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Effect of *Baculovirus penaei* on Growth and Survival of Experimentally Infected Postlarvae of the Pacific White Shrimp, *Penaeus vannamei*

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Abstract

In a series of experiments conducted to investigate age and size-dependent effects of the baculovirus BP on postlarvae of the Pacific white shrimp, *Penaeus vannamei*, six groups of specific pathogen-free shrimp of different ages (mysis 2–3 through PL 25) were exposed to the virus and cultured for 15 to 21 days. All BP-exposed groups of early postlarvae (PL 9 or younger) became heavily infected within 2–5 days of initial exposure to the virus, and some of those groups experienced high mortalities compared to the noninfected controls. Postlarvae that survived the infection had highly variable and significantly reduced growth, as determined by dry weight, compared to controls. Exposure of older postlarvae to BP produced a high prevalence of infection but with little effect on either survival or growth. One group of shrimp exposed to BP at PL 9 was cultured for 49 days. Postlarvae that survived the infection were significantly smaller than the noninfected controls for the first 4 weeks following exposure to the virus; however, the effect of BP on long-term growth of infected postlarvae appeared minimal. To determine the effect of BP on nutritionally stressed shrimp, groups of noninfected and previously infected postlarvae (PL 13–14) of similar size were deprived of food for 10 days. Less than 2% of the infected postlarvae survived the 10-day starvation period compared to 52% survival of the noninfected postlarvae.

Keywords: baculovirus, BP, *Penaeus vannamei*, postlarval shrimp, growth and survival

Introduction

The virus commonly known as BP, originally discovered, described, and named *Baculovirus penaei* by Couch (1974) and recently proposed as PvSNPV by Bonami et al. (1994), occurs in both wild and cultured penaeid shrimp throughout the Americas, including Hawaii. BP has been reported from 14 species of *Penaeus* (see Lightner and Redman, 1991; Lightner, University of Arizona, pers. comm.) and is known to cause serious epizootics with high mortality of both larval and postlarval stages in several species, including *P. vannamei* (see Overstreet et al., 1988; LeBlanc and Overstreet, 1990). In aquaculture operations, the virus causes economic losses from mass mortality in the hatchery phase as well as legal restrictions on transport of infected postlarvae for use in stocking grow-out ponds.

Mortality of penaeids from baculoviruses is not restricted to BP. The nonoccluded virus that causes baculoviral midgut gland necrosis (BMN) causes mortality of *Penaeus japonicus* experimentally infected as mysis or Day 2 postlarvae (PL 2), but not in that shrimp when infected as PL 9 (Sano et al., 1985). The widespread monodon baculovirus (MBV) infects all stages of *P. monodon* and causes mortalities of that species in juveniles and senescent adults (Lightner, 1988).

BP infects the epithelium of the hepatopancreatic tubules (HP) and anterior midgut; it produces polyhedra, or tetrahedral occlusion bodies, in the nuclei of infected cells. In the later stages of infection, the hypertrophied nucleus filled with free virons and virus containing polyhedra ruptures from the infected cells and the polyhedra pass through the intestine with the feces (Couch, 1991, and Fig. 5 therein). The amount of tissue destruction associated with the release of polyhedra depends on several factors, among which are the age when infected, size of infected shrimp, and severity of infection. Consequently, the loss of significant amounts of hepatopancreatic epithelium at critical points in larval or postlarval development results in a variety of adverse effects on the host (Couch, 1981).

The susceptibility of *P. vannamei* to BP infection and the effects of the infection on host survival are known to be age-dependent. Infections occurring during early larval development are often acute, with associated mortalities approaching 100% within 3 to 4 days after exposure to the virus (Overstreet et al., 1988). LeBlanc and Overstreet (1990) found that the virus has little effect on survival of postlarvae older than 63 days, whereas younger postlarvae occasionally experience high mortalities. Lightner (1983) reported that BP infections among postlarvae and juveniles are subacute or chronic and may result in reduced feeding and growth rates of the hosts. Specific information about the effects of BP on growth and survival of postlarval and juvenile *P. vannamei* is limited. The chronic or subacute effects of the virus have not been adequately documented, and the effects of BP on nutritionally stressed shrimp are unknown. The purpose of this study is to provide such information on both the acute and the subacute effects of BP on postlarval and juvenile *P. vannamei,* the stages that are typically stocked into nursery or grow-out ponds. That information is essential to a complete understanding of how BP acts as a disease agent in penaeid shrimp; consequently, it should be useful in management of aquaculture operations.

