ABSTRACT

Title of Thesis: QUANTIFYING DRIVERS OF MYCOBACTERIOSIS IN ATLANTIC STRIPED BASS AND CONSEQUENCES OF INCREASED NATURAL MORTALITY ON BIOLOGICAL REFERENCE POINTS

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The mycobacteriosis epizootic in Chesapeake Bay Atlantic striped bass (*Morone saxatilis*) is a concern for management because disease-associated mortality can negatively impact the coast-wide stock and its fisheries. As such, it is important to understand environmental drivers of disease dynamics and the consequences of increased disease-associated natural mortality on management reference points. Generalized linear models of fish health survey data collected in Maryland suggest water temperature, hypoxic volume, and fish condition influence disease presence in pre-migratory fish, and hypoxic volume, temperature, condition, age, and sex influence disease severity. Mortality approximately doubled across the range of hypoxic volume and temperature examined. A novel approach to calculating spawning potential ratio was explored to demonstrate the effect of increased natural mortality and compensation assumptions on resulting reference points, yield, and

spawning biomass. This work suggests striped bass recovery may require the

adoption of more conservative reference points in light of increased natural mortality.

QUANTIFYING DRIVERS OF MYCOBACTERIOSIS IN ATLANTIC STRIPED
BASS AND CONSEQUENCES OF INCREASED NATURAL MORTALITY ON
BIOLOGICAL REFERENCE POINTS

by

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Introduction

Infectious diseases can have substantial effects on the population dynamics of marine species through increased natural mortality, reduced growth, and decreased reproduction, resulting in a smaller population biomass (Jesse et al. In review). Diseases have been shown to have negative effects on ecologically and economically important fished species such as eastern oyster (*Crassostrea virginica*), American lobster (*Homarus americanus*), and snow crab (*Chionoecetes opilio*; Wilberg et al. 2011; Hoenig et al. 2017). Populations depleted by disease can lead to changes in community structure (Ward and Lafferty 2004), as well as affect recreational and commercial fisheries and the associated communities (Southwick Associates 2005, Lafferty et al. 2015). Because of these effects, disease is an important consideration for fisheries management.

Disease-associated effects on mortality, growth, and reproduction can also be affected by anthropogenic factors such as climate change (Roessig et al. 2004; Jesse et al. In review). Consequences of climate change (e.g. temperature and precipitation changes, CO_2 increases, storm activity) could have complex interactions with infectious diseases in marine systems depending on host susceptibility or resistance and pathogen virulence and abundance (Harvell et al. 2002, Burge et al. 2014). For

example, increased water temperature could increase the abundance of a disease, but it could also cause a species range shift to cooler waters away from the disease. Although some diseases may have little to no change in their effects under climate change, diseases in general are expected to increase in frequency and severity for most marine organisms (Harvell et al. 2002, Ward and Lafferty 2004, Burge et al. 2014).

One of the major diseases of concern in the Chesapeake Bay is mycobacteriosis, a chronic disease caused by *Mycobacterium* spp. that is found in many Chesapeake Bay fishes including Atlantic striped bass (*Morone saxatilis*) and Atlantic menhaden (*Brevoortia tyrannus*; Kane et al. 2007). Mycobacteriosis severity can vary depending on the mycobacteria species, host fish species, and if the fish is held in an aquarium or aquaculture facility versus the wild (Decostere et al. 2004, Gauthier and Rhodes 2009). Signs of disease generally include scale loss, pigment changes, abnormal behavior, emaciation, nodules or granulomas on internal organs, and external ulcerative lesions (Gauthier and Rhodes 2009). Disease is commonly characterized by granulomas on the spleen, liver, and kidneys before progressing to other organs in the body. In many cases there are no external signs of disease, but in severe cases ulcers can present on the body of the fish (Jacobs et al. 2009d). For striped bass, regression of disease state to a lesser state is rare in the wild and complete resolution of disease has not been seen in laboratory studies (Gauthier et al. 2003, Gauthier and Rhodes 2009, Groner et al. 2018).

Mycobacteriosis was first identified in the Chesapeake Bay striped bass population in 1997, although archived tissue samples tested positive as early as 1984 (Jacobs et al. 2009a). Since that time, prevalence of disease and presence of external lesions on Chesapeake Bay striped bass have increased compared to other wild populations (Jacobs et al. 2009d). Disease prevalence can vary widely by location and year, but can reach 60% or higher depending on the age of the fish (Appendix A ; Overton et al. 2003; Hoenig et al. 2017; Groner et al. 2018).

Mycobacteriosis has been shown to have both lethal effects (survival) and non-lethal effects (growth and reproduction) on striped bass . Tag-recapture studies found that mortality increases with increasing disease state and that moderately and severely diseased individuals have 84% and 54% yr^{-1} survival, respectively, relative to healthy fish (Hoenig et al. 2017). This translates to an approximate doubling of the natural mortality rate for fish with advanced stages of disease due to mycobacteriosis (Hoenig et al. 2017). Growth and maturity of individuals is also affected by mycobacteriosis. Disease-positive fish may grow slower and to a smaller size than disease-negative fish (Latour et al. 2012), and striped bass with mycobacteriosis mature earlier and at smaller sizes than those without disease (Gervasi et al. 2019). Growth differences due to disease were estimated to reduce the lifetime reproductive potential of striped bass by 3.3%, while natural mortality reduced reproductive potential by 74.5%, for a combined 75.1% loss in lifetime reproductive potential (Gervasi 2015). Thus, mycobacteriosis may have a substantial influence on the reproductive capabilities of the striped bass population.

The potential population-level effects of mycobacteriosis on striped bass is a serious concern to fisheries managers because striped bass are an important target of both recreational and commercial fisheries. The striped bass stock supports the largest recreational fishery in the US with approximately 38 million pounds harvested in 2017, and a smaller commercial fishery that harvested another 5 million pounds (National Marine Fisheries Service 2018). Given the Chesapeake Bay is the primary spawning and nursery ground for the US East Coast-wide stock, the apparent epizootic of mycobacteriosis is of management concern (Northeast Fisheries Science Center 2019). If fish from the Chesapeake Bay are experiencing increased morbidity and mortality because of the disease, it could negatively impact the coastwide stock and its fisheries (Richards and Rago 1999, Gauthier et al. 2008, Latour et al. 2012, Hoenig et al. 2017)

Despite the longevity and prominence of the disease, little is known about the potential drivers of mycobacteriosis. One possible driver for mycobacteriosis in striped bass is poor water quality and subsequent environmentally-induced metabolic stress (Coutant 1985). This hypothesis suggests that poor environmental conditions may compromise fish health and increase susceptibility and progression of disease. For example, the prevalence of mycobacteriosis in juvenile Atlantic menhaden was higher in rivers with lower pH and higher dissolved organic carbon (Kane et al. 2007). In regard to striped bass, water temperature and dissolved oxygen are the most likely water quality drivers of mycobacteriosis. In the summer, striped bass typically use the deeper portions of the Chesapeake Bay as a thermal refuge from the warmer

surface water (Coutant 1985); however, there are also hypoxic conditions in the bottom waters of the Chesapeake Bay in the summer (Testa et al. 2017). This means that striped bass may be forced into water outside of their preferred range of temperature and/or oxygen, which may consequently decrease the amount of suitable habitat resulting in stress and/or direct mortality from compromised conditions (Coutant 1985). This hypothesized temperature-oxygen "squeeze" can result in fish crowding in limited suitable locations which could increase susceptibility and progression of disease (Coutant 1985, Groner et al. 2018). Exposure to suboptimal conditions can also negatively affect metabolic performance for severely infected fish (Lapointe et al. 2014).

The temperature-oxygen squeeze hypothesis implies that striped bass have an optimal range for water conditions, and approaching or exceeding the limits of that optimal range can induce stress in the fish. Striped bass are fairly tolerant to water conditions, but they have been shown to demonstrate preferred ranges of water temperature and oxygen concentrations (Cimino et al. 2009). Juveniles are mostly found in the wild at temperatures of 24° C - 27° C in the spring and summer (Coutant et al. 1984). Adults prefer temperatures ≤ 25° C and choose cooler refuges in the summer from 22° C to 26° C (Davis and Parker 1990, Coutant and Benson 1990). Striped bass prefer concentrations of dissolved oxygen ≥ 2 - 3 mg/L, but ≥ 5 mg/L is optimal (Coutant and Benson 1990). More recent studies have shown that striped bass in the Chesapeake Bay selected the coolest water available without hypoxic conditions in

the summer. This may be evidence that the species' thermal range is more flexible than previously thought, but that oxygen tolerance is not (Kraus et al. 2015).

The second main driver proposed for mycobacteriosis in Chesapeake Bay striped bass is nutritional stress. This hypothesis suggests that food limitation and/or poor nutritional state can inflict metabolic strain on the fish, making them more susceptible to disease or increasing progression of disease (Jacobs et al. 2009b). After the striped bass stock collapsed in the 1970s, a fishing moratorium was imposed in several states, including Maryland, and the population rebounded to historically high abundance by the late 1990s (Northeast Fisheries Science Center 2019). At that time, it was thought that prey for striped bass declined to relatively low levels in Chesapeake Bay, possibly resulting in a trophic imbalance (Uphoff 2003, Jacobs 2007). Periods of reduced abundance of prey and increased abundance of striped bass may stress striped bass, possibly resulting in malnutrition and increased risk for mycobacteriosis. This hypothesis was tested experimentally, and found that nutritionally stressed fish had more severe mycobacteriosis infections and died within 4 to 6 weeks compared to no mortality for fish that were not nutritionally stressed (Jacobs et al. 2009b).

Quantifying the potential relationship between environmental conditions, fish health, and mycobacteriosis is complicated by the uncertain and complex nature of striped bass migratory behavior. Striped bass exhibit partial migration behavior, such that some fish stay in the Chesapeake Bay as year-round residents and others migrate out of the Chesapeake Bay to the coastal stock (Mansueti 1961, Kohlenstein 1981,

Wingate et al. 2011). The number of striped bass that migrate is thought to vary annually, and is determined by sex, maturity, size, density dependence, location, and year class strength (Secor and Piccoli 2007, Wingate et al. 2011, Secor et al. 2020). Historical tagging studies suggested that most males do not migrate into the coastal stock, some age two females migrate into the coastal stock, and approximately half of age three females migrate into the coastal stock (Kohlenstein 1981). Otolith microchemistry analysis found that female migration to the coastal stock was more gradual, occurring with maturity, and increasing with age (Secor and Piccoli 2007). In addition, otolith microchemistry suggests that a large portion of males also leave the Chesapeake Bay, and that some fish stay in the Chesapeake Bay as residents regardless of sex, age, or size (Secor and Piccoli 2007, Wingate et al. 2011). The most recent tagging study suggests that migration to the ocean increases sharply for striped bass of both sexes at lengths ≥80 cm total length and the age of 50% migration is 10.5-12.4 years (Secor et al. 2020). Given the uncertain and complicated migratory behavior of striped bass, making connections between environmental conditions and disease infection and progression may be confounded for migratory age fish that are exposed to conditions outside Chesapeake Bay.

Although mycobacteriosis has been studied extensively in the Virginia portion of the Chesapeake Bay (Hoenig et al. 2017, Groner et al. 2018) and the mainstem of the Chesapeake Bay (Gauthier et al. 2008, Latour et al. 2012, Gervasi et al. 2019), the relationship between water quality, fish health, and disease in striped bass has not been comprehensively analyzed in the Maryland portion of the Chesapeake Bay. My

goal for this project was to quantify water quality and fish health drivers of mycobacteriosis in striped bass in Maryland waters of the Chesapeake Bay. My objectives were to 1) quantify drivers of disease presence in pre-migratory fish, and 2) quantify drivers of disease progression to a severe state at which marked increases in mortality might occur.

Methods

Data

Two data sources were used to address my objectives. Disease and fish health information was collected as part of a long-term Maryland Department of Natural Resources (MDNR) fish health survey. Environmental conditions were characterized using estimates generated from a coupled hydrodynamic-biogeochemical model of Chesapeake Bay.

Striped bass disease and fish condition data

Striped bass mycobacteriosis data were obtained from the MDNR Fish and Wildlife Health Project in Maryland waters of the Chesapeake Bay for 1998-2015. Striped bass were sampled using a variety of gears such as hook and line, pound net, and beach seine from various locations around the Chesapeake Bay and its tributaries. Fish were collected from spring to fall through a combination of directed sampling trips and opportunistic data collection from other MDNR surveys and cooperating fishers. Fish samples were assigned to one of three broad regions based on location of

capture (Figure 1.1). At the Cooperative Oxford Laboratory, sex, weight, and total length were recorded for each fish. All fish were aged using an age-at-length key generated by the MDNR stock assessment program (Pers. Comm. Mark Matsche). Fish were examined for mycobacteria in spleen samples by testing for the presence of acid fast positive bacteria in granulomas. Beginning in 2007, each sample was also given an overall qualitative score indicating the total external lesion severity as a combination of pigmented foci and ulcers (0= no lesions, 1= mild, 2= moderate, 3=severe). Fulton's condition factor (K), the relationship between weight and length of a fish, was also calculated to describe the overall condition or health of each fish (Ricker 1975). Data collected in fall months (September-November) were selected for use in this analysis to avoid potential confounding of disease signal with seasonality of disease dynamics and change in sampling effort over time, and to examine the impact of summer water conditions (i.e., elevated temperatures and summer hypoxia).

Environmental data

Environmental data used in this study were generated by the Regional Ocean Modeling System with Row-Column Aesop (ROMS-RCA) which is a coupled hydrodynamic-biogeochemical model (Testa et al. 2014, Shen et al. 2020). The ROMS portion of the model simulates how the mainstem of the Chesapeake Bay responds to physical forces such as tides or wind, and the RCA portion of the model simulates the water quality response for the water column and sediment. The model full extent includes the Susquehanna River to the Mid-Atlantic Bight with 80 x 120 grid points (~1 km resolution) and 20 vertical layers, but in this study only the

Maryland portion of the Chesapeake Bay was used. The model was used to generate daily metrics representing dissolved oxygen and water temperature stressors from June-August 1998-2015. The predictions for dissolved oxygen are driven by photosynthetic production, air-water exchange, loading from external sources, algal respiration, and consumption in biogeochemical processes such as oxidation and nitrification (Testa et al. 2014). Predications of water temperature from the model are driven by sea surface temperature monitoring data, tributary discharge, and atmospheric forcings (Testa et al. 2014). The two metrics chosen were Hypoxic Volume and Heat Days. Hypoxic Volume was calculated as the average volume of bottom (below pycnocline) waters with dissolved oxygen concentration <3 mg/L. Heat Days was defined as the number of days that the average surface (above pycnocline) water temperature was > 25° C. The 25° C threshold was based on previous studies determining oxygen and temperature tolerance for striped bass (Coutant 1985, Coutant and Benson 1990). Environmental data were summarized across summer months (June-August) to represent the period during which dissolved oxygen and temperature are thought to impact habitat availability and metabolic stress in striped bass (Coutant 1985).

Modeling

Generalized linear models (GLMs; McCullagh and Nedler 1989) were constructed to test environmental and nutritional stress hypotheses and quantify the relationship between mycobacteriosis and environmental and nutritional drivers. To explore the impact of environmental and nutritional stressors on both contraction and progression

of the disease in individual fish, two different response variables were considered: 1)
disease presence in pre-migratory fish, and 2) disease severity based on external
lesions. Generalized additive models were explored as well, but strong nonlinear
relationships were not evident; therefore, GLMs are presented here for brevity.

Disease presence model

I explored factors associated with the presence of mycobacteriosis infection for pre-
migratory striped bass, defined as ages 0-1 (N=1,773), caught in the MDNR Fish
Health Survey during 1998-2015. Fish ages 2+ were excluded because they could
have altered exposure to mycobacteriosis or different stress levels outside of the
Chesapeake Bay as a result of migration to the ocean (Kohlenstein 1981). Alternate
migration scenarios were considered including increasing the age of emigration to as
high as 5 (Dorazio et al. 1994, Secor and Piccoli 2007) and using an 80 cm length
threshold for emigration (Secor et al. 2020). Results were quantitatively and
qualitatively similar between alternate migration models, so ages 0-1 models are
presented for brevity. Mycobacteriosis presence was modeled using a binomial GLM
with a logit link function,

$$\hat{Y} = \beta_0 + \beta_1 Hypoxic\ Volume + \beta_2 Heat\ Days + \beta_3 Fulton's\ K, \qquad (1.1)$$

where $\hat{Y}$ is the log-odds of a positive response, β_0 is the intercept, and β_{1-3} are the
regression coefficients for explanatory variables Hypoxic Volume, Heat Days, and
Fulton's K.

Disease severity model

I also explored factors associated with severe external lesions during 2007-2015.

Lesion severity scores of "no disease", "mild disease", and "moderate disease" were

combined for contrast with the "severe" disease designation to characterize the strong

negative effects (e.g., increased mortality) of mycobacteriosis in fish with severe

external disease (Hoenig et al. 2017, Groner et al. 2018). Fish ages 1+ (N=3,635)

were included in this analysis; age 0 fish were excluded because fish have only

recently been infected and the disease is not known to progress to the severe stage

that quickly. Mycobacteriosis severity was modeled using a binomial GLM with a

logit link function,

$$\hat{Y} = \beta_0 + \beta_1 Hypoxic\ Volume + \beta_2 Heat\ Days + \beta_3 Fulton's\ K$$
$$+ \beta_4 Age + \beta_5 Sex + \beta_6 Age * Sex, \tag{1.2}$$

where $\hat{Y}$ is the log-odds of a positive response, β_0 is the intercept, $\beta_{1\text{-}6}$ are the

regression coefficients for explanatory variables Hypoxic Volume, Heat Days,

Fulton's K, Age, Sex, and the interaction of Age and Sex.

Results

Disease presence model

Hypoxic Volume and Heat Days were significant factors in the disease presence

model (Table 1.1). The presence of disease in pre-migratory striped bass increased

with increasing Hypoxic Volume and increasing Heat Days (Figures 1.2. A-B).

Fulton's K was also a significant factor in the model ($P= 0.032$; Table 1.1). Disease presence for pre-migratory fish decreased with increasing fish condition (Figure 1.2. C).

Disease severity model

Significant factors in the disease severity model included Hypoxic Volume ($P= 0.002$), Heat Days ($P= 0.020$), Fulton's K ($P <0.0001$), Age ($P= 0.002$), and Sex ($P= 0.031$), but not the interaction between Age and Sex (Table 1.2). The occurrence of severe disease decreased with increasing Hypoxic Volume and increasing Heat Days (Figures 1.3. A-B). The occurrence of severe mycobacteriosis decreased with increasing fish condition (Figure 1.3. C) and increased with age (Figure 1.3. D). Males exhibited higher occurrence of severe disease than females (Figure 1.3. E); however, the interaction between age and sex showed a similar rate of progression to the severe disease state for each sex (Figure 1.3. F).

Discussion

Analysis of MDNR Fish Health Survey data provided evidence in support of both nutritional and environmental stress hypotheses, such that a combination of both factors may be associated with striped bass mycobacteriosis in Maryland waters of the Chesapeake Bay. It is likely that the two types of stressors are not mutually exclusive, and that both impact disease dynamics (Uphoff 2003, Jacobs et al. 2009b). Therefore, this study suggests that environmental and nutritional stress hypotheses are

not alternate hypotheses, but that both poor water conditions and fish condition are contributing to the striped bass mycobacteriosis epizootic.

Environmental Stress

The environmental stress hypothesis suggests that limited suitable habitat availability leads to metabolic stress and increased disease susceptibility in striped bass (Coutant 1985). In this analysis, disease presence in pre-migratory fish increased with increasing hypoxic volume and increasing heat days. Thus, poor summer water conditions in the Chesapeake Bay are likely contributing to disease presence through a combination of low dissolved oxygen and increased duration of days with high water temperature. This suggests that climate change could be an important factor in disease dynamics for striped bass, as temperature and hypoxia are expected to continue to increase in the Chesapeake Bay (Najjar et al. 2010).

Projections suggest that the Chesapeake Bay could see temperature increases of about 2-5.5° C by the end of the century (Najjar et al. 2010, Muhling et al. 2018). Also, hypoxic volume could increase by 10-30% from the end of the 20th century to the mid-21st century as a consequence of water column stratification, nutrient inputs, and temperature increase (Ni et al. 2019). It has also been suggested that sea level rise and nutrient input reductions will balance negative effects on dissolved oxygen in the future (Irby et al. 2018). Additionally, changes in temperature and hypoxia are connected because increasing temperature decreases solubility of the water and therefore exacerbates decreases in dissolved oxygen (Ni et al. 2019). Overall,

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warming is predicted to cause the largest effect on dissolved oxygen concentrations in the future (Irby et al. 2018, Ni et al. 2019), so it is likely that the two metrics represent the same problem and that both factors will continue to effect the dynamics of mycobacteriosis in striped bass.

Conversely, this analysis showed decreasing occurrence of severe mycobacteriosis under increasing hypoxic volume and increasing heat days. These results are counter to the expectations of the environmental stress hypothesis. However, tagging studies suggest that severe mycobacteriosis and increased water temperature could result in >90% mortality for striped bass in Virginia waters of the Chesapeake Bay (Groner et al. 2018); therefore, it is probable that the most severely diseased fish experience higher mortality in years with stressful summer water conditions. As such, severely diseased fish would be encountered at a lower rate in the fish health survey when environmental conditions are unfavorable, resulting in decreased occurrence of severe disease with unfavorable summer conditions. If mortality in severely diseased fish is triggered by environmental stress, relative mortality rates can be estimated for striped bass from the disease severity model (Table 1.2; Figure 1.3) under different hypoxic volume or heat days scenarios. Probability of severe disease occurrence can be calculated by transforming the log odds of severe disease from the GLM (McCullagh and Nedler 1989),

$$Probability = \frac{e^{\beta_0 + \beta_1 Hypoxic\ Volume + \beta_2 Heat\ Days + \beta_3 Fulton's\ K + \beta_4 Age + \beta_5 Sex + \beta_6 Age*Sex}}{1 + e^{\beta_0 + \beta_1 Hypoxic\ Volume + \beta_2 Heat\ Days + \beta_3 Fulton's\ K + \beta_4 Age + \beta_5 Sex + \beta_6 Age*Sex}}. \quad (1.3)$$

The estimated probability of severe disease is 0.061 at the lowest average hypoxic volume of 8.127 km^3, and decreases by about half to 0.031 for the highest average hypoxic volume of 10.6 km^3. If the decrease in severe disease under worsening environmental conditions is due to disease-associated mortality, this implies a doubling of mortality from the lowest average hypoxic volume to the highest generated by the ROMS-RCA model. Similarly, lowest average heat days of 115 was associated with a probability of severe disease of 0.057 compared to a probability of 0.031 for the highest average heat days of 136. Again, this corresponds to an approximate doubling in mortality over the range of average heat days generated from the ROMS-RCA model. Altogether, this model suggests a pronounced effect of temperature and hypoxia on mortality of severely diseased fish.

