

# Hormonal manipulations of growth rate and its influence on predator avoidance – foraging trade-offs

M.V. Abrahams and T.C. Pratt

**Abstract:** Theoretical investigations into the impact that predators exert on prey species suggest that two parameters, growth rate and mortality rate, should be the most influential in determining when animals should risk exposure to a predator in order to achieve higher feeding rates. While these two parameters have usually been assumed to be environmentally determined, we used thyroid hormone (3,3',5-triiodo-L-thyronine ( $T_3$ )) to manipulate growth rates and examine the behavioural consequences associated with these manipulations. In two experiments, we examined how the growth rate of fathead minnows (*Pimephales promelas*) is affected by treatment with  $T_3$ , and used the results from this experiment to make a priori predictions about their relative willingness to risk exposure to a predator in order to receive increased feeding rates. The first experiment demonstrated that  $T_3$  significantly reduced the growth rates of fathead minnows compared with an unmanipulated control. When groups were compared in their relative willingness to risk exposure to a predator, manipulated growth rates in the first experiment were an accurate predictor of behaviour; groups with relatively high growth rates were more willing to risk exposure to a predator. These results are consistent with the theoretical expectation that growth rates should be an important factor determining decisions that involve trade-offs.

**Résumé :** Les recherches théoriques sur l'impact qu'exercent les prédateurs sur les diverses espèces de proies indiquent que deux variables, le taux de croissance et le taux de mortalité, semblent être les facteurs les plus déterminants du choix des animaux de risquer de s'exposer à un prédateur pour parvenir à un taux d'alimentation plus élevé. Ces deux variables sont généralement considérées comme régies par l'environnement, mais nous avons utilisé de l'hormone thyroïdienne (3,3',5-triiodo-L-thyronine ( $T_3$ )) pour manipuler les taux de croissance et pour examiner les changements de comportement en réaction à ces manipulations. Au cours de deux expériences, nous avons examiné comment le taux de croissance de Têtes-de-boule (*Pimephales promelas*) est affecté par le traitement hormonal et nous avons ensuite utilisé les résultats pour faire des prédictions a priori au sujet du « bon vouloir » relatif des poissons de risquer de s'exposer à un prédateur pour parvenir à des taux d'alimentation plus élevés. La première expérience a démontré que  $T_3$  modifie significativement les taux de croissance des Têtes-de-boule par comparaison à ceux de poissons témoins non manipulés. Lorsque les groupes étaient comparés quant à leur disposition relative à risquer de s'exposer à un prédateur, les taux de croissance manipulés dans la première expérience permettaient de prédire avec justesse leur comportement; les groupes à taux de croissance relativement élevés étaient plus enclins à subir les risques de prédation. Ces résultats sont en accord avec les hypothèses selon lesquelles les taux de croissance sont un important facteur décisionnel quand il s'agit de faire des compromis.

[Traduit par la Rédaction]

## Introduction

It has been recognized that many decisions made by prey species, including those made about mate choice, parental care, and social structure, incorporate the risk of predation. Perhaps the most studied aspect of this decision making is the impact that risk of predation has upon foraging decisions. As a consequence, there has been considerable interest in developing models to investigate its role. Theorists

have employed both analytical methods (Werner and Gilliam 1984; Gilliam and Fraser 1987; Abrams 1991; Leonardsson 1991) and dynamic state variable techniques (Mangel and Clark 1986, 1988; Ludwig and Rowe 1990; Houston et al. 1993). All these models have predicted a solution generally similar to that originally proposed by Werner and Gilliam (1984), namely that under certain conditions and subject to some assumptions, animals should choose to feed in locations that minimize the ratio of mortality rate ( $\mu$ ) to growth rate ( $g$ ). Gilliam and Fraser (1987) have provided experimental support for a modified form of this model.

Most experiments that have investigated how animals incorporate predation risk into their foraging decisions have manipulated food and risk levels associated with different feeding locations (for a review see Lima and Dill 1990). These experiments demonstrated that animals will signifi-

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cantly alter their behaviour in response to a perceived risk of predation, and have led some authors to suggest that the ecological impact of predation risk may sometimes exceed the direct effect of predation mortality (Kotler and Holt 1989).