Materials and Methods

Age and Size-Dependent Effects of BP on Growth and Survival

The study consists of a series of 6 experiments in which groups of specific pathogen-free (SPF) *P. vannamei* (see Wyban, 1992) of different ages (mysis 2–3 through PL 25) were experimentally infected with BP. The source of shrimp, age at infection, stocking densities, date and duration of experiment after initial viral exposure, temperature and salinity of cultures, and presence of infectious hypodermal and hematopoietic necrosis virus (IHHNV) in BP-exposed shrimp are listed in Table 1. Spawns of shrimp originating as SPF stocks from BP-free facilities in Hawaii were routinely monitored for BP before supplying other facilities from which we obtained shrimp. A subsample of 20 shrimp from each of the 6 experimental groups was initially examined for the presence of BP polyhedra in the HP following the diagnostic procedures for fresh shrimp described by Overstreet et al. (1988). Lack of BP in any of the control groups confirms the BP-free status of our experimental stocks. Shrimp used in most of these experiments were also checked for the presence of IHHNV using a gene probe (Lightner et al., 1992) developed by researchers at the University of Arizona. Because this probe was not available during the earlier phases of this study, data on the IHHNV status for all experimental groups are incomplete.

Table 1. Culture Parameters for BP-Exposed and Nonexposed "Control" Treatments for Six Experimental Groups of *Penaeus vannamei*

Stage at infection and source of shrimp	Date and duration of experiment (days postinfection)	IHHNV status	Initial stocking density (shrimp per liter)	Salinity (ppt)	Temperature (°C)
Mysis 2–3 (SC)	7–92 (15)	Negative	20.0	30 ± 1	28 ± 1
PL8–9 (H)	7–92 (21)	Negative	8.0	25 ± 1	27 ± 1
PL B–9 (SC)	7–91 (21)	N/A	8.0	25 ± 1	27 ± 1
PL 8–9 (T)	3-93 (21°)	Negative	8.0	25 ± 1	27 ± 1
PL 14–16 (H)	4–91 (21)	N/A	5.0	20 ± 1	27 ± 1
PL23–25 (T)	4–93 (21)	Negative	2.5	20–15[b]	27 ± 1

Note: Source of larvae: SC, South Carolina; H, Hawaii; T, Texas. N/A, data not available.

a. At Day 21, control and BP-exposed cultures were restocked at a density of 4.0 postlarvae per liter and maintained for a total of 49 days postinfection.

b. Salinity was gradually reduced during the course of the experiment.

The shrimp used in each experiment were originally obtained as nauplii from one of three sources: (1) The Oceanic Institute, Honolulu, Hawaii, (2) Harlingen Shrimp Farm, Los Fresnos, Texas, and (3) Waddell Mariculture Center, Bluffton, South Carolina. Nauplii were placed in 200-liter rectangular glass aquaria containing 150 liters of 30-ppt salt water produced from hw-Marinemix (Hawaiian Marine Imports, Houston, Texas) and deionized water. Larvae were reared to the desired age at 27 ± 1°C on a diet consisting of the diatom *Chaetoceros gracilis* during protozoeal stages 1–3 and brine shrimp during protozoeal stage 3–postlarvae.

With the exception of the experiment with PL 14–16, all exposures were conducted in two 200-liter glass aquaria containing 150 liters of salt water. Shrimp in one of those

aquaria were exposed to BP by introducing 8 mg per liter of homogenized BP-infected postlarvae directly into the culture. The second aquarium, which served as the source of the negative control group of shrimp, was administered an identical amount of homogenized BP-free shrimp tissue. An identical second dose of BP-infected or uninfected tissue was introduced 24 hr after the initial dose. The strain of BP used in this study was originally collected in Ecuador (see Overstreet et al., 1988) from wild broodstock and pond-reared juveniles of *P. vannamei* and then passed through numerous lots of hatchery-spawned larval *P. vannamei* experimentally infected at the Gulf Coast Research Laboratory. Approximately 36 hr after the initial introduction of viral material, 50 to 100 BP exposed and corresponding negative control shrimp were removed from the 200-liter cultures, counted, and placed in separate 19-liter glass aquaria containing 12 liters of water from the original culture. These aquaria were maintained under conditions identical to the primary cultures and were used to estimate survival in the original cultures at the termination of the experiment. The experiment with the PL 14–16 group was conducted in a series of six 38-liter glass aquaria, each containing 20 liters of salt water. Three replicate aquaria were administered BP-infected tissue, and three others served as negative controls. The method and quantity of tissues administered to both the exposed and the control aquaria were identical to those used for the 200-liter cultures. Survival was determined at the end of the experiment by counting the number of shrimp remaining in each of the six aquaria. All cultures were fed newly hatched brine shrimp and Zeigler pellets (Zeigler Bros., Gardners, Pennsylvania) *ad libitum.* Temperature and salinity of all cultures were routinely monitored, and excess food and feces were siphoned from the cultures daily. Water was never exchanged; it passed through biological sponge filters and received constant aeration. The culture tanks were monitored daily for mortality, and dead shrimp were removed.