The possibility of increased striped bass mortality under elevated temperature is supported by tag-recapture studies in Virginia which showed that mortality increased for both healthy and diseased striped bass with increasing average summer sea surface temperature (Groner et al. 2018). Tagging model estimates suggested that healthy, mild, and moderately diseased fish experienced 25-75% mortality at temperatures of 26-29° C, respectively, whereas severely diseased fish experienced 64-91% mortality across the same temperature range. The increased mortality observed in both Maryland and Virginia at higher water temperatures supports the hypothesis that striped bass are living at the edge of their thermal tolerance, and indicates that increases in temperature could have detrimental effects, especially for diseased fish.

In addition to temperature, this study suggests a relationship between hypoxic volume and increased mortality in striped bass in Maryland waters of the Chesapeake Bay. An effect of low dissolved oxygen on striped bass mortality has not been observed in Virginia waters likely due to lesser occurrence of hypoxia in the lower portion of the Chesapeake Bay (Hagy et al. 2004, Groner et al. 2018). Striped bass that occupy Maryland waters may experience different disease dynamics given they are subject to both elevated summer temperatures and increased hypoxia. Tagging studies have shown that striped bass avoid hypoxic areas (Kraus et al. 2015) and subsequently, striped bass may be occupying higher temperature water to avoid low oxygen areas. Laboratory studies of striped bass aerobic performance showed that higher temperatures decreased the ability of fish to tolerate hypoxic conditions (Lapointe et al. 2014). Thus, prolonged exposure to high temperature waters and crowding in the remaining available habitat likely all contribute to mycobacteriosis disease dynamics in Maryland and elevated mortality among fish in a severe disease state.

Nutritional Stress

The nutritional stress hypothesis suggests that malnutrition is an increased risk factor for mycobacteriosis in striped bass. This analysis showed that occurrence of severe disease increased with decreasing fish condition (represented by Fulton's K), which indicates nutritional stress may impact disease dynamics. Overton et al. (2013) also found that disease-positive fish in the Maryland portion of the Chesapeake Bay had reduced condition factor; in particular, fish with external lesions had significantly lower body condition.

However, we cannot be certain if poor condition is a cause or a symptom of the disease severity. Fish compromised by the disease may have reduced ability to forage successfully, which could worsen their condition and lead to further increases in disease severity. Previous studies have associated a decline in adult striped bass preferred prey, including Atlantic menhaden, bay anchovy (*Anchoa mitchilli*), and spot (*Leiostomas xanthurus*), with a shift to other food sources in conjunction with the mycobacteriosis epizootic from the mid 1990s -2007 (Jacobs 2007, Jacobs et al. 2009b). From 1997 to present, though, Atlantic menhaden abundance in Chesapeake Bay surveys has remained stable or even increased (SEDAR 2020). Spot abundance estimated from the Chesapeake Bay Multispecies Monitoring and Assessment Program (ChesMMAP) peaked in 2007, but returned to similar levels from before the peak before slowly decreased to a fairly stable lower abundance (Bonzek et al. 2019). Bay anchovy abundance in the Chesapeake Bay has not been updated since 2000 (Jung and Houde 2004). Diet analysis of striped bass from the ChesMMAP survey from 2002-2007 (around the time of Jacobs et al. 2009 nutrition hypothesis studies) showed 41.3% of diet was fish while 31.7% was crustaceans (Bonzek et al. 2008), while the 2002-2018 report showed an increase in fish consumption to 54.9%, particularly bay anchovy, and a decrease in crustacean consumption to 20.8%, particularly for mysid shrimp (Bonzek et al. 2019). This suggests that forage fish availability may not be as large of a contributing factor as hypothesized for disease severity in the Chesapeake Bay. Updated forage fish availability information (especially bay anchovy) and diet analyses would need to be considered to more

properly test if forage availability is contributing to disease progression in adult striped bass.

In contrast, this analysis showed disease presence in pre-migratory fish decreased with decreasing fish condition, represented by Fulton's K. This does not support the nutritional stress hypothesis, but is potentially explained by the fact that the model only includes ages 0-1 fish. Pre-migratory fish in poor condition may also have decreased survival when diseased at this young age, and, therefore, may appear less frequently in the survey. This might suggest that young fish are vulnerable to increased mortality from mycobacteriosis as well as adult fish. Another possible explanation is that Fulton's K is not the best measure of fish condition, especially for age 0-1 fish. One potential problem with Fulton's K is that it may not reflect well the nutritional status of the fish, but rather only the robustness of the fish (Nash et al. 2006). Lipid concentration is thought to be an appropriate guide for nutritional health in striped bass, and a body fat index (BFI) is a better indicator of lipids than Fulton's K (Jacobs et al. 2013). The MDNR Fish Health survey data did not include a BFI score for the majority of pre-migratory fish; however, an alternate model that utilized the subset of records with BFI scores showed similar results to models that included Fulton's K; therefore, Fulton's K was likely a reasonable measure of fish condition to use in this analysis.

The disease severity model confirmed the chronic and progressive nature of mycobacteriosis for striped bass (i.e., severe disease incidence steadily increased with

age; Figure 1.3). Also, disease dynamics differed between sexes in a manner reflective of sex-specific migratory behavior. Male striped bass demonstrated higher occurrence of severe disease; however, progression of the disease was similar between the sexes. Males likely exhibit higher occurrence of severe disease because male fish are longer-term residents in the Chesapeake Bay (Kohlenstein 1981, Secor et al. 2020), and, therefore, have more exposure to localized environmental and nutritional stressors that are thought to exacerbate the disease (Coutant 1985, Uphoff 2003). Previous studies reported a decrease in apparent disease prevalence for older female striped bass, which was attributed to disease-associated mortality (Gauthier et al. 2008; MDNR unpubl. data; Mark Matche, pers. comm.). However, in Maryland, the decrease in prevalence among older females was found to be an artifact of low sample size for older fish; as more samples of older females were collected over time, a decline in prevalence was no longer apparent. This study and other more recent studies have not been able to corroborate disease progression differences between sexes (Groner et al. 2018).

Several simplifying assumptions were made in this study due to data limitations. First, I assumed that ROMS-RCA estimates of hypoxic volume and heat days were a reasonable representation of water conditions across the whole Maryland portion of the Chesapeake Bay even though it does not include tributaries; this assumption was supported by the results of alternate presence and severity models that used empirical temperature and DO data from the Chesapeake Bay Program (including tributaries) which produced similar results. Also, some potential environmental drivers could not

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be included in the models because of correlations with other environmental variables, For example, river flow, algal biomass, and wind are all correlated with hypoxic volume, but could play a role in disease dynamics. Additionally, this study was limited in spatial resolution of fish health survey data. Low sample sizes in some region/year combinations and imprecise collection location data prohibited inclusion of a detailed spatial element in this analysis. Collection of more spatially explicit data in the future could help better identify explicit drivers of mycobacteriosis in the Chesapeake Bay. It is also important to note that interpretation of these results is limited to the Maryland portion of the Chesapeake Bay. The study of striped bass mycobacteriosis dynamics in Chesapeake Bay would benefit from collaborative data collection and analysis between both Maryland and Virginia.

I explored nutritional and environmental stress hypotheses in this study because those two drivers have the most support, but there are other potential drivers that could be contributing to mycobacteriosis in Chesapeake Bay striped bass. First, the mode of transmission is unknown for mycobacteriosis in the Chesapeake Bay and could be an important factor in disease dynamics (Jacobs et al. 2009d). Other fish species, including aquarium and aquaculture fish populations with mycobacteriosis epizootics, have documented disease transmission from direct contact, contaminated feed, mother to egg transmission, and intermediate host species (Decostere et al. 2004). A study of mycobacteria from various Chesapeake Bay fish found that striped bass and Atlantic menhaden share isolates of mycobacteria potentially supporting a hypothesis for infection of striped bass through ingestion of infected prey (Stine et al. 2010).

Another possible driver of striped bass disease dynamics is elevated mycobacteria abundance in the water column which could be caused by water quality drivers such as high total suspended solids, low dissolved oxygen, and/or increasing salinity, and increase susceptibility of infection (Appendix B). Although this study suggests a strong linkage between environmental conditions and disease state in striped bass, additional research is needed to understand the underlying mechanism of infection.

Management Implications

Recent concerns about the current overfished and overfishing status of the striped bass stock have given rise to questions about the coastwide impact of the mycobacteriosis epizootic in Chesapeake Bay (Northeast Fisheries Science Center 2019). The Chesapeake Bay is the primary spawning and nursery grounds for the coastwide stock (Kohlenstein 1981, National Marine Fisheries Service 2018). Therefore, increases in disease-associated mortality in the Chesapeake Bay could have broad, population-level impacts.

This study highlighted the relationship between environmental conditions in the Chesapeake Bay and disease presence in pre-migratory fish (Table 1.1; Figure 1.2. A-B), and demonstrated increased mortality in striped bass with severe disease under compromised environmental conditions (Table 1.2; Figure 1.3. A-B). Given striped bass in Chesapeake Bay are likely living at the edge of their thermal tolerance in summer (Coutant 1985, Groner et al. 2018), increases in temperature or decreases in suitable habitat due to hypoxia could have detrimental effects, especially on the pre-

migratory stock and severely diseased spawners. Moving forward, it will be important to continue Chesapeake Bay-wide water quality monitoring and to consider hypoxic volume and water temperature in Maryland striped bass habitat management.

The other main spawning and nursery areas for striped bass should also be examined for disease outbreak potential and appropriately monitored. Although both the Delaware Bay and the Hudson River estuary have large amounts of nutrient input, hypoxia is not a current problem (Howarth et al. 2000, Sharp 2010). Both of these producer areas for striped bass have experienced problems with nutrient input in the past, mostly from wastewater, but have seen major improvements in dissolved oxygen concentration and water quality in recent decades (Najjar et al. 2009). The Chesapeake Bay has not had these same improvements, likely because the nutrient inputs to the Chesapeake Bay are inorganic in nature and have not been reduced to the same extent as other estuaries (Najjar et al. 2009). This may be due in part to the fact that the Delaware Bay water column is well mixed compared to the Chesapeake Bay which has more persistent stratification in the summer (Sharp 2010, Li et al. 2016). The Hudson River estuary is also fairly turbid and has short residence time (Howarth et al. 2000). Although, the Delaware Bay and Hudson River estuaries are both expected to increase in temperature with future climate change (Najjar et al. 2009, Seekell and Pace 2011), a future mycobacteriosis epizootic may not be a concern for these producer areas because striped bass may be able to find thermal refuge in deeper waters, which is not possible in the Chesapeake Bay due to hypoxia.

If mycobacteriosis is causing elevated mortality in diseased fish, as suggested by this study and tagging studies in Virginia, assumptions about natural mortality in the striped bass stock assessment will need to be reconsidered. The most recent stock assessment for striped bass recognized the potential impact of disease and began the process of exploring population-level impacts of increased mortality due to mycobacteriosis (Northeast Fisheries Science Center 2019). A new assessment model[1] was constructed that incorporated migration and separated the Chesapeake Bay and Coastal stocks. In this model, natural mortality was assumed to increase by +0.12 for fish age 3+ in the Chesapeake Bay; natural mortality assumed at older ages reverted to the original lower rate when fish migrated out of the Chesapeake Bay. This study supports the need for a two-stock assessment model. However, this study suggests mortality rates may double among severely diseased adult fish in Maryland waters when summer water conditions are poor. To properly incorporate increased natural mortality due to mycobacteriosis in future assessments, additional Chesapeake Bay-wide studies quantifying the rate of change in mortality with disease progression at age in migratory fish may be required. Also, further investigation is needed of innovative ways in which habitat quality (i.e., water temperature and hypoxic volume) can be integrated into the assessment as it relates to survival of fish in each disease state (e.g. Wilberg et al. 2013).

In addition to considering alternate assessment frameworks, striped bass management in the Chesapeake Bay may have to undergo large changes to respond adequately to

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[1] This model did not pass peer review and is not currently used for management, but it will likely be used in future assessments as model development progresses.

the mycobacteriosis epizootic and preserve the fishery. Ecosystem-based management for striped bass should be considered, in which both habitat and disease dynamics are integrated quantitatively in assessment and management. This could include the development of models that estimate the annual variability in natural mortality at age as a function of summer temperature, hypoxic volume, and disease severity for incorporation in the stock assessment model. However, this will require continued monitoring of habitat conditions and striped bass survival rates as well as estimation of age- or size-specific disease prevalence and severity rates across the Chesapeake Bay. More extensive and updated studies on the abundance of striped bass forage (especially their most abundant prey, bay anchovy) should be undertaken, as well, and, if necessary, multispecies interactions should be considered in management. Finally, alternate reference points may need to be considered for striped bass that incorporate the apparent increase in disease-associated natural mortality in order to prevent overfishing and further declines in biomass.

Tables

Table 1.1. Parameter estimates for a binomial generalized linear model of mycobacteriosis presence in pre-migratory striped bass in Maryland waters of the Chesapeake Bay from 1998-2015 as a function of environmental and fish condition.

Variable	Estimate	Std. Error	Z	*P*
Intercept	-13.541	1.254	-10.799	< 0.0001
Hypoxic volume	0.589	0.063	9.305	< 0.0001
Heat days	0.045	0.009	5.131	< 0.0001
Fulton's K	0.116	0.054	2.149	0.032

Table 1.2. Parameter estimates for a binomial generalized linear model of mycobacteriosis severity in age 1+ striped bass in Maryland waters of the Chesapeake Bay from 2007-2015 as a function of environmental and fish condition.

Variable	Estimate	Std. Error	Z	P
Intercept	5.221	2.069	2.523	0.016
Hypoxic volume	-0.282	0.090	-3.125	0.002
Heat days	-0.029	0.013	-2.326	0.020
Fulton's K	-0.391	0.076	-5.140	< 0.0001
Age	0.273	0.072	3.777	0.002
Sex	0.861	0.399	2.159	0.031
Age*Sex	0.029	0.078	-0.370	0.712

Figures
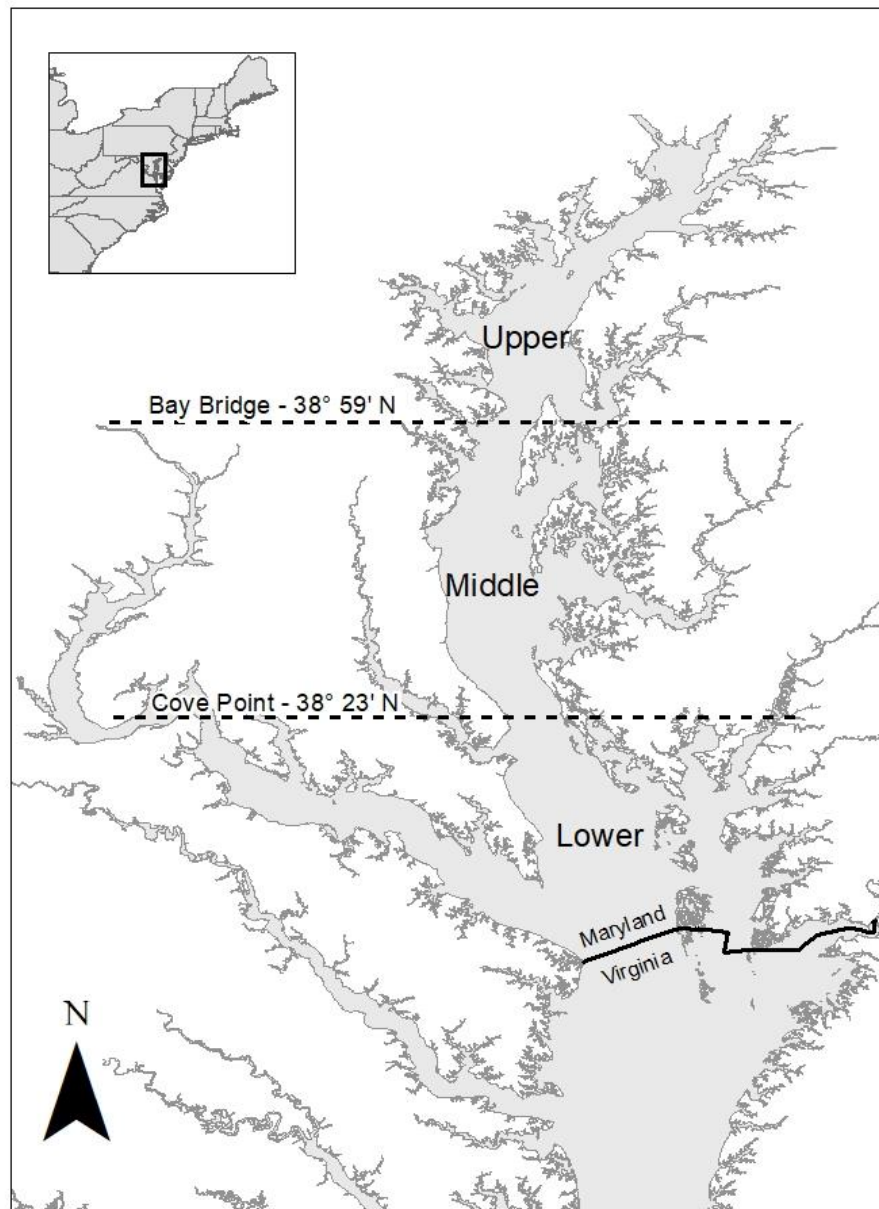
Figure 1.1. Regions for striped bass collection by MDNR Fish and Wildlife Health Project in Maryland waters of the Chesapeake Bay.
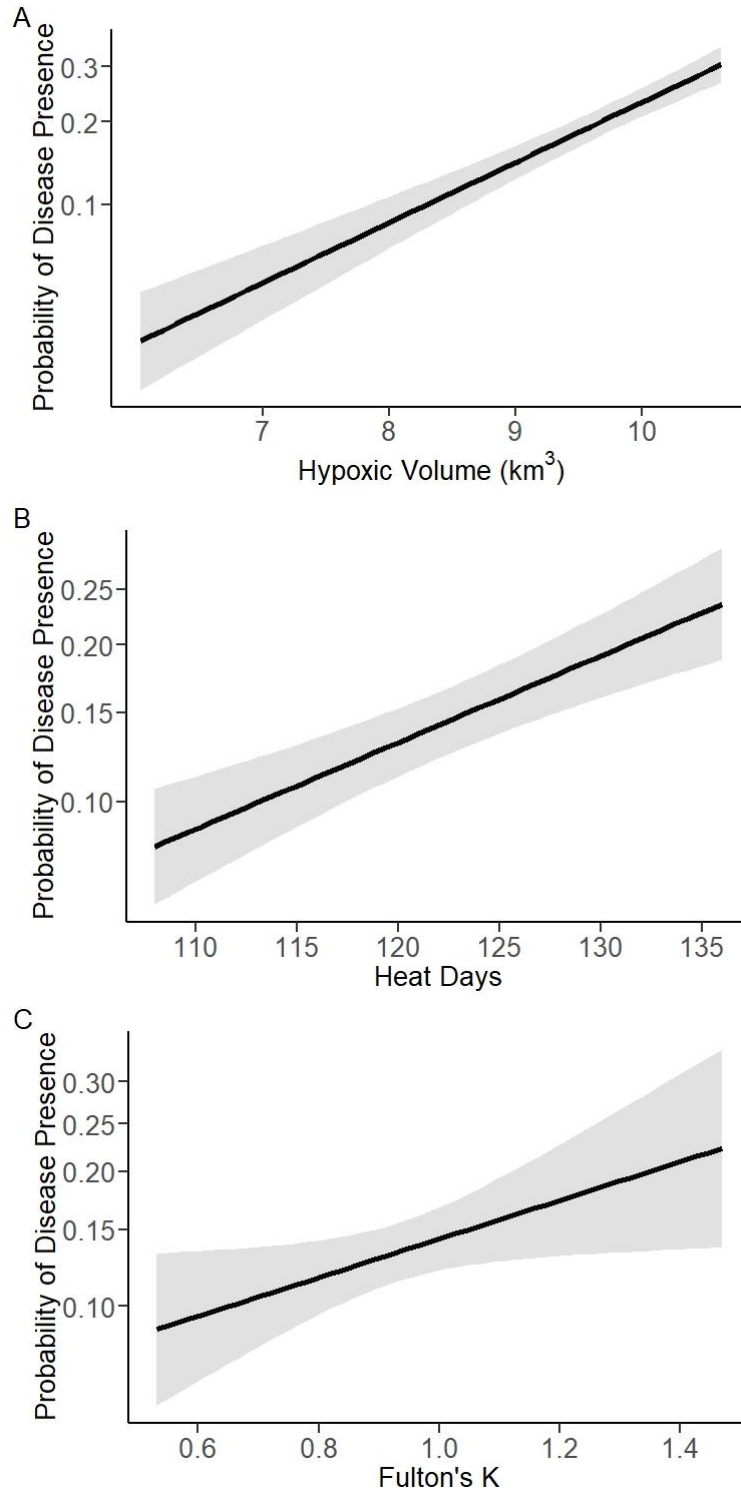
Figure 1.2. Partial effects of (A) Hypoxic Volume, (B) Heat Days, and (C) Fulton's K on disease presence in pre-migratory striped bass from 1998-2015 in Maryland waters of the Chesapeake Bay.
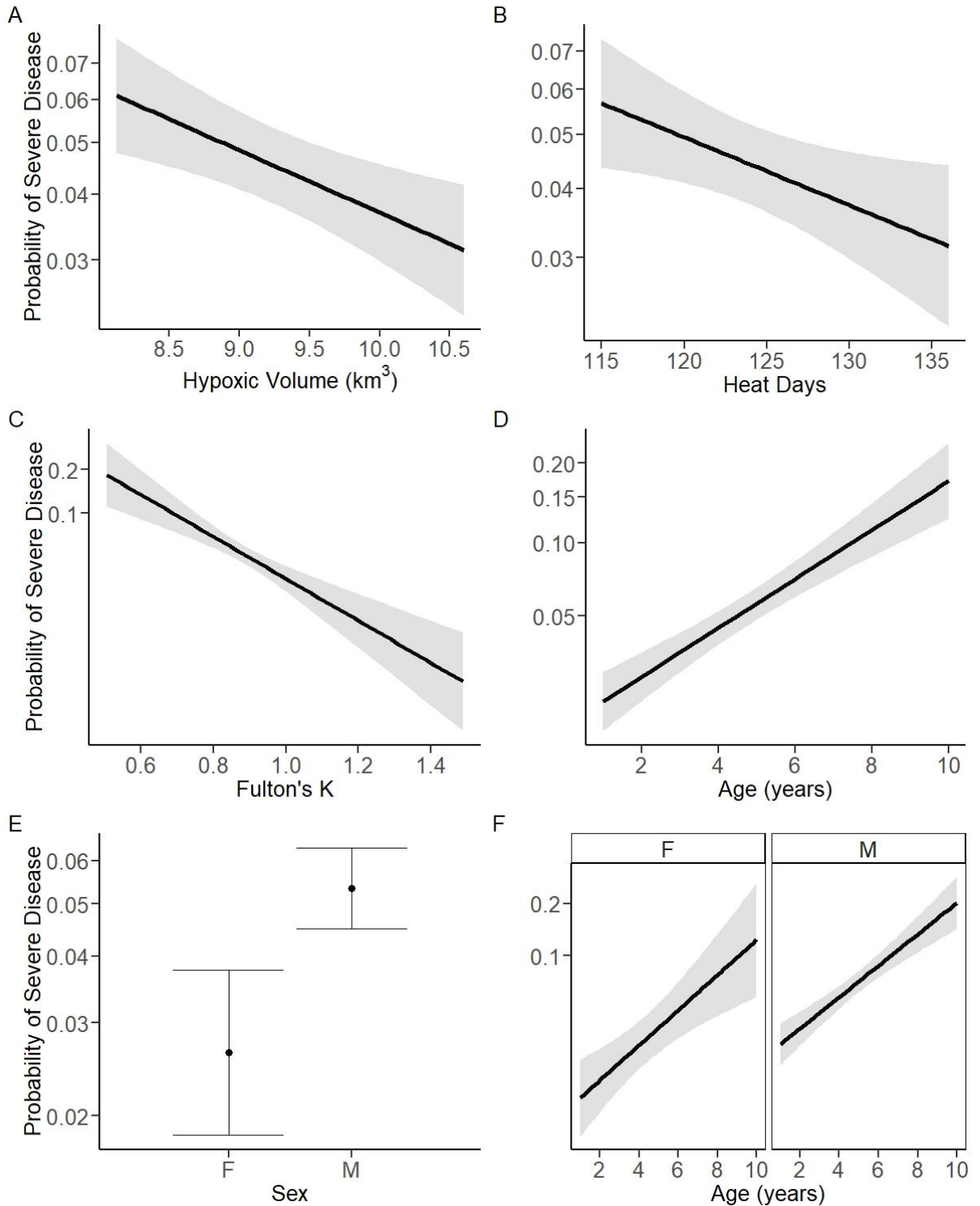
Figure 1.3. Partial effects of (A) Hypoxic Volume, (B) Heat Days, (C) Fulton's K, (D) Age, (E) Sex, and (F) Age and Sex on severe disease in age 1+ striped bass from 2007-2015 in Maryland waters of the Chesapeake Bay.