Based upon theoretical work, it is clear that there are two general parameters that are important in determining whether an animal should be willing to risk exposure to a predator: parameters that affect mortality rates (i.e., the probability of being killed by a predator over a discrete period) and those that affect growth rates (e.g., the life-history characters of the animal, particularly the fitness benefits associated with food). Experimental work has demonstrated that reduced vulnerability to predators, such as increased size (Johnsson 1993) or the presence of antipredator morphology (McLean and Godin 1988; Abrahams 1995), results in animals being relatively more willing to risk exposure to a predator. The benefits of food are more difficult to determine. In some experiments, starved or parasitized animals were relatively more willing to risk exposure to a predator (Dill and Fraser 1984; Godin and Sproul 1988). In other experiments, food altered the relative willingness to risk exposure to a predator (Cerri and Fraser 1983; Fraser and Huntingford 1986; Gilliam and Fraser 1987; Holbrook and Schmitt 1988; Abrahams and Dill 1989), although the results varied depending upon the design of the experiment, the species, and the sex.

To better understand how the relationship between energy and fitness affects antipredator behaviour, we tested the hypothesis that hormonal manipulations that alter the relative benefits associated with feeding opportunities should correspondingly alter the relative willingness to risk exposure to a predator. Hormones are well known to alter the growth rates of fishes (Donaldson et al. 1979). Since increased growth rates are generally of value in aquaculture, most reports in the literature are focused on increased growth rates. For example, a diet supplemented with synthetic androgens doubled the growth rate of carp (*Cyprinus carpio*; Lone and Matty 1983) and increased rainbow trout (*Oncorhynchus mykiss*) growth rates up to 69% over 60 days (compared with unmanipulated controls; Matty and Cheema 1978). Thyroid hormone, particularly the active form ( $T_3$ ), is also known to enhance the growth rates of fish (Higgs et al. 1982). The mechanisms underlying such increased growth rates include increased appetite, alteration of anabolic pathways (possibly with synergistic effects with other hormones (Higgs et al. 1982)), and increased proteolytic enzyme activity resulting in increased digestion and assimilation of food (Weatherly and Gill 1987). These results suggest that hormonal manipulations can alter animal characteristics beyond those shaped by natural selection.

These characteristics can then be examined experimentally to determine whether relative growth rates can predict the relative willingness to risk exposure to a predator. Assuming that individuals make decisions about feeding in dangerous areas based upon the perceived differences in the benefits gained from feeding less the cost of risking exposure to a predator (which is functionally equivalent to Werner and Gilliam's 1984 model; Brown 1992), it is then possible to simulate habitat-selection decisions by these individuals when they must choose between two feeding sites,

one that contains some risk of predation and another that provides food with no risk.

## Materials and methods

The care of the animals and the procedures employed in these experiments were in accordance with the principles and guidelines of the Canadian Council on Animal Care (The University of Manitoba Animal Care Protocol No. FRF96-044/1).

For these experiments, the active form of thyroid hormone, 3,3',5-triiodo-L-thyronine ( $T_3$ ), was used to manipulate growth rates. Thyroid hormone (unlike other hormones that affect growth rates, such as growth hormone) is not species specific and is readily available and relatively inexpensive to use. It is also possible to administer it non-invasively with ambient water, significantly simplifying experimental procedures. In addition, unlike steroids, relatively few side effects are associated with the administration of  $T_3$  (Higgs et al. 1982).

Mature fathead minnows (*Pimephales promelas*) were collected in minnow traps during early June 1994 at The University of Manitoba Delta Marsh field station (located at the south end of Lake Manitoba). Eggs were stripped from ripe females and fertilized with sperm that had been removed from the testes of males. Fertilized eggs were then reared in aerated water (thermally regulated at 21°C) in 3-L glass aquaria, and the hatched larvae were allowed to grow into fully developed juvenile fish. Initially, these juvenile fish were fed live baby brine shrimp (*Artemia salina*) ad libitum; after they were approximately 4 weeks old, they were switched to an ad libitum diet of frozen adult brine shrimp.