Samples of 15–20 individuals were assessed for BP infection from the 200-liter BP-exposed and control cultures several times during and at the termination of each experiment. Prevalence of infection among the PL 14–16 group was determined only at the termination of the experiment. Individual shrimp were examined for the presence of viral polyhedra following the diagnostic procedures described by Overstreet et al. (1988). At the termination of each experiment, each individual from a sample of 25–30 shrimp from both the exposed and control cultures was measured (total length, TL) and weighed wet. Those individual shrimp were then dried at 60°C for 48 hr and reweighed to obtain their dry weight.

Long-Term Effects of BP on Growth

A portion of one of the PL 8–9 groups of postlarvae, the one obtained from Texas, was maintained in culture for 49 days after initial exposure (postinfection, p.i.) to BP. Samples each consisting of a minimum of 30 individuals were collected from both the exposed and the control cultures on Days 3, 7, 14, 21, 28, 35, and 49 p.i. Total length, as well as wet and dry weight, was determined for individual shrimp in each sample. Prevalence of infection in a sample of 20 shrimp from both the BP-exposed and the control tanks was monitored daily for the first 3 days after introduction of the virus and then twice a week until termination of the experiment. Because of high mortality during the first week following introduction of the virus in the BP-exposed culture, stocking densities in both the exposed and control groups were readjusted to 4.0 shrimp per liter at Day 21 p.i. Routine removal of

shrimp for weight determinations and BP diagnosis resulted in an average reduction of the stocking densities from 4.0 to 0.5 shrimp per liter by Day 49 p.i. Salinity of the water for both the exposed and the control cultures was also gradually reduced by the addition of deionized water from 25 ppt to 15 ppt between Days 14 and 35 p.i.

Effect of BP on Survival of Nutritionally Stressed Postlarvae

Upon termination of the short-term growth study using mysis 2–3 from South Carolina, we determined the prevalence of infection in an isolated group of postlarvae (PL 13–14) within a standardized size range of 8–10 mm TL (estimated to be 0.3 to 0.5 mg dry weight per postlarva) from both the control and the infected cultures. Based upon examination of the entire HP from each of 20 individuals from each group, we found the negative control group to be free of BP polyhedra, while the infected group exhibited a 90% prevalence of infection. Subsequently, postlarvae from control and infected groups were placed into plastic trays with 18 compartments, one postlarva per compartment. Three trays were stocked with the previously infected postlarvae and three trays with noninfected control postlarvae. The trays were placed in a constant temperature incubator and maintained for 10 days at 27°C and 25.0 ± 0.5 ppt under constant light. During this 10-day period, postlarvae in both the control and the infected groups were not fed. Trays were checked daily for the presence of dead shrimp, at which time 50% of the water in each compartment was exchanged.

Analysis of Data

Size of negative control and BP-exposed shrimp during growth studies was quantified as the mean and standard deviation of the dry weights obtained from subsamples of each treatment group. Mean and standard deviation were also determined for survival data collected during the starvation experiment. Size differences between control and BP-exposed treatments in growth experiments and differences in survival during the starvation experiments were tested for significance using the Student t test. Differences in survival between control and BP-exposed treatments in growth experiments were tested for significance using the χ^2 statistic.

Results

Age and Size-Dependent Effects of BP on Growth and Survival

Significant differences were observed in the short-term growth of some of the six groups of postlarval shrimp used in this phase of the study (Table 2). Significantly ($\alpha = 0.05$) lower final mean weights were observed for BP-exposed groups in comparison to control treatments when infections were initiated at either mysis 2–3 or PL 8–9. For the group of shrimp infected at PL 14–16, two replicates had slightly higher final mean weights compared to controls, but the mean weight in a third replicate was lower than the means for all control replicates. The group of shrimp exposed to BP at PL 23–25 had slightly higher final weights compared to the control treatment. The differences, however, in growth observed between BP-exposed and control treatments in neither the PL 14–16 nor PL 23–25 experimental groups were significant. Highly variable growth in postlarvae, as exemplified by the

greater standard deviations relative to the mean of infected versus control treatment groups, was especially evident in all three groups of shrimp from different sources infected with BP at PL 8–9.