Chapter 2: Consequences of increased natural mortality on biological reference points: a case study for Atlantic striped bass (*Morone saxatilis*)

Introduction

Natural mortality (M) is defined as mortality from non-fishing sources and is typically assumed to be a constant value in fisheries stock assessment models (Quinn and Deriso 1999). However, some populations have displayed evidence of natural mortality changing over time such as disease outbreaks in eastern oyster (*Crassostrea virginica*; Wilberg et al. 2011; Damiano and Wilberg 2019) and Pacific herring (*Clupea pallasii*; Marty et al. 2003; Marty et al. 2010), lack of stock recovery under low fishing pressure in Atlantic weakfish (*Cynoscion regalis*; Northeast Fisheries Science Center 2009), or possible increased predatory consumption in Atlantic herring (*Clupea harengus*; Deroba 2015; Northeast Fisheries Science Center 2018). Misspecification of natural mortality in stock assessments can lead to biased estimates of stock size (Deroba and Schueller 2013). Although time-varying natural mortality has been incorporated into some stock assessment models (Lapointe et al. 1989, 1992, Clark 1999, Deroba and Schueller 2013, Johnson et al. 2015), accounting for a change in natural mortality in management benchmarks is challenging because most biological reference point models assume equilibrium conditions, meaning that the population is in a steady-state where conditions are not changing. For example,

environmental fluctuations could result in population variability that is not accounted

for in equilibrium calculations (Clark 1991, Legault and Palmer 2015).

One of the most common options for developing biological reference points for

management is to utilize dynamic pool models that estimate yield per recruit (YPR)

and spawning stock biomass per recruit (SSB/R; Quinn and Deriso 1999). YPR

models estimate the yield an individual fish produces in its lifetime by tracking the

exponential decline in abundance across a cohort and estimating the yield generated.

Cohort abundance is calculated as:

$$N_{a+1} = N_a e^{-M_a + F_a}, \tag{2.1}$$

where a is age, N_a is abundance-at-age, M_a is natural mortality-at-age, and F_a is

fishing mortality-at-age. YPR is then calculated as the sum of fishery yield-at-age

across all ages:

$$YPR = \sum_a \frac{F_a}{M_a + F_a}(1 - e^{-M_a + F_a})N_a W_a \tag{2.2}$$

where W_a is weight-at-age. A typical fishing mortality reference point generated from

YPR models is F_{max}, the fishing mortality rate that maximizes yield per recruit.

SSB/R models estimate the spawning potential of an individual fish in its lifetime by

converting cohort abundance-at-age estimated from the exponential mortality

equation (Eq 2.1) to spawning biomass by incorporating weight-at-age and percent

maturity-at-age (Mat_a). SSB/R is defined as the sum of $s$pawning stock biomass-at-

age across all ages:

$$SSB/R = \sum_a Mat_a N_a W_a \,. \tag{2.3}$$

Reference points are generated from SSB/R models by first calculating the spawning potential ratio (SPR), which compares SSB/R under fished (SSB/R$_F$) and unfished conditions (SSB/R$_{F=0}$):

$$SPR = \frac{SSB/R_F}{SSB/R_{F=0}} \,. \tag{2.4}$$

SSB/R models can be used to calculate biological reference points, such as F$_{X\%}$, the fishing mortality rate that reduces SSB/R to some percent (X%) of the unfished level (Clark 1991).

Per recruit models typically assume a constant growth, maturity, and mortality schedule in order to quantify how much lifetime yield and spawning potential is reduced by fishing (Shepherd and Pope 2008). However, when a population experiences a sustained increase in natural mortality, per recruit-based reference points can be modified to account for the change in natural mortality. There are two ways SPR-based reference points are typically calculated when natural mortality has increased. The first approach is to maintain the status quo in which SPR is calculated with the original, lower natural mortality, often because the magnitude of increase in natural mortality is considered to be not well quantified. With this approach, fishing at an unsustainable level could occur because the additional mortality is not taken into consideration when setting management benchmarks (Legault and Palmer 2015).

The second approach to calculating SPR when natural mortality has increased is to update both the fished and unfished portions of the SPR equation (Eq 2.4) with the new estimate of natural mortality. Increased natural mortality implies fish die at a younger age, and so the stock can be fished at a higher rate to harvest the fish before they die from natural causes. Using Approach 2 to incorporate increased natural mortality implies that a population is able to compensate for increased mortality due to a release of density dependent processes that typically limit productivity of the population, including competition and predation (Rose et al. 2001). Fisheries models assume that sustainable harvest is possible in a population because a population can compensate for increased mortality in a variety of ways such as higher recruitment, faster growth, and maturity at younger ages (Morgan et al. 2014). However, there are limits to the ability of a population to compensate; increasing natural mortality in both the fished and unfished portions of SPR models may overestimate the population's ability to compensate. Approach 2 could have serious repercussions for stocks that are already facing declines if compensation has not occurred and the productivity of the stock has not increased concurrently to offset losses from increased natural mortality.

A novel third approach to calculating SPR is to incorporate the new, higher natural mortality estimate in the calculation of fished SSB/R, but to retain the original lower natural mortality in the calculation of unfished SSB/R (Eqn. 2.4). This approach allows for comparison of SSB/R under both fishing and increased natural mortality conditions with the unfished SSB/R under original natural mortality conditions,

thereby assuming that the productivity of the stock has not increased to counteract the increase in natural mortality. By assuming the population has not developed additional compensatory mechanisms, this third approach should result in more conservative reference points when there has been an increase in natural mortality. This approach could help avoid many of the risks associated with the prior two approaches.

A case study for Atlantic striped bass

The consequences of increased natural mortality in per recruit models can be explored using a case study of the mycobacteriosis epizootic in Atlantic striped bass (*Morone saxatilis*) in the Chesapeake Bay. Mycobacteriosis, a chronic disease caused by *Mycobacterium* spp., was first identified in the Chesapeake Bay striped bass population in 1997 (Jacobs et al. 2009a), and there is evidence that natural mortality has increased since that time (Northeast Fisheries Science Center 2019). Tag-recapture studies have estimated an approximate doubling of natural mortality, from the previously assumed rate of 0.15 to 0.29 year^{-1} when disease-associated morality is added to pre-mycobacteriosis estimates of natural mortality (Hoenig et al. 2017). Regression of mycobacteriosis is uncommon, and the disease is typically assumed to be fatal (Gauthier and Rhodes 2009, Groner et al. 2018). Natural mortality due to mycobacteriosis was estimated to reduce the lifetime reproductive potential of striped bass by 74.5% (Gervasi 2015). Thus, increased natural mortality from mycobacteriosis may have a substantial influence on the striped bass population.

Mycobacteriosis is a serious concern to fishery managers because striped bass support both recreational and commercial fisheries, and the Chesapeake Bay is the primary spawning and nursery ground for the coast-wide stock (Richards and Rago 1999, National Marine Fisheries Service 2020). Understanding of disease dynamics is complicated by the fact that striped bass exhibit partial migration behavior, such that some fish stay in the Chesapeake Bay as year-round residents and others migrate out of the Chesapeake Bay to the ocean (Mansueti 1961, Kohlenstein 1981, Wingate et al. 2011). The proportion of fish that emigrate from the Chesapeake Bay stock to the coastal ocean stock increases with age and length, which vary by sex (Secor et al. 2020). As such, increased natural mortality for striped bass in the Chesapeake Bay could negatively impact both the Chesapeake Bay and coastal ocean stock and their associated fisheries (Richards and Rago 1999, Gauthier et al. 2008, Hoenig et al. 2017).

The most recent stock assessment for striped bass explored the incorporation of increased natural mortality due to mycobacteriosis with an alternate assessment model; however, neither the base model nor the biological reference points currently used for management account for increased natural mortality in Chesapeake Bay fish (Northeast Fisheries Science Center 2019). The empirical reference points used to determine stock status are ad hoc and based on the spawning stock biomass in 1995, when the stock was declared recovered. Threshold spawning stock biomass is defined as the 1995 spawning stock biomass, and target biomass is defined as 125% of the 1995 spawning stock biomass; F reference points are those that achieve the threshold

and target spawning stock biomass. These empirical reference points could be problematic because they do not account for increases in natural mortality due to the mycobacteriosis epizootic or other potential changes to the stock that may have occurred since 1995. Considering the current overfished and overfishing stock status designations, development of new biological reference points is still a management priority for striped bass (ASMFC 2020). However, the best approach for incorporating recent increased natural mortality needs to be carefully considered before new biological reference points are proposed for use in management.

My goal for this project was to examine the consequences of a sustained increase in natural mortality on biological reference points using striped bass and mycobacteriosis as a case study. My objectives were to 1) compare three alternative approaches to incorporating increased natural mortality in SPR-based reference points, 2) demonstrate how reference points, equilibrium yield, and equilibrium stock size are affected by the way in which increased natural mortality is incorporated in SPR models, and 3) demonstrate the potential consequences of failing to account for increased M in striped bass biological reference points.

Methods

Three deterministic yield and spawning stock biomass per recruit models were developed to compare alternative approaches for calculating SPR when there has been a distinct, sustained increase in natural mortality in a population. Stock-recruitment functions were incorporated to provide estimates of yield and stock size

over a range of fishing mortality values for each of the models. Also, a suite of biological reference points generated from the three SPR calculation approaches and the resulting equilibrium yield and spawning stock biomass were compared. Models were parameterized to examine the case study of increased natural mortality in Atlantic striped bass due to mycobacteriosis (Hoenig et al. 2017, Groner et al. 2018). Alternate scenarios were generated to examine how several assumptions about magnitude of disease-associated natural mortality and disease progression with migration influence the SPR, equilibrium yield, and spawning stock biomass resulting from the three alternative SPR modeling approaches.

Stock-Recruitment Estimation

Stock-recruitment functions were estimated for striped bass to calculate median yield and spawning stock biomass curves. Annual abundance-at-age on January 1 and female spawning weight-at-age from the most recent striped bass stock assessment (Northeast Fisheries Science Center 2019) were used to calculate spawning stock biomass-at age (Eq 2.1.1). This, in combination with age-1 recruitment estimates from 1982-2017 (Northeast Fisheries Science Center 2019), were used to estimate the parameters of both Beverton-Holt (Eq 2.1.2) and Ricker stock-recruitment functions (Eq 2.1.3) within a maximum likelihood framework (Eq 2.1.4). The resulting stock-recruitment curves are shown in [Appendix C](#) (Figure C-1 top).

YPR and SSB/R Models

Per recruit models were structured and parameterized to reflect life history and fishery dynamics of striped bass ages 1 to 15+ as estimated in the most recent stock assessment (Table 2.2-2.3; Northeast Fisheries Science Center 2019). Fishing mortality-at-age was calculated for each of the two regions (Chesapeake Bay vs ocean) as the product of fishing mortality and region-specific selectivity-at-age (Eq 2.4.1 and 2.4.2). Fishing mortality was apportioned to each of the two regions using a ratio of 0.3, the ratio of Chesapeake Bay to ocean fishing mortality averaged across the last three years of the assessment (2015-2017). Selectivity-at-age estimates were taken from the most recent selectivity time block for each region in the assessment model (1996-2017 in Chesapeake Bay; 1997-2017 in the ocean). Selectivity of the fishery in Chesapeake Bay was dome-shaped, whereas ocean fishery selectivity was asymptotic with 50% selected at approximately age 6 (Table 2.2, Figure 2.1).

Region-specific fishing- and natural mortality-at-age were summed after applying age-specific migration to calculate total mortality-at-age (Eqs 2.4.3-2.4.5). Natural mortality-at-age was assumed to be 0.14 yr^{-1} greater for fish ages 3+ occupying Chesapeake Bay (Hoenig et al. 2017; Northeast Fisheries Science Center 2019; Table 2.2). The rate of migration from Chesapeake Bay to the ocean was age-dependent with 50% migration at approximately age 9 (Table 2.2).

Abundance-at-age was calculated assuming exponential mortality within a cohort and the infinite series adjustment for the plus group (Eqs 2.4.6 and 2.4.7). YPR was

calculated for each region and summed to generate an estimate of total coastwide yield (Eq 2.4.8). SSB/R in the fished and unfished condition were calculated as described in Eq 2.3; natural mortality values used in each equation are described in Table 2.2. Catch weight-at-age and spawning weight-at-age were calculated as the mean of stock-wide estimates from 2015-2017, and coastwide maturity-at-age estimates were taken from the assessment (Northeast Fisheries Science Center 2019).

SPR Calculation Approaches

Three approaches to calculating SPR (Eq 2.4) in light of increased natural mortality were explored. The approaches differ in the natural mortality rates assumed in the numerator and the denominator of the SPR calculation (Eq. 2.4). The status quo (SQ) approach assumed no increase in natural mortality-at-age in both the numerator (SSB/R_F) and denominator ($SSB/R_{F=0}$) portions of the SPR equation, assuming the increase was unknown or not being addressed in the analysis. Approach 1 incorporated increased natural mortality-at-age in both the numerator (SSB/R_F) and denominator ($SSB/R_{F=0}$) of the SPR equation, updating the entire SPR calculation to reflect the increase in natural mortality. Approach 2 incorporated increased natural mortality in the numerator (SPR_F), but not the unfished portion of the SPR equation. This approach treats the increased natural mortality as if it was fishing mortality. The three alternative SPR calculation approaches were compared using natural mortality scenario M1 (Table 2.5, as it most closely resembled the assumptions made in the two-stock model explored in the most recent striped bass stock assessment.

Biological Reference Points

Biological reference points included F_{MSY} (the fishing mortality rate that achieves maximum sustainable yield) were calculated using yield curves generated from both Beverton-Holt and Ricker stock-recruitment functions by applying the method from Shepherd (1982; Eqs.2.1.5- 2.1.9). Additionally, F_{max}, the fishing mortality rate that maximizes YPR, was calculated. Finally, SPR-based $F_{X\%}$ reference points including, $F_{40\%}$, $F_{30\%}$, and $F_{20\%}$ were calculated, which represent the fishing mortality rates that reduce the SSB/R to 40%, 30%, and 20% of the unfished level, respectively (Clark 1991, 2002). The equilibrium yield and spawning stock biomass associated with each of these reference points were calculated as well (Shepherd 1982; Eqs. 2.1.5- 2.1.9).

Equilibrium Yield and Biomass

Equilibrium conditions for striped bass were calculated using spawning stock biomass, yield per recruit estimates, and stock-recruitment function parameters. Equilibrium spawning stock size (Eqs 2.1.5 and 2.1.6), as well as equilibrium recruitment (Eqs 2.1.7 and 2.1.8), and equilibrium median yield (Eq 2.1.9) were then calculated using both Beverton-Holt and Ricker stock-recruitment parameters as described by Shepherd (1982). Equilibrium calculations were also made with Chesapeake Bay specific stock-recruitment relationships, and are presented in Appendix C because inputs were taken from an alternate stock assessment model that is still in development.

Natural Mortality Scenarios

Four natural mortality scenarios were considered that represent different hypotheses

about how natural mortality-at-age has increased due to mycobacteriosis and how the

disease progresses with migration (Table 2.5). These scenarios were compared to the

status quo (SQ) which assumed no increase in natural mortality:

> SQ. Status Quo (No increase in M)
> M1. Age 3+ experience increased M, revert to lower M with migration
> M2. Age 1+ experience increased M, revert to lower M with migration
> M3. Age 3+ experience increased M, does not revert to lower M with migration
> M4. Age 1+ experience increased M, does not revert to lower M with migration.

The SQ scenario is comparable to the natural mortality used in the current stock

assessment, which does not incorporate increased natural mortality due to

mycobacteriosis in the Chesapeake Bay. Scenario M1 most closely resembles the

increased natural mortality vector explored in an alternative model in the most recent

stock assessment for striped bass (Northeast Fisheries Science Center 2019). In this

scenario, an increase in Chesapeake Bay natural mortality (M^B) of +0.14 yr^{-1} (Hoenig

et al. 2017) was applied only to pre-migratory fish ages 3+ that are residents of

Chesapeake Bay[2]; upon migration to the ocean, pre-mycobacteriosis natural mortality

rates were applied (Table 2.3.4). This scenario assumes mycobacteriosis is a chronic

disease that progresses slowly, that conditions in the Chesapeake Bay are responsible

for disease-associated increase in natural mortality (Coutant and Benson 1990, Jacobs

2007, Groner et al. 2018), and that there is regression of the disease with

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[2] The stock assessment uses +0.12 for increased mortality due to mycobacteriosis, but the data used to estimate that number is from a preliminary report generated in 2012, which was updated to +0.14 in Hoenig et al. (2017).

outmigration. Scenario M2 differed from M1 only in that increased natural mortality begins at age 1 instead of age 3. To date, the influence of disease on mortality rates for fish below age 3 has not been studied; however, fish as young as age 0 contract the disease (Appendix A) such that increased mortality cannot be ruled out. Scenario M3 also assumes natural mortality for fish age 3+ is elevated due to mycobacteriosis, but that there is no regression of the disease such that fish continue to experience elevated natural mortality rates even after migrating out of the Chesapeake Bay. Given there is limited evidence of disease regression in striped bass (Groner et al. 2018), it is possible that fish outside of the Chesapeake Bay still experience increased natural mortality from disease or disease-associated symptoms. Finally, scenario M4 represents the worst case scenario in which natural mortality is elevated by disease for age 1+ fish and does not decrease with migration out of the Chesapeake Bay. Under all four natural mortality scenarios, Chesapeake Bay striped bass are subject to higher natural mortality when they enter the fishery compared to the ocean fishery which selects for older individuals that experience lower natural mortality (Figure 2.1). SPR, YPR, biological reference points, equilibrium yield, and equilibrium spawning stock biomass were calculated for each natural mortality scenario and SPR approach combination.

Results

Alternative SPR Calculation Approaches

SPR and resulting biological reference points were affected by the way in which increased natural mortality was incorporated into its calculation. At a fishing

mortality rate of zero, SPR for Approach 1 was the same as the status quo then

remained slightly above the status quo as fishing mortality increased (Figure 2.2).

However, SPR calculated using Approach 2 was less than half that of both the status

quo and Approach 1 at low fishing mortality, converging on similar SPR values as

fishing mortality increased. By comparison, YPR was independent of SPR

calculations. Thus, YPR was the same for both SPR Approaches 1 and 2, but lower

than the status quo because total mortality was higher which reduces total yield

(Figure 2.2 bottom). The maximum YPR for status quo was 0.36, while for

Approaches 1 and 2 it was 0.22, representing a 39% reduction under higher natural

mortality conditions.

Alternative SPR calculation approaches resulted in large differences in fishing

mortality biological reference points for striped bass (Table 2.6). F_{MSY} was 8-34%

lower when increased natural mortality was incorporated into SPR calculations

(Approaches 1 and 2), the associated yield was 51-57% lower, and spawning stock

biomass was 26-55% lower depending on stock-recruitment relationship assumed.

Conversely, F_{max} was 101% higher with the incorporation of increased natural

mortality in YPR calculations and was greater than the F that was estimated to cause

extirpation for both Beverton-Holt and Ricker stock recruitment relationships. For

Approach 1, $F_{20\%}$, $F_{30\%}$, and $F_{40\%}$ were 17-21% higher, and depending on the stock-

recruitment relationship, yield was 42-67% lower, and spawning stock biomass was

46-74% lower than status quo for Approach 1. For Approach 2, $F_{20\%}$, $F_{30\%}$, and $F_{40\%}$

and associated yield were 58-92% lower, while spawning stock biomass remained the same as status quo.

For Approaches 1 and 2, striped bass MSY was more than 50% lower than that of the status quo scenario regardless of the stock-recruitment relationship assumed (Figure 2.3). Although MSY was similar across stock-recruitment relationships, when a Beverton-Holt relationship was assumed, MSY was achieved at a lower fishing mortality rate than when a Ricker relationship was assumed (Figure 2.3). Similarly, spawning stock biomass was lower for Approaches 1 and 2 compared with the status quo, although the magnitude of the difference depended on the assumed stock-recruitment relationship (Figure 2.4).

Natural Mortality Scenarios

The four alternative scenarios describing how mycobacteriosis impacts natural mortality in Chesapeake Bay striped bass produced similar results, but differed from the status quo natural mortality scenario by up to 168% for reference points, 82% for yield, and 86% for spawning stock biomass. For Approach 1, the SPR curves for the alternative natural mortality scenarios were all slightly above the status quo (Figure 2.5 top), but for Approach 2, the SPR curves were lower than the status quo for all natural mortality scenarios (Figure 2.5 bottom), as seen in the original base runs of the model. At low fishing mortality the Approach 2 SPR was lowest for scenarios M3 and M4 (scenarios in which natural mortality did not decrease with migration); however, at high fishing mortality (F>0.4) all natural mortality scenarios produced

similar SPRs (Figure 2.5 bottom). For Approach 2, maximum SPR ranged between

0.435 and 0.184 compared to the status quo of 1. YPR was also similar among natural

mortality scenarios at low fishing mortality (Figure 2.6). However, as fishing

mortality increased, YPR for alternative natural mortality scenarios diverged from the

status quo with scenarios M2 and M4 (elevated natural mortality for ages 1+)

exhibiting the lowest YPR (Figure 2.6). Maximum YPR ranged between 0.22 and

0.15 for natural mortality scenarios compared to the higher status quo maximum YPR

of 0.36.