At this time, the juvenile fish were randomly separated into four different 30-L aquaria, each of which received a different treatment: a control and three concentrations (12.5, 25, and 50  $\mu\text{g}\cdot\text{L}^{-1}$ ) of  $T_3$ . The concentrations of  $T_3$  used in this study were within the range of dosages employed elsewhere (see Higgs et al. 1982).  $T_3$  was not administered to these fish until they were fully developed, to avoid the developmental abnormalities that have been associated with this type of hormonal manipulation (Higgs et al. 1982). To generate the 50  $\mu\text{g}\cdot\text{L}^{-1}$  concentration, 0.1551 g of purified  $T_3$  was added to 100 mL of 0.1 M NaOH and 1 mL of this solution was added to 30 L of water in the holding aquarium. Serial dilutions with 0.1 M NaOH were used to generate the 25 and 12.5  $\mu\text{g}\cdot\text{L}^{-1}$  concentrations. The control group was exposed to a 0.1 M NaOH solution. As  $T_3$  is known to be a very adhesive molecule (Imarisio and Greco 1964), it was assumed that hormone concentrations would diminish over time. To minimize this effect and to prevent accumulations of nitrogenous waste, each aquarium received a complete change of water and 1 mL of the appropriate treatment solution every week. Throughout these experiments, the laboratory photoperiod was adjusted weekly to correspond to natural conditions.

### Experiment 1: the effect of $T_3$ on growth rates

The goal of this experiment was to use  $T_3$  to modify the growth rates of fathead minnows. The results of this experiment were then used to make a priori predictions about behaviour in experiment 2.

After the fish were 10 weeks old, 20 individuals of approximately similar size (five from each of the four different treatments) were isolated and their growth monitored under controlled conditions. Each individual was placed in a separate 3-L aquarium. Each aquarium contained only an air stone to minimize the adhesion of  $T_3$  to surfaces within the aquarium. In addition, once per week each aquarium was completely drained and refilled with water and the  $T_3$  solution to maintain constant treatment levels. All aquaria were in a laboratory where the temperature was regulated to provide a water temperature of approximately 20°C; the photoperiod was also regulated to approximate that experienced by fish in the

**Table 1.** Mean wet mass (g) of the fish used in experiment 2.

Treatment	Trial group					
	1	2	3	4	5	6
Control	1.013 (r)	0.989 (b)	0.570 (y)	0.535 (r)	0.603 (w)	0.343 (w)
T <sub>3</sub> (µg·L <sup>-1</sup> )						
12.5	0.815 (b)	0.830 (y)	0.575 (w)	0.456 (b)	0.626 (b)	0.369 (b)
25	0.817 (y)	0.803 (r)	0.534 (b)	0.748 (w)	0.623 (y)	0.468 (y)
50	0.689 (w)	0.859 (w)	0.564 (r)	0.490 (y)	0.444 (r)	0.471 (r)

**Note:** Letters in parentheses indicate tag colours that were used to identify treatment conditions within each trial group: r, red; b, blue; y, yellow; and w, white).

wild at the same time of year (initially, 14 h light : 10 h dark, adjusted weekly).

Experiments were initiated by anaesthetizing the fish with water containing 0.37 mL 2-phenoxyethanol·L<sup>-1</sup> and obtaining their wet mass and fork length. Over two feedings per day, each fish received 20% of its wet mass in frozen brine shrimp (mean wet mass of 0.003 g; variance = 0.000772 g). At the beginning of each day, unconsumed food was removed, allowing us to determine how much food was consumed by each individual.

The minnows were anaesthetized and reweighed every 2 weeks. Their diet was modified to maintain it at 20% of their most recent mass. After 6 weeks, the fish were reweighed once per week. The growth of these fish was monitored for 13 weeks. Upon completion of this experiment, the fish were returned to the general holding tanks.

The effect of T<sub>3</sub> on the growth rate of these fish was determined by plotting the size of the fish under the different treatment levels against time. The slope of this relationship for each fish was used as the measure of treatment-specific growth rate. These data were analysed employing the repeated measures approach described by von Ende (1993).

### Experiment 2: the effect of T<sub>3</sub> on behaviour

The results of experiment 1 were used to test the prediction that relative growth rates should be positively correlated with the relative willingness to risk exposure to a predator. This was done by presenting a new group of hormonally manipulated minnows with a situation in which they could either risk exposure to a live predator in order to gain access to additional food or remain safe from the predator and feed at a reduced rate (e.g., a two-patch ideal free distribution with predation risk). Under these conditions and if individuals have different growth rates, the composition of the group of individuals willing to risk exposure to feed in the presence of the predator will be dominated by those that have the highest relative growth rates, a result similar to that predicted for the unequal competitors ideal free distribution model (Houston and McNamara 1988).

