Genetic Effects for Response to Live Edwardsiella ictaluri, Killed E. ictaluri, and Stress in Juveniles from All Crosses Among USDA 103, USDA 102, and Norris Channel Catfish Ictalurus punctatus Strains

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Abstract.—Juveniles from all possible crosses among USDA 102, USDA 103, and Norris channel catfish Ictalurus punctatus strains were compared for: 1) survival and anti-Edwardsiella ictaluri antibody after exposure to live E. ictaluri bacterium (isolate S97-458); 2) antibody level after injection with formalin-killed E. ictaluri (S97-458); and 3) pre-stress, post-stress, and stress-recovery serum cortisol levels. Purebred and crossbred USDA 102 strain fish had higher survival (mean of five genetic groups = 87%) and lower anti-E. ictaluri antibody (mean optical density (OD) of five genetic groups = 0.167) 30 d after live E. ictaluri challenge than purebred Norris and USDA 103 strains and their crosses (means of four genetic groups = 60% survival and 0.210 OD antibody level). Significant general combining ability, line effects, and heterosis indicated that the USDA 102 strain contributed additive and dominance genetic effects for increased survival and lower antibody level after live E. ictaluri challenge. Antibody response to formalin-killed, intraperitoneally injected E. ictaluri was not different among genetic groups (overall mean = 0.198 OD). Serum cortisol was measured prior to (pre-stress), immediately after (post-stress), and 2 h after (stress-recovery) a standard stressor. Serum cortisol level was highest in post-stress fish (35.8 ng/mL), intermediate in stress-recovery fish (10.9 ng/mL), and lowest in pre-stress fish (6.5 ng/mL), but was not different among genetic groups within a stress time period. Results indicate that differences exist among genetic groups of channel catfish for survival and antibody production after live E. ictaluri challenge, but these differences were not related to antibody response to killed E. ictaluri or serum cortisol levels.

Enteric septicemia of catfish (ESC), caused by the bacterium Edwardsiella ictaluri, is a major disease problem for the farmed-raised catfish industry, and reducing the incidence of ESC will benefit catfish producers. Advances in antibiotic therapy, vaccine development, management strategies, and selective breeding have played roles in controlling diseases in other livestock species (Gogolin-Ewens et al. 1990; Noordhuizen and Welpelo 1996; Barrow 1997; Klasing 1998; Lamont 1998; Soller and Andersson 1998) and similar approaches may be useful for controlling ESC in farm-raised catfish.

Channel catfish strains and families within strains differ in mortality after exposure to Edwardsiella ictaluri (Wolters and John- son 1994, 1995) suggesting that genetic variation for ESC resistance exists and improvement of resistance through selective breeding may be possible. However, information on the genetic architecture (additive or dominance effects) needed to identify breeding strategies to improve ESC resistance is limited. In addition, little is known about the immunological and physiological basis for the differences in ESC resistance observed among catfish strains and families.

A negative correlation between percent survival and anti-E. ictaluri antibody level has been observed in E. ictaluri challenges
of different genetic groups of channel catfish (Wolters and Johnson 1994). The negative correlation between survival and antibody level may be due to a dose response (greater bacterial proliferation/increased antigen resulting in greater antibody production in susceptible groups) or it may be related to differences in antigen processing/presentation among resistant and susceptible groups. Understanding the basis for the negative correlation between survival and antibody level could be useful in developing breeding strategies to improve resistance to *E. ictaluri*.

Environmental stress has a detrimental effect on immune function in channel catfish (Wise et al. 1993; Ciembor et al. 1995) and differences in ESC resistance among genetic groups of catfish may be associated with differences in stress response among groups. If there are genetic differences in stress response, it may be possible to reduce the incidence of ESC by selecting for catfish with reduced stress response.

Improving ESC resistance of catfish through selective breeding will require a better understanding of the genetics of ESC resistance in catfish. The objectives of this study were to compare all possible crosses among USDA 102, USDA 103, and Norris strains of channel catfish and estimate genetic effects for: 1) survival and anti-*E. ictaluri* antibody production after exposure to live *E. ictaluri*; 2) antibody level after injection with killed *E. ictaluri*; and 3) pre-stress, post-stress, and stress-recovery cortisol levels.