Striped bass biological reference points differed across natural mortality scenarios

(Table 2.7). Beverton-Holt-based F_{MSY} estimates were fairly stable under different

natural mortality scenarios; however, Ricker-based F_{MSY} estimates decreased up to

59% as natural mortality increased. In contrast, F_{max} was 99-168% higher for

increased natural mortality scenarios compared to the status quo, and increased with

increasing natural mortality. Approach 2 SPR-based reference points, including $F_{40\%}$,

$F_{30\%}$, and $F_{20\%}$, were lower under increased natural mortality scenarios. $F_{X\%}$ reference

points decreased with each of the M1-M4 scenarios, resulting in several negative

fishing mortality reference points.

Additionally, both yield and spawning stock biomass were substantially lower for

alternative natural mortality scenarios relative to status quo (Figures 2.7 and 2.8).

MSY decreased 51-82% from status quo depending on the natural mortality scenario

and stock-recruitment relationship assumed (Figure 2.7). Spawning stock biomass

estimates varied between stock-recruitment functions, but maximum spawning stock biomass decreased 30-86% from status quo depending on the natural mortality scenario and stock-recruitment relationship assumed (Figure 2.8). Among alternate natural mortality scenarios, the highest yield and spawning stock biomass resulted from the M1 scenario and the lowest resulted from the M4 scenario, regardless of stock-recruitment relationship assumed.

Discussion

This study demonstrates that selection of an appropriate SPR calculation approach should be guided by the degree to which compensation for increased natural mortality has been demonstrated to have occurred. Productivity of a stock determines how much fishing can occur before the population is overexploited. Generally, populations with higher natural mortality die at a younger average age and therefore exhibit life history traits that are characteristic of higher stock productivity, such as higher recruitment, faster growth, and maturity at younger ages (Morgan et al. 2014). When natural mortality increases in a population, that increase in mortality may or may not be offset by changes in life history that increase population productivity. Therefore, the selection of reference points for populations with increased natural mortality should be based on the level of compensation that is thought to have occurred and the management goals for the stock.

This analysis of three different approaches to calculating SPR provided evidence that traditional methods may result in some biological reference points that are too

aggressive when there has been an increase in natural mortality and no evidence of compensation (Table 2.6). The incorporation of increased natural mortality in general (Approaches 1 and 2) resulted in lower F_{MSY} compared to status quo, as well as lower yield and SSB. Conversely, F_{max} was unreasonably high, resulting in either extremely low yield and SSB or extinction. Approach 1 (incorporating increased natural mortality in both the fished and unfished portion of the SPR calculation) resulted in elevated $F_{X\%}$ reference points, but lower yield and SSB than status quo. It is also important to note that $F_{30\%}$ and $F_{20\%}$ for Approach 1 were above F_{MSY}. Approach 2 (using increased natural mortality in only the fished portion of the SPR calculation) resulted in more conservative biological reference points relative to the other SPR approaches, but were associated with much lower yield to achieve higher SSB.

There are management risks associated with applying each of the SPR approaches explored in this study. The risk of status quo management, when in reality there has been a change in natural mortality, is that the fishing mortality rate reference point may be too aggressive (Table 2.6). However, natural mortality is difficult to estimate well (Vetter 1988), and detecting changes in natural mortality over time is challenging, particularly when life history-based approaches are used (e.g. Pauly 1980; Hoenig 1983). Additionally, stocks for which we have evidence of increased natural mortality may continue to be managed using biological reference points that assume natural mortality has not changed because of the complications for reference point calculations (Legault and Palmer 2015). This study highlights the fact that the status quo approach can result in overestimates of fishing rate reference points, total

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yield, and SSB (Figures 2.2-2.6) relative to approaches that incorporate increased

natural mortality without assumptions of compensation. As such, increased natural

mortality should be an important consideration when generating reference points for

management.

SPR calculation Approach 1 is potentially problematic for management if natural

mortality has increased but compensation has not occurred because it results in

fishing mortality rate reference points that are likely too high to be sustainable long-

term (Legault and Palmer 2015). This approach also results in lower SSB, which

could be a concern for stocks that are already designated overfished. Approach 2 is

advantageous in that there is no assumption of increased productivity in the stock, and

SPR-based reference points change in a more logical direction (i.e., substantially

decreased $F_{X\%}$ reference points; Table 2.6) when mortality has increased in a

population without evidence of compensation. Although the more conservative

fishing mortality reference points associated with Approach 2 should avoid

overexploitation in the long-term and adhere to a more precautionary management

approach that maintains a larger stock size (Gabriel and Mace 1999), they may be too

conservative to achieve management goals for some fisheries. One of the

management risks associated with this approach is potentially forgone yield if the

increase in natural mortality has been overestimated or if some degree of unaccounted

for compensation has occurred. In order to maintain relatively high SSB with

Approach 2 $F_{X\%}$ reference points, fishing mortality must be very low (or may result in

fishery closures in some of the alternate M scenarios), which may not be ideal for fisheries for which maximizing yield is the main goal (Tables 2.6 and 2.7).

Another management considered that should be taken into account when natural mortality has increased is the yield and SSB associated with reference points. Examining the yield and SSB across the three approaches, there is not always a clear advantage of using Approach 2 because similar yield and SSB can be achieved with status quo or Approach 1 if the percent of unfished biomass is increased. For example, Approach 2 $F_{20\%}$ produces similar yield and SSB to Approach 1 $F_{40\%}$ (Table 2.6), so indicating that comparable precaution can be taken when natural mortality has increased by using traditional SPR methods with more conservative Fx% levels. This study shows that a range of similar yield and SSB levels may be achieved from different SPR calculation approaches and the tradeoffs among resulting reference points generated should be carefully examined as part of the process of proposing new management benchmarks when natural mortality has increased in a population.

Incorporate time-varying natural mortality in reference points can have a large impact on resulting management advice. For example, the 2016 weakfish stock assessment estimated a time-varying natural mortality that increased over time, and SPR-based reference points were estimated using the time-series average natural mortality (ASMFC 2016). This new average M was an increase from the previously assumed natural mortality. While the authors recognized that using the highest natural mortality estimated from the stock assessment model to generate reference points

would not achieve conservation-minded goals, the F30% reference point resulting

from the average natural mortality increased from previous estimates. (Northeast

Fisheries Science Center 2009, ASMFC 2016). If natural mortality has increased

without compensation in this population, using average natural mortality in reference

point calculations may be too aggressive an assumption to recover this stock.

Although the per recruit models in this study are deterministic and do not account for

population stochasticity, they provide a general framework for understanding the

potential consequences of incorporating natural mortality in SPR-based reference

point calculations. In reality, stochastic events such as recruitment pulses or pulse

mortality events may positively or negatively affect stock response to changes in

fishing mortality and baseline M in the short term. Overall, though, this study

demonstrated that SPR calculation Approach 2 provides a valuable precautionary tool

for setting biological reference points when natural mortality has increased and there

has been no evidence of compensation.

A case study for Atlantic striped bass

This case study for Atlantic striped bass evaluated the potential consequences of

adopting different methods for incorporating increased M in biological reference

points. In the Chesapeake Bay, empirical evidence suggests that additional

compensation has not occurred. No significant change has been observed in

maturation rate of diseased fish (Gervasi et al. 2019), and growth of diseased fish was

shown to be compromised (i.e., reduced size-at-age; Latour et al. 2012). As there is

no evidence that compensation has occurred in the striped bass stock, SPR-based

reference points generated using Approach 2 could be a valuable tool for use in

management. All the Approach 2 $F_{X\%}$ reference points would require large reductions

in fishing mortality from the status quo when elevated natural mortality is taken into

account; however, the SSB would be relatively large. For striped bass, maintaining a

large SSB may be a more important consideration for management as there is

widespread stakeholder interest in the catch and release fishery and trophy-sized fish.

Other reference points could be considered for striped bass, but they may not achieve

the goals for the stock, such as F_{MSY}, which results in a higher yield at a relatively low

stock size (Table 2.6). If Approach 1 reference points are considered, management

may wish to pursue a higher percent of unfished biomass than what is currently being

achieved to avoid the lower SSB associated with this approach. Using Approach 1

without increasing the percent of unfished biomass could be troubling given the

striped bass stock is overfished at this time (although not on the verge of a collapse as

in the 1970s; Northeast Fisheries Science Center 2019). Reference points generated

using a more precautionary approach would be a prudent decision for striped bass that

might help recover the stock to its biomass target.

Current reference points used in striped bass management are based on estimated SSB

in 1995 when the stock was declared recovered but before the onset of the

mycobacteriosis epizootic. The current F threshold was estimated in the most recent

stock assessment to be 0.240 yr^{-1} and the F target was estimated to be 0.197 yr^{-1}.

These fishing mortality rates approximately correspond with SPRs of 18.8-21.1%

under Approach 2 assumptions of increased M and no compensation. In most cases, F20% is considered too aggressive for a long-lived finfish stock such as striped bass, and F35-40% or higher is more typical used to avoid overexploitation (Clark 2002). Using Approach 2, F_{30} and $F_{40\%}$ were estimated to be 0.044 and 0.119 yr^{-1}. The estimated fishing mortality rate in 2017 was 0.307 yr^{-1} which is substantially higher than the biological reference points suggested by Approach 2 and is approximately equal to $F_{16\%}$. By not accounting for increased M since 1995, the current F reference points are likely too high to achieve stock SSB goals. This study demonstrated that the current empirical reference points used in management may be too aggressive to achieve the biomass goals for striped bass.

The most recent stock assessment included an SPR analysis for both the base model used in management and an alternate model that incorporated migration. The SPR analysis for the base model resulted in what was thought to be unreasonably high female SSB, so the reference points generated were not considered reliable. However, the alternate model that incorporated migration suggested that the 1995 SSB threshold corresponded to approximately SPR20%, a reference point usually considered too aggressive for long-lived finfish. This study also suggested that the current empirical F threshold of 0.240 $year^{-1}$ is roughly equivalent to F20% under Approach 2 assumptions of increased M and no compensation.

Alternate scenarios M1-M4, representing different assumptions of how mycobacteriosis influences natural mortality-at-age, resulted in similar lower

estimates of SPR, YPR, yield, and SSB compared with the status quo scenario of no increase in natural mortality. This indicates that the decision to incorporate increased M in SPR-based striped bass reference points can have a large impact on the stock size and its fisheries. Among alternate M scenarios, M1 (the natural mortality scenario that most closely resembled the assumptions made in an alternate stock assessment model used in the last assessment to explore the impact of mycobacteriosis) produced the most optimistic results (Figures 2.5-2.8). This suggests that, if the assumptions made in the alternate assessment model about the age at which increased natural mortality begins or the dampening influence of coastal migration on natural mortality is incorrect, the effect of mycobacteriosis could be worse than currently described in the assessment. For example, F20% estimated using Approach 2 was 0.517 year^{-1} for scenario M1 vs 0.041 year^{-1} for the worst case scenario M4 (Table 2.7). An additional concern is that F40% was estimated to be negative for all scenarios except M1, F30% was estimated to be negative for M3 and M4, and F20% was estimated to be negative for M4 (Table 2.7). This implies that, under several alternate M scenarios, SSB targets may not be achievable, even if the fishery were to close.

Legault and Palmer (2015) recommend estimating F_{MSY} for multiple stock-recruitment relationships and evaluating the tradeoffs between yield and SSB when developing biological reference points in light of changes in M. This striped bass case study indicated that the two stock-recruitment relationships estimated fairly similar overall yield and SSB. However, when a Beverton-Holt relationship was assumed,

MSY was achieved at a lower fishing mortality rate than when a Ricker relationship was assumed, indicating that incorrect choice of stock-recruitment relationship could result in overestimation of F_{MSY} reference points (Figure 2.3). A Beverton-Holt relationship is more typically assumed for striped bass, which also appears to be a more precautionary choice for F_{MSY}.

Given the overfished status of the stock, managers are currently considering a transition from the use of empirical reference points to biological reference points (ASMFC 2020). If SPR-based reference points are adopted, the way in which increased M due to mycobacteriosis is incorporated should be carefully considered. This study showed that increased M from mycobacteriosis made a substantial difference in the resulting reference points, yield, and SSB for striped bass. Failing to account for increased M in SPR-based reference points or assuming that compensation has occurred could result in overly aggressive management advice that will not help the stock recover. Because compensation has not occurred in the population (Latour et al. 2012, Gervasi et al. 2019), recovery of striped bass may benefit from the adoption of SPR-based biological reference points that assume M has increased with no concurrent increase in productivity (Approach 2).

Tables

Table 2.1 Stock recruitment and yield model equations.

	Equation	Description
2.1.1	$$S_y = \sum_a Mat_a W^{SSB}{}_{y,a} N_{y,a}$$	Spawning stock biomass
2.1.2	$$R = \frac{\alpha S}{\beta + S}$$	Beverton-Holt recruitment
2.1.3	$$R = \alpha S e^{-\beta S}$$	Ricker recruitment
2.1.4	$$LL = \sum_y \ln(\sigma) + \frac{0.5(obsR - predR)^2}{\sigma^2}$$	Loglikelihood function for recruitment
2.1.5	$$S^* = \alpha(S/R) - \beta$$	Beverton-Holt equilibrium spawning stock biomass
2.1.6	$$S^* = \frac{\log(\alpha) + \log(S/R)}{\beta}$$	Ricker equilibrium spawning stock biomass
2.1.7	$$R^* = \frac{\alpha S^*}{\beta + S^*}$$	Beverton-Holt equilibrium recruitment
2.1.8	$$R^* = \alpha S^* e^{-\beta S^*}$$	Ricker equilibrium recruitment
2.1.9	$$Y^* = YPR \; x \; R^*$$	Equilibrium yield

Table 2.2. Symbols and description of variables used in per recruit models.

Symbol	Description
a	age
y	year
F	Fishing mortality
B	Chesapeake Bay stock
O	Coastal ocean stock
r	Fishing mortality ratio for Chesapeake Bay to ocean
s	selectivity
m	Migration rate from Chesapeake Bay to ocean
M	Natural mortality
Z	Total mortality
N	Abundance
W	Weight
SSB	Female spawning stock biomass
YPR	Yield per recruit
Mat	Proportion mature
SSB/R	Spawning stock biomass per recruit
SPR	Spawning potential ratio
S	Stock size
R	Recruitment
LL	Log-likelihood
σ	Log-likelihood parameter
S^*	Equilibrium stock size
R^*	Equilibrium recruitment
Y^*	Equilibrium yield
α	Stock recruitment parameter
β	Stock recruitment parameter

Table 2.3. Life history and fishery parameters used in striped bass per recruit models. Catch weight-at-age and spawning weight-at-age were calculated as the mean of stock-wide estimates from 2015-2017. Maturity-at-age are stock-wide estimates. Regional selectivity-at-age estimates represent the most recent selectivity time block (1996-2017 in Chesapeake Bay; 1997-2017 in the ocean). Assumed Chesapeake Bay natural mortality-at-age represented as ocean natural mortality-at-age with additional mycobacteriosis-induced mortality of 0.14 yr^{-1} for ages 3+ (Hoenig et al. 2017; Northeast Fisheries Science Center 2019).

Age	Catch weight	Spawning weight	Maturity	Chesapeake Bay selectivity	Ocean selectivity	Migration rate	Chesapeake Bay natural mortality	Ocean natural mortality
1	0.18	0.14	0.00	0.01	0.00	0.01	1.13	1.13
2	0.40	0.34	0.00	0.07	0.02	0.02	0.68	0.68
3	0.92	0.76	0.00	0.32	0.09	0.04	0.59	0.45
4	1.48	1.29	0.09	0.78	0.21	0.06	0.47	0.33
5	2.39	2.07	0.32	0.99	0.37	0.10	0.39	0.25
6	3.48	3.12	0.45	0.99	0.53	0.16	0.33	0.19
7	4.59	4.26	0.84	0.95	0.67	0.24	0.29	0.15
8	5.68	5.37	0.89	0.91	0.77	0.35	0.29	0.15
9	6.80	6.50	1.00	0.86	0.85	0.48	0.29	0.15
10	8.39	8.02	1.00	0.82	0.90	0.61	0.29	0.15
11	9.59	9.26	1.00	0.78	0.94	0.72	0.29	0.15
12	10.86	10.47	1.00	0.74	0.96	0.81	0.29	0.15
13	12.81	12.39	1.00	0.71	0.97	0.88	0.29	0.15
14	14.55	14.12	1.00	0.67	0.98	0.93	0.29	0.15
15+	16.12	16.12	1.00	0.64	0.99	0.95	0.29	0.15

Table 2.4. Per recruit model equations.

	Equation	Description
2.4.1	$F^B{}_a = s^B{}_a F r$	Fishing mortality-at-age for Chesapeake Bay
2.4.2	$F^O{}_a = s^O{}_a F(1-r)$	Fishing mortality-at-age for ocean
2.4.3	$F_a = F^B{}_a(1-m) + F^O{}_a m$	Fishing mortality-at-age with migration rates from Chesapeake Bay
2.4.4	$M_a = M^B{}_a(1-m) + M^O{}_a m$	Natural mortality–at-age with migration rates from Chesapeake Bay
2.4.5	$Z_a = F_a + M_a$	Total mortality-at-age
2.4.6	$N_{a+1} = N_a e^{-Z_a}$	Abundance-at-age
2.4.7	$N_{15+} = N_{14} \dfrac{e^{-Z_a}}{1 - e^{-Z_a}}$	Abundance of plus group (ages 15+)
2.4.8	$YPR = \sum_a \dfrac{F^B{}_a + F^O{}_a}{Z_a}(1 - e^{-Z_a})N_a W_a$	Yield per recruit for Chesapeake Bay and ocean

Table 2.5. Natural mortality-at-age (M; yr^{-1}) vectors used in model scenarios compared to status quo (no natural mortality increase; SQ). Scenarios include alternate natural mortality 1 (striped bass age 3+ experience increased natural mortality in the Chesapeake Bay and revert to lower natural mortality with migration to the ocean; M1); alternate 2 (striped bass age 1+ experience increased natural mortality in the Chesapeake Bay and revert to lower natural mortality with migration to the ocean; M2); alternate 3 (striped bass age 3+ experience increased natural mortality in the Chesapeake Bay and ocean; M3); and alternate 4 (striped bass age 1+ experience increased natural mortality in the Chesapeake Bay and ocean; M4).

Age	SQ	M1	M2	M3	M4
1	1.13	1.13	1.268	1.13	1.27
2	0.68	0.68	0.817	0.68	0.82
3	0.45	0.585	0.585	0.59	0.59
4	0.33	0.461	0.461	0.47	0.47
5	0.25	0.376	0.376	0.39	0.39
6	0.19	0.308	0.308	0.33	0.33
7	0.15	0.256	0.256	0.29	0.29
8	0.15	0.241	0.241	0.29	0.29
9	0.15	0.223	0.223	0.29	0.29
10	0.15	0.205	0.205	0.29	0.29
11	0.15	0.189	0.189	0.29	0.29
12	0.15	0.176	0.176	0.29	0.29
13	0.15	0.167	0.167	0.29	0.29
14	0.15	0.160	0.160	0.29	0.29
15+	0.15	0.156	0.156	0.29	0.29

Table 2.6. Reference points and associated yield and spawning stock biomass (SSB) estimates for each spawning potential ratio modeling approach and stock-recruitment relationship: Beverton-Holt (B-H) and Ricker. Per recruit modeling approaches include status quo (no increase in natural mortality; SQ), Approach 1 (increased natural mortality in both fished and unfished spawning stock biomass per recruit, SSB/R; A1), and Approach 2 (increased natural mortality in fished SSB/R only; A2). Dashed lines represent negative values of yield and SSB.

Approach	SQ		A1		A2	
SR relationship	B-H	Ricker	B-H	Ricker	B-H	Ricker
F_{MSY}	0.36	0.58	0.33	0.38	0.33	0.38
Yield	55.35	55.85	23.64	27.47	23.64	27.47
SSB	166.28	100.26	74.06	73.90	74.06	73.90
F_{max}	0.605		1.213		1.231	
Yield	50.32	55.80	-	-	-	-
SSB	86.23	95.63	-	-	-	-
F40%	0.231		0.271		0.0176	
Yield	52.21	38.26	23.25	25.53	3.98	2.92
SSB	248.72	182.27	89.86	98.65	248.72	182.27
F30%	0.336		0.400		0.088	
Yield	55.24	47.49	23.31	27.42	14.44	12.41
SSB	178.45	153.38	59.30	69.76	178.45	153.38
F20%	0.517		0.627		0.217	
Yield	53.05	55.25	18.69	18.89	22.16	23.08
SSB	108.18	112.67	28.74	29.05	108.18	112.67

Table 2.7. Instantaneous fishing mortality rate (yr^{-1}) reference points estimated using spawning potential ratio calculation Approach 2 and alternate stock-recruitment curves: Beverton-Holt (B-H) and Ricker. See Tables 2-5 and 2-6 for full description of natural mortality scenarios and SPR approaches, respectively. Dashes represent values that are unobtainable (i.e., they require negative values of fishing mortality).