To create this situation, a 200-L (120 × 30 × 45 cm) glass aquarium was divided in half by a partition of horizontal glass rods set at 1-cm intervals. These rods allowed the fathead minnows access to both sides of the tank, while restricting a piscivorous yellow perch (*Perca flavescens*) to one side of the aquarium.

Automated feeders were placed on either side of the partition at a distance of 8 cm from it. Both feeders provided 0.25 g of frozen brine shrimp at a constant rate for 20 min (see Abrahams 1989 for details). The food was provided through five holes spread over 25 cm, preventing any individual from defending the food. Because the feeders delivered food at the same absolute rate, the spatial distribution of the fish determined the individual feeding rate (i.e., the spatial distribution of the fish should match the spatial distribution of the food, sensu Abrahams and Dill 1989). Therefore, if fewer individuals elected to feed in the presence of the predator, the individual feeding rate would be greater in that location.

Three individuals from each treatment group (control, and 12.5, 25, and 50 µg T<sub>3</sub>·L<sup>-1</sup>) were placed in the test aquarium for a total of 12 individuals per trial. These individuals were selected so that all were of approximately equal size (Table 1). To distinguish treatment groups within the apparatus, individuals received a red, white, yellow, or blue tag that was a modified version of one described by Chapman and Bevan (1990). Each treatment group received a randomly determined colour prior to each experiment (Table 1).

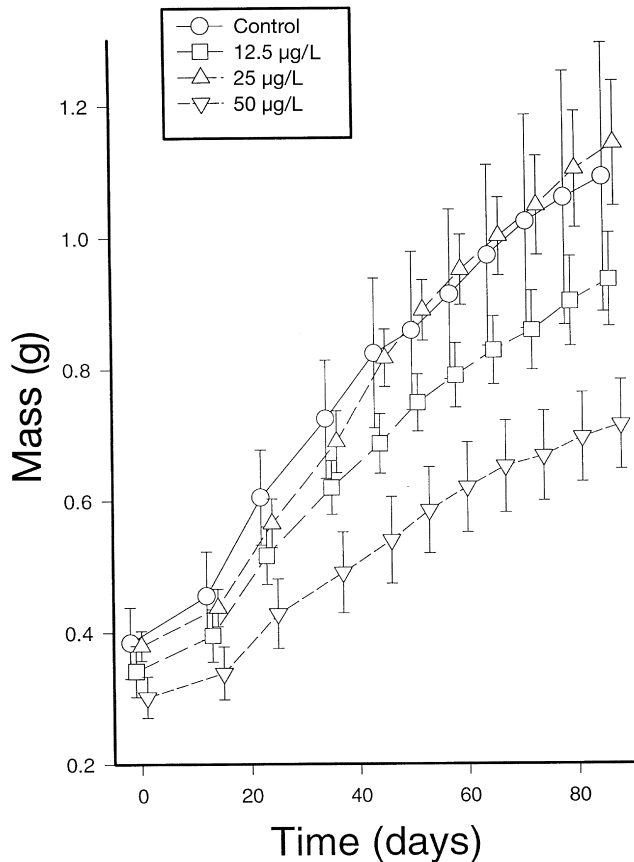
The tagged fish were placed together in the test apparatus and fed exclusively by the automated feeders for 5 days. This provided the fish with sufficient time to recover from tagging, learn to use the feeders, and acclimatize to the experimental apparatus. During experiments, fish lived in the test apparatus, obviating the need for any additional time in which to become familiar with the experimental conditions.

To conduct experiments, one side of the aquarium was randomly selected to be the dangerous side and one of three yellow perch (wet masses of 12.85, 9.97, and 8.92 g) was then placed in that side of the apparatus. Equal amounts of food were placed in the feeders and the flow of food started. Trials (20 min) were monitored remotely by a 8-mm video camera that was positioned to record both sides of the apparatus. To facilitate identifying individuals according to their coloured tags, the apparatus was illuminated by overhead fluorescent and halogen lights and a neutral gray background was placed behind the apparatus. Upon termination of the trial, the feeders were removed and opaque partitions were placed 15 cm from each end of the aquarium to conceal the predator. Trials were conducted three times a day, with the trials being separated by at least 3 h. A particular treatment was conducted for 2 days. After that time, the position of the predator was reversed and the procedure repeated. Upon completion, the fish used in the trial were removed from the apparatus and a new group added. Six different groups of fish were used for this experiment.