Table 2. Size/Age-Dependent Effects of BP on the Growth and Survival of Postlarval (PL) *Penaeus vannamei*

Stage at infection and source of shrimp	Preinfection dry weight (average mg per PL)	Treatment[a]	Final dry weight (average mg per PL)	Maximum prevalence of BP (%)	Final prevalence of BP (%)	Survival (%)
Mysis 2–3 (SC)	0.06 ± 0.01	I[b]	0.33 ± 0.08	100	70	71[c]
		U	0.79 ± 0.54	0	0	98
PL 8–9(H)	0.20 ± 0.09	I[b]	1.40 ± 1.74	100	78	89
		U	3.32 ± 2.37	0	0	95
PL 8–9 (SC)	0.18 ± 0.05	I[b]	2.75 ± 3.04	95	80	95
		U	4.67 ± 2.79	0	0	98
PL 8–9 (T)	0.20 ± 0.06	I[b]	2.61 ± 3.03	100	75	66[c]
		U	5.80 ± 3.52	0	0	100
PL 14–16 (H)	0.64 ± 0.23	I-1	22.96 ± 10.25	N/A	28	96
		I-2	22.12 ± 11.71	N/A	36	97
		1-3	16.43 ± 6.65	N/A	32	94
		U-1	20.56 ± 8.56	N/A	0	98
		U-2	19.76 ± 6.50	N/A	0	94
		U-3	20.04 ± 6.75	N/A	0	95
PL 23–25 (T)	2.51 ± 1.46	I	31.25 ± 23.54	80	49	94
		U	28.90 ± 17.36	0	0	100

Note: Mean weights and standard deviations (SD) were determined from a sample of 25 or more individuals, and the source of shrimp was SC, South Carolina; H, Hawaii; T, Texas.

a. Treatment: I, infected; U, uninfected. N/A, data not available.

b. Denotes significant difference between paired treatments by Student's *t* test at $\alpha = 0.05$.

c. Denotes significant difference between paired treatments by $\chi2$ statistic at $\alpha = 0.05$.

Progression of the viral infection, determined by the presence of viral polyhedra, was monitored during each experiment, with the exception of that of the PL 14–16 group. In those experiments polyhedra appeared 18 to 24 hr after initial introduction of viral material, reached a maximum prevalence of infection 3 to 7 days p.i., and then gradually decreased in prevalence through the remainder of the culture period.

BP was also associated with lower survival in infected shrimp than in the controls in several experiments. Significantly lower survival ($\alpha = 0.05$) compared to controls was observed in the group infected at mysis 2–3 and one of the groups infected at PL 8–9. Massive mortality in those two groups was evident by the accumulation of dead shrimp 4 to 7 days p.i.; only an occasional dead shrimp was observed after that period.

Long-Term Effects of BP on Growth

Shrimp in the BP-exposed treatment group were noticeably smaller than those in the control group by Day 3 p.i. (Fig. 1). The difference became significant ($\alpha = 0.05$) by Day 7 and

remained so through Day 28 of the study. Standard deviation of the mean weight for the infected samples during that period was higher than the corresponding controls, reflecting the highly variable growth of infected individuals. An analysis of size class distribution during the course of the experiment (Fig. 2) demonstrates that the BP-exposed treatment group became negatively skewed compared to the control by Day 7 p.i. because of the proportionally higher number of small individuals. By Days 35 and 49 p.i., differences in size between BP-exposed and control shrimp were no longer significant.

Viral polyhedra first appeared 24 hr after initial introduction of the virus and reached 100% prevalence of infection by 48 hr p.i. By day 21, prevalence of shrimp with polyhedra fell to 75% and steadily decreased to 6% by Day 49. At Day 35 p.i. prevalence of infection by size class of shrimp was determined (Fig. 3), with the highest value (85%) in the smallest weight group. A substantially smaller size of the unmeasured HP of infected individuals compared to that of uninfected individuals of the same size was common in most individuals examined between Days 7 and 28 p.i.
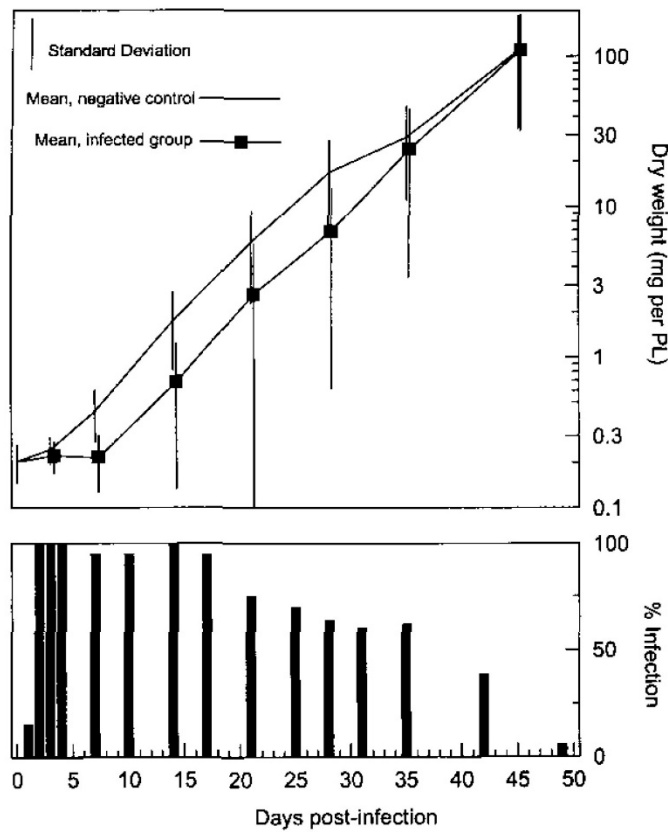

