diseases have been a major cause of the variation in shrimp production (Moss, 2002). The rapid spread of infectious diseases has been ascribed to environmental degradation and the use of wild larvae as the seed for grow out farms (Gjedrem and Fimland, 1995) and also to the introduction of more intensive production systems with high densities of shrimps in the ponds. Among the many pathogens known to affect shrimps (Lightner and Redman, 1998) White Spot Syndrome Virus (WSSV) has been one of the most detrimental and widely disseminated pathogens of farmed shrimps.

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doi:10.1016/j.aquaculture.2005.02.011

WSSV was first detected in Taiwan in 1992 (Chou et al., 1995). Since then white spot disease spread rapidly to most Asian countries including Japan, Thailand and India (Inouye et al., 1994; Chen, 1995; Flegel and Alday-Sanz, 1998; Wongteerasupaya et al., 1995; Sahul Hameed et al., 1998) and by 1996 most farming regions in South-East Asia were affected (Flegel and Alday-Sanz, 1998). In the western hemisphere the virus was first detected in 1995 in cultured *Litopenaeus setiferus* in Texas (USA) (Lightner, 1996) and in wild populations of *L. setiferus* and *Litopenaeus duodarum* in the same region in 1997 and 1998 (Lightner, 1999). At the same time the first WSSV outbreak in farmed *P. vannamei* and *P. stylirostris* appeared in South Carolina (USA) and was associated with 95% cumulative losses (Lightner, 1999). By early 1999, WSSV had spread to farmed *P. vannamei* in Central America where it caused high mortalities (Jory and Dixon, 1999). The economic losses caused by WSSV in Asia and in Latin America have been enormous with a large number of farms being put out of production and many jobs lost making this disease one of the worst pandemics in aquaculture (Hill, 2001).

In Colombia, WSSV was first positively identified on the Pacific coast in 1999 and mortalities in infected ponds often exceed 95%. Currently the survival in commercial ponds averages about 20%, as compared to nearly 60% before the arrival of WSSV. However, the disease did not spread to the Colombian Atlantic coast and until now there have not been fatalities due to WSSV in this part of the country. This is likely due to the higher water temperatures on the Atlantic coast: Vidal et al. (2001) convincingly demonstrated that WSSV does not kill *P. vannamei* when water temperatures are above 32 °C.

On the Atlantic and the Pacific coast, different biosecurity measures have been applied to control the disease. On the Atlantic coast closing the life cycle and stocking with WSSV PCR negative seed have helped maintain the region free of the disease. However the main reason of the absence of WSSV fatalities is most likely the high water temperatures on the Atlantic coast, since the same measures coupled with lower stocking densities did not effectively control the epidemic on the Pacific coast.

Selective breeding is an attractive option for long term disease control (Gjedrem, 1983; Chevassus and Dorson, 1990; Fjalestad et al., 1993; Bedier et al., 1998; Roch, 1999; Bachere, 2000; Gjedrem, 2000) specially when no treatment is available and all attempts to interfere with the unrestrained occurrence and spread of the disease have failed. Several studies have estimated genetic variation for disease resistance in fish (Gjedrem et al., 1991; Beacham and Evelyn, 1992; Bailey et al., 1993; Gjedrem and Gjøen, 1995; Gjøen et al., 1997; Henryon et al., 2002). There are also a few studies in shrimp (Fjalestad et al., 1999; Argue et al., 2002), but none of these studies address resistance to WSSV.

In any selective breeding program it is important to know the magnitude of genetic correlations among important traits in order to optimize the selection work and control possible correlated responses. In *P. vannamei* the genetic correlation between resistance to Taura Syndrome virus (TSV) and growth was found to be unfavorable (Fjalestad et al., 1999; Argue et al., 2002) whereas Gitterle et al. (2005) reported a favorable genetic correlation between growth rate and pond survival.

In 1997 CENIACUA, in collaboration with AKVAFORSK, initiated a breeding program for *P. vannamei* in Colombia to improve growth rate and pond survival using a between and within family selection procedure. Initially the most important disease was the Taura syndrome, but high levels of resistance were rapidly obtained in the domesticated populations through individual selection. However, the focus of disease resistance breeding shifted to resistance to WSSV due to the devastation of the shrimp industry on the Pacific Coast of Colombia after the arrival of White Spot Syndrome Virus (WSSV) in Colombia in 1999.

This paper presents results from the selective breeding program for *P. vannamei* in Colombia in terms of genetic variation for harvest weight and resistance in juveniles to WSSV and their genetic correlation.

2. Materials and methods

2.1. Genetic material and production of families

The data were obtained from animals of the two genetic lines from the on-going programme that was