It is well known that fathead minnows contain alarm substance (a chemical that elicits a fright reaction), and this chemical is released when fish are physically damaged (see review by Smith 1997). It is our view that the function of this chemical is to alert the fish to the presence of a predator. Since the predator was obviously present in the apparatus, we assumed that the release of the chemical would provide no additional information, and so the choice was made not to change the water if a fish was captured by a predator.

The videotapes were analysed by identifying the location of fish from the different treatment groups every 30 s from the time food started to enter the apparatus. The mean result of 40 observations represented a single observation for a trial. The average number of individuals from each treatment group feeding in the presence of the predator was determined. A risk score was assigned to each treatment group within each trial group. This score ranged from a value of four for the treatment group that had the greatest number of individuals feeding in the presence of the predator, to a value of one for the treatment group that had the least. The mean of the six

**Fig. 1.** The mean size ( $\pm$ SE) of fathead minnows for the duration of the growth experiment. The legend indicates the treatment that each group of fish received: control or 12.5, 25, or 5  $\mu\text{g T}_3\cdot\text{L}^{-1}$ .



risk scores for each group (three trials per day for 2 days) was used to provide a single observation for each treatment group at each predator position.

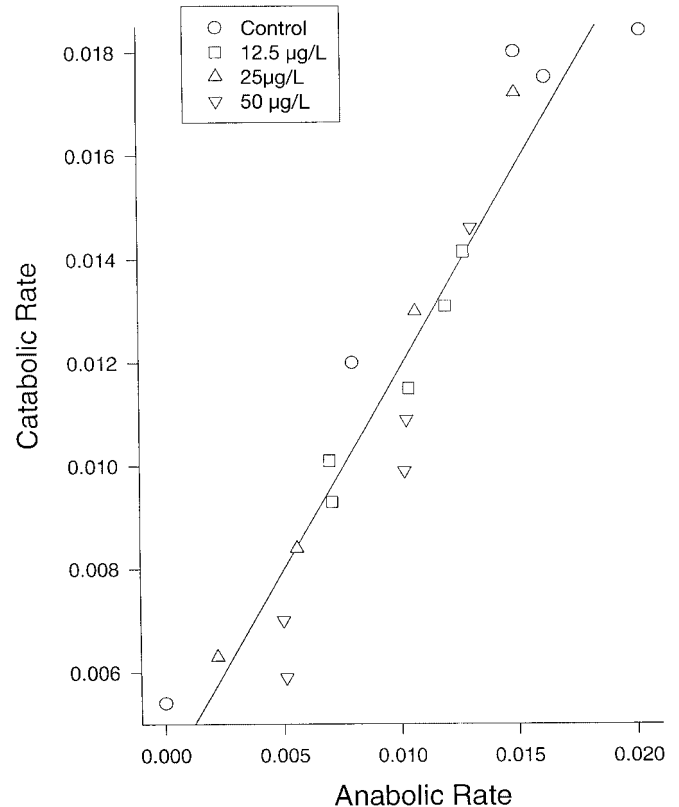
## Results

### Experiment 1: growth rate

During this experiment, one fish in the 25  $\mu\text{g T}_3\cdot\text{L}^{-1}$  treatment group died and one in the control group was excluded as an outlier, since its final size was 2.5 times larger than that of the mean for the group. As a result, 18 fish were used for the analysis (five each for the 50 and 12.5  $\mu\text{g T}_3\cdot\text{L}^{-1}$  treatments and four each for the control and 25  $\mu\text{g T}_3\cdot\text{L}^{-1}$  treatments).

