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# Predator risk cues and prey physiological state in an intertidal food web

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## PREDATOR RISK CUES AND PREY PHYSIOLOGICAL STATE IN AN INTERTIDAL FOOD WEB

A thesis presented

by

## Timothy Robert Dwyer

to The Department of Biology

## In partial fulfillment of the requirements for the degree of Master of Science

in the field of

Biology

Northeastern University Boston, Massachusetts August, 2009

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by

Timothy Robert Dwyer

ABSTRACT OF THESIS

Submitted in partial fulfillment of the requirements for the degree of Master of Science in the field of Biology in the Graduate School of Arts and Sciences Northeastern University, August, 2009

#### ABSTRACT

Non-consumptive effects exerted by predators on their prey can have far reaching implications for the structure of ecological communities and functioning of ecosystems. In addition to risk cues detected from nearby predators, physiological state of individual prey animals may influence the sign and magnitude of trait-mediated indirect interactions. Using a tri-trophic intertidal food web, I tested the influence of prey state as represented by injury history on behavior and resource allocation in an intermediate prey species, the marine snail *Nucella lapillus*. Despite the presence of shell injury in natural populations of this and other intertidal gastropods, injured *N. lapillus* showed no differences in feeding rate, energy assimilation efficiency or growth in morphological traits relative to uninjured conspecifics. I also compared the relative influence of predator risk cues account for the great majority of reductions in resource and energy consumption while physiological prey state has negligible influence. Although repaired shells were significantly weaker when tested in compression than uninjured shells, injury status did not influence mortality in snails exposed to predation in the field. These data indicate that predation risk is a major driver of resource consumption and community effects while prey state as represented by shell injury makes little contribution.

## DEDICATION

To my academic advisor, Geoffrey C. Trussell,

for whom the ends always justify the means.

#### ACKNOWLEDGEMENTS

Although we're often taught to think of ourselves as independent entities, my success, such that it is, comes to pass only with the efforts and support of many people. Had I really tried to do everything alone, I would not have made it even this far. The family, friends and families of friends that all helped me a little bit along the way are too numerous to mention individually but a few stand out:

For their unwavering support over the past five years I sincerely thank my parents, Robert and Katherine, my sister, Elisabeth, my grandmother, Julia, my aunts, Patricia and Candace, my uncle, Bernard and my brother-in-law, David. Though I don't express it often enough I am grateful on a daily basis for the accident of birth through which we are related.

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With that said, the principle lesson that I take from this experience is summed up in the first

verse to a poem I've always liked:

Now this is the Law of the Jungle - as old and as true as the sky; And the Wolf that shall keep it may prosper, but the Wolf that shall break it must die. As the creeper that girdles the tree-trunk, the Law runneth forward and back -For the strength of the Pack is the Wolf, and the strength of the Wolf is the Pack.

- Rudyard Kipling

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#### OVERVIEW

Molluscs and the hard shells that protect many of them have long intrigued natural scientists in pursuit of diverse research lines. Taxonomists and evolutionary biologists have taken advantage of a fossil record replete with shells to develop and promote theory on phylogenetic relationships and the processes underlying them. Biochemists and medical researchers continue to identify and isolate rare compounds from toxic species to expand pharmaceutical libraries. Physiologists explore properties of the copper-based respiratory pigments in molluscan blood and investigate narrower questions regarding the mechanisms and energetic costs of shell deposition. Geologists and climatologists rely on the calcium carbonate makeup of mollusc shells to infer long term climate trends from oxygen isotope ratios. Archaeologists and anthropologists depend on middens of discarded shells to resolve diets and other habits of ancient peoples. The list goes on.

To ecologists studying organization of pre-historic and geologically recent communities and ecosystems, gastropod molluscs in particular have proven themselves useful subjects for investigating interactions among species and their abiotic environment. Gastropods are often small, slow-moving and easy to capture. They are relatively cheap and easy to maintain. They have life histories amenable to experimentation on ecologically-relevant time scales. Moreover, they often occupy intermediate positions in many marine and freshwater food webs. It is this role that has facilitated broad acceptance of behavior and other plastic phenotypic traits as important factors contributing to community and ecosystem structure.

In this context I make use of a distinctive aspect of gastropod morphology - permanent evidence of injury - in an attempt to further our understanding of the role that trait-mediated interactions play in ecological communities. Fear of being eaten influences the behavior and morphology of many (if not most) potential prey animals. It is a safe assumption that where predators are abundant, many individuals in a population of prey animals will potentially come into direct physical contact with them. However, few predators are 100% successful and it follows that individual prey animals which have somehow escaped death will come away injured. When effects of these injuries persist and a large number of prey animals in a population have suffered them, the cumulative effects of individual injuries may begin to shape higher levels of ecological organization.

Unfortunately, in most prey animals obvious evidence of non-lethal predation is lost when wounds heal. Subtle, residual changes in behavior and other traits may be difficult if not impossible to detect. Gastropod shells, however, bear the scars of injury for the life of the animal (and often well beyond). In this work I use this characteristic to take some early steps into exploring the implications that unsuccessful predation may have for community and ecosystem organization.

Though shell injury in the fossil record has been a cornerstone of evolutionary biological research, its presence often goes overlooked in ecological research. Shell injury is thought to be a nearly ubiquitous feature of living populations but few scientific studies specifically identify it. Chapter I seeks to verify its presence in a typical Gulf of Maine marine intertidal community and to provide indications of its variability among snail species and over time. This work provides the ecological relevance supporting Chapter II, which addresses questions concerning the role that failed predation may play in contributing to the trait-mediated effects that influence intertidal community structure.

#### <u>CHAPTER I</u>

#### Shell Injury in Intertidal Snails

#### INTRODUCTION

Predation is an important source of mortality in many animal taxa, shaping morphologies and behaviors on evolutionary time scales. In Holocene species, many phenotypic traits are presumably the outcome of selective 'arms races' wherein selective pressures promote defensive prey adaptations and predator coevolution (Vermeij 1982a, 2002). Although predation may have profound impacts on prey densities and related population dynamics, individual predators frequently fail to subdue intended prey (Vermeij et al. 1981, Vermeij 1982a, b). Surviving prey animals are then able to impart to their offspring successful predator-avoidance attributes taking the form of both constitutive defenses - such as armor or toxicity - or plastic defenses - such as behavior (Trussell 1996, Trussell and Smith 2000, Schoeppner and Relyea 2008). It follows that at least some individual members of many populations are likely to be survivors of non-lethal predation (Blundon and Vermeij 1983). These individuals may display residual effects from predation attempts in the form of permanently-induced defenses (Lively 1986, Trussell and Etter 2001) or visible injury (Vermeij et al. 1981, Juanes and Smith 1995).

Effects from past predation have the potential to alter predator-prey, prey-resource and competitive interactions (Agrawal 2001, Maginnis 2006). Modified predator-prey relationships have been shown to shape food web dynamics, community structure and ecosystem processes (Werner and Peacor 2003, Peacor and Werner 2004, Miner et al. 2005, Trussell et al. 2008). If a sizeable proportion of a given population or community has escaped predation, lingering effects from these attacks may have ecological impacts beyond the individual (Harris 1989). However, examinations of population-wide occurrences of non-lethal predation are difficult because in most taxa, physical evidence of these events disappears as animals heal or regenerate (Harris 1989, Maginnis 2006).

Repaired mollusc shells bear the scars of shell injury throughout the life of the animal and beyond. Hence, molluscs - and specifically gastropods - are an appropriate taxon for exploring questions of non-lethal predation effects on ecological communities. Paleobiologists have often compared gastropod, bivalve and nautiloid shell repair frequencies in the fossil record to contemporary populations in order to draw inferences about paleoecological conditions and the selective processes shaping shell form (Vermeij et al. 1981, Schmidt 1989, Vermeij 2002, Zuschin et al. 2003). Though the usefulness of shell damage frequency as a means of quantifying predation intensity has been met with skepticism (Schoener 1979, Cadée et al. 1997), the relative efficiency of shell-crushing or shell-boring predators within a given assemblage may be more reliably assessed (Schmidt 1989, Dietl and Alexander 2009). Where durophagous predators often fail, fossil assemblages and Holocene populations should exhibit higher frequencies of shell repair. Conversely, where predators are relatively effective, shell repair should be infrequent or even nonexistent, though the complete absence of predators could account for this latter situation (Vermeij et al. 1981, Schindel et al. 1982, Vermeij 1982a, Vermeij et al. 1982, Zuschin et al. 2003).

Repaired shell may functionally compare to uninjured shell in three ways (Watabe 1983). The animal may alter shell architecture or deposition of materials in response to previous injury, improving resistance relative to undamaged shell. Conversely, physiological limits of calcium and protein deposition may limit repair of micro-structural imperfections, leading to an irregular repair which prevents the shell from achieving prior resistance levels (Andrews 1934, Watabe 1983). Finally, the repair may be structurally seamless, or the previous two mechanisms may interfere to produce a repaired shell functionally identical in resistance to an uninjured shell. What few experimental studies have addressed this issue in gastropods disagree on the anti-predator effectiveness of repaired shells (Zipser and Vermeij 1980, Blundon and Vermeij 1983, Greenfield et al. 2002).