Scenario	SQ	M1	M2	M3	M4
F_{MSY} B-H	0.36	0.33	0.27	0.37	0.29
F_{MSY} Ricker	0.58	0.38	0.28	0.35	0.24
F_{max}	0.605	1.21	1.23	1.60	1.63
F40%	0.231	0.018	-	-	-
F30%	0.336	0.088	0.012	-	-
F20%	0.517	0.217	0.114	0.089	-

Figures
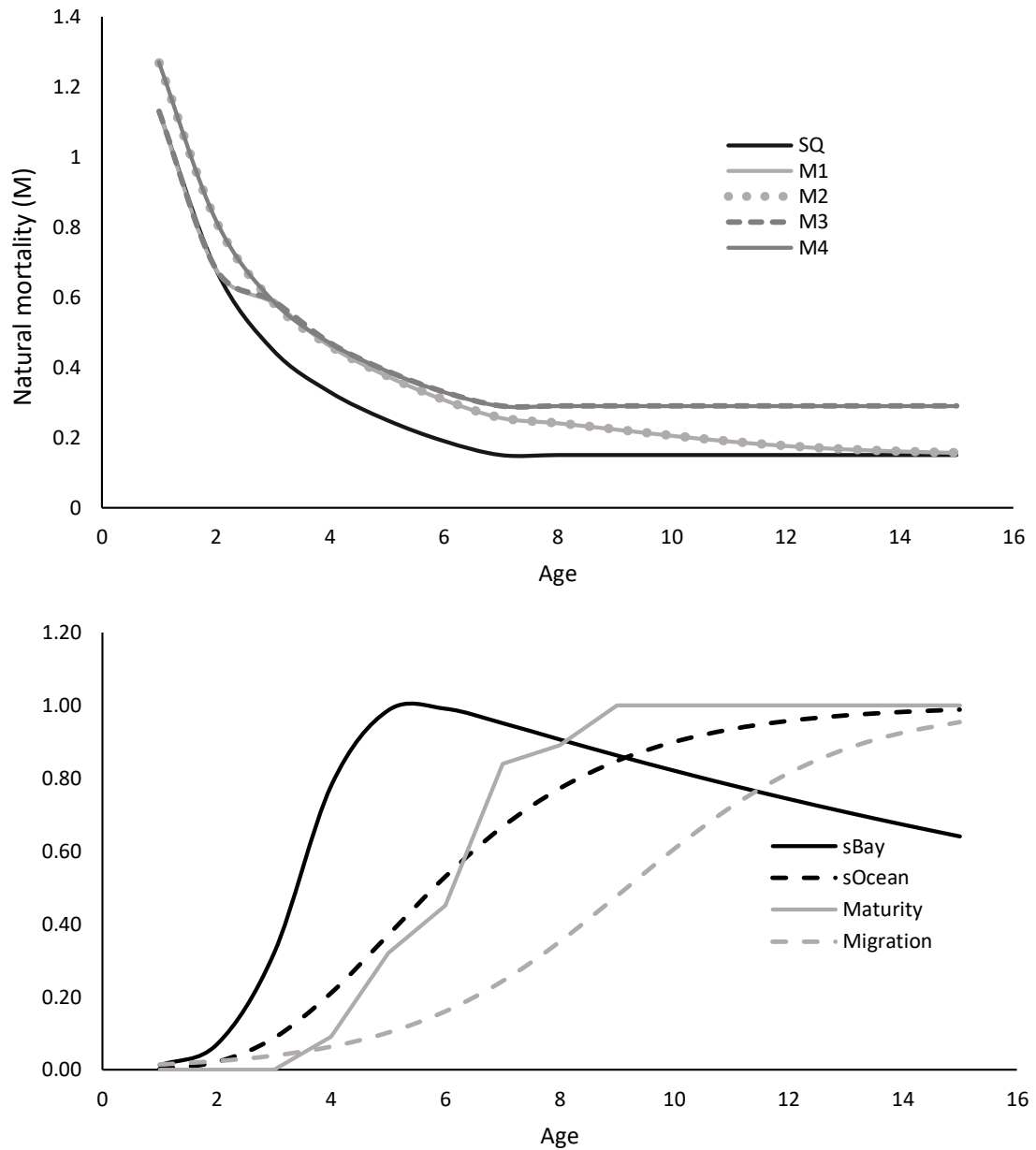
Figure 2.1. Natural mortality (M) ogives assumed in alternate scenarios compared to the status quo (SQ) M ogive (top) and regional selectivity-at-age (s), maturity-at-age, and migration proportion-at-age (bottom). Scenarios include striped bass age 3+ experience increased natural mortality in the Chesapeake Bay and revert to lower natural mortality with migration to the ocean (M1), striped bass age 1+ experience increased natural mortality in the Chesapeake Bay and revert to lower natural mortality with migration to the ocean (M2), striped bass age 3+ experience increased natural mortality in the Chesapeake Bay and ocean (M3), and striped bass age 1+ experience increased natural mortality in the Chesapeake Bay and ocean (M4).
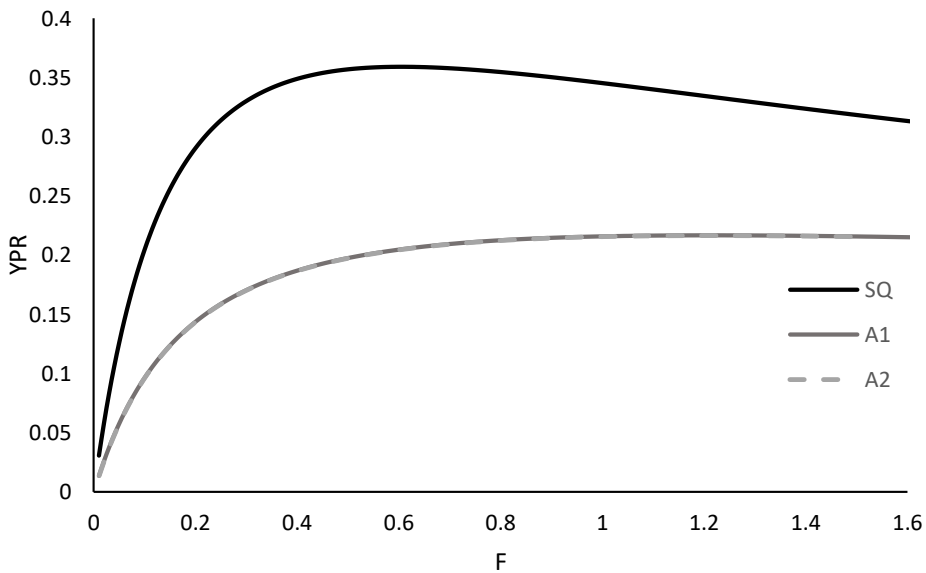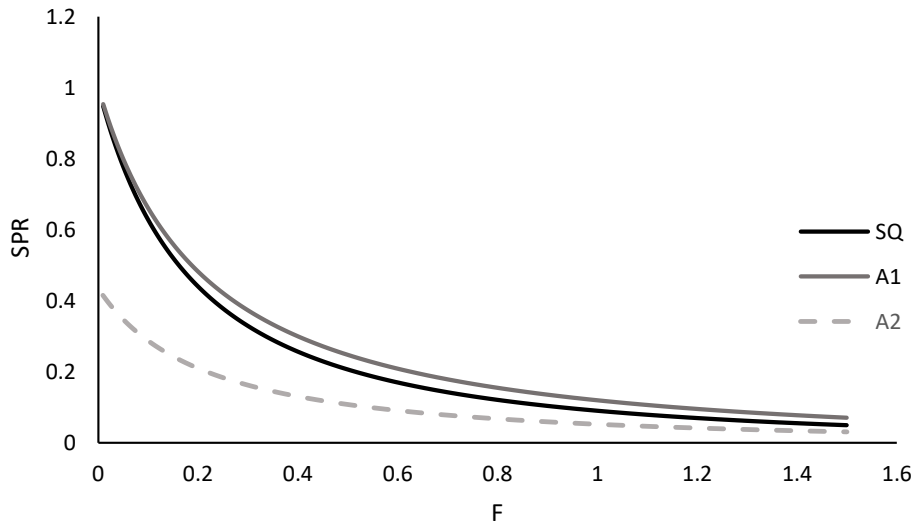
Figure 2.2. Spawning potential ratio (SPR; top) and yield per recruit (YPR; bottom) for striped bass calculated assuming status quo SSB/R calculation (SQ; no increase in natural mortality), Approach 1 (A1; increased natural mortality in both fished and unfished SSB/R), and Approach 2 (A2; increased natural mortality in only fished SSB/R) across a range of fishing mortality rates (F)
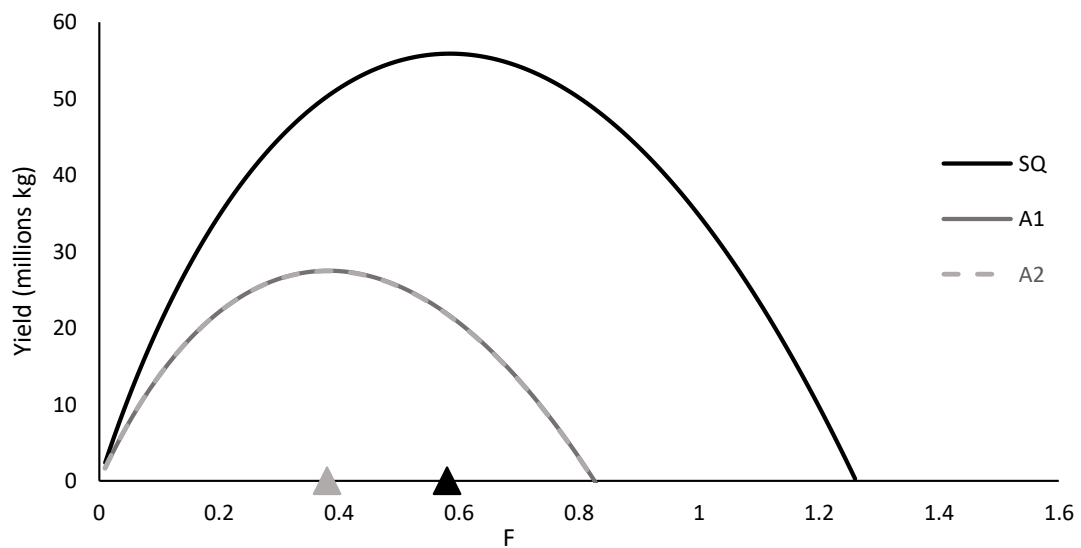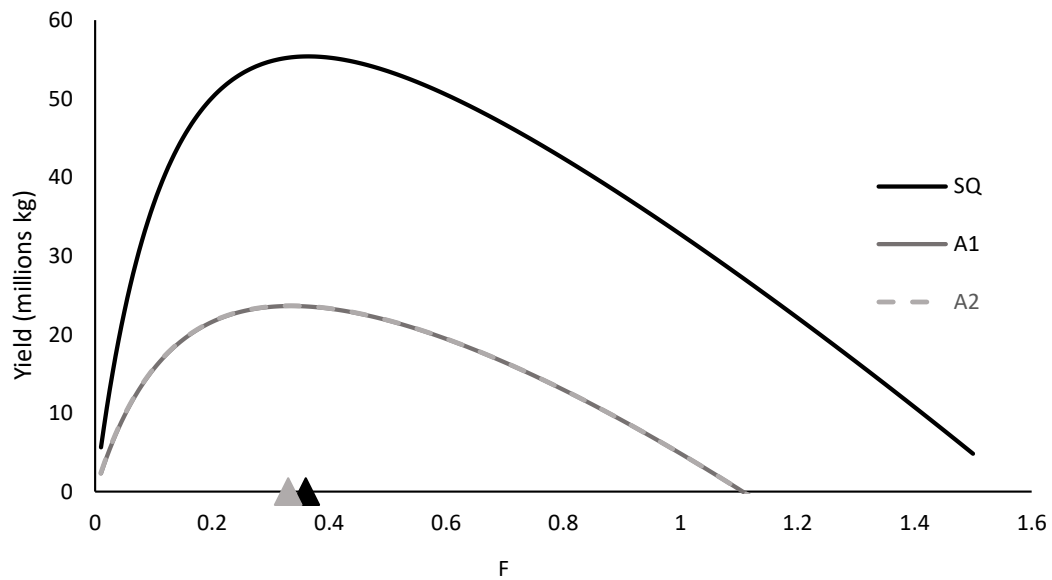
Figure 2.3. Striped bass equilibrium yield for status quo (SQ) and Approaches 1 and 2 (A1, A2) using Beverton-Holt (top) and Ricker (bottom) stock-recruitment functions across a range of fishing mortality rates (F). The fishing mortality rates that achieve maximum sustainable yield for SQ (black triangle) and A1 and A2 (grey triangle) are indicated. See Figure 2-1 for a full description of SPR approaches.
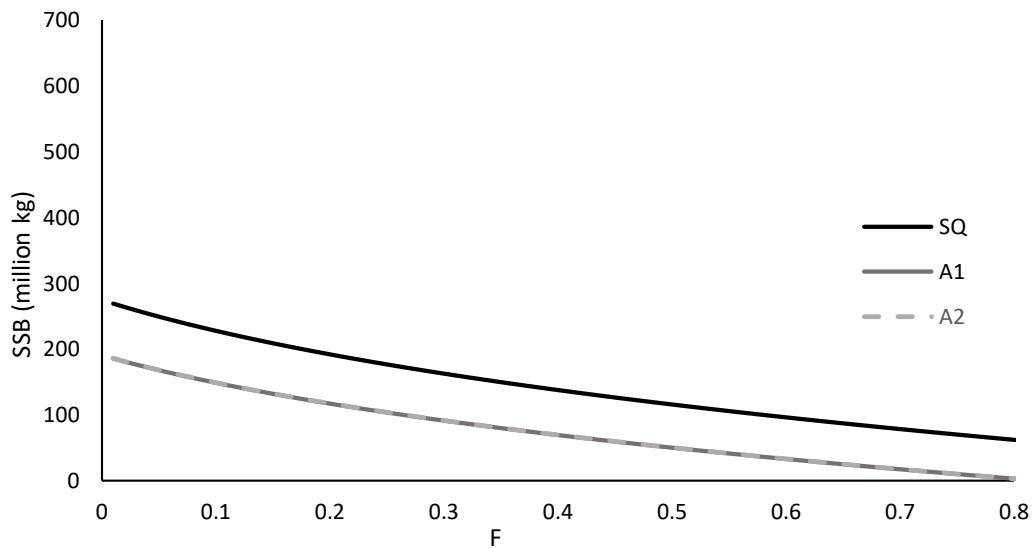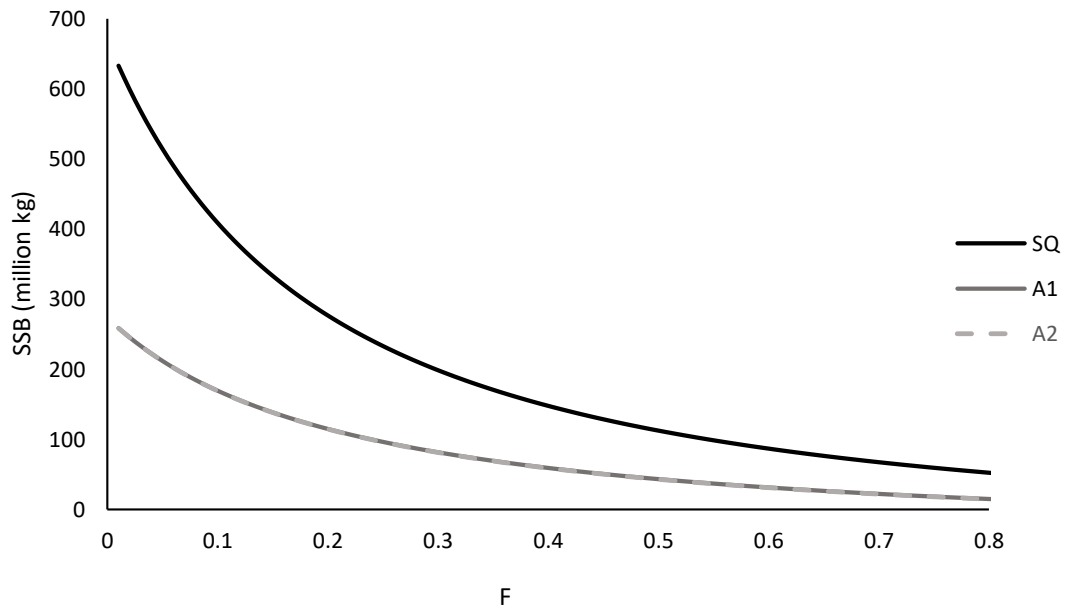
Figure 2.4. Striped bass equilibrium spawning stock biomass (SSB) for status quo (SQ) and alternate approaches 1 and 2 (A1, A2) using Beverton-Holt (top), Ricker (bottom) stock-recruitment estimates and M1 scenario for A1/A2 across a range of fishing mortality rates (F). See Figure 2-1 for a full description of SPR approaches.
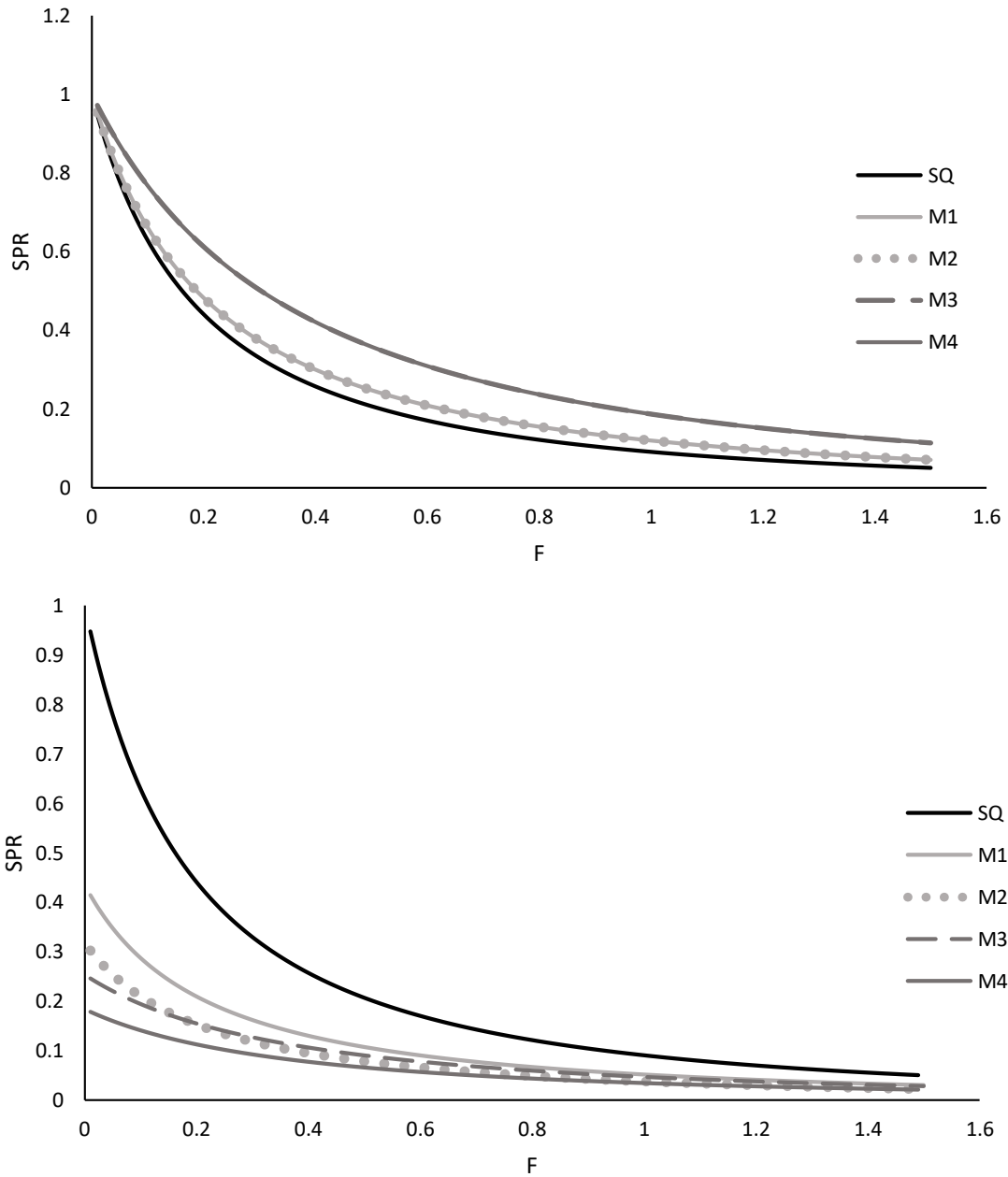
Figure 2.5. Spawning potential ratio (SPR) curves comparing status quo (SQ) natural mortality assumptions to alternate natural mortality scenarios using SPR calculation Approach 1 (A1; top) and Approach 2 (A2; bottom) across a range of fishing mortality rates (F). For full description of approaches, see Figure 2-1 and for natural mortality scenarios, see Figure 2-3.
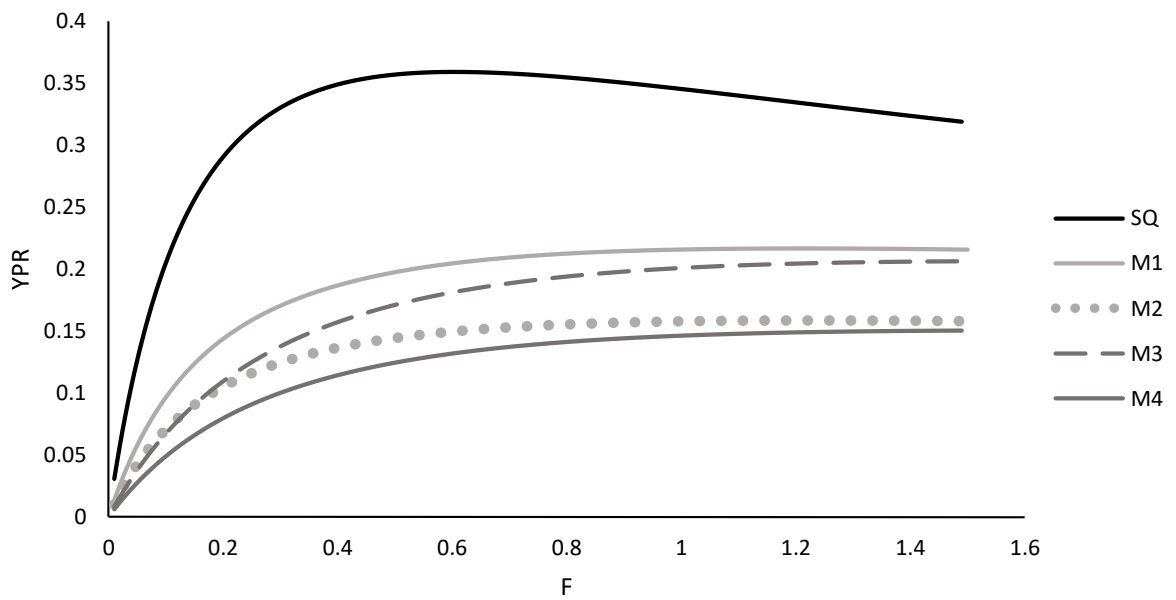
Figure 2.6. Yield per recruit (YPR) curves comparing status quo (SQ) to natural mortality scenarios used in SPR calculation Approaches 1 and 2 across a range of fishing mortality rates (F). For full description of SPR approaches, see Figure 2-1, and for natural mortality scenarios, see Figure 2-3.
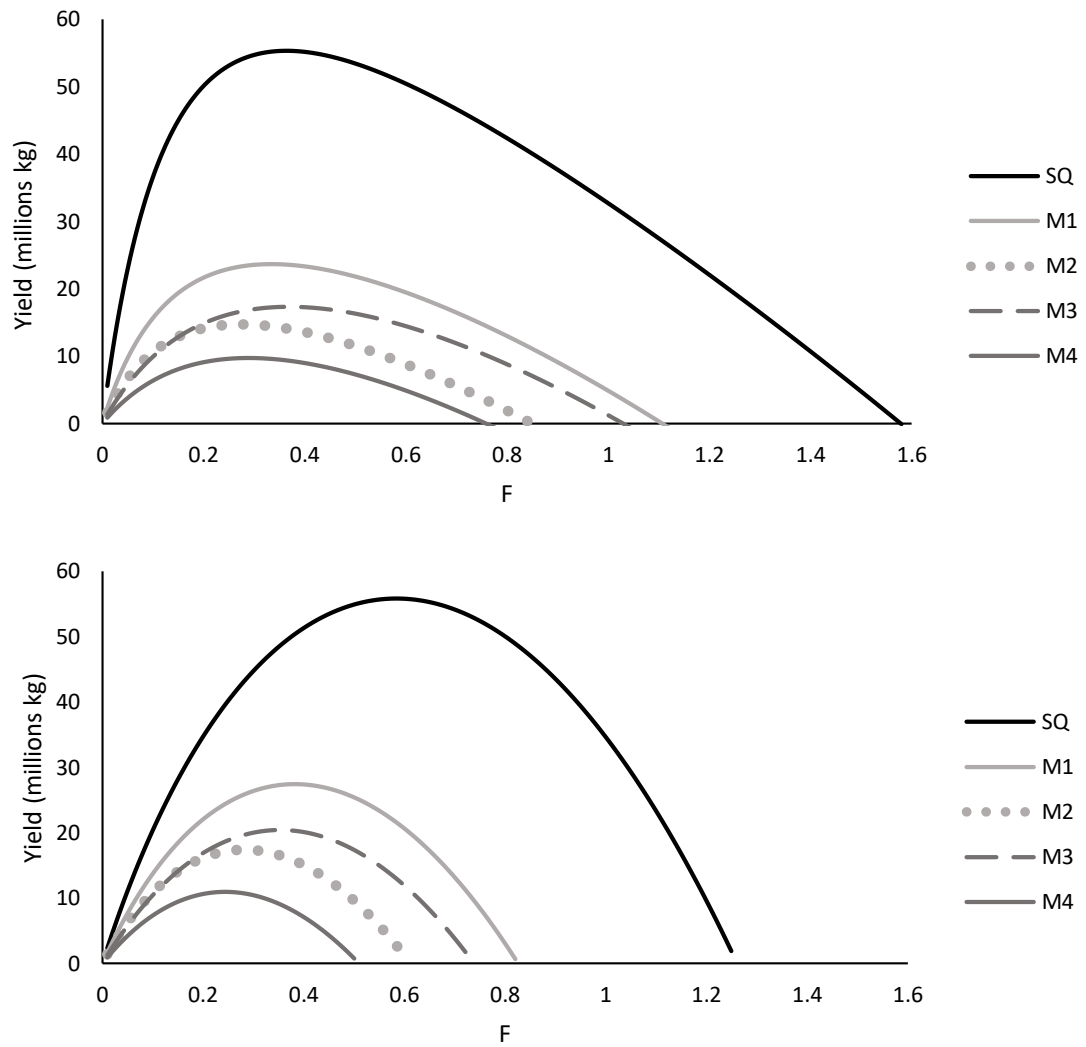
Figure 2.7. Striped bass equilibrium yield comparing status quo (SQ) to natural mortality scenarios for Beverton-Holt (top), Ricker (bottom) stock-recruitment functions across a range of fishing mortality rates (F). For full description of natural mortality scenarios, see Figure 2-3.
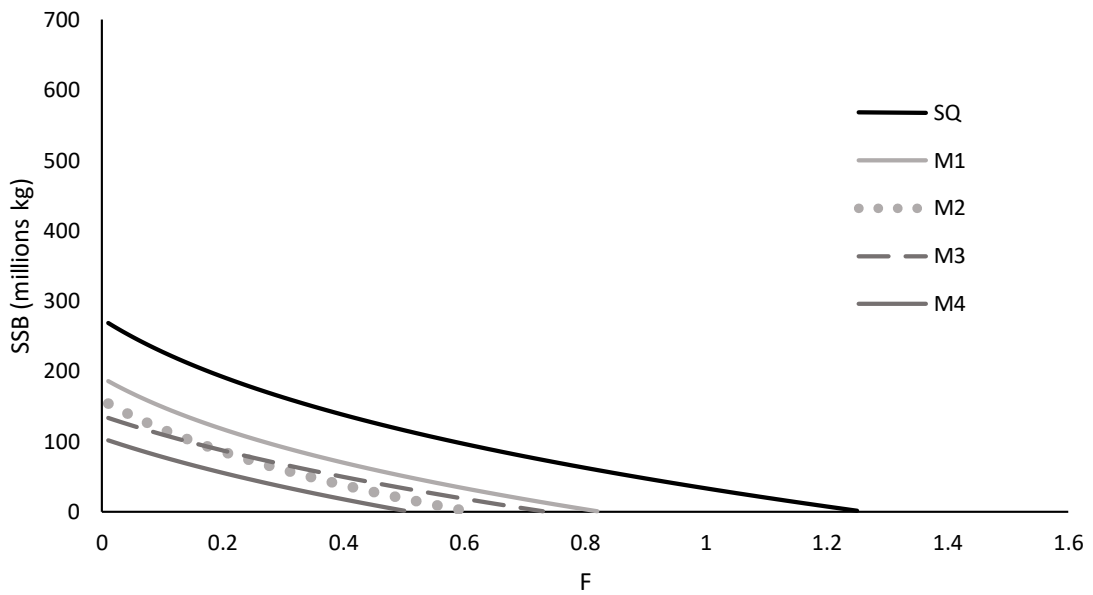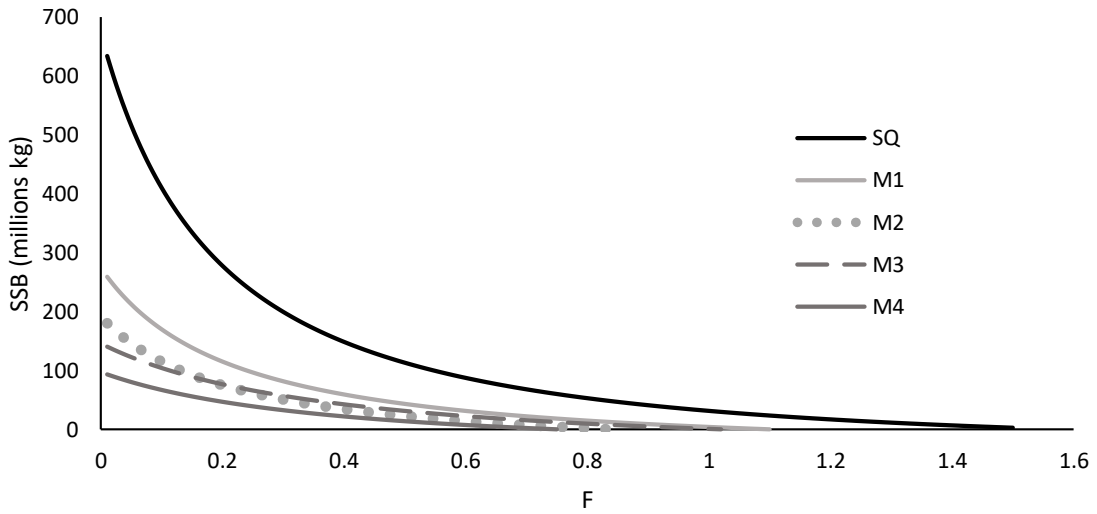
Figure 2.8. Striped bass spawning stock biomass (SSB) comparing status quo (SQ) to natural mortality scenarios for Beverton-Holt (top), Ricker (bottom) stock-recruitment functions across a range of fishing mortality rates (F). For full description of natural mortality scenarios, see Figure 2-3.