Daily growth rates were determined by plotting size at age for each fish and fitting a straight line through the data. The slope of the line was used to describe the daily growth rate. While growth is normally assumed to follow some curvilinear pattern (e.g., a von Bertalanffy curve), these straight lines were a very good fit to the data (average  $r^2 = 0.972$ ). The effect of  $\text{T}_3$  was to reduce the average growth rates for fathead minnows from a rate of  $8.69 \times 10^{-3} \text{ g}\cdot\text{day}^{-1}$  under the control treatment to  $5.05 \times 10^{-3} \text{ g}\cdot\text{day}^{-1}$  under the 50  $\mu\text{g T}_3\cdot\text{L}^{-1}$  treatment (repeated measures ANOVA,  $F_{[3,15]} = 3.31$ ,  $P = 0.05$ ; Fig. 1). The growth rate of the control group was significantly higher than that of the groups receiving the

**Fig. 2.** The effect of thyroid hormone on anabolic and catabolic rates in the fish studied. Anabolic and catabolic rates were not measured directly but were determined iteratively based on the growth data for each fish. The legend indicates the treatment that each fish received (control or 12.5, 25, or 50  $\mu\text{g T}_3\cdot\text{L}^{-1}$ ) and the straight line through these data was fitted by least squares.



12.5 and 50  $\mu\text{g T}_3\cdot\text{L}^{-1}$  treatments, when  $\alpha = 0.05$ . No significant difference existed between the growth rates of the control and 25  $\mu\text{g T}_3\cdot\text{L}^{-1}$  treatment groups.

Based on the methods described by From and Rasmussen (1984), we used our growth data to determine iteratively the effect that hormonal manipulations had on the catabolic and anabolic components of metabolism for each fish. A plot of catabolic rate versus anabolic rate demonstrated that all fish fell very close to a line fitted by least squares (Fig. 2;  $r^2 = 0.9121$ ). This result indicates that increased growth rates for these fish must be accompanied by increased metabolic rates. The impact of hormonal manipulations upon this relation was less clear. Fish from the control group spanned the full range of combinations observed in this experiment, supporting the observation of a higher variation in growth rates in the control group than in the hormonally manipulated groups. However, specific hormone treatments had no consistent effect upon this relationship.

$\text{T}_3$  treatment significantly affected the average amount of unconsumed food (one-way ANOVA,  $F_{[3,14]} = 4.819$ ,  $P = 0.016$ ). Groups with relatively slower growth left more unconsumed food (i.e., they ate less food). The ratio of mass gain to mass of food consumed provided a description of the conversion efficiency between food consumed and biomass acquired.  $\text{T}_3$  treatment had no significant influence on con-

**Table 2.** Comparison of the ability of fish in the various treatment groups to convert food into biomass.

Treatment	No. of fish/group	Conversion efficiency <sup>a</sup>	SE
Control	4	8.5322	1.1441
T <sub>3</sub> (μg·L <sup>-1</sup> )			
12.5	5	8.6679	0.4891
25	4	9.2043	0.3714
50	5	7.8026	0.5098

<sup>a</sup>Conversion efficiency was calculated by dividing the increase in wet mass of the fish by the mass of the brine shrimp consumed and multiplying the result by 100.

version efficiency (Table 2; one-way ANOVA,  $F_{[3,14]} = 0.78$ ,  $p = 0.53$ ) and the difference in growth rates measured in this experiment was probably generated by changes in appetite.

### Experiment 2: antipredator behaviour

Based on the results of experiment 1, it was predicted that the control and 25 μg T<sub>3</sub>·L<sup>-1</sup> groups should be the most willing to risk exposure to a predator and the 50 μg T<sub>3</sub>·L<sup>-1</sup> group the least willing, with the 12.5 μg T<sub>3</sub>·L<sup>-1</sup> group intermediate.

During this experiment, 72 fish were exposed to a predator for 4 days each. The predators captured three individuals, one from the control group and one each from the 12.5 and 50 μg T<sub>3</sub>·L<sup>-1</sup> treatment groups.