Whether in response to the repair process or to the unsuccessful predation event itself, the animal potentially experiences an altered perception of its risk environment. Given that the evidence of injury is permanent, a history of non-lethal predation may correspond to long-term phenotypic changes, particularly in morphology or behavior. In communities where a substantial proportion of prey species have been attacked and injured, phenotypic responses to injury may have implications for community structure. If frequencies of unsuccessful predation change over time, these community effects may intensify or diminish and new ones may emerge.

The ecological impacts of non-lethal predation have been little studied. However, injury has been documented in a few Holocene marine intertidal gastropod populations, signifying that non-lethal shell-injuring factors continue to operate. Working with *Littorina rudis* on the coast of England,

Raffaelli (1978) observed high injury frequencies - the proportion of population exhibiting damage - on wave-exposed shores with intermediate substrate particle sizes. On Guam, Zipser and Vermeij (1980) found that the number of scars on repaired *Conus sponsalis* increased with size while the size-frequency distribution of injured snails did not change over the course of a 30 d mark-recapture study. Blundon and Vermeij (1983) noted the presence of repaired *Littorina* (= *Littoraria*) *irrorata* in Virginia marshes. Working in northern California, Geller (1983) found that repair frequencies in *Nucella emarginata* were dependent on microhabitat; snails found in mussel beds were more frequently damaged. Schmidt (1989) quantified shell injury frequencies in populations of 11 gastropods in northern Mexico and observed that damage frequencies varied with microhabitat and shell morphology. Cadée et al. (1997) returned to the same location and confirmed Schmidt's findings noting that injury frequencies had not changed significantly in the intervening decade. In Georgia marshes, Greenfield et al. (2002) collected injured *L. irrorata* to use in crab predation experiments. *L. irrorata* was also the focal animal of surveys conducted by Dietl and Alexander (2005) in North Carolina marshes. They concluded that dense stands of marsh grasses impeded movement of predators and this interacted with tidal submersion time to influence distribution of damage frequencies in snails throughout the marsh.

Given the paucity of evidence for the presence of non-lethal injury in Holocene marine gastropod populations, the present work addresses aspects of its occurrence in three species of gastropods that are abundant on rocky shores of the Northwest Atlantic: *Nucella lapillus, Littorina obtusata* and *Littorina littorea*. Specifically, a series of surveys quantifies population densities of these three snails, identifies the proportion of shells bearing repair scars and compares sizes of repaired and uninjured individuals. Additionally, I quantify changes in these characteristics over the course of a summer growth season. The goal of this effort is to demonstrate the utility of gastropods as a model system for later empirical work exploring the ecological implications of non-lethal predation.

### METHODS

#### Study site

On a stretch of rocky shoreline (~400 m) near New Harbor, Maine (43.875°N, 69.490°W), I conducted a series of surveys to establish the presence of non-lethal damage in a natural snail population as well as any change in its proportion over time. This site consists of a series of granitic

benches exposed to moderate wave action from the open Atlantic. The local intertidal community is characteristic of moderately exposed, Gulf of Maine communities. Substrate at low tidal heights is dominated by a matrix of turf-building red macroalgae (*Chondrus crispus*) and young-of-the-year blue mussels (*Mytilus edulis*) with ephemeral macroalgae (*Ulva spp.*, *Chaetomorpha linum*) present in the spring and absent by autumn. Dense canopies of rockweed (*Fucus spp.*) and knotted wrack (*Ascophyllum nodosum*) dominate intermediate tidal heights, transitioning to a continuous zone of barnacles (*Semibalanus balanoides*) at upper tidal heights.

#### Survey techniques

To quantify injury rates among snails across tidal heights and over time, PVC quadrats (15 x 15 cm) were haphazardly tossed at three different tidal heights (mean low water, mean high water and intermediate) along the previously described rocky shoreline during daytime low tides. In late June (N = 183 quadrats) and early October (N = 180 quadrats) 2008, I collected and preserved all live individuals of *Nucella lapillus*, *Littorina obtusata*, and *L. littorea* (the three dominant snail species) > 5 mm shell length found within each quadrat. Later, in the laboratory I identified snails to species and scored shells for presence or absence of unambiguous signs of prior injury. For a given species, damage frequency was defined as the number of individuals exhibiting at least one scar divided by total number of snails of this species in each quadrat.

To examine patterns in snail size over time with respect to shell damage history, a second series of surveys was conducted in June and October along approximately 400 m of shoreline at the same location. For a given snail species, I haphazardly selected single snails at ~1 m increments along the shoreline at all tidal heights, examining them for shell scars. If the shell had previously been damaged, I collected it, then moved ~1 m and haphazardly collected an undamaged snail. Where the first snail I selected was not damaged, I moved on and repeated the process. Later, I measured total shell length (apex to tip of the siphonal canal) using Mitutoyo electronic calipers (± 0.01 mm). I was principally interested in presence or absence of shell damage within these populations, hence I did not differentiate shells with multiple scars from those with single scars. Nor did I try to establish any timeline or pathology for this damage based on the relative position or the nature of scarring along the body whorl (Zuschin et al. 2003).

#### Statistical analyses

Physiological tolerances to temperature, desiccation and food scarcity differ dramatically among intertidal snail species (Menge 1976, Osborne 1977, Menge 1991). As these limiting factors become more intense with increasing tidal height, natural densities of each snail species I collected differ as well. Additionally, variation among species in shell architecture and microstructure may affect predator success in snails of similar size and microhabitat (Currey 1983, Schmidt 1989, Kohn 1999). Hence, I ignored potential interspecific differences and focused on within-population changes in density and frequency of shell injury. To explore changes in densities of each of the three snail species and the entire 3-snail ensemble, I performed separate two-way ANOVAs that treated survey date and tidal height as fixed effects. Density data were Log-transformed when necessary to meet the test assumption of homogeneity of variances (Zar 1999). I should note that *L. saxatilis* was present at my survey location in the high and low intertidal zones. However, extremely low densities (< 5 m<sup>-2</sup>) on both sampling dates and at all tidal heights dissuaded me from analyzing density and shell damage frequency for this species.

A similar approach was used to examine changes in frequency of damaged snails within each population. As estimates of shell damage frequency can vary dramatically when densities are very low, I excluded quadrats with few individuals (< 3 snails) of a given species. Unfortunately, this approach greatly reduced sample sizes to the point where comparisons of tidal height would have been uninformative. Therefore, these data were pooled. Similarly, very few quadrats contained individuals of all three snail species. Hence, comparisons of damage frequency among species would violate test assumptions of data independence and I performed separate analyses for each snail species using single-factor ANOVAs. Data were arcsine-transformed where appropriate to satisfy test assumptions. Even after transformation, variances remained highly unequal (Levene's test, P < 0.0001). However, ANOVA is robust to departures from the assumption of homogeneity of variances when sample sizes are equal (Zar 1999). These data were subsampled specifically to maintain equal sample sizes within each snail species. Hence, I carried out analyses in light of this violation. I analyzed size survey data for each snail species using two-factor ANOVAs that treated survey date and injury history as fixed effects. Data were Log-transformed where appropriate to satisfy test assumptions.

#### RESULTS

Two-way ANOVA revealed that the density of the three-snail ensemble declined by 68.4% (206 m<sup>-2</sup> in June, 65 m<sup>-2</sup> in October) during the summer months. I likewise noted a significant decline in densities of *Nucella lapillus* (by 77.6%, 335 - 75 m<sup>-2</sup>) and *Littorina obtusata* (by 68.0%, 161 - 52 m<sup>-2</sup>) between June and October, excluding tidal height. While *L. littorea* densities declined as well (by 44.5%, 124 - 69 m<sup>-2</sup>), this change was not significant (Figure 1, Table 1). With respect to tidal height, *N. lapillus* densities decreased (88.4%, 413 - 48 m<sup>-2</sup>) between Low and High microhabitats. Following a similar pattern, *L. littorea* densities dropped by 90.0% (200 - 20 m<sup>-2</sup>). *L. obtusata*, all but absent at Low tidal heights, was abundant in the Mid intertidal (133 m<sup>-2</sup>) with its density increasing 1.4-fold between Mid and High (185

 $m^{-2}$ ) tidal heights. From Low to High tidal heights total snail density fell off by 59.3% (209 - 85  $m^{-2}$ ). Highly significant interaction terms (P < 0.0001) between survey date and tidal height suggested that the rate of summer decline in *N. lapillus*, *L. obtusata* and the combined snail ensemble was influenced by tidal height. In general, rates of decline were greatest at low and intermediate tidal heights (Figure 1).

Shell damage frequencies within the entire snail ensemble increased 3.4-fold (n = 162) between June and October 2008 (Figure 2, Table 2). I observed similar increases in injury frequency within populations of *Nucella lapillus* (3.5-fold, n = 86) and *Littorina obtusata* (9.1-fold, n = 48). Injury frequency in the *L. littorea* population increased modestly (1.3-fold, n = 58), though this change was not significant. I observed no change in mean size of *L. littorea* with respect to survey date or damage history. Both *Nucella* and *L. obtusata* populations displayed modest-but-significant increases in mean shell length over the summer months (1.1- and 1.2-fold, respectively; Table 3). However, a history of injury apparently bore no influence on mean size of any of these snail species (Figure 3, Table 3). **DISCUSSION** 

My data suggest that non-lethal shell injury is a noteworthy characteristic of at least one assemblage of marine intertidal snails in New England. Though I extensively surveyed only one locality along the coast of Maine (-400 m of coastline), my findings agree with previous works (Geller 1983, Schmidt 1989, Cadée et al. 1997) which enumerated shell damage frequencies in a number of intertidal

snail species and microhabitats. Densities of the predatory whelk *Nucella lapillus* and the herbivorous periwinkle *Littorina obtusata* declined sharply over the summer months between late June and early October 2008. I observed variable degrees of decline across tidal-heights microhabitats as well. Concurrent with these density reductions, *N. lapillus* and *L. obtusata* suffered higher frequencies of non-lethal shell injury in October. Interestingly, *L. littorea* densities and injury frequencies remained statistically similar between these two dates.