Appendices

Appendix A: Mycobacteriosis prevalence and severity trends for striped bass in Maryland waters of the Chesapeake Bay

Disease data are commonly characterized as an apparent prevalence, calculated as the number of individuals that test positive for disease out of the sample collected, thought to be representative of the whole population. Apparent prevalence of mycobacteriosis in striped bass from Maryland waters of the Chesapeake Bay exhibits a general increasing trend from 1997-2017, reaching over 60% in recent years (Figure 1-A). Prevalence by age and sex depicts an increasing trend as well, with the exception of low sample size age-sex combinations, and older age groups attain almost 80% prevalence of disease (Figure 2-A). It is important to note that there does not appear to be lower prevalence for older females as previously seen (Gauthier et al. 2008), which was likely the result of low sample size for older age classes of female fish.

For mycobacteriosis in striped bass, apparent prevalence will vary based on whether visceral (spleen testing) or dermal (external examination) methods were used to determine disease status, as fish test positive in spleen samples before they show external symptoms due to the slow progression of mycobacteriosis (Gauthier et al. 2003). Increased catchability for diseased individuals has been proposed to result in inaccurately high apparent prevalence in striped bass (Groner et al. 2018).

Additionally, an unbalanced sampling design could alter the apparent prevalence if there are spatial or temporal differences are not taken into account.

The proportion of fish at each disease severity stage can be calculated by dividing the number of fish in a stage by all the diseased fish in the sample. The proportion of diseased fish that show any form of external lesions (mild-severe) decreases in more recent years from a peak around 50% of diseased fish showing external lesions in 2012 to only about 20% in 2017 (Figure 3-A). The main difference appears to be in the mildly diseased fish, which could be a result of human bias in external lesion analysis, changing sampling locations, or improved testing methods to identify diseased fish. Severity proportion by age increases until age 5 where it appears to level off to age 10+, with some variability around ages 7-9 likely due to low sample sizes (Figure 4-A). This shows support for the chronic and progressive nature of mycobacteriosis in striped bass.
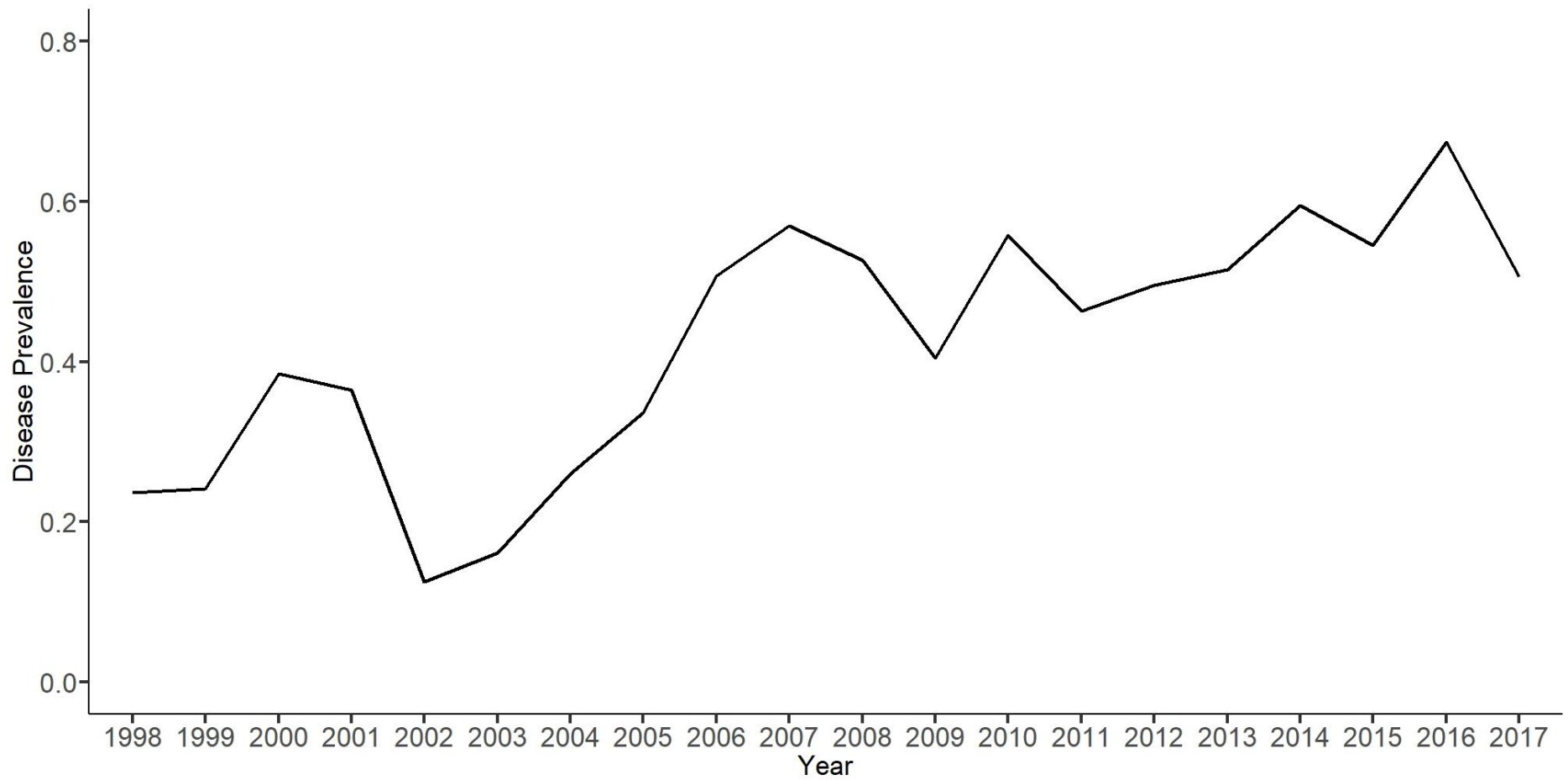
Figure A-1. Annual mycobacteriosis prevalence for striped bass collected from Maryland waters of the Chesapeake Bay as part of the Maryland Department of Natural Resources Fish Health Program from September-November 1998-2017.
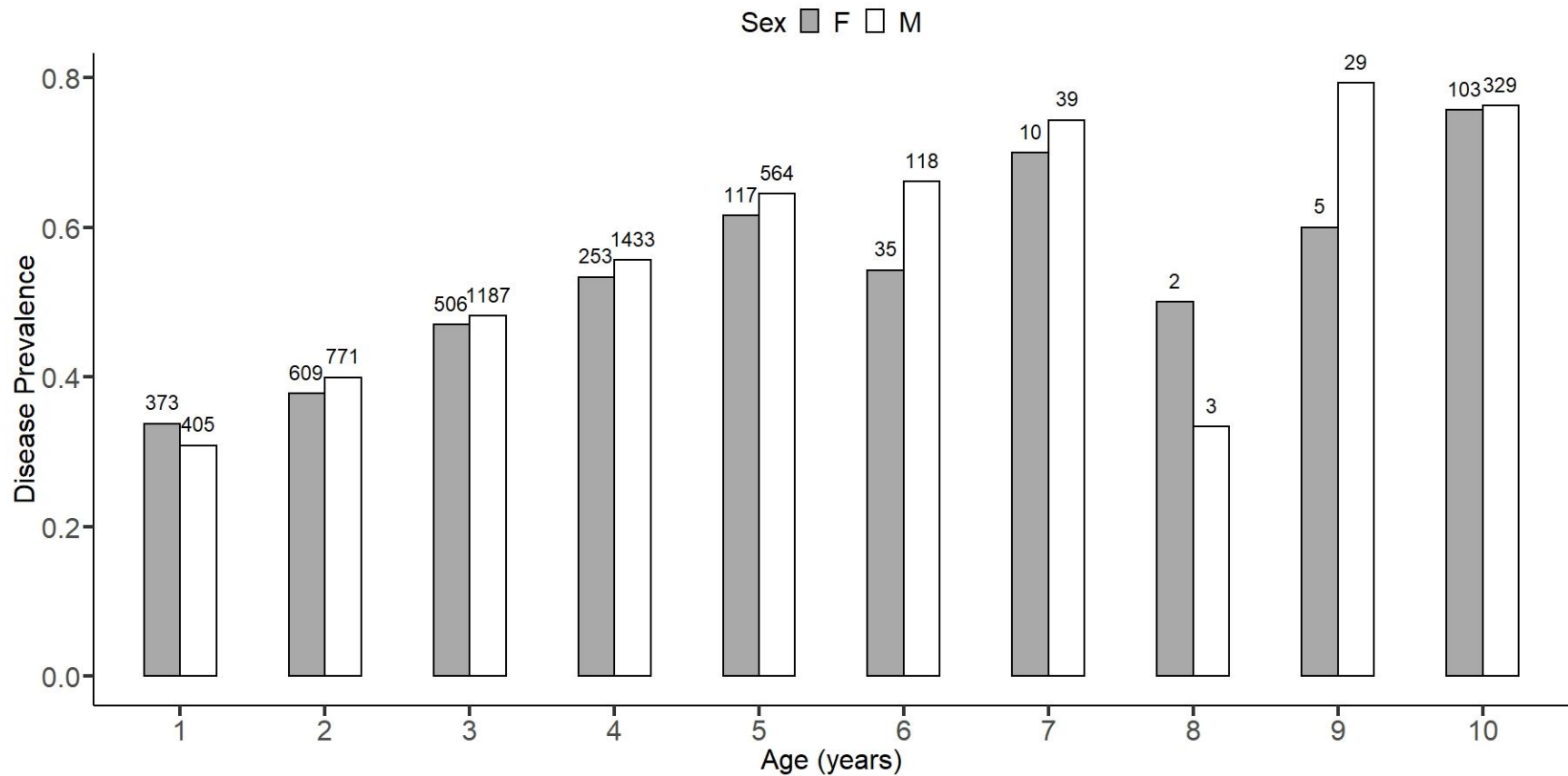
Figure A-2. Mycobacteriosis prevalence for striped bass ages 1-10+ collected from Maryland waters of the Chesapeake Bay as part of the Maryland Department of Natural Resources Fish Health Program from September-November 1998-2017. Grey bars represent female striped bass and open bars represent male striped bass. Sample sizes are denoted above bars.
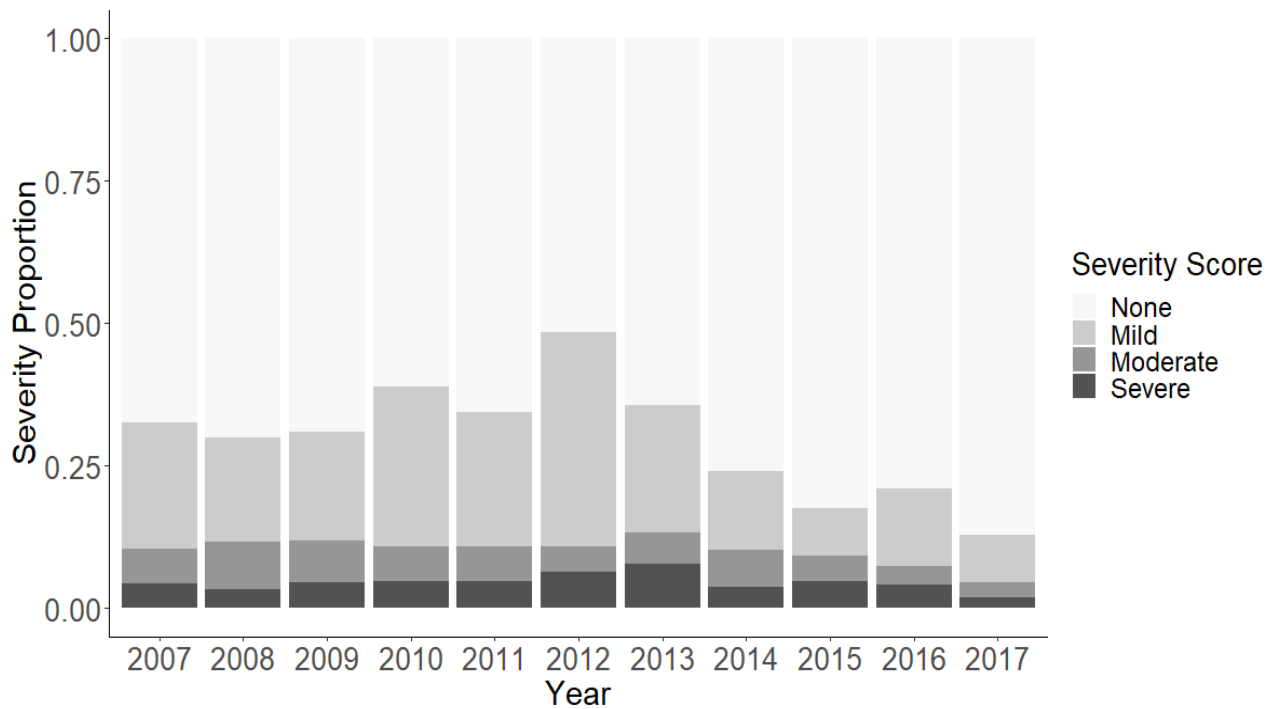
Figure A-3. Annual proportion of striped bass by mycobacteriosis severity score for fish collected from Maryland waters of the Chesapeake Bay as part of the Maryland Department of Natural Resources Fish Health Program from September-November 2007-2017.
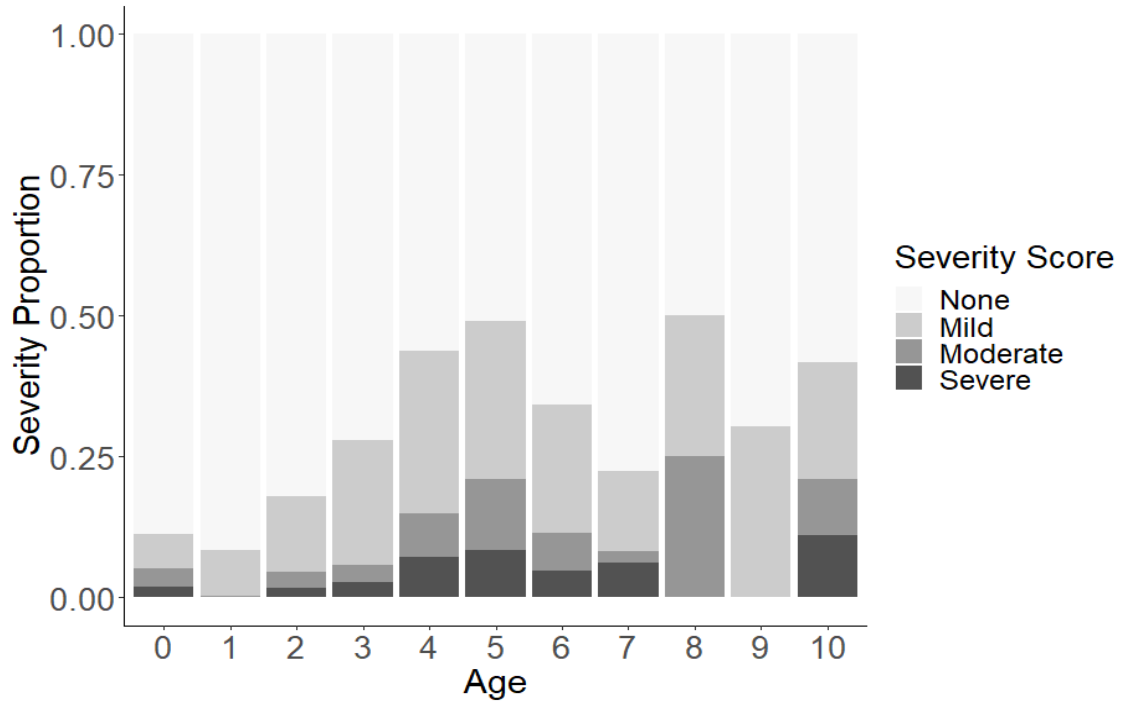
Figure A-4. Proportion of striped bass ages 0-10+ by mycobacteriosis severity score for fish collected from Maryland waters of the Chesapeake Bay as part of the Maryland Department of Natural Resources Fish Health Program from September-November 2007-2017.