**Materials and Methods**

**Fish and Matings**

All possible crosses were made among USDA 102, USDA 103, and Norris strains of channel catfish. The USDA 102 strain was developed through family selection for improved reproductive characteristics, growth, and disease resistance from fish obtained from the Uvalde National Fish Hatchery, Uvalde, Texas, USA. Norris strain fish were purchased as broodfish from Norris Fish Farm in Cash, Arkansas, USA.

In March, nine 0.04-ha ponds were each stocked with 15 male and 30 female catfish of the appropriate genetic group to produce offspring from the three purebred strains and six reciprocal F1 crossbred groups. Five milk cans were placed in each pond to provide spawning habitat and cans were checked for egg masses two times each week from mid-May through mid-June. Egg masses were brought into the hatchery and each full-sib family was reared in a separate 150-L fiberglass tank supplied with flow-through well water (26 C) and aeration. Fish were fed a 55% protein sinking trout feed (Ziegler Brothers, Inc., Gardner, Pennsylvania, USA) until 45 d post-hatch, fed a mixture of trout feed and 36% protein, 3-mm floating pellet (SF Services, Greenville, Mississippi, USA) from 45 to 70 d post-hatch, and the 36% diet after 70 d post-hatch. Fish densities were reduced to 200–300 fish per tank at 50 d post-hatch. At approximately 120 d post-hatch (range 110–131 d post-hatch), 43 full-sib families (4–5 families/genetic group) were randomly chosen for use in experiments.

**Live E. ictaluri Challenge**

The live *E. ictaluri* challenge was conducted in three replicate concrete raceways (7-m long × 2.1-m wide) supplied with aeration and flow-through well water (26 C) at a water depth of 30 cm. Each raceway contained 43 PVC, mesh baskets (Delta Net and Twine, Greenville, Mississippi, USA) with a mesh size (0.5 cm × 0.5 cm) large enough to allow water flow, but small enough to retain fish. One hundred and fifty juveniles from each of 43 full-sib families (4–5 families/genetic group) were used in the challenge. Groups of 50 fish from each family were weighed as a group and each
group of 50 fish was stocked into a separate basket in each raceway. After a 21-d acclimation period, water depth was reduced to 8–10 cm, water flow was discontinued, and sufficient BHI broth containing virulent E. ictaluri (isolate S97-458) was added to yield $1 \times 10^6$ bacteria/mL. The E. ictaluri isolate used in the challenge was isolated from catfish from a commercial farm experiencing an outbreak of ESC, was passed by injection into live fish and isolated from dying fish to assure virulence. Plate counts were used to estimate bacterial concentration in broth culture. Broth was poured along the length of the raceway and aeration was continued to insure dispersion and mixing of the bacteria. Fish were held in static water for 6 h and then flow was resumed and water depth was returned to 30 cm.

Fish were fed once daily to satiation starting the day after exposure. Number of mortalities was recorded daily through 30 d post-challenge, and cause of death was determined for random samples of moribund fish. Cause of death was determined by necropsy including inoculation of kidney tissue on brain-heart infusion agar for confirmation of the presence of E. ictaluri. An ELISA (Klesius 1993) was used to determine serum antibody levels to E. ictaluri exoantigen (reported as optical density) from three pre-challenge fish/family and 15 fish/family at 30 d post-challenge. OD readings from the ELISA have been shown to be linearly related to antibody levels under the conditions used (Klesius 1993). Conditions used for the ELISA have been described previously (Wolters et al. 1996). Reference control serum samples (four positive and four negative for anti E. ictaluri antibody) were included on each plate to account for inter-plate variation in the assay.

Killed E. ictaluri Injection

A subset of 37 of the 43 families used in the live challenge was used to determine antibody response to formalin-killed E. ictaluri (isolate S97-458). Fish were stocked into two replicate aquaria per family at 8–10 fish/aquaria and allowed to acclimate for 2 wk. E. ictaluri from the same isolate used in the live challenge was grown in BHI broth for 24 h at 25 C, killed by addition of 1% formalin, harvested by centrifugation at 2,500 rpm for 10 min, washed and harvested twice by centrifugation with 100 mL of phosphate buffered saline pH 7.2 (PBS), and resuspended in PBS. Bacterial concentration in the suspension was determined using a hemocytometer and microscope to count cells in diluted samples. The bacterial suspension was diluted to a concentration of approximately $1 \times 10^6$ cells/mL with filter sterilized PBS.