Density differences among tidal heights in these intertidal snail species are not surprising given their well-known and distinct realized niches (Menge 1976, 1983). Detailed descriptions of community ecology on New England rocky shores are available elsewhere (Menge 1976, Lubchenco and Menge 1978, Etter 1988a, 1989) and are beyond the scope of this effort, however several apparent patterns bear discussing. The increases in summer rates of decline in *Nucella lapillus* and *Littorina obtusata* densities from High to Low tidal heights agree with previous works demonstrating increased predation intensity along a decreasing gradient in environmental rigor (Menge 1978a, b, 1983). In the high intertidal, where temperature and desiccation stress is consistently intense and food availability low, snail densities remain low from June to October. At low and intermediate tidal heights these stressors are not as severe and greater snail densities in June decline towards the end of the summer. While availability of algal food remains high into autumn for *L. littorea* and *L. obtusata*, declines in *N. lapillus* densities may coincide with declines in mussel and barnacle food resources, though this was not measured.

Autumn migrations into sheltered crevices and the subtidal zone may likewise potentially explain this pattern. In Massachussetts, *Nucella lapillus* ceases feeding and seeks shelter beginning in November, eventually emerging in April (Etter 1989). Presumably, the earlier onset of winter explains late September migrations in Nova Scotian populations of *Nucella lapillus* (Hughes 1972). Early October surveys at an intermediate latitude may have captured the beginnings of this migration in *Nucella lapillus*. However, I did not observe similar seasonal declines in *L. littorea*, which also seeks shelter in low intertidal crevices or the shallow subtidal during winter months (Williams 1964). Conversely, I detected a seasonal decline in *L. obtusata* which remains in the intertidal during winter months,

sheltering in macroalgal holdfasts (Williams 1990). Seasonal migrations may therefore only partially explain observed patterns in snail densities.

It is possible that low densities observed in October are related to heavy wave action. Indeed, two weeks prior to my October 10-12 survey dates, wave heights 3-fold greater (2.35 m) than 2008 mean daily summer wave heights (0.79 m, June 1 - August 31) were recorded at a regional offshore weather station (GoMOOS 2009). Despite recent intense wave activity, I observed a large quantity of snail shell fragments persisting in the thick matrix of turf-forming algae (*Chondrus crispus*) and small mussels (*Mytilus edulis*) which dominated the low intertidal zone. I was not able to quantify mass and proportion of species making up these deposits, but snail shells were easily distinguished from consumed young-of-the-year mussels and the overall abundance of snail shell fragments was striking. This location lacks the intermediate-sized particles or dense jetsam responsible for wave-driven shell damage (Raffaelli 1978). Here, wave action would likely remove whole snails to lower tidal heights or into the subtidal whereas durophagous (shell crushing) predators typically attack prey in situ, leaving shells behind. Based on these observations, I suggest that observed density patterns are possibly explained by a number of different mechanisms, with predation being one potential contributor.

Density declines corresponding with increases in shell injury frequency over time are suggestive of increased predation intensity but decreased predator effectiveness. Vermeij (1982c) theorized that high shell damage frequencies meant that predators were ineffective at subjugating prey relative to assemblages where damage was infrequent. Although the applications of this theory for quantitative estimates of predation intensity across geologic time and paleoecological microhabitats have met with resistance (Schoener 1979, Cadée et al. 1997), the basic premise is widely accepted (Kohn 1999). An influx of small or inexperienced predators at high densities during the summer growing season could explain the declines in snail density with increasing shell injury for *Nucella lapillus* and *Littorina obtusata*. Between these two species, shell shape and microstructure differences may account for observed qualitative differences in injury frequency.

The proteinaceous fraction of *Littorina obtusata* shells is much greater than that of *Nucella lapillus* and this material is superior in stopping crack propagation relative to the inorganic CaCO<sub>3</sub> which makes up the majority of *N. lapillus* shells (Kohn 1999). Over the summer, change in shell

damage frequency was greater in *L. obtusata* (+9.1-fold) than among *N. lapillus* (+3.5-fold) and although these were not examined statistically, they suggest that *L. obtusata* survives more frequently. *L. littorea*, whose shells also contain a high proteinaceous fraction and which typically achieves a large size, did not experience significant population declines or changes in shell damage frequency. *N. lapillus* may be at a distinct disadvantage if shell microstructure plays a major role in resistance to durophagous predators on these shores (Kohn 1999).

I did not actively monitor activities of known snail predators at this location and thus can not attribute shell injury to any one source. A variety of animals are known to consume intertidal snails and many of these do so by breaking the shell; decapod crustaceans are chief among these. The American lobster (*Homarus americanus*), rock crab (*Cancer irroratus*), jonah crab (*Cancer borealis*) and green crab (*Carcinus maenas*) are local subtidal and intertidal predators who gain access to soft snail tissue using chelae (claws) to gradually clip away - or outright crush - shell defenses. Though Etter (1988b) observed very few instances of decapod predation on gastropods during extensive surveys of the submerged intertidal, controlled experiments repeatedly confirm decapod ability to handle snail prey (Kitching et al. 1966, Elner and Hughes 1978, Hughes and Elner 1979, Lawton and Hughes 1985, Hughes and Seed 1995, Seed and Hughes 1995, Behrens Yamada and Boulding 1996). This mode of shell damage therefore remains a plausible mechanism explaining observed injury frequencies.

Mean size of *Nucella lapillus*, *Littorina obtusata* and *L. littorea* individuals did not appear to be influenced by injury history, and with the exception of *L. littorea*, increased only slightly between sampling dates. These findings suggest that injured snails are no more or less likely to succumb to abiotic or biotic sources of mortality than uninjured snails, a finding which agrees with one possible outcome of shell repair. Resistance to crushing may be augmented, diminished or unaffected by postinjury repair (Watabe 1983). At first glance these data seem to indicate that repaired shells are just as resistant to durophagous predation as undamaged shells. However, post-injury behavior may change to compensate for increased or decreased predation resistance. My survey technique only collected snails foraging away from crevices and other shelter habitats. In quadrat surveys, if injured snails of a given size spend more time sheltering, they would disproportionately go undetected relative to similarlysized, uninjured snails. However, I was actively looking for injured snails and may simply have

collected snails that had left shelter on rare forays, skewing results of mean sizes. Additionally, the principle local intertidal decapod predator, the green crab, evaluates gastropod prey based on size rather than vulnerability (Seed and Hughes 1995). These crabs also prefer gastropod prey well below the 'critical size' above which shells can not be crushed (Zipser and Vermeij 1978, Lawton and Hughes 1985, Seed and Hughes 1995). If repaired shell is either only slightly inferior or slightly superior to uninjured shell, decapod predators attacking snails with overwhelming force may be successful regardless of shell injury status. However, it is unclear whether other known snail predators adopt similar foraging strategies.

Some marine birds, including common eiders (*Somateria mollissima*), possess a grinding gizzard in which mollusc shells are broken down before being excreted. It is common for snails to pass through their digestive track relatively intact and therefore plausible that common eiders serve as another shell-injuring vector (Cadée 1994, Zuschin et al. 2003). Common eiders inhabit the survey area yearround in sizeable flocks (Peterson 1980, personal observation) and must be considered a potential source of non-lethal damage. Other shore birds which lack gizzards, namely gulls (*Larus spp.*), have been observed dropping larger snails onto rocks while airborne or simply swallowing them whole (Osborne 1977, Zuschin et al. 2003). Although it is possible that gull foraging is a source of some mortality, low observed population densities seem an unlikely cause of such drastic declines in snail densities and increases in damage frequencies.

Fish, including cunner (*Tautogolabrus adspersus*) and various flatfishes (family *Pleuronectidae*), have been known to consume snails (Bigelow and Schroeder 1953, Etter 1988b, Cadée et al. 1997). Cunner in particular possess a hard mouth-plate and grinding pharynx capable of crushing snail shells (Bigelow and Schroeder 1953) and are also abundant in the shallow subtidal during summer months (personal observation). Although earlier surveys of snail density at nearby locations (< 5 km distant) concluded that fish and bird predation were not significant sources of mortality among intertidal populations (Osborne 1977), these remain plausible shell-injuring factors.

#### Implications

Although observed shell damage at this location is potentially attributable to non-lethal predation, the exact predators and their specific strategies remain unclear. Regardless of the

mechanism of injury, a noteworthy proportion of individuals of the three dominant snail species at my representative survey site bear repair scars. Prior non-lethal predation may trigger morphological or behavioral modifications that compensate for changes in shell resistance. Such changes would likely lead to modified interactions between the snails and their predators, resources and competitors, altering food web dynamics and related community structure. My data indicate that the proportion of injured snails in populations of certain species increases throughout the summer growing season. If this proportion becomes high and injury-driven morphological or behavioral modifications are drastic, late-summer intertidal community structure may be heavily influenced by indirect effects of non-lethal predation. Therefore, snail injury history seems plausible as a driver of predator-prey interactions and community dynamics. In the second chapter, I test questions of behavioral and functional responses of snails to shell injury using laboratory and field manipulations.