Appendix B: Influence of water quality on *Mycobacterium* spp. abundance in Maryland waters of the Chesapeake Bay

Introduction

Environmental mycobacteria are known to cause a wide variety of diseases in both humans and animals (Wagner and Young 2004). Additionally, many fish species have experienced disease caused by *Mycobacterium* spp., including aquaculture, aquarium, and wild populations from freshwater, brackish, and marine environments (Decostere et al. 2004). In the Chesapeake Bay, the current mycobacteriosis epizootic in Atlantic striped bass (*Morone saxatilis*) is of particular concern because of potential increased mortality and lifetime reproductive losses, as well as the ecological and economic importance of the species (Gervasi 2015, Hoenig et al. 2017). The mode of infection and transmission of mycobacteriosis in striped bass is not well understood, and, as a result, further investigating *Mycobacterium* in the water column may be imperative to understanding this disease.

Although environmental mycobacteria are thought to be ubiquitous, increased abundance of *Mycobacterium* in the water column could increase susceptibility to disease infection. In general, environmental *Mycobacterium* growth has been linked to lower pH, higher organic materials, suspended particulates, and other water quality features associated with degraded habitats and climate change (Kane et al. 2007). *Mycobacterium* concentrations in the Maryland coastal bays have also been linked to

low dissolved oxygen and high nutrient loads characteristic of eutrophication, as well as lower salinity (Jacobs et al. 2009e). Furthermore, in the Chesapeake Bay, many heterotrophic bacteria that cause diseases are likely to increase in the face of warming water temperatures due to climate change (Najjar et al. 2010). Quantifying water quality drivers of *Mycobacterium* spp. abundance in Maryland waters of the Chesapeake Bay may shed light on disease dynamics in striped bass.

Methods

Data

Maryland Department of Natural Resources (MDNR) monitored *Mycobacterium* spp. in the water column starting in 2006, primarily during the months of April, June, July, August, and October, at a subset of Chesapeake Bay Program Water Quality monitoring sites (Figure 1-B). A sample was collected at each site from the surface of the water column and each sample was tested for *Mycobacterium* abundance (cells/mL). The test did not specify the species of *Mycobacterium*. At the same time, water samples were collected and tested for a variety of other features including dissolved oxygen, temperature, pH, nutrients, etc. Chesapeake Bay Program Water Quality data was downloaded from the Chesapeake Bay Program DataHub[3] and matched with the corresponding *Mycobacterium* spp. abundance information by monitoring station and sampling date.

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[3] http://data.chesapeakebay.net/WaterQuality, accessed 2/5/2019

Modeling approach

A binomial generalized linear model with a logit link function was constructed to evaluate the relationships between *Mycobacterium* spp. abundance and water quality in Maryland waters of the Chesapeake Bay from 2007-2017. *Mycobacterium* is considered ubiquitous across aquatic environments, so the response variable was the presence of elevated *Mycobacterium* abundance above the dataset's 4th quantile (140 cells/mL; Jacobs et al. 2009b). Total suspended solids (mg/l; TSS), dissolved oxygen (mg/l; DO), total nitrogen (mg/l; TN), and salinity (ppt) were chosen as explanatory variables based on previous studies of *Mycobacterium* spp. environmental drivers (Falkinham et al. 2004, Jacobs et al. 2009c); however, variables of water temperature, pH, and total phosphorus included in previous studies were not considered due to collinearity with dissolved oxygen and total nitrogen. The model also included latitude and longitude to account for spatial patterns in *Mycobacterium* abundance across the study area. Seasonal and long term trends in *Mycobacterium* abundance were examined by adding month and year as factors. Elevated *Mycobacterium* abundance was then modeled as:

$$\hat{Y} = \beta_0 + \beta_1 TSS + \beta_2 DO + \beta_3 TN + \beta_4 Salinity + \beta_5 Lat + \beta_6 Long + \beta_7 Month + \beta_8 Year, (1)$$

where $\hat{Y}$ is the log-odds of a positive response, β_0 is the intercept, β_{1-7} are the regression coefficients for explanatory variables.

Results

Results suggested that TSS, DO, and salinity were significant predictors of elevated
Mycobacterium abundance (Table 1-B). The occurrence of elevated *Mycobacterium*
increased with increasing TSS (Figure 2-B. A), decreasing DO (Figure 2-B. B), and
increasing salinity (Figure 2-B. C). There was no evidence of a relationship between
elevated *Mycobacterium* and total nitrogen (mg/l, TN; Figure 2-B. D). Latitude was
marginally significant in the model (Table 1-B). Lower latitudes showed higher
occurrence of elevated *Mycobacterium* abundance (Figure 2-B. E), but there was no
evidence of a longitudinal effect (Figure 2-B. F). Although sampling was not
conducted in all months, April showed lower occurrence of elevated *Mycobacterium*
than most summer and fall months (Figure 2-B. G). Elevated *Mycobacterium*
abundance steadily increased from 2007 to a peak in 2011, followed by a period of
decline leading to a time series low in 2015, followed by another increase from 2016-
2017 (Figure 2-B. H).

Discussion

This study shows that the occurrence of elevated *Mycobacterium* is likely influenced
by water quality in Maryland waters of the Chesapeake Bay. Elevated *Mycobacterium*
may occur more frequently in environments with high total suspended solids, low
dissolved oxygen, and/or increasing salinity. If these favorable conditions increase,
the occurrence of elevated *Mycobacterium* may also increase in the Chesapeake Bay.
Consequently, striped bass occupying environments that have elevated

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Mycobacterium may experience increased risk of mycobacteriosis infection and the negative effects that come along with the disease.

A similar study was conducted in the Maryland coastal Bays examining *Mycobacterium* abundance and distribution (Jacobs et al. 2009e). Elevated *Mycobacterium* abundance was also defined as the 4th quartile, but in the coastal bays the level was much lower, only 45 cells/ml versus 140 cells/ml in the Chesapeake Bay. Consistent with the lower occurrence of elevated *Mycobacterium* found in April in the Chesapeake Bay, the coastal bays also demonstrated depressed abundance in the spring and higher abundance in the summer and fall. Mean abundance increased with latitude in the coastal bays, but occurrence of elevated *Mycobacterium* decreased with latitude in the Chesapeake Bay.

If mycobacteriosis in striped bass is driven by elevated *Mycobacterium* in the environment, these comparisons have interesting implications for striped bass. It is important to highlight that the Chesapeake Bay has higher *Mycobacterium* abundance and a prominent mycobacteriosis epizootic, which is potential evidence that the elevated abundance plays a role in infection. Also, the elevated abundance mostly occurs in the summer and fall when there is concern about environmental conditions exacerbating disease in striped bass in the Chesapeake Bay (Coutant 1985). It is also interesting that elevated abundance is more prominent in lower latitudes within the Chesapeake Bay, which could potentially point toward increased disease in Virginia

waters. Future analyses should include data from the Virginia portion of the Chesapeake Bay as well.

Jacobs et al. (2009) also looked at environmental drivers of *Mycobacterium* in the Maryland coastal bays. Their analysis concluded that elevated *Mycobacterium* abundance occurs in nutrient-rich, low dissolved oxygen, low salinity environments in the coastal bays. The Chesapeake Bay also showed elevated *Mycobacterium* abundance in low dissolved oxygen environments, but there was no significant relationship with total nitrogen, representing nutrients in the Chesapeake Bay. Salinity had the opposite relationship with occurrence of elevated *Mycobacterium* in the Chesapeake Bay; however, the coastal bays have a very different salinity gradient. The coastal bays model showed occurrence of elevated *Mycobacterium* decreasing from 18-36 ppt, while the Chesapeake Bay modeled increased from 0-22 ppt. This indicates that there could be an ideal salinity range for mycobacteria, perhaps around 20 ppt, or a nonlinear response, but a targeted study could explore this idea more comprehensively for the whole Chesapeake Bay. Total suspended solids were positively correlated with *Mycobacterium* abundance in the coastal bays, which is mirrored in the Chesapeake Bay models, but it was not included in the coastal bays elevated abundance models. Environmental mycobacteria are typically slow growing, free-living, saprophytes that tend to be found at the water-air interface because of a hydrophobic outer cell wall (Falkinham 2009). The positive relationship with TSS could be because *Mycobacterium* are living off detritus particles in the water. Overall, attempts at limiting low dissolved oxygen and high suspended solids in the

Chesapeake Bay may help decrease the occurrence of elevated *Mycobacterium* and potentially decrease mycobacteriosis in striped bass.

It is important to reiterate that the species of *Mycobacterium* was not specified in this study. *Mycobacterium shottsii* and *M. pseudoshottsi* are the most commonly isolated species of mycobacteria in Chesapeake Bay striped bass, but there are many more species of mycobacteria found in the environment (Stine et al. 2009). As a result, we cannot confirm if the *Mycobacterium* spp. in this study is likely to cause mycobacteriosis in striped bass or if those species would have the same relationships with water quality in the Chesapeake Bay. Additionally, the mode of infection for mycobacteriosis in striped bass is unknown (Jacobs et al. 2009d). Elevated *Mycobacterium* in the water column is one possible hypothesis, but infection could feasibly come from mother to egg transmission, contaminated food sources, or other methods (Decostere et al. 2004).

Tables

Table B-1. Parameter estimates for occurrence of elevated mycobacteria abundance in Maryland waters of the Chesapeake Bay from 2007-2017 from a binomial generalized linear model with logit link function.

Variable	Estimate	Std. Error	Z	*P*
Intercept	21.073	20.659	1.02	0.308
TSS	0.022	0.008	2.853	0.004
DO	-0.186	0.059	-3.164	0.002
TN	0.060	0.236	0.253	0.801
Salinity	0.062	0.024	2.556	0.011
Latitude	-0.394	0.222	-1.771	0.077
Longitude	0.129	0.214	0.602	0.547
May	0.466	0.546	0.853	0.394
June	1.250	0.389	3.211	0.001
July	1.569	0.301	5.214	< 0.001
August	1.597	0.386	4.133	< 0.001
September	0.324	0.569	0.57	0.569
October	2.215	0.321	6.907	< 0.001
2008	1.494	0.330	4.53	< 0.001
2009	2.522	0.362	6.976	< 0.001
2010	2.714	0.338	8.023	< 0.001
2011	4.624	0.407	11.358	< 0.001
2012	3.307	0.418	7.905	< 0.001
2013	1.458	0.504	2.893	< 0.001
2014	2.609	0.458	5.699	< 0.001
2015	-0.622	0.796	-0.781	0.435
2016	0.758	0.510	1.487	0.137
2017	3.465	0.428	8.09	< 0.001

Figures

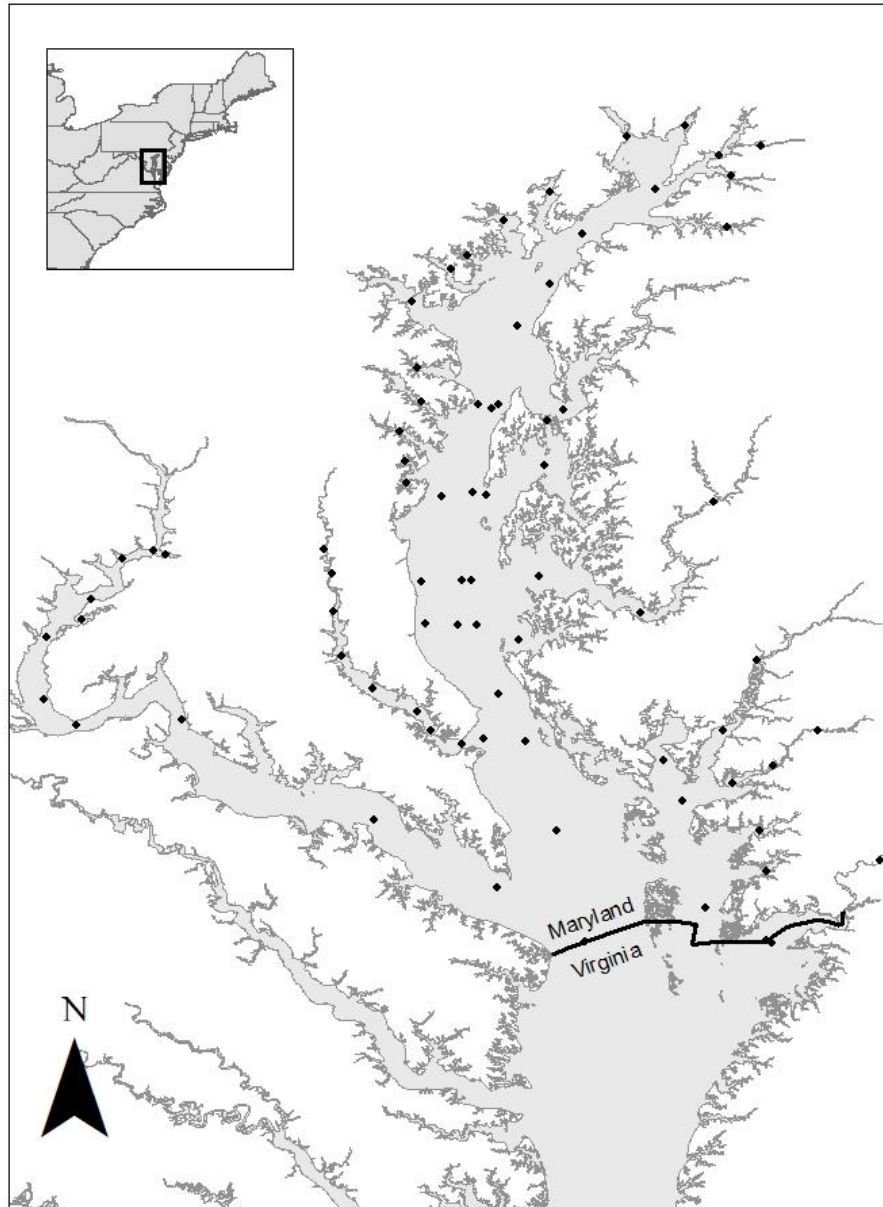Figure B-1. Subset of Chesapeake Bay Program Water Quality monitoring sites used for Mycobacterium spp. collections from 2006-2017.
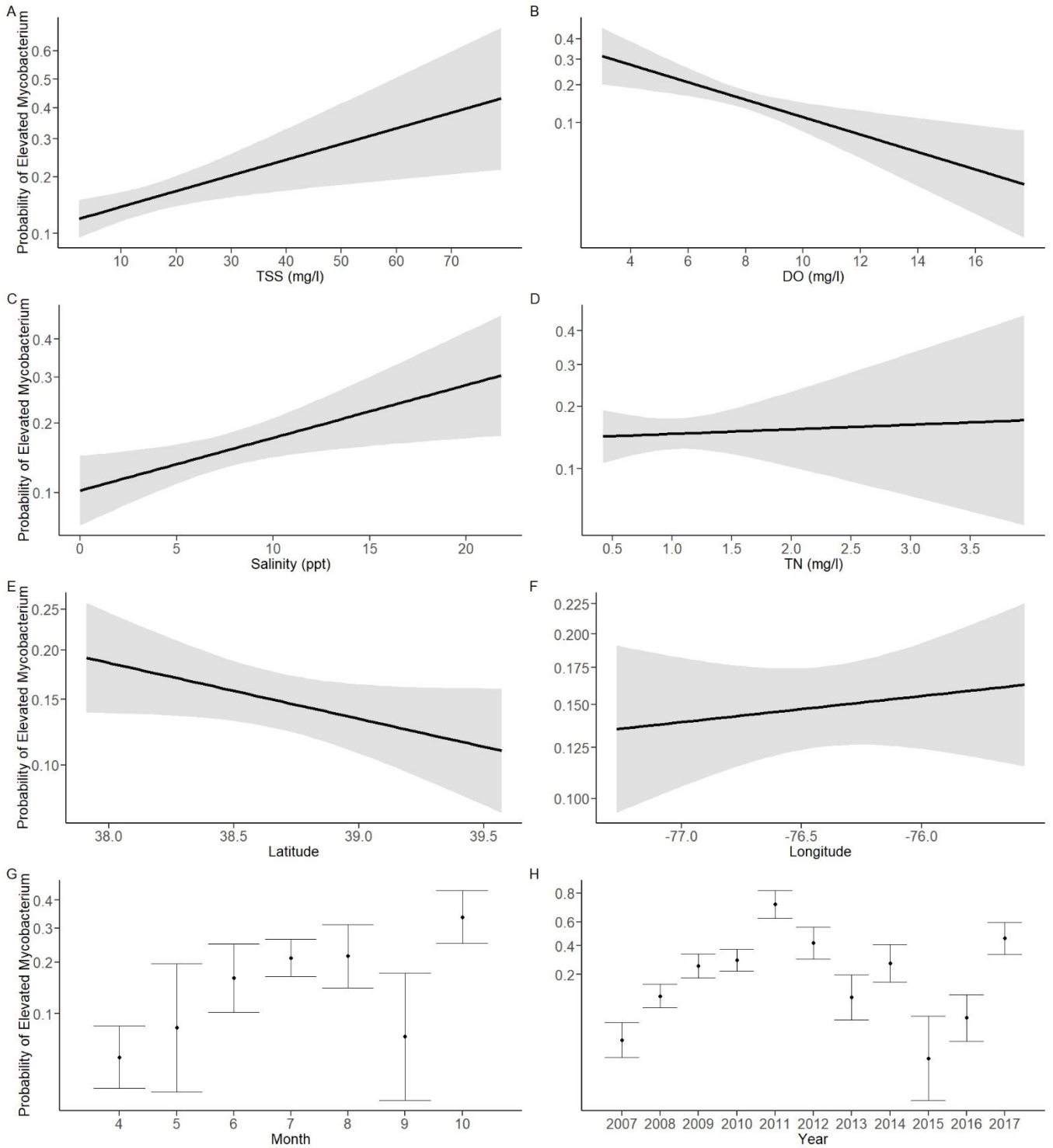
Figure B-2. Partial effect of (A) total suspended solids (TSS), (B) dissolved oxygen (DO), (C) salinity, (D) total nitrogen (TN), (E) latitude, (F) longitude, (G) month, and (F) year on occurrence of elevated Mycobacterium abundance from 2007-2017 in Maryland water of the Chesapeake Bay.

Appendix C: Comparison of Chesapeake Bay-specific and coast-wide per recruit analysis and biological reference points

The most recent assessment for striped bass (Northeast Fisheries Science Center 2019) explored both a single coast-wide stock model (base) and an alternate assessment model that explicitly estimated Chesapeake Bay and coastal ocean sub-stock dynamics with migration between them. The alternate model produced Chesapeake Bay- and ocean-specific estimates of spawning stock biomass and recruitment. The analyses explored in Chapter 2 are repeated here using Chesapeake Bay specific stock-recruitment parameters. Note, however, that the alternate model upon which these estimates were based did not pass peer review and is not currently used for management. As such, the Chesapeake Bay stock-recruitment curve calculated from the alternate assessment model that includes migration should be considered for comparison purposes only and is not suitable for management use at this time.

The stock-recruitment relationships generated using Chesapeake Bay estimates of recruitment and spawner biomass were substantially different from those using coast-wide estimates (Table C-1; Figure C-1). As expected, the alternate model generated lower spawning stock biomass in Chesapeake Bay relative to the coast-wide stock, but it estimated Chesapeake Bay recruitment levels that were similar to that of the coast-wide stock in the base model. Also, the alternate assessment model estimated recruitment at or near zero SSB, resulting in steeper curves for the Chesapeake Bay

stock-recruitment relationship (Figure C-1). This means that the Chesapeake Bay

stock is estimated to be more productive than the coast-wide stock according to the

alternate two-stock model. The high log-likelihood of the Ricker curve generated

using Chesapeake Bay-specific estimates indicates that there is not sufficient

evidence for a Ricker stock-recruitment relationship, so it was not explored further in

this analysis (Table C-1).

In contrast with stock-wide results, F_{MSY} for the Chesapeake Bay was substantially

higher under increased natural mortality Approaches 1 and 2 than status quo (Table

C-2). This was due to the shape of the Chesapeake Bay stock-recruitment curve

which estimated high recruitment at very low SSB (Figure C-1); therefore, high yield

was estimated even at high F values that likely have very low SSB (Figure C-2). F_{max}

and FX% reference points do not include stock-recruitment and thus were not

affected.

Similar to F_{MSY} results, equilibrium estimates of yield and SSB associated with

Chesapeake Bay F_{MSY} (Table C-2) were considerably different from stock-wide

calculations (Table 2.6). Chesapeake Bay yield and SSB calculated with F_{max} for

increased natural mortality approaches (Table C-2) were higher than what is seen in

the stock-wide yield curve (Table 2.6). Again, this is likely because the stock-

recruitment curve for the Chesapeake Bay is problematic and the migration

assessment model may not be providing realistic estimates of sub-stock dynamics.

Chesapeake Bay equilibrium yield and SSB calculated for each FX% (Table C-2) were similar in magnitude to those calculated with stock-wide estimates (Table 2.6).

Alternate natural mortality scenarios explored using Chesapeake Bay-specific information (Figure C-3) produced similar results to the stock-wide analysis (Figure 2.7-2.8). The magnitude of change between scenarios was also similar for both the coast-wide and Chesapeake Bay sub-stock yield and SSB; although the maximum SSB was slightly lower for the Chesapeake Bay, and as noted, the yield remained high at high F values. Chesapeake Bay-specific Beverton-Holt F_{MSY} increased from status quo in natural mortality scenarios (Table C-3), unlike stock-wide Beverton-Holt F_{MSY} which was similar to status quo (Table 2.7); however, both were fairly consistent across scenarios, indicating that assumptions about how mycobacteriosis influences natural mortality-at-age may not be vital to F_{MSY} calculations.

Tables

Table C-1. Estimated median Beverton-Holt (B-H) and Ricker stock-recruitment relationship parameters using coast-wide and Chesapeake Bay-specific estimates.

Parameter	Coast-wide B-H	Coast-wide Ricker	Chesapeake Bay B-H	Chesapeake Bay Ricker
alpha	192.76	4.212	143.66	41.438
beta	32.371	0.00996	0.777	0.0643
sigma	0.348	0.348	0.464	0.749
LL	-19.487	-19.466	-9.375	7.389

Table C-2. Chesapeake Bay-specific reference points and associated yield and spawning stock biomass estimates for each spawning potential ratio modeling approach. Per recruit modeling approaches include status quo (no increase in natural mortality; SQ), Approach 1 (increased natural mortality in both fished and unfished SSB/R; A1), and Approach 2 (increased natural mortality in fished SSB/R only; A2).