Fish were tranquilized, weighed as a group, and injected intraperitoneally with a 1-mL syringe equipped with a 20-ga needle to give a dose of $\sim 1 \times 10^6$ formalin-killed E. ictaluri cells/g of fish body weight. Injection volume was calculated based on mean weight of fish in each tank (all fish in a tank received the same injection volume). Syringes were inverted between fish to mix the cell suspension. Blood samples were collected 21 d after injection with killed E. ictaluri and serum anti-E. ictaluri antibody levels were determined by ELISA for 16 fish/family.

Stress and Cortisol Levels

Pre-stress, post-stress, and stress-recovery serum cortisol was measured for fish from 41 of the 43 families used in the live challenge. Fish from each family were stocked into two replicate aquaria (10–12 fish/aquarium) and allowed to acclimate for 2 wk. After the acclimation period, four fish/aquarium were quickly netted, immediately placed in water containing anesthetic (200-ppm MS-222), and blood was collected from the caudal vasculature with a 1-cc syringe equipped with a 20-ga needle. These samples represented pre-stress fish. Three days later the remaining fish in each aquaria were subjected to a low water stress which involved draining the water in the
tank and holding the fish in minimal water (approximately 2 cm) for 1 h (Wise et al. 1993). At the end of 1 h fish were netted, placed in anesthetic, and blood was collected from the caudal vasculature. Samples collected immediately after the 1-h stress represented post-stress fish. An additional 16–20 fish from each family were stocked into two replicate aquaria (8–10 fish/aquarium) and allowed to acclimate for 2 wk. After the acclimation period, fish were exposed to the same standard stress for 1 h, pre-stress water levels were re-established for a 2-h recovery period, and then fish were removed, tranquilized, and bled. These fish represented the stress-recovery group. All blood samples were placed on ice immediately after collection, centrifuged, and serum was collected and frozen at −80°C until cortisol assays were performed. Serum cortisol levels were determined for 12–16 fish/family for post-stress and stress recovery fish and four fish/family for pre-stress fish with a radioimmunoassay kit (Magic Cortisol Assay, Chiron Diagnostics Corp., Pittsburg, Pennsylvania, USA). Serum cortisol levels are reported as ng/mL. Cortisol assays and development of standard curves were according to the kit manufacturer’s instructions.

Data Analysis

Genetic group means for percent mortality and antibody level after live E. ictaluri challenge were compared by ANOVA using a complete block design that included raceway as a block effect, fixed effect of genetic group, and random effect of family within genetic group. Genetic group means for antibody level to formalin-killed E. ictaluri and pre-stress, post-stress and stress-recovery serum cortisol levels were compared by ANOVA using a model which included fixed effect of genetic group, random effect of family within genetic group, and random effect of replicate aquaria within family within genetic group. Mean square error for family within genetic group was used as the error term in tests of genetic group means. Family age and mean weight were included as covariates in the initial analysis of survival, antibody levels, and serum cortisol, but were not significant and not included in the final models.

Estimates of average, line and specific heterosis; general combining ability; line effects; maternal effects; and reciprocal effects were determined by forming contrasts among means of appropriate genetic groups according to the genetic model of Eisen et al. (1983). Mean square error for family within genetic group was used in testing significance of estimates of genetic effects. The Mixed procedure of SAS (SAS version 8, SAS Institute, Cary, North Carolina, USA) was used for ANOVA and estimation of genetic effects.

Pearson product-moment correlations (SAS version 8, SAS Institute, Cary, North Carolina, USA) among family means and among genetic group means were determined for survival after live E. ictaluri challenge, antibody level after live challenge, antibody level after injection with killed E. ictaluri, pre-stress cortisol, post-stress cortisol, and recovery-stress cortisol levels. Estimates of genetic effects, differences among genetic groups means, and correlations among traits were declared significant at $P < 0.05$. A macro written for Proc Mixed (Pdmix800, Arnold M. Saxton, University of Tennessee) was used to do multiple-range tests on genetic group means.