Figure 1. Effect of BP on growth rate of postlarval *Penaeus vannamei* over a 49-day culture period. The Y-axis for dry weight is presented as a logarithmic scale.
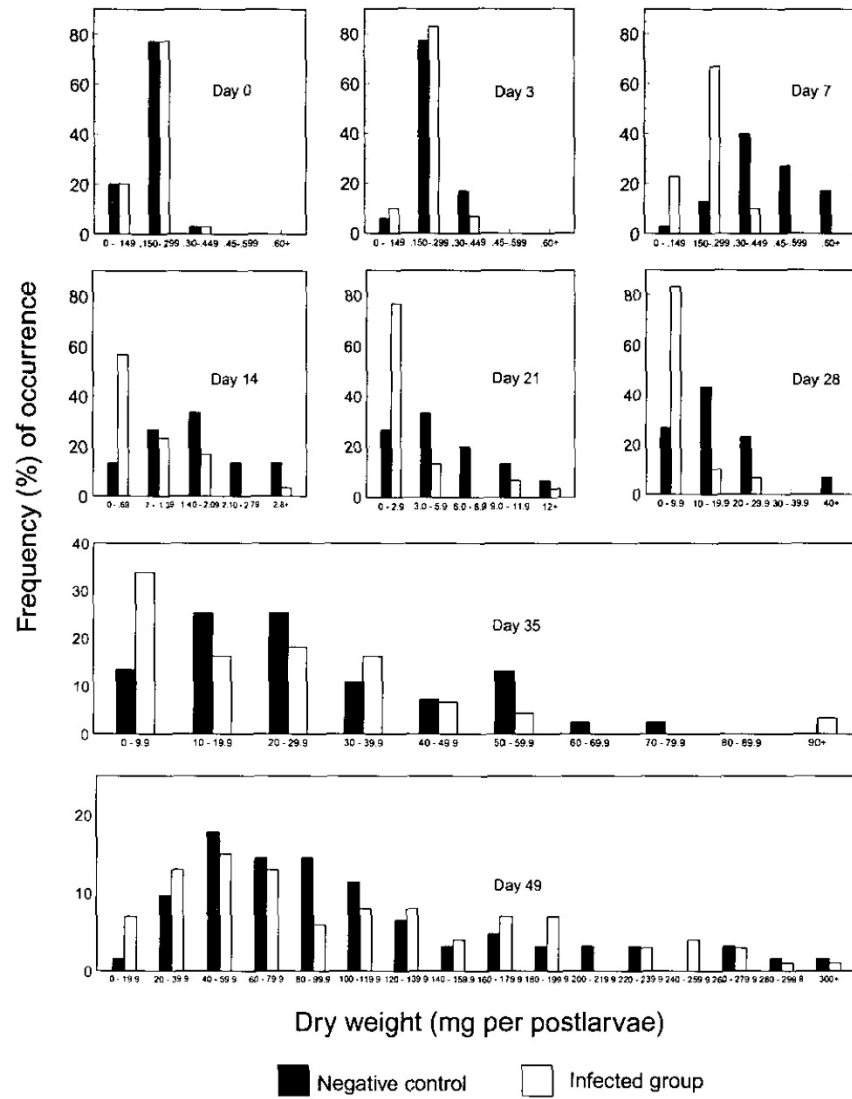
Figure 2. Weight-frequency distribution of BP-exposed and nonexposed (control) post-larvae at 0 (preinfection), 3, 7, 14, 21, 28, 35, and 49 days postexposure. Samples taken between Day 0 and Day 28 consisted of 30 BP-exposed and 30 nonexposed shrimp, Day 35 consisted of 92 BP-exposed and 83 nonexposed shrimp, and Day 49 consisted of 98 BP-exposed and 82 nonexposed shrimp.
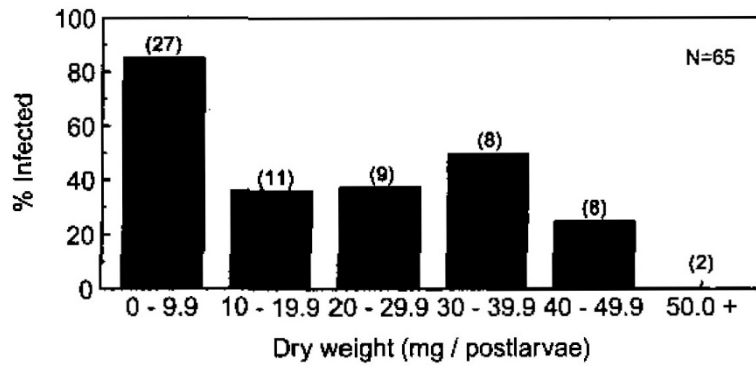
Figure 3. Prevalence of BP-infected larvae by weight class 35 days postexposure. *N*, total number of shrimp examined. The number in parentheses represents the actual number of shrimp in each weight class.