		•		
Source	df	MS	F	Р
Nucella lapillus [Log(X+1)]	5	41.11	51.01	<0.0001
Survey Date	1	29.26	36.30	<0.0001
Tidal Height	2	79.48	98.60	<0.0001
Date X Tidal Height	2	7.64	9.48	<0.0001
Error	357	0.81		
Littorina obtusata [Log(X+1)]	5	52.00	90.02	<0.0001
Survey Date	1	50.28	87.05	<0.0001
Tidal Height	2	91.16	157.80	<0.0001
Date X Tidal Height	2	14.21	24.61	<0.0001
Error	357	0.57		
Littorina littorea [Log(X+1)]	5	16.48	16.29	<0.0001
Survey Date	1	2.47	2.44	0.1192
Tidal Height	2	39.92	39.45	<0.0001
Date X Tidal Height	2	0.02	0.02	0.9849
Error	357	16.49		
All snails [Log(X+1)]	5	14.90	31.67	<0.0001
Survey Date	1	40.59	86.28	<0.0001
Tidal Height	2	16.37	34.41	<0.0001
Date X Tidal Height	2	0.80	1.70	0.1838
Error	357	0.47		

Table 1: Summary of two-way ANOVAs on the effect of survey date (June, October) and tidal height (High, Mid, Low) on population densities of three snail species. Data transformations noted in brackets.

Table 2: Summary of one-way ANOVAs testing the effect of survey date (June, October) on shell damage frequencies in three snail species. Data transformations noted in brackets.

Source	df	MS	F	Р	
Nucella lapillus [arcsine]					
Survey Date	1	1.07 X 10⁻ <sup>9</sup>	8.47	0.0051	
Error	60	1.27 X 10 <sup>-10</sup>			
Littorina obtusata [arcsine]					
Survey Date	1	1.86 X 10⁻⁰	14.05	0.0008	
Error	28	1.32 X 10 <sup>-9</sup>			
Littorina littorea					
Survey Date	1	0.005	0.36	0.5509	
Error	53	0.014			
All Snails [arcsine]					
Survey Date	1	2.34 X 10 <sup>-9</sup>	21.84	<0.0001	
Error	99	1.07 X 10 <sup>-10</sup>			

Source	df	MS	F	Р	
Nucella lapillus [Log(X)]	3	0.081	2.46	0.0669	
Survey Date	1	0.154	4.66	0.0332	
Injury History	1	0.08	2.49	0.1177	
Date X Injury	1	0.009	0.27	0.6025	
Error	94	0.033			
Littorina obtusata [Log(X)]	3	0.041	4.49	0.0069	
Survey Date	1	0.106	11.52	0.0013	
Injury History	1	0.009	1.03	0.3136	
Date X Injury	1	0.007	0.72	0.3988	
Error	54	0.009			
Littorina littorea	3	5.15	0.23	0.8765	
Survey Date	1	11.41	0.50	0.4799	
Injury History	1	3.87	0.17	0.6802	
Date X Injury	1	1.95	0.09	0.7700	
Error	62	22.59			

Table 3: Summary of two-way ANOVAs testing the effect of survey date (June, October) and shell injury history (Damage, No Damage) on mean individual size in three snail species. Data transformations noted in brackets.



**FIGURE 1.** Population densities of *Nucella lapillus* (A), *Littorina obtusata* (B), *Littorina littorea* (C) and all snails (D) across date and tidal height microhabitats on a rocky shoreline in Maine. Mean  $\pm$  1 SE. Untransformed data are presented for clarity.



**FIGURE 2.** Shell injury frequencies within single populations of three intertidal snail species and the combined snail community over time. Single-factor ANOVA, mean  $\pm$  1 SE, \* denotes significance at P < 0.001, NS denotes no significant difference.



**FIGURE 3.** Mean size of individuals of injured and uninjured *Nucella lapillus* (A), *Littorina obtusata* (B) and *Littorina littorea* (C) across dates on a rocky shoreline in Maine. Mean  $\pm$  1 SE. Untransformed data are presented for clarity.

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#### CHAPTER II

#### Risk Cues and Prey State in an Intertidal Food Web

#### INTRODUCTION

The non-lethal effects of predators on prey traits are increasingly identified as contributors to variability in dynamics, structure and processes of many communities and ecosystems (Dill et al. 2003, Werner and Peacor 2003, Schmitz et al. 2008). The study of these trait-mediated interactions (TMIs) has added to an understanding of predator-prey and competitive relationships traditionally focused on theoretical and empirical approaches highlighting relative population densities as major drivers of this variability (Trussell et al. 2002, Preisser et al. 2005, Agrawal et al. 2007). In a variety of systems, workers have demonstrated that the simple presence of predators - largely independent of density - often causes prey to express defensive strategies including changes in behavior (Huang and Sih 1990, Marko and Palmer 1991, Trussell et al. 2003), morphology (Harvell 1984, Trussell 2000, Iwami et al. 2007), physiology (Rovero et al. 1999) or life history (Lively 1986, Crowl and Covich 1990). In turn, these 'induced defenses' have been shown to alter prey-resource interactions in a direction and magnitude similar to that of density-mediated trophic cascades (density-mediated indirect interactions) (Schmitz et al. 2004, Preisser et al. 2005). Ecologists have begun to synthesize these approaches by addressing simultaneous influences of trait- and density-mediated effects on food web dynamics (Peacor and Werner 2000, Bolker et al. 2003, Trussell et al. 2008).

In rocky shore food webs, predators can have strong, positive indirect effects on prey resources through trait- (Trussell et al. 2002, Trussell et al. 2006a) and density-mediated (Trussell et al. 2006a) pathways. Decapod crustacean predators in these systems may greatly alter community composition (Lubchenco and Menge 1978, Menge 1983) and energy flow (Trussell et al. 2006b, 2008). They achieve this by changing intermediate prey behavior through fear responses (Trussell et al. 2004) or controlling prey density through direct consumption (Trussell et al. 2006a) thereby releasing prey resources from consumer pressure. More broadly, empirical tests of density- and trait-mediated indirect interactions (DMIIs and TMIIs, respectively) are typically performed by exposing prey animals to predator risk cues while prey densities are manipulated to simulate density variation in natural populations. However, logistical constraints often require common but less conspicuous aspects of prey populations other than

these two main effects to be left out of experimental designs. In this study, I examine the role of one such overlooked factor in a system where TMIIs are known to operate.

Variation in physiological condition of individual prey animals - prey state - has the potential to play a role in the risk/reward optimization that ultimately affects fitness and can influence community structure (Mangel and Clark 1986, Luttbeg et al. 2003). Although individual condition has been the subject of much experimental and theoretical work as it relates to life-history theory (McNamara and Houston 1996), it has received considerably less attention in the context of community ecology. Theory predicts that predators should target relatively young, sick, slow or otherwise vulnerable prey items in order to minimize energy expenditures (Murray 2002, Perlman and Tsurim 2008). These prey should similarly alter their perception of predation risk based on their physiological condition. Noteworthy observational and experimental efforts in this area use state variables such as relative prey size (Freeman 2006, Ovadia & Schmitz 2002, 2004), hunger level (Palmer 1990, Heithaus et al. 2007) and reproductive status (Murray 2002) to draw inferences about the broader community effects of prey state. However, each of these factors can only affect a subset of a given population at any one time. Young animals are typically smaller, poor competitors typically go hungry and females typically bear the major physiological costs of reproduction. Unlike other state variables, non-lethal injury through unsuccessful predation, accident or abiotic action has the potential to strike any member of a population, irrespective of overall condition. Hence, this particular state variable may be an important influence on prey behavior, TMIIs and the broader structure of communities and ecosystems.

Study of non-lethal injury has devoted considerable attention to the physiological phenomenon of autotomy, largely in reptilian vertebrates, marine & terrestrial arthropods and echinoderms (Harris 1989, Juanes and Smith 1995, Maginnis 2006). The ability to disconnect an appendage along an evolutionarily predetermined breakage plane provides the short-term benefits of evading predators, escaping difficult molting events or rapidly closing wounds. It also carries long-term costs; chief among these are reduced mobility, loss of stored energy reserves and energetic costs of regenerating lost appendages (reviewed in Maginnis 2006). Hence, autotomy represents a convenient phenomenon for exploring restrictions of mobility and their impact on species interactions. However, autotomizing animals can choose the precise point and timing of limb separation during predator attacks; natural

selection may likely have acted to dampen costs of limb loss. Hence, autotomy may not be a valid general proxy for non-lethal injury because many animals lack the ability to choose the location, timing and severity of injury.

A more broadly applicable model for testing indirect community effects of injury and predation risk may lie in the repaired shells of gastropod molluscs. Repair scars from shell injuries persist throughout the life of these animals and represent a permanent change in prey state relative to predamage condition. Most animals have little control over the body location where wounds are suffered; likewise shell injury may occur at any point on the shell, not just at specific breakage points. Whether the cause of shell injury stems from abiotic conditions, such as intense wave action, or from encounters with durophagous (shell-breaking) predators, damaged gastropods potentially perceive a permanent change in shell integrity, even following complete repair.