Results

Mean weight of genetic groups at live-challenge ranged from 6.8 g to 9.8 g and did not differ among genetic groups (Table 1). Two families did not have sufficient fish to be used in the stress test and six families did not have sufficient fish to be used in the killed E. ictaluri injection test.

Live E. ictaluri Challenge

Mean survival after live E. ictaluri challenge was different among genetic groups (range 51.1–92.8%, Table 1). Purebred
USDA 102 strain and the four F1 crosses involving the USDA 102 strain generally had better survival (mean of five genetic groups = 86.8%, range 79.0–92.8%) than purebred Norris, USDA 103, and their crosses (mean of four genetic groups = 60.1%, range 51.1–70.8%). USDA 102 strain had favorable general combining ability (12.8%), line heterosis (12.2%), and line effects (14.9%) for survival (Table 2). Specific heterosis was positive (15.5%) for crosses among USDA 103 and Norris strains, and USDA 103 maternal effects were negative (−10.3%) for survival.

Within each type of control serum sample (positive vs. positive, negative vs. negative) mean OD readings for anti-

\[ E. \text{ ictaluri} \]

antibody were not significantly different among plates, indicating that plate-to-plate variation in the assay was negligible. All pre-challenge fish tested were negative for anti-

\[ E. \text{ ictaluri} \]

antibody (OD < 0.07). Means for anti-

\[ E. \text{ ictaluri} \]

antibody levels were different among genetic groups (range 0.139–0.232 OD, Table 1). Purebred USDA 102 strain and the four F1 crosses involving the USDA 102 strain generally had lower antibody levels (mean of five genetic groups = 0.167 OD, range 0.139–0.192 OD) than purebred Norris, USDA 103 and their crosses (mean of four genetic groups = 0.211 OD, range 0.187–0.232 OD). General combining ability for antibody level was negative (−0.041 OD) for the USDA 102 strain and positive for the Norris strain (0.042 OD) (Table 2). Specific heterosis was positive (0.036 OD) for antibody level in crosses among USDA 103 and Norris strains and negative (0.030 OD) for antibody level in crosses among USDA 102 and USDA 103 strains. Correlations among survival and antibody level after live challenge were negative based on family means (\( r = -0.43, P = 0.009 \)) and genetic group means (\( r = -0.74, P = 0.02 \)).

**Killed E. ictaluri Challenge**

Mean antibody level after injection with killed \[ E. \text{ ictaluri} \] was not different among genetic groups (overall mean = 0.198, range of genetic groups = 0.180–0.211) (Table 1). Correlations among antibody response to killed \[ E. \text{ ictaluri} \] and live challenge survival or antibody level were not significant for either genetic group means or family means.

### Table 1. Least square means (± average standard errors) for live weight, survival after challenge with live \[ E. \text{ ictaluri} \], antibody level after challenge with live \[ E. \text{ ictaluri} \], antibody level after injection with formalin killed \[ E. \text{ ictaluri} \], and pre-, post-, and recovery-stress cortisol levels.