Effect of BP on Survival of Nutritionally Stressed Postlarvae

The survival of starved BP-infected compared with starved uninfected postlarvae over a 10-day period was significantly lower ($\alpha = 0.05$) by the second day of starvation and remained significantly lower through the duration of that period (Fig. 4). From Days 3 through 6 of starvation, the mean survival of the negative control group remained relatively constant (92–94%), while the survival of the infected group fell rapidly from 81 to 36%. By Day 10 of starvation, only 1 postlarva survived of the original 54 stocked, compared to 28 of the 54 stocked as the control group.
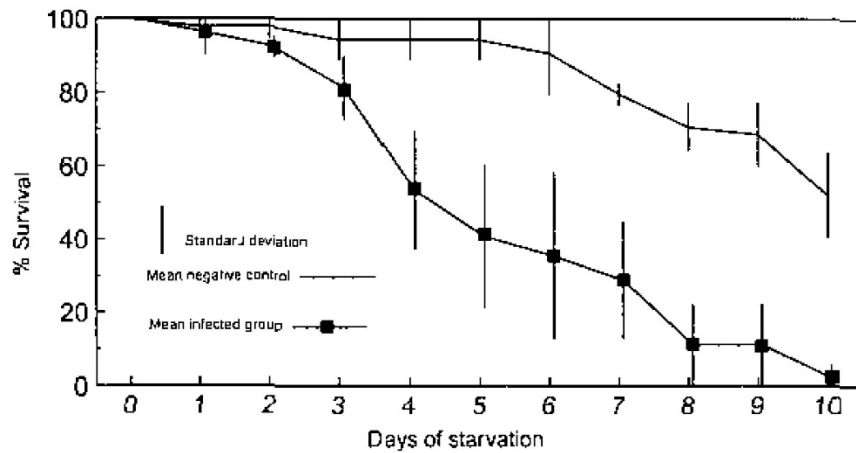

Figure 4. Survival of BP-infected and noninfected postlarvae of *Penaeus vannamei* during a 10-day starvation period.

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Discussion

Results of this study corroborate the findings of previous studies that the pathogenic effects of BP are somewhat dependent on the age of *P. vannamei* when the shrimp are initially infected, and those results provide additional details of this relationship. Overstreet et al. (1988) reported that the prevalence of infection and mortality of shrimp experimentally infected with BP at the protozoeal or mysis stages approached 100%, whereas older shrimp were more difficult to infect. LeBlanc and Overstreet (1990) experimentally infected seven groups of *P. vennamei* at ages ranging from 3 to 454 days after reaching postlarvae. Three-day-old postlarvae (PL 3) became 100% infected within 9 days after exposure to the virus, and those shrimp experienced 100% mortality by 14 days p.i. Older postlarvae (PL 39) exposed to BP developed a 30% prevalence of infection and did not experience the extensive mortality observed in the younger group. Based on those data and our preliminary observations, we designed experiments to investigate infections during the first few months of postlarval development. Since previously published studies were restricted to the pathogenic effects of BP in terms of prevalence of infection, extent of polyhedra in the individual HP, and occurrence of significant mortality in cultured populations, we stressed the effects on shrimp growth immediately following BP infection, long-term growth of infected postlarvae, and depletion of energy reserves.