A history of shell damage may improve, worsen or have no impact on resistance, depending on the repair mechanism (Watabe 1983). The few experimental studies that have addressed this issue in gastropods (Zipser and Vermeij 1980, Blundon and Vermeij 1983, Geller 1990, Greenfield et al. 2002) disagree on the anti-predator effectiveness of repaired shells. Zipser and Vermeij (1980) were unable to detect any difference in mortality rates or size-frequency distributions of experimentally damaged and repaired *Conus sponsalis* in a mark-recapture experiment. Conversely, Geller (Geller 1990) found that experimentally damaged *Nucella emarginata* fared significantly worse than undamaged snails in a similar mark-recapture study. In marsh periwinkles (*Littorina* (= *Littoraria*) *irrorata*), shells collected in the field and quantitatively crushed showed that repaired shells were as strong as undamaged shells from the same populations (Blundon and Vermeij 1983). However, an experiment where field-collected marsh periwinkles were offered to blue crabs (*Callinectes sapidus*) found that snails without repair scars were consumed more frequently (Greenfield et al. 2002).

Regardless of the functional outcome of repair, an injured animal potentially experiences an altered perception of its risk environment. A history of injury may correspond to long-term phenotypic changes, particularly in morphology or behavior. Phenotypic changes have the potential to affect the strength of interactions between gastropods and their predators as well as gastropods and their prey.

In localities where a substantial proportion of prey species are injured, phenotypic responses to injury may have implications for community structure and ecosystem processes.

This study explores the importance of prey state as represented by shell injury in influencing prey traits and prey resource densities in the context of a tri-trophic food chain where TMIIs have been documented. In a laboratory mesocosm experiment, I allowed injured and uninjured snails (*Nucella lapillus*) to feed on mussels (*Mytilus edulis*) in the presence and absence of risk cues emitted by predatory crabs (*Carcinus maenas*). Specifically, I measured growth, energy assimilation efficiency and shell strength to determine the relative contributions of risk cue and prey state to overall indirect effects of predators. Finally, I tested the functional influence of shell repair on mortality rates by subjecting *Nucella* to natural predation conditions in the field.

#### MATERIALS AND METHODS

#### Effects of risk cues and prey state: experimental design

In a laboratory mesocosm experiment, I tested the effects of predator risk cues and prior nonlethal damage on growth, energy assimilation efficiency and shell strength of *Nucella lapillus* in a simple food chain. The green crab (*Carcinus maenas*) served as a source of chemical risk cues for the carnivorous dogwhelk (*Nucella*) which itself fed on a basal resource, the blue mussel (*Mytilus edulis*). I randomly applied two experimental treatments, predator cue ('Crab' & 'No Crab') and artificially manipulated prey shell condition ('Damage' & 'No Damage') to separate containers in a fully-crossed design with 8 replicates per treatment level.

Each replicate mesocosm consisted of a clear plastic container (27 X 15 X 5 cm) divided into two sections. The first section (11 X 15 X 5 cm) was designed to hold crabs (crab chamber) and was attached to an independent water supply via a small hose. This chamber had a clear plastic roof and held either green crabs fed 3 *Nucella* each week ('Crab' treatment level) or just 3 *Nucella*, changed each week ('No Crab' treatment level). The second chamber (16 X 15 X 5 cm) housed both mussels and *Nucella* (snail chamber) and was separated from the crab chamber by a perforated divider across which flowed water. A roof of plastic mesh (3.75 X 2.90 mm) permitted water to exit the container through this chamber. I placed each container within a larger basin (35 X 15 X 5 cm) to prevent contamination of 'No Crab' replicate containers with crab chemical cues. This design ensured the constant

downstream transport of crab chemical cues into the snail chamber while preventing physical contact between crabs and experimental snails.

I collected young *Nucella* (14.07 ± 0.11 mm shell length [mean ± SE]) and small mussels from the same rocky shore where I surveyed damage frequencies (Chapter I) near New Harbor, Maine. These were immediately transported to flow-through seawater tables at Northeastern University's Marine Science Center in Nahant, Massachusetts. Mussels were sorted to size (1 - 2 cm shell length) and then added to replicate containers. Predator cues are known to affect mussel physiology (Rovero et al. 1999), and over time, this may alter energy content of tissue consumed by snails. To minimize this potential effect, I stocked each replicate with 50 mussels initially and added 50 mussels each week for the duration of the experiment. All containers were cleaned and maintained weekly.

After starving snails for seven days to standardize hunger, I damaged one half of experimental animals by removing shell along the apertural lip using a small pair of bone shears and taking care not to remove material from the siphonal canal as this would have affected length measurements. After morphological measurements were made, Damage and No Damage snails were immediately added to appropriate containers. Ten snails were added to each replicate container, approximating *Nucella* densities in the field (-420 m<sup>-2</sup>). Three of these snails were numbered and changes in their morphological traits were tracked. The experiment was conducted over 60 d between late July and late September 2008 when water temperatures were at or near their yearly maximum (18.2  $\pm$  0.3°C [mean  $\pm$  SE]).

#### Snail morphometrics

I was interested in the effects of predator risk cues and physiological state on snail foraging behavior. These snails have been shown to alter energy allocation among shell and tissue mass growth in the presence of predation risk. Hence I measured the strength of these two indirect effects using changes (growth) in three snail morphological traits (shell length, shell mass and body tissue mass) as proxies for risk response behavior. First, I measured maximum shell length (apex to tip of the siphonal canal) at the outset and end of the experiment using Mitutoyo electronic calipers (± 0.01 mm). To separate changes in shell mass and body tissue mass without sacrificing snails, I applied the buoyant weighing technique first used by Palmer (1982) and modified by Trussell (1996). In brief, live snails

were first weighed in air and then while submerged in room-temperature seawater using an electronic balance (Mettler-Toledo PG 503  $\pm$  0.0001g). Snails of various sizes from the same population were destructively measured wherein submerged shell mass (65 - 5229 mg wet tissue) was related to true, dry shell mass using linear regression (Eq 1). Subtracting true shell mass from mass in air yielded wet body tissue mass which was likewise compared to destructively-determined dry body tissue mass with linear regression (Eq 2).

(Eq 1) dry shell mass = 1.5632604 (wet shell mass) - 4.022792 (R<sup>2</sup> = 0.9998, P < 0.0001, N = 48)

(Eq 2) dry tissue mass = 0.3179636 (wet tissue mass) - 9.437759 (R<sup>2</sup> = 0.9804, P < 0.0001, N = 48)

I attempted to standardize the amount of shell material removed from snails in Damage treatments; snails were clipped, returned to water for > 4 hr and the weighing procedure repeated (shell mass removed =  $9.1 \pm 0.5\%$  [mean  $\pm$  SE]). Snails in No Damage treatments were similarly handled. The weighing process was repeated at the end of the experiment. Mean growth in shell length, shell mass and tissue mass was obtained by subtracting initial values from their respective final values. Across all treatments, there were no significant pre-damage differences in shell length (P = 1.00), shell mass (P > 0.98) and tissue mass (P = 1.00) nor did the amount of shell removed among Damage treatment levels differ significantly (P > 0.15).

To determine relative shell strength in compression, I used an Instron dynamic testing machine (model 5582,  $\pm$  0.0001 N) to record the force necessary to crush shells. Frozen snails were thawed in air for one hour and then placed aperture-down on a stationary steel plate while a second, opposing plate was lowered from above at a rate of 10 mm min<sup>-1</sup>. Shells emitted an obvious cracking sound at the moment of failure and I verified maximum compressive load (N) by examining load:deformation curves produced for each snail. This test was used simply to approximate the relative strength of shells among

experimental treatments. Hence, I did not attempt to mimic different green crab shell breaking strategies (Boulding and LaBarbera 1986) nor their respective force characteristics.

#### Foraging and indirect effect sizes

Mussels served as the food resource for experimental snails. When consumed, their empty shells bear obvious bore holes inflicted by snails. I recorded the number of mussels consumed in each mesocosm and used this value to calculate per capita snail foraging rates. Frequently, leftover mussel shells showed no obvious signs of predation ( $18.4 \pm 0.01\%$  [mean  $\pm$  SE]) such that cause of death, either from predation or natural mortality, could not be determined. I considered these mussels to be consumed because *Nucella* have been shown to enter shells without boring and to scavenge upon dead mussel flesh (Rovero et al. 1999).

In estimating the proportional effects of predator risk cues (risk cue effect, RCE) and prey physiological condition (physiological state effect, PSE) on per capita amount of mussel resources (R) consumed by snails, I followed the approaches of Peacor & Werner (2004) and Trussell et al. (2006b, 2008).

(Eq 3) RCE = Risk Cue Effect = 1 - ( $R_{Crab, No Damage}/R_{No Crab, No Damage}$ )

(Eq 4) PSE = Physiological State Effect = 1 - ( $R_{Damage, No Crab}/R_{No Damage, No Crab}$ )

These proportions give a reasonable indication of the magnitude of the impact that each stressor has on foraging behavior. Numerators in each of these proportions were the number of mussels consumed per capita for the appropriate treatment combination in each replicate container. Denominators were the number of mussels consumed per capita, averaged across all replicates of the appropriate treatment level. Hence, resulting values range between 0 and 1 with a value of 0.0 indicating no reduction on snail foraging attributed to a given stressor and 1.0 indicating a 100% reduction (Trussell et al. 2008). This generates a value for overall effect of risk cue or prey state on snails in each replicate container, yielding eight estimates of each effect size that can then be analyzed with ANOVA.