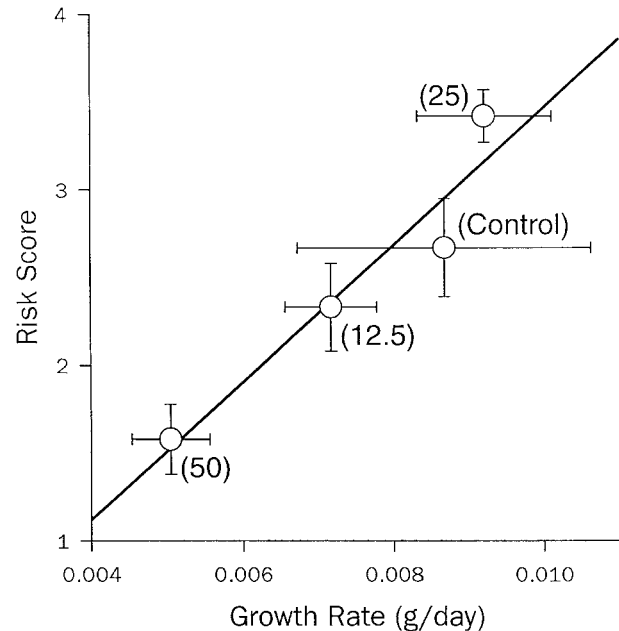
Hormonal treatment had a significant influence on the relative willingness of individuals to risk exposure to the predator (Friedman's ANOVA,  $\chi^2_r = 10.1$ ,  $df = 3$ ,  $P = 0.018$ ). Of particular interest is that the relative willingness of the different treatment groups to risk exposure to the predator exactly matched the relative growth rates generated by these different hormonal treatments in the first experiment (Fig. 3). Employing Monte Carlo simulation, we determined the probability of observing this type of match between growth rates and behaviour by random event to be 0.04. It is therefore statistically improbable that the relation between relative antipredator behaviour and relative growth rates was random.

Two possible confounding factors that may influence these results are fish size and tag colour. The treatment groups with the most rapid growth rates may also have been larger in size and therefore less susceptible to attack by the predator. However, no significant correlation existed between these two parameters (Spearman's rank correlation coefficient,  $r_s = -0.08$ ,  $df = 24$ ,  $P = 0.71$ ) indicating that the observed behavioural results were not a consequence of size variation. Similarly, a Friedman's test indicated that tag colour was not related to the willingness to risk exposure to a predator ( $\chi^2_r = 4.85$ ,  $P = 0.18$ ).

### Discussion

The response by our fish to the thyroid hormone treatment generated a complex triphasic response and generally reduced the growth rates of our fathead minnows. Biphasic responses to thyroid hormone are known from other studies (Higgs et al. 1982), and our more complex response may be

**Fig. 3.** The effect of growth rate on the relative willingness to risk exposure to a predator as measured by a risk score (see text for details). These data illustrate that groups with higher growth rates are relatively more willing to risk exposure to a predator in order to gain access to additional food. The straight line was fitted through the mean data by the least squares method and the error bars correspond to 1 SE. The labels in parentheses indicate the experimental treatment (control or 12.5, 25, or 50 μg T<sub>3</sub>·L<sup>-1</sup>) associated with each coordinate.



due to differences in our methodology. We provided the hormone treatment to the fish via water rather than by food or injection. This was necessary because of the very small size of our fish. Because of the adhesion of T<sub>3</sub> molecules to glass, we changed the hormone solution every week. This process may have introduced some pulsing of the dose levels that may have influenced the growth rates of our fish. This may also have resulted in a reduction in growth rates rather than the increase in growth rates normally associated with elevated levels of thyroid hormone (Higgs et al. 1982). Despite this result, that we were able to generate changes in growth rates of these fish was all that was necessary to test the influence of altered growth rates on antipredator behaviour.

Theorists have predicted that, with some simplifying assumptions, populations should occupy those habitats that minimize the ratio of mortality rate to growth rate (see, among others, Werner and Gilliam 1984). Even though this theoretical approach has been widely regarded as the best explanation to describe the spatial distribution of populations where habitat quality includes the risk of predation, there has been no support to indicate that this was also how individuals incorporated the risk of predation into their foraging decisions. Gilliam and Fraser (1987) were able to demonstrate theoretically that, when animals were also provided with a refuge that provided complete safety from predators and no food, minimizing the ratio of mortality rate ( $\mu$ ) to growth rate ( $g$ ) could be simplified to minimizing the ratio

of mortality rate to feeding rate. Their experiments supported their theory. Here we have demonstrated that an experimental manipulation that alters growth rate also changes the relative willingness of individuals to risk exposure to a predator in a manner that is consistent with the  $\mu/g$  model. Furthermore, these experiments also suggest that linked hormonal control of growth rate and behaviour may be the proximate mechanism that generates this result.