Pathogenicity of BP in *P. vannamei* appears to undergo a change from the host-age range of mysis 2–3 to PL 23–25. This trend is not readily evident from data on prevalence of infection or mortality alone, but it is best demonstrated by data on growth. By combining our observations with those of previous studies (Overstreet et al. 1988; LeBlanc and Overstreet, 1990), we noticed a distinct age-dependent pattern of disease. The effect of BP on larvae and early postlarvae is often acute, and it frequently, but not always, concludes with mortality. Should postlarvae become infected with BP at approximately PL 8–9, the age at which they are commonly stocked into nursery or grow-out ponds, the response by the shrimp to the virus is primarily subacute, resulting in slow and highly variable growth immediately following infection. Exposure of older postlarvae to BP may produce a high prevalence of infection, associated with little recognizable effect on either survival or growth rates. Juveniles and subadults are less susceptible to infection, and they may become completely resistant to infection. This pattern of pathogenicity is by no means invariable, and factors such as culture conditions, virulence of virus, and host nutritional condition at the time of infection may have considerable influence on the pattern of disease. For example, LeBlanc and Overstreet (1990) reported 100% mortality of a 63-day-old group of postlarvae 16 days after exposure to BP, although the maximum prevalence of infection recorded was only 42% at 14 days.

The overall detrimental effect of BP on long-term growth of postlarvae infected at PL 8–9 appears to be minimal. Infected postlarvae showed little or no growth for a short period of time immediately following exposure to the virus, and moderate mortality (estimated to be about 25%) occurred from 5 to 7 days following exposure to the virus. Of particular interest is the substantial difference in weight-frequency of BP-exposed and control treatment groups by Day 7. The selective absence of large individuals among the BP-exposed group suggests that the virus is initially most pathogenic to faster-growing individuals.

We have made similar unpublished observations for protozoeae of *P. vannamei* exposed to BP. After 7 days p.i., growth observed in the infected culture group closely paralleled and eventually equaled that of the uninfected culture group; still, growth within the infected group remained highly variable, as indicated by the standard deviations. Some variability, as indicated by the PL 8–9 tests, may relate to source of larvae, date when tested, and probably other factors.

The significant reduction in prevalence of viral infection that we observed from 21 to 49 days p.i. corroborates similar findings reported by LeBlanc and Overstreet (1990). The reduction in prevalence of infection with time in our study generally corresponded with the appearance by Day 35 of a few large, rapidly growing individuals in the BP-exposed cultures. Weight-frequency analysis of the prevalence of infection in the exposed group at Day 35 showed that the largest individuals had the lowest prevalence of infection. Those data support our unpublished observations that once BP-infected shrimp have lost or significantly reduced the extent of their infection, they have the potential for accelerated growth.

Based on our results we suggest that BP can substantially reduce energy reserves in postlarval *P. vannamei* and that reduction results in high mortality of infected individuals in response to nutritional stress. The HP, the primary organ infected by BP, is the major site of lipid storage in decapods (Gibson and Barker, 1979). O'Leary and Matthews (1989) reported that the highest level of triacylglycerides, the class of lipids utilized primarily as energy reserves, in *P. monodon* occurs in the HP. Vogt (1992) observed that in advanced stages of a different baculovirus infection (MBV), *P. monodon* exhibited reduced lipid reserves. He speculated that lipids in the HP were being utilized to supply energy for viral replication. We have also observed a similar reduction in the number and size of lipid droplets in the HP cells of BP-infected larvae and postlarvae of *P. vannamei* immediately following exposure to the virus. The association between lipid reserves and viral replication may be related to our observation that among larvae and early postlarvae, the larger or more rapidly growing individuals, those which are likely to have substantial energy reserves, develop a patent infection faster and are initially more susceptible to the harmful effects of the virus than are slower-growing individuals. However, once the infection is established and lipid reserves are depleted, small shrimp are most susceptible to the harmful effects of the virus, especially under conditions of nutritional stress.

Reduction in the size of the HP in infected postlarvae compared to that in uninfected individuals from the same size group probably resulted from the destruction of infected HP cells as nuclei containing polyhedra are released into the lumen of the midgut. We have made similar observations for protozoea and mysis stages infected with BP. The loss of hepatopancreatic tissue further reduces the capacity of the HP to store or utilize energy reserves and probably contributes to the high mortality observed among nutritionally stressed postlarvae.

The age-dependent pathogenic response to BP observed in this and previous studies may also be related to the ontogeny of the HP in penaeid shrimps. Lovett and Felder (1989) reported the period from PL 1 to PL 10 as a "critical" time during which high rates of mortality are often observed in cultured penaeid shrimps. During this period, the entire digestive system is undergoing extensive morphogenesis, and the ratio of HP to body weight is

extremely low. In cultured *Penaeus setiferus*, there is no significant change in the volume of the HP from mysis 2 through PL 4, and the rate of increase in the HP volume does not equal that of the body until about PL 10, when tubule ramification of the HP becomes significant. We noticed a similar pattern of HP development in *P. vannamei* (see Overstreet et al., 1988). The loss of a significant portion of the HP resulting from viral infection during this critical period of development may account for much of the acute pathogenicity observed among larval and early postlarval shrimp. The relationship between development of the HP in larval shrimp and acute effects of BP was first proposed by Couch (1981). Infections occurring after this period may be less acute because of the accelerated growth of the HP and the ability of that organ to quickly replace virally damaged cells. Age-dependent growth data from this study generally support this hypothesis.