<table>
<thead>
<tr>
<th>Traits</th>
<th>Weight (g)</th>
<th>Live challenge survival (%)</th>
<th>Live challenge Ab level (OD)</th>
<th>Killed challenge Ab level (OD)</th>
<th>Pre-stress cortisol (ng/mL)</th>
<th>Post-stress cortisol (ng/mL)</th>
<th>Recovery-stress cortisol (ng/mL)</th>
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<tbody>
<tr>
<td>USDA 102 × USDA 102</td>
<td>7.9</td>
<td>89.7&lt;sup&gt;A&lt;/sup&gt;</td>
<td>0.169&lt;sup&gt;A&lt;/sup&gt;</td>
<td>0.180</td>
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<td>79.0&lt;sup&gt;A,B,C&lt;/sup&gt;</td>
<td>0.160&lt;sup&gt;A&lt;/sup&gt;</td>
<td>0.191</td>
<td>8.8</td>
<td>38.4</td>
<td>11.8</td>
</tr>
<tr>
<td>USDA 102 × Norris</td>
<td>8.3</td>
<td>83.9&lt;sup&gt;A,B,C&lt;/sup&gt;</td>
<td>0.176&lt;sup&gt;A&lt;/sup&gt;</td>
<td>0.194</td>
<td>7.6</td>
<td>33.6</td>
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<tr>
<td>USDA 103 × USDA 102</td>
<td>9.5</td>
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<td>0.139&lt;sup&gt;A&lt;/sup&gt;</td>
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<td>3.4</td>
<td>38.8</td>
<td>12.0</td>
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<td>Norris × USDA 102</td>
<td>9.8</td>
<td>88.7&lt;sup&gt;A&lt;/sup&gt;</td>
<td>0.199&lt;sup&gt;B&lt;/sup&gt;</td>
<td>0.210</td>
<td>5.9</td>
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<td>65.2&lt;sup&gt;C,D&lt;/sup&gt;</td>
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<td>11.5</td>
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<td>51.1&lt;sup&gt;D&lt;/sup&gt;</td>
<td>0.187&lt;sup&gt;C&lt;/sup&gt;</td>
<td>0.211</td>
<td>2.6</td>
<td>38.1</td>
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<td>Norris × USDA 103</td>
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<td>0.232&lt;sup&gt;D&lt;/sup&gt;</td>
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<td>37.5</td>
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<td>USDA 103 × Norris</td>
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<td>70.8&lt;sup&gt;B,C,D&lt;/sup&gt;</td>
<td>0.227&lt;sup&gt;C,D&lt;/sup&gt;</td>
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</tbody>
</table>

* Within a column, genetic group means with different superscript letters are different at \( P < 0.05 \).
Cortisol

Cortisol levels were lowest in pre-stress fish (overall mean = 6.5 ng/mL, range 2.6–10.5 ng/mL), highest in post-stress fish (overall mean = 35.8 ng/mL, range 32.6–38.8 ng/mL), and intermediate in stress-recovery fish (overall mean = 10.9 ng/mL, range 3.5–14.8 ng/mL). Serum cortisol was not different among genetic groups within each stress treatment. Correlations of pre-stress, post-stress, and stress-recovery serum cortisol levels with antibody level and survival after live *E. ictaluri* challenge were not significant for either genetic group means or family means. Genetic effects were not estimated for body weight, antibody level after injection with killed *E. ictaluri*, or for cortisol levels since genetic groups did not differ for these traits.

Discussion

The results of this study indicate that genetic differences exist among channel catfish strains and strain crosses for resistance to live *E. ictaluri*, the bacterium that causes enteric septicemia of catfish (ESC). Purebred and crossbred USDA 102 strain channel catfish generally had better survival following live *E. ictaluri* challenge than the other genetic groups tested. Previous studies have demonstrated that the USDA 102 strain had better resistance to *E. ictaluri* than other channel catfish strains (Wolters and Johnson 1994) and channel catfish × blue catfish hybrids (Bosworth et al., submitted companion paper). The positive general combining ability, line heterosis, and line effects for post-challenge survival indicated that the USDA 102 strain contributed favorable additive and dominance ge-
netic effects (Eisen et al. 1983) for survival. The results demonstrate differences exist among strains for ESC resistance and it may be possible to improve ESC resistance through identification and commercial use of superior strains. Favorable additive and dominance effects for ESC resistance in the USDA 102 strain indicate that using this strain as a purebred line or in crossing with other strains tested should result in improved resistance to ESC. However, other traits such growth rate, fillet yield, and reproductive success need to be considered in order to develop improved germplasm with a favorable composite phenotype for commercial use.

The negative correlation between survival and anti-*E. ictaluri* antibody we observed following the live challenge has been reported in other *E. ictaluri* challenges of channel catfish (Wolters and Johnson 1994). A similar negative correlation between antibody response and survival (high antibody response/low survival) has been reported in divergently selected mouse lines in challenges with certain intracellular bacterial pathogens (*Salmonella, Listeria* etc., reviewed by Mouton et al. 1988). Resistant mouse lines have lower antibody response in response to these pathogens and also to standard antigens such as sheep red blood cells. The differences among lines for antibody response appears to be related to differences in antigen processing and presentation by the macrophages (Mouton et al. 1984; Consales et al. 1990).