The results of these experiments also have implications for better understanding how the risk of predation may impact natural populations. Within a population, individuals that receive relatively greater fitness benefits from food are predicted to be more likely to risk exposure to a predator in order to increase their feeding rate. Assuming that all individuals are equally vulnerable to these predators, these groups should suffer higher mortality rates from predators. Our experiments demonstrated that unmanipulated fish had the greatest variation in growth rates, indicating that this variability may naturally exist within a population. Maintenance of this variability may reflect fluctuations in the predator pressure and food availability experienced by these populations. Periods of high food availability and high predator density may provide a selective advantage to those less willing to risk exposure to predators. Low food availability and high predator density may shift the advantage to those willing to incur a higher risk associated with their foraging behaviour.

Ketterson and Nolan (1992) have argued that hormones form the mechanistic basis for trade-offs in life histories. Hormones often produce changes in more than one trait; if one trait is beneficial and the other is detrimental, the level of the hormone within the organism will reflect the optimal trade-off between these conflicting demands. Furthermore, given the ease with which hormones can be manipulated, it is relatively easy to identify the costs associated with deviation from optimum. For example, Derting (1989) elevated thyroid hormone ( $T_4$ ) levels of young cotton rats (*Sigmodon hispidus*) and compared the growth rates of manipulated individuals with unlimited and restricted access to food; the obvious result was observed: groups where food was unrestricted had higher growth rates than groups where food was restricted. The effect of  $T_4$  was to elevate the metabolic rate of young cotton rats. Under restricted feeding conditions, this increased cost resulted in reduced growth relative to the unmanipulated control group. However, when feeding was unrestricted, the increased activity levels allowed them to grow at rates exceeding that of the control group.

When fathead minnows were exposed to different levels of thyroid hormone, growth rates at two of the treatment levels were reduced relative to the control group. The mechanism that appears to be responsible for the difference in growth rates was a reduction in appetite. The treatment groups with the slowest growth also consumed the least food. Associated with this difference in growth rate was a difference in behaviour. Groups with the most suppressed appetite were also the groups least likely to risk exposure to a predator in order to gain access to additional food. If this hormone does affect the link between growth and behaviour, it is not unreasonable to assume that other treatments that increase growth rates may result in individuals that are relatively more willing to risk exposure to a predator.

Results similar to those of our experiment have also been observed in salmonids. Jönsson et al. (1996) demonstrated that rainbow trout (*Oncorhynchus mykiss*) treated with growth hormone were more willing to feed in close proximity to a predator than unmanipulated fish. Johnsson et al. (1996) demonstrated a similar result for brown trout (*Salmo trutta*). These authors have interpreted their results to indicate that levels of growth hormone represent a balance between the positive effects of growth and the increased mortality associated with changes in antipredator behaviour. Within a hatchery, where there are no predators, they argue that this balance will be altered (Johnsson et al. 1996).

Lachmansingh and Rollo (1994) have investigated the life-history consequences associated with accelerated growth rates by investigating the time budgets of a transgenic strain of "supermice" (*Mus domesticus*, strain Tg(MT-1,rGH), Bri2). This particular strain of mouse contains multiple copies of rat growth hormone genes that significantly elevate growth hormone levels (Palmiter et al. 1982) and double growth rates and body sizes. These results occur despite having a lower mass-specific feeding rate than unmanipulated mice (Kajiura and Rollo 1994). They also observed that the increased growth of the supermice comes at the expense of reduced behavioural expenditures. Compared with unmanipulated mice, transgenic mice spent proportionately more time asleep and less time in locomotion, drinking, and grooming, but were not different in the amount of time spent feeding. Lachmansingh and Rollo (1994) concluded that, in light of ecological demands, the behavioural consequences of increased growth prevent wild populations from achieving the growth rate of transgenic supermice.

In conclusion, the experiments reported in this paper demonstrate that the relative willingness of different animals to risk exposure to a predator in order to gain access to additional food may be partially predicted by their life-history characters (i.e., those individuals or species that derive greater benefits from a resource should be willing to incur a greater cost). While this result seems intuitive, it has implications for predator-prey interactions. An increased willingness to risk exposure to a predator means that these animals should be less willing to forfeit profitable activities in order to reduce the probability of death from a predator. As a consequence, these individuals or species should be less susceptible to the indirect effects of predator intimidation.

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