References

Bonami, J. R., Bruce, L. D., Poulos, B. T., and Lightner, D. V. 1994. Partial characterization and cloning of the genome of PvSNPV (BP-type virus) pathogenic for *Penaeus vannamei*. Submitted for publication.

Couch, J. A. 1974. An epizootic nuclear polyhedrosis virus of pink shrimp: Ultrastructure, prevalence, and enhancement. *J. Invertebr. Pathol.* 24, 311–331.

Couch, J. A. 1981. Viral diseases of invertebrates other than insects. *In* "Pathogenesis of Invertebrate Microbial Diseases" (E. W. Davidson, Ed.), pp. 127–160. Allenheld, Osmum Publishers, Totowa, NJ.

Couch, J. A. 1991. Baculoviridae: Nuclear polyhedrosis viruses. 2. Nuclear polyhedrosis viruses of invertebrates other than insects. *In* "Atlas of Invertebrate Viruses" (J. R. Adams and J. R. Bonami, Eds.), pp. 205–225. CRC Press, Boca Raton, FL.

Gibson, R., and Barker, P. L. 1979. The decapod hepatopancreas. *Oceanogr. Mar. Biol.* 17, 285–346.

LeBlanc, B. D., and Overstreet, R. M. 1990. Prevalence of *Baculovirus penaei* in experimentally infected white shrimp *(Penaeus vannamei)* relative to age. *Aquaculture* 87, 237–242.

Lightner, D. V. 1983. Diseases of cultured penaeid shrimp. *In* "CRC Handbook of Mariculture," Vol. 1, "Crustacean Aquaculture" (J. P. McVey, Ed.), pp. 289–320. CRC Press, Boca Raton, FL.

Lightner, D. V. 1988. MBV virus disease of penaeid shrimp. *In* "Disease Diagnosis and Control in North American Marine Aquaculture," 2nd ed. (C. J. Sindermann and D. V. Lightner, Eds.), pp. 22–25. Elsevier, Amsterdam.

Lightner, D. V., and Redman, R. M. 1991. Host, geographic range, and diagnostic procedures for the penaeid virus diseases of concern to shrimp culturists in the Americas. *In* "Frontiers in Shrimp Research" (P. DeLoach, W. J. Dougherty, and M. A. Davidson, Eds.), pp. 173–196. Elsevier, Amsterdam.

Lightner, D. V., Poulos, B. T., Bruce, L., Redman, R. M., Mari, J., and Bonami, J. R. 1992. New developments in penaeid virology: Applications of biotechnology in research and disease diagnosis

for shrimp viruses of concern in the Americas. *In* "Diseases of Cultured Penaeid Shrimp in Asia and the United States" (W. Fulks and K. L. Main, Eds.), pp. 233–253. The Oceanic Institute, Honolulu, HI.

Lovett, D. L., and Felder, D. L. 1989. Ontogeny of gut morphology in the white shrimp, *Penaeus setiferus* (Decapoda, Penaeidae). *J. Morphol.* 201, 253–259.

O'Leary, C. D., and Matthews, A. D. 1989. Lipid class distribution and fatty acid composition of wild and farmed prawn, *Penaeus monodon* (Fabricius). *Aquaculture* 89, 65–81.

Overstreet, R. M., Stuck, K. C., Krol, R. A., and Hawkins, W. E. 1988. Experimental infections with *Baculovirus penaei* in the white shrimp, *Penaeus vannamei* (Crustacea: Decapoda), as a bioassay. *J. World Aquaculture* Soc. 19, 175–187.

Sano, T., Nishimura, T., Fukuda, H., Hayashida, T., and Momoyama, K. 1985. Baculoviral infectivity trials on kuruma shrimp larvae, *Penaeus japonicus*, of different ages. *In* "Fish and Shellfish Pathology" (A. E. Ellis, Ed.), pp. 397–403. Academic Press, New York.

Vogt, G. 1992. Transformation of the anterior midgut and hepatopancreas cells by monodon baculovirus (MBV) in *Penaeus monodon* postlarvae. *Aquaculture* 107, 239–248.

Wyban, J. A. 1992. Selective breeding of specific pathogen-free (SPF) shrimp for high health and increased growth. *In* "Diseases of Cultured Penaeid Shrimp in Asia and the United States" (W. Fulks and K. L. Main, Eds.), pp. 257–268. The Oceanic Institute, Honolulu, HI.