#### Energy assimilation efficiencies

Using previously identified relationships between shell length and dry tissue mass for *Mytilus edulis* in the Gulf of Maine and energetic equivalents (Anala 1974), I was able to estimate per capita energy intake of snails over the 60 d duration of the experiment. I should note that in recent years *Mytilus trossulus* has been detected near my mussel collection sites among historical monocultures of *Mytilus edulis* and are estimated to make up as much as 5% of the mussel population in this region (P. Yund, personal communication). However, as *M. trossulus* was likely present in my mussel collections in small proportions if at all, I proceeded with assimilation calculations based on energetic equivalents for *M. edulis* alone. Shell lengths of all consumed mussels in each replicate were measured and converted to dry tissue mass equivalents (in milligrams) and then into energetic equivalents (20.28 J mg<sup>-1</sup>; Anala 1974). Total energy consumed in each replicate was divided by the total number of snails per replicate, yielding per capita energy consumption.

A similar technique was used to estimate energetic equivalents of *Nucella* shell and body tissue. I converted estimated (wet) shell mass to true (dry) shell mass equivalents (Eq 1, above) and then to energy (0.381 J mg<sup>-1</sup> shell; Burrows and Hughes 1990). Similar mass-to-energy conversions were made for *Nucella* body tissue (Eq 2, above, 22 J mg<sup>-1</sup> tissue; Burrows and Hughes 1990). Energy converted to biomass over the course of the experiment was determined by simply subtracting initial energy values for shell and tissue from final values. I then calculated "snail growth efficiency" following Trussell et al. (Trussell et al. 2006b, 2008). This is defined as the proportion of energy obtained from mussels converted by snails into tissue and shell biomass (per capita tissue & shell produced, in joules)/(per capita mussel tissue consumed, in joules).

#### Prey state and relative mortality on a rocky shoreline

To track the relative resistance of damaged and undamaged shells to durophagous predators, I tethered live *Nucella* to intertidal substrate during three separate trials in October 2008. This experiment consisted of a single treatment with three levels: undamaged snails, recently damaged snails and snails that were experimentally injured but allowed to repair completely. Snails were collected from the same rocky shoreline in Maine noted above, transported to Massachusetts and sorted by shell length (16.43  $\pm$  0.17 mm [mean  $\pm$  SE]). One week prior to tethering, shells of 'Repair' snails

were damaged along the apertural lip using bone shears, returned to a holding tank and allowed to repair. One day prior to tethering, 'Damage' snails were injured in an identical manner; 'No Damage' snails were similarly handled, though not injured. On a protected shoreline at Galloupes Point, Swampscott, Massachusetts (42.463°N, 70.897°W) at which *Nucella* are present and green crab densities are locally high, I used a marine epoxy (KOP-COAT A-788 splash zone compound) and nylon line (0.2 m length) to affix snails to rocky substrate. Tethers were placed haphazardly and spaced approximately 0.5 - 0.75 m apart in the lower-mid intertidal zone on a continuous granitic shelf dominated by the macroalga *Ascophyllum nodosum*. On October 10 and 21 2008, 39-50 individuals of each treatment level were deployed in the field for approximately 48 hours (4 tidal cycles). On 3 October, Repair snails were unavailable and only No Damage and Damage snails were used. During retrieval, snails of each treatment were scored as either dead or alive. Where tethers were missing or were not attached to any shell or fragment, mortality could not be verified and these individuals were excluded from analysis. *Statistical analyses: effects of risk cues and prey state* 

Changes in *Nucella* morphology (shell length, shell mass and body tissue mass) were analyzed using two-way ANCOVAs that treated risk cue (Crab, No Crab) and prey condition (Damage, No Damage) as fixed effects. Initial, post-injury values for each response trait served as covariates. As there were multiple snails in each mesocosm I treated replicate containers as random effects nested within each treatment in these analyses. Response variables were Log+100 transformed to satisfy the model assumption of homogeneity of variances (Zar 1999)(Levene's test; all P >0.209).

I analyzed differences in shell strength in compression (maximum compressive load) and snail growth efficiency (shell growth efficiency, tissue growth efficiency and their sum - total growth efficiency) using two-way ANOVAs that treated risk cue and prey condition as fixed effects and replicate containers as random effects nested within these two treatments. Where necessary, response variables were arcsine transformed to satisfy assumptions of ANOVA (Levene's test; all P > 0.021). With respect to shell strength, I was chiefly interested in the absolute value among treatments because this is the value that determines overall resistance to predation. Hence, I did not adjust for final differences in size, mass or other potential traits which may have influenced strength on a per-unit basis. The size of the effects on both the number of mussels consumed and the amount of energy

acquired were analyzed with one-way ANOVA that treated effect size (Risk Cue Effect, Physiological State Effect) as a fixed effect. Data were arcsine transformed to satisfy test assumptions (Levene's test; all P > 0.019).

#### Statistical analyses: prey state and relative mortality on a rocky shoreline

Environmental conditions varied greatly during my three tethering trials. Statistical comparisons of relative mortality among damage treatments across these dates would be uninformative and hence, were not performed. Within experimental runs, I analyzed different shell injury treatments in terms of relative survival rates using the chi-square test. Where the experimental trial had only two treatment levels (3 October), adjustments were made using the Yates correction for continuity (Zar 1999).

#### RESULTS

#### Effects of risk cues and prey state

*Nucella* raised in the presence of predator risk cues exhibited very little growth, adding only 12.1% as much body tissue (Figure 1), 22.2% as much shell and 19.7% as much overall length as controls. At the end of 60 d these snails exhibited only 38.0% as much body tissue mass (ANOVA,  $F_{31,59} = 10.58$ , P < 0.0001) and were only 3.5% as efficient at translating acquired energy into body tissue (Figure 2) as controls raised in the absence of these cues (Table 1). Predator risk cues did not seem to affect shell deposition efficiency. Prey physiological state had no detectable effect on any of these traits, nor was there any significant interactive effect between predation risk and prey condition (Table 1). Predator risk cues had a 23.9-fold greater influence on indirect effects reducing *Nucella* mussel consumption (77.7 ± 2.5% [mean ± SE]) than did prey state (3.2 ± 2.7% [mean ± SE]). Risk cues accounted for 71.8 ± 1.9% (mean ± SE) of indirect effects on decline in energy intake while prey state explained 3.4 ± 2.0% (mean ± SE), a 21.1-fold difference (Figure 3).

In compression, shells that had been injured and repaired over the 60 d of this experiment were only 77.4% as strong as snails that had never been injured. Snails exposed to predation risk were likewise only 68.5% as strong as snails that were not exposed (Table 1), though these exposed snails achieved only 72.6% of the shell length (ANOVA,  $F_{31,59} = 11.28$ , P < 0.0001) and 40.5% of shell mass (ANOVA,  $F_{31,59} = 10.47$ , P < 0.0001) of controls at the end of 60 d. A non-significant interaction term (Table 1) prevented the use of *post hoc* multiple comparisons; it appears that the main effects of predation risk and prey condition on absolute shell strength are additive (Figure 4).

#### Prey state and relative mortality on a rocky shoreline

Separate chi-square analyses of tethered snail mortality on 3 October ( $X^2 = 0.184$ , df = 1, P = 0.67), 10 October ( $X^2 = 2.211$ , df = 2, P = 0.33) and 21 October ( $X^2 = 0.103$ , df = 2, P = 0.95) indicate no significant differences among damage treatments. Ignoring unquantified variability in biotic and abiotic conditions at the tethering site between tethering trials and pooling my data, no relationship emerges ( $X^2 = 1.41$ , df = 2, P = 0.49). Predators, most likely green crabs (*Carcinus maenas*) showed no preference for *Nucella* in a particular condition. Depending on the trial, I recovered between 45 - 75% of snails deployed. Some tethers remained in place but held no snail or shell fragment. Including these, my effectiveness at relocating tethering sites ranged between 69 - 81%.

#### DISCUSSION

The significance of prey state as a factor influencing community structure and ecosystem processes by altering predator-prey interactions has received little empirical attention to date. A large body of work exists that recognizes prey state variables as important drivers of population dynamics, life history processes and optimal foraging behavior. However, explicit tests linking identified prey state effects on individual fitness with broader community impacts in the context of predation remain few (but see Ovadia and Schmitz 2002, 2004, 2007). Using a single, discrete variable - injury presence or absence - as a measure of prey state, I tested the effect of individual physiological condition in the context of a tri-trophic food chain where risk of predation has been shown to alter community dynamics. Despite its prevalence in natural communities and observed weakening influence on shell strength, shell injury had no discernable impact on prey behavior or trait-mediated indirect interactions.