Our results (no differences among genetic groups for antibody response to killed *E. ictaluri* and lack of significant correlations between killed *E. ictaluri* antibody response and live *E. ictaluri* antibody response or survival) suggest that the negative antibody-survival correlation observed after live challenge was not due to differences in antigen processing/presentation among resistant and susceptible genetic groups. It is possible that the correlation between antibody level and survival simply reflects a dose response, i.e., susceptible fish have more bacteria and therefore produce more antibodies. Other studies have demonstrated a dose response between *E. ictaluri* antigen level and antibody response (Vinitnantharat and Plumb 1992; Terhune 1999). Our data indicate that selection for antibody response to killed *E. ictaluri* would not result in correlated improvement for resistance to live *E. ictaluri*. However, our results are based on a single concentration of killed *E. ictaluri* and antibody measured at one point in time. Further work on antibody response to both live and killed *E. ictaluri* or standard antigens could provide useful insights into immune response and resistance of channel catfish to *E. ictaluri*.

Serum cortisol levels we observed followed the expected pattern: lowest prior to stress, highest immediately after stress, and intermediate after recovery from stress. Pre-stress and post-stress serum cortisol levels we observed were similar to those reported for catfish by Davis et al. (1984, 1994). We found no differences in cortisol levels among the genetic groups tested indicating that the differences in post-challenge survival among genetic groups were not related to differences in stress response.

Selection for increased resistance to *E. ictaluri* through indirect selection for decreased stress response (measured as serum cortisol) does not appear to be a good strategy given the lack of difference among genetic groups for the standardized stress test used. Reports of genetic variation in stress response and correlated responses for disease resistance in other fish species are variable. Heritabilities for stress induced cortisol levels were negligible in Atlantic salmon and low to moderate in rainbow trout (Fevolden et al. 1991). Disease challenges of rainbow trout and Atlantic salmon divergently selected for low and high stress response had inconsistent results, low stress lines were more susceptible to some bacterial pathogens but were not different or more susceptible than high stress lines for other bacterial pathogens (Fevolden et al. 1992, 1993). A short-term, standardized
stressor like the one we used may not be representative of the duration or severity of stressors present under culture condition. Although more complicated, long-term stress tests may provide insight into the effects of stress on immune function, they would be difficult and costly to measure on large numbers of fish.

The potential effects of feeding activity of fish during *E. ictaluri* challenges on subsequent mortality and immune response need to be considered in interpretation of challenge results. Wise and Johnson (1998) demonstrated a positive correlation between feeding rate during challenge and ESC mortalities, and commercial catfish farmers typically restrict feed during ESC outbreaks. In contrast, Lim and Klesius (2003) reported that catfish fed daily before and after an ESC challenge had better post-challenge survival than fish that were feed-restricted before and/or after the challenge. We fed the fish in this study to satiation before and after the challenge. We fed the fish in this study to satiation before and after the challenge and the genetic effects for *E. ictaluri* resistance may, at least partially, reflect genetic differences in feed consumption among groups. Although no data was collected on feeding activity in this study, the USDA 103 strain has been shown to consume a higher percentage of body weight than other channel catfish strains (Silverstein et al. 1999). The relatively poor post-challenge survival of the USDA 103 strain may be related to its higher feed consumption. Understanding the environmental and genetic interactions between *E. ictaluri* resistance and feed intake has important implications for interpreting results of *E. ictaluri* challenges and developing improved catfish for commercial use. Given the conflicting results on effects of feeding on catfish mortalities associated with ESC challenges, additional research is needed to determine the relationship between feeding and ESC.

In summary, the USDA 102 strain contributed additive and dominance genetic effects for increased survival and reduced antibody level after exposure to live, virulent *E. ictaluri*. There were no differences among genetic groups tested for cortisol levels or antibody response to formalin-killed *E. ictaluri*. Genetic improvement of *E. ictaluri* resistance is possible but should be based on survival and antibody response following live challenge. Indirect selection for antibody response to formalin-killed *E. ictaluri* or serum cortisol following stress is not likely to improve resistance to *E. ictaluri*.

**Literature Cited**