A sizeable body of work has demonstrated behavioral and morphological effects that crab predation risk cues have on foraging behavior of *Nucella lapillus* (Vadas et al. 1994, Trussell et al. 2003, Trussell et al. 2006a, Trussell et al. 2006b, 2008), its congeners (Appleton and Palmer 1988, Palmer 1990, Marko and Palmer 1991) and other intertidal gastropods (Trussell et al. 2003). In general, these studies document a marked and immediate decline in snail activity levels when chemical cues

from certain known predator species (Marko and Palmer 1991) or injured conspecifics (Appleton and Palmer 1988, Vadas et al. 1994) are introduced into the test environment. Snails reduce foraging or increase hiding, thereby relieving snail resources from consumptive pressure. These prey resources themselves experience a corresponding increase in survivorship. Over time, reduced activity and/or physiological responses to risk cues correspond with morphological changes in size, shape and shell architecture & ornamentation (Palmer 1990). These ecophenotypic adjustments by the prey ostensibly improve their resistance to predators. The now-altered predator-prey interaction serves as the pathway by which TMIIs are transmitted from crab predators to snail resources and lower trophic levels. State variables have the potential either to amplify or dampen the effects of predator risk cues on snail behavior and in this capacity, they bear exploration as drivers of community dynamics and limiters of energy flow.

I selected shell injury as a prey state variable because it likely represents a discrete and permanent change in the defensive capacity provided by shell architecture. Other studies addressing prey state included continuous or ultimately uncontrolled state variables such as age (Cam et al. 2002), energy reserves (McNamara and Houston 1987, Shertzer and Ellner 2002), reproductive status (Murray 2002) or parasite load (Bustnes and Galaktionov 2004). Although each seems a valid indicator of prey state, standardizing these for use in experiments may prove difficult, especially as many of these variables interact. For example, age, ability to store energy, reserve levels and reproductive capacity are likely linked. Inferences drawn from experimental outcomes attributed to these continuous variables may therefore be inaccurate, particularly when they are carried on to explain structure and processes of broader communities and ecosystems. Conversely, standardized injury to the shell has the potential to serve as a state variable relatively free of these encumbrances for a number of reasons. Within 36 hr of shell lip injury, I witnessed my experimental Nucella covering the gap in apertural lip with a thin layer of new material, irrespective of risk environment. Within 7 d in nearly all snails, this repair achieved qualitatively similar thickness and durability to surrounding, undamaged shell. However, the repair scar remained visible in all injured snails throughout the subsequent 53 d of trial. My experimental damage technique is therefore instantaneous and rapidly addressed by the animal but persists in the history of the shell.

Shell material itself is not vascularized and is dominated by cheaply produced inorganic CaCO<sub>3</sub> (Palmer 1983, Avery and Etter 2006). Additionally, the energetically expensive proteinaceous fraction of *Nucella* shell is typically small (Palmer 1983) and its absolute thickness does not vary dramatically among a range of ecophenotypes (Avery and Etter 2006). Hence, shell makes up a relatively low proportion (< 5%) of total metabolic costs (Palmer 1992). Unlike autotomy, which often reduces foraging efficiency and is linked to costs of regeneration (Maginnis 2006), interpretations arising from experimental use of shell damage are not as likely to be confounded with other prey state variables.

My experiment detected no effect of shell damage on growth in *Nucella* shell mass, tissue mass or overall length, nor did I see any effect on resource (mussel) consumption when compared to appropriate control treatments. As expected, and in agreement with previous works (Palmer 1990, Marko and Palmer 1991, Trussell et al. 2003, Trussell et al. 2006a, Trussell et al. 2006b), predation risk cues brought about notable reductions in each of these traits. Taken together, these data suggest that snails which have experienced shell damage are no more sensitive to risk than snails with no history of injury. Overall, effect sizes indicate that state-dependant influences on trait-mediated reductions in mussel consumption and energy intake are negligible (3.2% & 3.4%, respectively) while reductions mediated by risk cues are extreme (78% & 72%, respectively).

While my data revealed marked reductions in tissue growth efficiency and total efficiency in the presence of predation risk, prey state had no noticeable effect. As previously suggested (Trussell et al. 2006b, 2008), several aspects of prey response to predation risk could play a role in limiting efficient energy translation into body tissue. Mussels respond to both *Nucella* cues and attacks by increasing heart rate (Rovero et al. 1999) which can be used as a metric of metabolic rate. If *Nucella* exhibits a similar response, maintenance metabolic demands in risky environments would command a greater proportion of energy budget that might otherwise be used for growth. If this elevated metabolism persisted for the duration of cue presence, over time the implications for growth efficiency would likely be profound. Additionally, the cues themselves may cause reallocation of metabolic resources to producing defensive structures independent of food intake (Palmer 1990).

Differences in activity levels could likewise explain this pattern. *Nucella* foraging behavior can be quite variable (reviewed in Crothers 1985). Several studies have addressed activity patterns in this

genus while under predation risk. Working in a vertical flume setup, Vadas et al. (Vadas et al. 1994) found that over short (3 hr) time scales a greater proportion of mussel-fed snails remained stationary in a submerged sheltered habitat when predator and damaged conspecific cues were present. Of those that left shelter under predation threat, juveniles (< 20 mm SL) were significantly more likely to retreat to shelter above the water line while adults (20 - 36 mm SL) were not. Trussell et al. (Trussell et al. 2008) determined that following three days of risk cue exposure, the proportion of *Nucella* feeding differed little among cue-exposed and risk-free treatments. Together, these studies suggest that juvenile *Nucella* feeding rates decline briefly at the outset of cue exposure and soon return to precue activity levels. Hence, maintaining activity levels while enduring increased metabolic costs over long periods is one potential mechanism explaining my observed growth efficiency reductions.

Allometric scaling of metabolic rate with body mass may also contribute to declines in efficiency. Ontogenetic increases in an animal's mass correspond with an increase in overall metabolic rate but a *decline* in metabolic rate per unit mass (metabolic intensity) (Czarnolęski et al. 2008). Consumption rates also decline with growth, though not at the same rates (Ovadia and Schmitz 2007). Though not a universal phenomenon (Glazier 2006) there is evidence that allometric scaling equations can be applied to molluscs (Vladimirova 2001), gastropods (Czarnolęski et al. 2008) and *Nucella lapillus* in particular (Bayne and Scullard 1978). Shell material, once produced, requires no metabolic maintenance and I observed no differences in efficiency of shell material production. This suggests that overall differences in efficiency stem from dissimilarities in soft tissue maintenance costs and reinforces the applicability of allometric scaling of metabolic rates as an underlying mechanism. Therefore, it seems plausible that differences in body tissue mass - either through this relationship alone or interacting with other potential mechanisms mentioned above - could contribute to observed differences in growth efficiency. However, I can not state with certainty the exact mechanism responsible for this pattern.

Prey state as represented by shell damage had no influence on my metrics of behavior, indirect effect size, or growth efficiency. This is surprising to me given compression testing after 60 d revealed that damaged shells - all of which repaired rapidly - were only 77% as strong as undamaged shells. Given that repaired shells are at a functional disadvantage independent of predation risk, I could

reasonably expect to see some type of compensatory behavioral or morphological change following damage, but none emerged. There are several plausible explanations for this. Risk cue-raised *Nucella* are known to add defensive structural elements to their shell in the form of larger teeth and a thicker lip at the aperture, even without any energy intake (Palmer 1990). Because green crabs often attack larger shells by focusing on the aperture (lip or columella) rather than crushing the body whorl (Hughes and Elner 1979, Lawton and Hughes 1985, Seed and Hughes 1995), my snails may have mounted a morphological response to this specific predator by bolstering defenses at the apertural lip. Snails in my predation risk treatment remained relatively small ( $15.44 \pm 0.21$  mm final SL [mean  $\pm$  SE]), and large green crabs access prey of this size by crushing the body whorl (Hughes and Elner 1979), negating any advantage of improved defenses at the apertural lip. If predator-specific changes to shell architecture are occurring, they would likely relate to snail size. In Crab/Damage treatments - where snails were smallest and weakest - I might have expected to see further reductions in foraging or increases in overall shell mass or length as a general defense strategy, but I did not. In any case, any advantage to shell resistance of unquantified induced defensive traits would likely have been detected by my compression testing methods.

The values obtained from compressive load testing are applicable because of the manner in which gastropods repair damaged shell when it occurs at the apertural lip. *Nucella* shell consists of two layers; an inorganic outer section consisting of aragonite crystals and a thin inner layer made up of crystals imbedded in an overlapping lattice of protein (Palmer 1983, Avery and Etter 2006). When broken clean through at the lip, the calcium-secreting epithelium of the mantle can not simply join new material into the existing crystalline and protein matrices (Andrews 1934, Watabe 1983). Instead, the mantle deposits another double layer of inorganic and organic material just proximal and interior (in cross-section) to the broken shell edge. For a short distance, the shell is four layers thick but as new shell is extended beyond the edge of the break, it resumes a two-layer microstructure. The added shell is unable to bind seamlessly with material at the broken edge, forming the scar and, potentially, a weak point in the structure (Andrews 1934). Though a scar is visible externally, it may be seamless internally, raising the possibility that the snail detects the scar only for the duration of the repair.

marsh periwinkle *Littorina* (= *Littoraria*) *irrorata* using similar techniques (Blundon and Vermeij 1983). However, the authors note the high content of flexible, proteinaceous material in these shells and speculate that outcomes may be otherwise for taxa with differing shell microstructure. Similar repair microstructure has been observed among marine and terrestrial gastropod taxa (Andrews 1934, Geller 1982, Palmer 1983) and weakness at the point of repair would likely override benefits of other architectural defenses. My compressive load testing methods would likely detect these weaknesses.

My tethering experiment evaluated the implications for weaker shell when confronted with predation under field conditions. Interestingly, here too I failed to see an effect of prey state; mortality rates were no different among uninjured, fully repaired and recently-damaged shells. Shellinjuring forces at my tethering location are ambiguous, though observed green crab densities are locally high at this site and it is likely that these predators were largely responsible for observed mortality. The most likely explanation for the lack of an effect is that many crab species, including green crabs, are known to feed on molluscan prey that are well below critical size, e.g. the maximum size they are capable of subjugating (Zipser and Vermeij 1978, Lawton and Hughes 1985, Seed and Hughes 1995). Green crabs foraging on mobile prey with patchy distributions, such as *Nucella*, often abandon prey items that are difficult to access (Hughes and Elner 1979). In the context of optimal foraging theory, predators maximize energy intake per unit effort by selecting prey that can be located and opened quickly but still provide sizeable energy gain (Elner and Hughes 1978, Sih 1980). An addendum to this explanation is that the risk of injury to crab chelae and loss of function and energy intake during the recovery period may prevent crabs from attacking prey closer to the critical size (Juanes and Smith 1995, Seed and Hughes 1995). My tethered snails were of intermediate size (16.43 ± 0.17 mm SL[mean ± SE]) but still below the minimum shell length at which large green crabs consistently open thin-shelled Nucella morphs (Hughes and Elner 1979, their figure 1). Hence, differences in shell strength on the order of 20% are unlikely to influence green crab predation mortality rates in snails of this size.

It seems prey state as represented by shell injury has little-to-no importance as either a transmission or interference mechanism for trait-mediated, predator-prey effects on community dynamics or ecosystem processes because injury state did not influence behavior, efficiency or

survival. With the present data, I am unable to address questions of density-mediated effects, though previous work on *Nucella* foraging in experimental mussel beds showed no impact of conspecific density on resource consumption (Trussell et al. 2008). However, in interactions where predators evaluate their prey based on structural vulnerability the implications of density-mediated interactions may be strong (Hughes and Seed 1995). Given shell damage frequencies seen in my survey data, I would not expect that distributions and abundances of rocky intertidal resources would be altered through existing trait-mediated interactions by major changes in the proportion of *Nucella* with shell damage, though this may not hold true for other intertidal snail species. Similarly, I would not expect shell injury to have an impact on energy transfer among trophic levels. Tissue growth efficiency did not vary among damage treatment levels, nor did I observe any effect on absolute tissue growth rates. As suggested previously, higher order predators can potentially limit food chain length by limiting prey growth efficiency, thereby decreasing energetic quality of their prey and the total transfer of energy among trophic levels (Trussell et al. 2006b, 2008). Additionally, prey animals trading ultimate fitness for immediate survival by remaining in refugia and foregoing feeding have the potential to further limit total energy transfer (see Trussell et al. 2006b, 2008 for further discussion on this subject), yet shell injury does not appear to be a driving factor in these relationships.

If anything, the lack of any noticeable effect of the marked reductions in shell resistance related to shell injury on interspecific interactions reinforces the importance of anti-predator behavior in community and ecosystem organization. Predation risk environment had the greatest indirect effect on foraging rates and growth efficiencies in *Nucella*. Snails in risk cue treatments fed and grew little. Though this lack of growth may decrease fitness due to reduced reproductive output, it may also greatly improve their odds of surviving for a number of reasons. First, small size provides access to a greater number of refuge habitats. Second, energetic requirements are lower in an absolute sense, and prey need not venture into risky environments to forage as often. Third, green crabs have claws of intermediate dexterity relative to other decapods (Seed and Hughes 1995) and the likelihood of being dropped during an attack and escaping is greater for smaller snails, though this will vary with relative size of the predator. Finally, by remaining small, snails may cause generalist predators including green

crabs to shift their prey preference and concentrate on other local species such as mussels and barnacles (Seed and Hughes 1995).

My work supports earlier conclusions on the role of prey state in influencing TMIIs in that prey physiological condition does not seem to affect broader community or ecosystem structure and processes (Ovadia and Schmitz 2002, 2004, 2007). These earlier works used herbivore size as a state variable to help determine whether mathematical models and computer simulations needed to include parameters of individual phenotype to predict direction and strength of community and ecosystem dynamics. However, size is often confounded with a number of other continuous variables including reproductive state, competitive ability, social status and energy reserves. Interference among these may have produced the lack of a state-dependent effect seen in other studies. I attempted to address these questions by simplifying state-dependant interactions using shell injury status as a discrete variable, relatively independent of other indicators of prey state. Further experiments are needed that disentangle the interrelationships between size and other common state variables before ecologists can safely discount condition of individuals when considering community and ecosystem dynamics.

**Table 1:** Summary of two-way ANCOVAs and ANOVAs testing the effect of risk cue (Crab, No Crab) and prey state (Damage, No Damage) on growth (ANCOVAs), effect sizes and ecological efficiency (ANOVAs) in *Nucella lapillus*. Covariates are initial trait values taken immediately following experimental injury. Data transformations are noted in brackets.

Source	df	MS	F	Р	
Tissue mass growth [Log(X+100)]	33	0.23	12.45	<0.0001	
Risk	1	4.89	214.94	<0.0001	
State	1	0.003	0.14	0.7144	
Risk X State	1	0.01	0.42	0.5209	
Replicate (nested)	28	0.02	1.25	0.2349	
Covariate [Log(X+100)]	1	0.27	14.41	0.0004	
Slope	1	< 0.001	0.004	0.9477	
Frror	57	0.19	0.001	017117	
	57	0.17			
Shell mass growth [Log(X+100)]	33	0.20	10.89	<0.0001	
Risk	1	5.39	211.44	<0.0001	
State	1	< 0.001	0.006	0.9379	
Risk X State	1	< 0.001	0.02	0.8988	
Replicate (nested)	28	0.03	1 47	0.1318	
Covariate $[l og(X+100)]$	1	0.008	0.44	0.1910	
	1	0.000	1 22	0.3073	
Frror	57	0.02	1.52	0.2333	
LITOI	57	0.02			
Shell length growth [] og(X+100)]	22	25.06	15 98	<0.0001	
	1	738.68	361.85		
State	1	0.01	0.01	0.0001	
Diale V State	1	0.01	0.01	0.9340	
RISK A State Deplicate (rested)	1	0.03	0.20	0.0103	
Replicate (nested)	28	2.05	1.31	0.1928	
Covariate [Log(X+100)]	1	8.41	5.30	0.0242	
Slope	1	0.85	0.54	0.4651	
Error	57	1.5/			
Effect sizes					
Mussel consumption [arscine]		2.04	100.10	0.0004	
Model	1	2.06	409.12	<0.0001	
Error	13	0.005			
Energy acquisition [arcsine]					
Model	1	1.86	521.49	<0.0001	
Error	13	0.003			
		• • • •		0.0004	
Issue efficiency [arcsine]	30	0.11	3.74	<0.0001	
Risk	1	2.30	76.75	< 0.0001	
State	1	0.01	0.32	0.5737	
Risk X State	1	0.01	0.35	0.5570	
Replicate (nested)	27	0.03	1.02	0.4559	
Error	57	0.03			
			–		
Shell efficiency	30	< 0.001	1.17	0.2977	
Risk	1	< 0.001	4.34	0.0459	
State	1	0.001	0.79	0.3813	
Risk X State	1	< 0.001	0.18	0.6704	
Replicate (nested)	27	< 0.001	1.07	0.4071	
Error	57	< 0.001			

**Table 1 (continued):** Summary of two-way ANCOVAs and ANOVAs testing the effect of risk cue (Crab, No Crab) and prey state (Damage, No Damage) on growth (ANCOVAs), effect sizes and ecological efficiency (ANOVAs) in *Nucella lapillus*. Covariates are initial trait values taken immediately following experimental injury. Data transformations are noted in brackets.

Source	df	MS	F	Р	
Total growth efficiency [arcsine]	30	0.12	3.74	<0.0001	
Risk	1	2.45	72.2	<0.0001	
State	1	0.01	0.21	0.6498	
Risk X State	1	0.01	0.28	0.6006	
Replicate (nested)	27	0.03	1.05	0.4184	
Error	57	0.03			
Absolute shell strength	30	6264.2	2.54	0.0013	
Risk	1	60186	22.34	<0.0001	
State	1	27793	10.3	0.0032	
Risk X State	1	8521.3	3.16	0.0857	
Replicate (nested)	27	2704.1	1.10	0.3769	
Error	56	2468.5			



**FIGURE 1.** Behavioral metric of damaged and undamaged *Nucella lapillus* raised in the presence and absence of predator risk cues (Crab, No Crab). Mean ( $\pm$  1 SE) growth in body tissue mass over 60 d. Untransformed data are presented for clarity. Growth in shell mass and shell length showed similar patterns and are not depicted.



**FIGURE 2.** Effects of predator risk cues and prey physiological state on snail body tissue growth efficiency (J converted to tissue/J of mussel tissue consumed)(mean  $\pm$  1 SE). Untransformed data are presented for clarity. Shell growth efficiency was not influenced by risk cue or prey state while total growth efficiency (tissue growth efficiency + shell growth efficiency) showed a similar pattern to tissue growth efficiency.



**Figure 3.** Influence of risk cue (RCE) and physiological state (PSE) on total indirect effects on mussel consumption and energy acquisition. Untransformed data are presented for clarity.



**FIGURE 4.** Effects of predator risk cues and prey physiological state on absolute (size-independent) shell strength in compression (Mean  $\pm$  1 SE). Shell strength is defined as maximum load at point of shell failure.

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