Approach	SQ	A1	A2
F_{MSY}	0.59	1.18	1.18
Yield	51.13	30.04	30.04
SSB	90.07	21.48	21.48
F_{max}	0.605	1.213	1.213
Yield	51.12	30.01	30.01
SSB	87.61	20.70	20.70
F40%	0.231	0.271	0.0176
Yield	43.81	23.37	3.34
SSB	208.71	90.32	208.71
F30%	0.336	0.400	0.088
Yield	48.40	26.55	12.65
SSB	156.34	67.54	156.34
F20%	0.517	0.627	0.217
Yield	50.98	29.11	21.30
SSB	103.97	44.77	103.97

Table C-3. Instantaneous fishing mortality rate (yearr^{-1}) reference points estimated using spawning potential ratio calculation Approach 2. See Tables 2.5 and 2.6 for full description of natural mortality scenarios and SPR approaches, respectively.

Scenarios	SQ	M1	M2	M3	M4
F_{MSY} Bay B-H	0.59	1.18	0.99	1.28	1.22

Figures
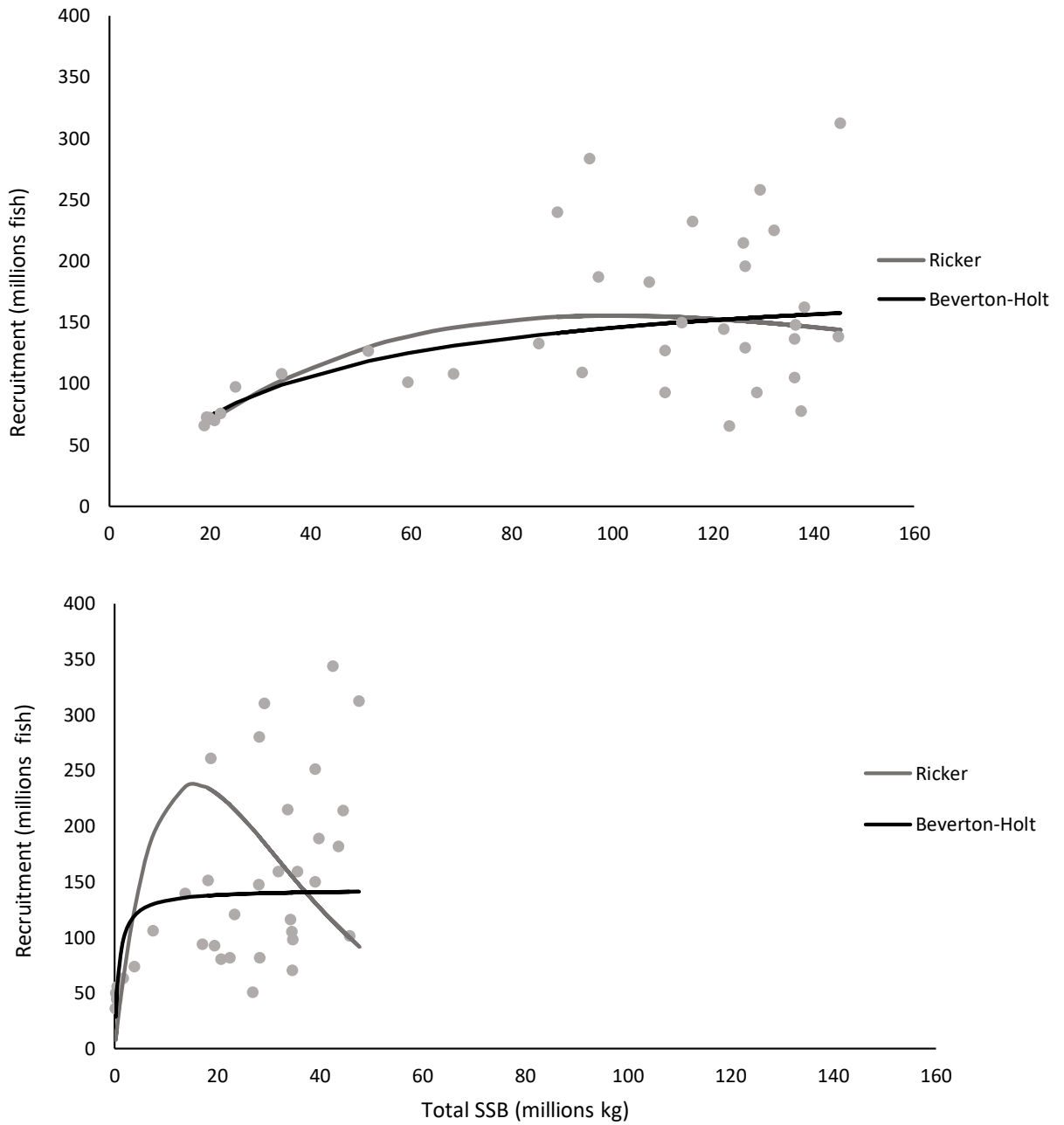
Figure C-1. Stock-recruitment curves generated from coast-wide estimates from the base assessment model (top) and Chesapeake Bay estimates from the alternate model with migration between two sub-stocks (bottom).
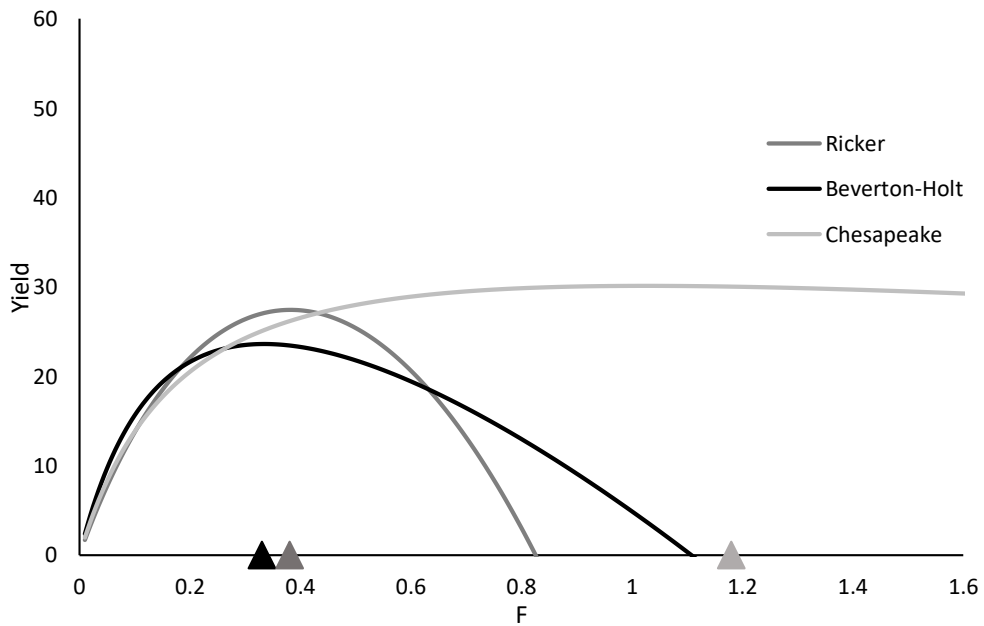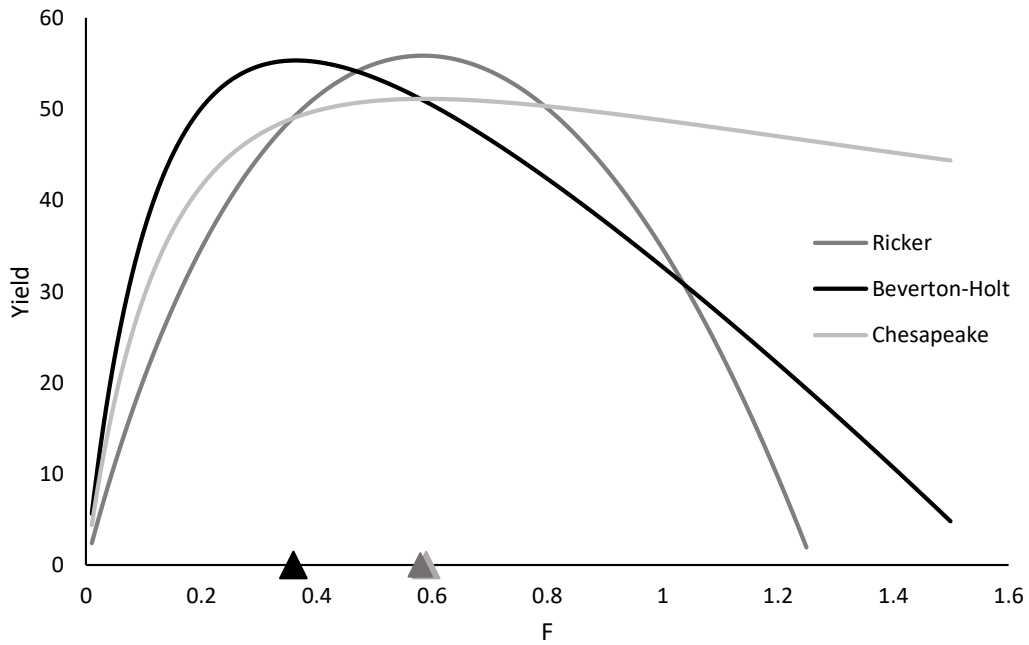
Figure C-2. Striped bass yield for status quo (top) and Approaches 1 and 2 (bottom) using coast-wide Beverton-Holt, coast-wide Ricker, and Chesapeake Bay-specific Beverton-Holt stock-recruitment estimates. F_{MSY} for coast-wide Beverton-Holt (black triangle), coast-wide Ricker (dark grey triangle), and Chesapeake Bay Beverton-Holt (light grey) are indicated. See Figure 2.1 for a full description of SPR approaches.
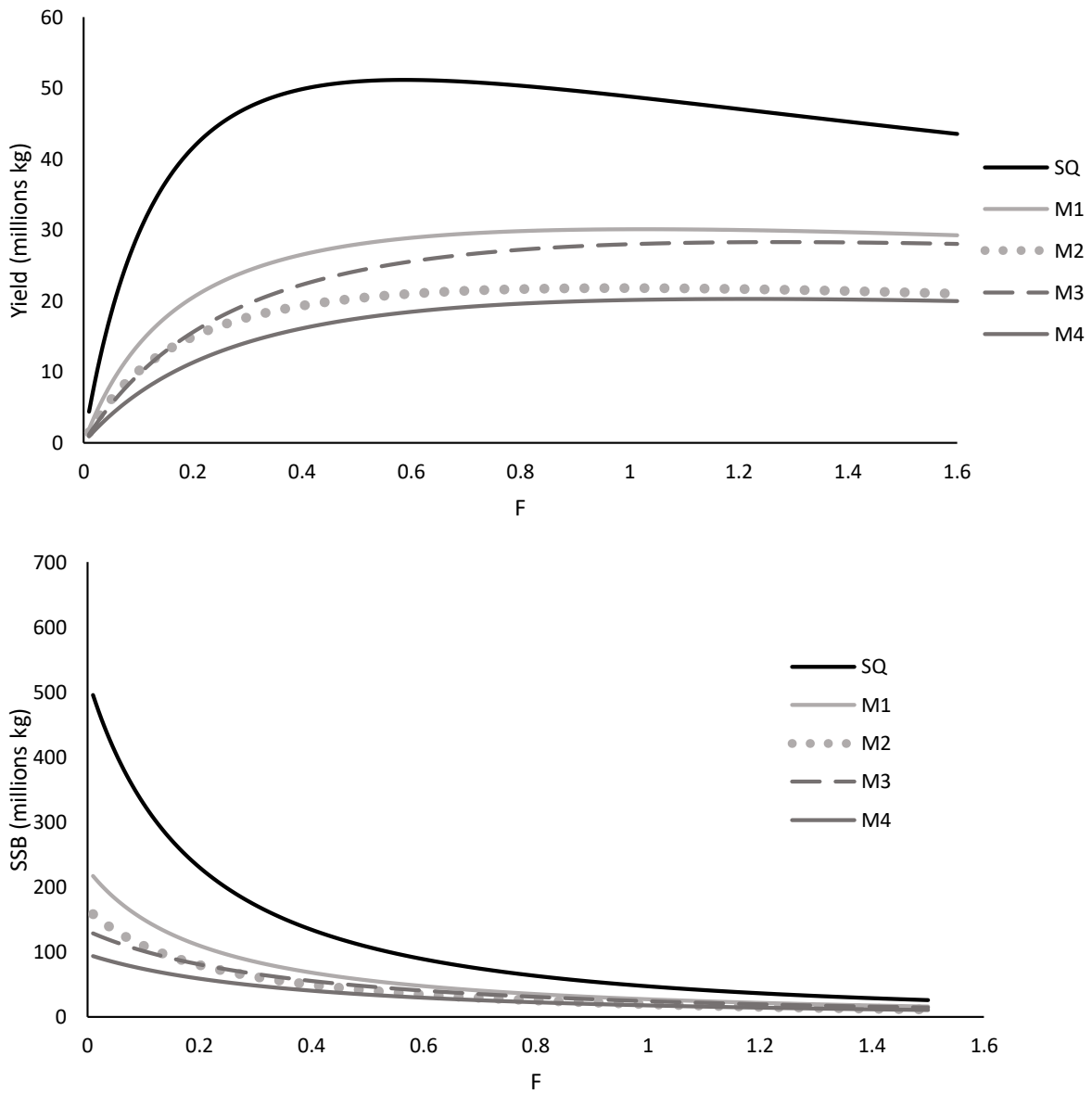
Figure C-3. Striped bass yield (top) and SSB (bottom) comparing status quo (SQ) to natural mortality scenarios generated using a Chesapeake Bay-specific Beverton-Holt stock-recruitment function. For full description of natural mortality scenarios, see Figure 2.3.

References

ASMFC. 2016. Weakfish Benchmark Stock Assessment and Peer Review Report.

ASMFC. 2020. 2020 Spring Meeting Webinar Summary.

Bonzek, C. F., J. Gartland, D. J. Gauthier, and R. J. Latour. 2019. Annual Report - 2018 Data collection and analysis in support of single and multispecies stock assessments in Chesapeake Bay : The Chesapeake Bay Multispecies Monitoring and Assessment Program.

Bonzek, C. F., R. J. Latour, and J. Gartland. 2008. FINAL REPORT - 2007 Data collection and analysis in support of single and multispecies stock assessments in Chesapeake Bay : The Chesapeake Bay Multispecies Monitoring and Assessment Program.

Burge, C. A., C. Mark Eakin, C. S. Friedman, B. Froelich, P. K. Hershberger, E. E. Hofmann, L. E. Petes, K. C. Prager, E. Weil, B. L. Willis, S. E. Ford, and C. D. Harvell. 2014. Climate Change Influences on Marine Infectious Diseases: Implications for Management and Society. Annual Review of Marine Science 6:249–277.

Cimino, J., M. Fabrizio, K. Culzoni, D. Gauthier, J. Jacobs, M. Johnson, E. Martino, N. Meserve, S. Minkkinen, D. Secor, A. Sharov, J. Uphoff, and W. Vogelbein. 2009. Ecosystem-Based Fisheries Management for Chesapeake Bay: Striped Bass Background and Issue Briefs.

Clark, W. G. 1991. Groundfish exploitation rates based on life history parameters. Canadian Journal of Fisheries and Aquatic Sciences 48:734–750.

Clark, W. G. 1999. Effects of an erroneous natural mortality rate on a simple age-structured stock assessment. Canadian Journal of Fisheries and Aquatic Sciences 56:1721–1731.

Clark, W. G. 2002. F 35% Revisited Ten Years Later . North American Journal of Fisheries Management 22:251–257.

Coutant, C. C. 1985. Striped Bass, Temperature, and Dissolved Oxygen: A Speculative Hypothesis for Environmental Risk. Transactions of the American Fisheries Society 114:31–61.

Coutant, C. C., and D. L. Benson. 1990. Summer Habitat Suitability for Striped Bass in Chesapeake Bay - Reflections on a Population Decline. Transactions of the American Fisheries Society 119:757–778.

Coutant, C. C., K. L. Zachman, D. K. Cox, and B. L. Pearman. 1984. Temperature Selection by Juvenile Striped Bass in Laboratory and Field. Transactions of the American Fisheries Society 113:666–671.

Damiano, M. D., and M. J. Wilberg. 2019. Population dynamics of eastern oysters in the Choptank River Complex, Maryland during 1989–2015. Fisheries Research 212:196–207.

Davis, K. B., and N. C. Parker. 1990. Physiological stress in striped bass: effect of acclimation temperature. Aquaculture 91:349–358.

Decostere, A., K. Hermans, and F. Haesebrouck. 2004. Piscine mycobacteriosis: A literature review covering the agent and the disease it causes in fish and humans. Veterinary Microbiology 99:159–166.

Deroba, J. J. 2015. Atlantic herring operational assessment report 2015.

Deroba, J. J., and A. M. Schueller. 2013. Performance of stock assessments with misspecified age- and time-varying natural mortality. Fisheries Research 146:27–40.

Dorazio, R. M., K. A. Hattala, C. B. McCollough, and J. E. Skjeveland. 1994. Tag Recovery Estimates of Migration of Striped Bass from Spawning Areas of the Chesapeake Bay. Transactions of the American Fisheries Society 123:950–963.

Falkinham, J. O. 2009. The biology of environmental mycobacteria. Environmental Microbiology Reports 1:477–487.

Falkinham, J. O., G. L. Nichols, J. Bartram, A. Dufour, and F. Portaels. 2004. Natural ecology and survival in water of mycobacteria of potential public health significance. Pages 15–25 *in* S. Pedley, J. Bartram, G. Rees, A. Dufour, and J. Cotruvo, editors. Pathogenic Mycobacteria in Water: A Guide to Public Helath Consequences, Monitoring and Management. IWA Publishing, London, UK.

Gabriel, W. L., and P. M. Mace. 1999. A review of biological reference points in the context of the precautionary approach. Proceedings of the fifth national NMFS stock assessment workshop: providing scientific advice to implement the precautionary approach under the Magnuson-Stevens fishery conservation and management act. NOAA Tech Memo NMFS-F/SPO-40:34–45.

Gauthier, D. T., R. J. Latour, D. M. Heisey, C. F. Bonzek, J. Gartland, E. J. Burge, and W. K. Vogelbein. 2008. Mycobacteriosis-associated mortality in wild striped bass (Morone saxatilis) from Chesapeake Bay, U.S.A. Ecological applications : a publication of the Ecological Society of America 18:1718–27.

Gauthier, D. T., and M. W. Rhodes. 2009. Mycobacteriosis in fishes: A review. Veterinary Journal 180:33–47.

Gauthier, D. T., M. W. Rhodes, W. K. Vogelbein, H. Kator, and C. A. Ottinger. 2003. Experimental mycobacteriosis in striped bass Morone saxatilis. Diseases of Aquatic Organisms 54:105–117.

Gervasi, C. L. 2015. The reproductive biology of Striped Bass (Morone saxatilis) in the Chesapeake Bay , with consideration of effects due to mycobacteriosis.

Gervasi, C. L., S. K. Lowerre-Barbieri, W. K. Vogelbein, J. Gartland, and R. J. Latour. 2019. The reproductive biology of Chesapeake Bay striped bass with consideration of the effects of mycobacteriosis. Bulletin of Marine Science 95:117–137.

Groner, M., J. Hoenig, R. Pradel, R. Choquet, W. Vogelbein, D. Gauthier, and M. Friedrichs. 2018. Dermal mycobacteriosis and warming sea surface temperatures are associated with elevated mortality of striped bass in Chesapeake Bay. Ecology and Evolution 8:9384–9397.

Hagy, J. D., W. R. Boynton, C. W. Keefe, and K. V. Wood. 2004. Hypoxia in Chesapeake Bay, 1950–2001: Long-term change in relation to nutrient loading and river flow. Estuaries 27:634–658.

Harvell, C. D., C. E. Mitchell, J. R. Ward, S. Altizer, A. P. Dobson, R. S. Ostfeld, and M. D. Samuel. 2002. Climate warming and disease risks for terrestrial and marine biota.

Hoenig, J. M. 1983. Empirical use of longevity data to estimate mortality rates. Fishery Bulletin 81:898–903.

Hoenig, J. M., M. L. Groner, M. W. Smith, W. K. Vogelbein, D. M. Taylor, D. F.

Landers, J. T. Swenarton, D. T. Gauthier, P. Sadler, M. A. Matsche, A. N. Haines, H. J. Small, R. Pradel, R. Choquet, and J. D. Shields. 2017. Impact of disease on the survival of three commercially fished species: Ecological Applications 27:2116–2127.

Howarth, R. W., D. P. Swaney, T. J. Butler, and R. Marino. 2000. Climatic control on eutrophication of the Hudson River estuary. Ecosystems 3:210–215.

Irby, I. D., M. A. M. Friedrichs, F. Da, and K. E. Hinson. 2018. The competing impacts of climate change and nutrient reductions on dissolved oxygen in Chesapeake Bay. Biogeosciences 15:2649–2668.

Jacobs, J. M. 2007. Mycobacteriosis in Chesapeake Bay Striped Bass (Morone saxatilis): the interaction of nutrition and disease.

Jacobs, J. M., R. M. Harrell, J. Uphoff, H. Townsend, and K. Hartman. 2013. Biological Reference Points for the Nutritional Status of Chesapeake Bay Striped Bass. North American Journal of Fisheries Management 33:468–481.

Jacobs, J. M., D. W. Howard, M. Rhodes, M. W. Newman, E. B. May, and R. M. Harrell. 2009a. Historical presence (1975-1985) of mycobacteriosis in chesapeake bay striped bass morone saxatilis. Diseases of Aquatic Organisms 85:181–186.

Jacobs, J. M., M. Rhodes, A. Baya, R. Reimschuessel, H. Townsend, and R. M. Harrell. 2009b. Influence of nutritional state on the progression and severity of mycobacteriosis in striped bass Morone saxatilis. Diseases of Aquatic Organisms 87:183–197.

Jacobs, J. M., M. Rhodes, B. Sturgis, and B. Wood. 2009c. Influence of Environmental Gradients on the Abundance and Distribution of Mycobacterium spp. in a Coastal Lagoon Estuary. Applied and Environmental Microbiology 75:7378–7384.

Jacobs, J. M., C. B. Stine, A. Baya, and M. L. Kent. 2009d. A review of mycobacteriosis in marine fish.

Jacobs, J., M. Rhodes, B. Sturgis, and B. Wood. 2009e. Influence of Environmental Gradients on the Abundance and Distribution of Mycobacterium spp. in a Coastal Lagoon Estuary. Applied and Environmental Microbiology 75:7378–7384.

Johnson, K. F., C. C. Monnahan, C. R. Mcgilliard, K. A. Vert-pre, S. C. Anderson, C. J. Cunningham, F. Hurtado-ferro, R. R. Licandeo, M. L. Muradian, K. Ono, C. S. Szuwalski, J. L. Valero, A. R. Whitten, and A. E. Punt. 2015. Time-varying natural mortality in fisheries stock assessment models: identifying a default approach. ICES Journal of Marine Science 72:137–150.

Jung, S., and E. D. Houde. 2004. Production of bay anchovy Anchoa mitchilli in Chesapeake Bay : application of size-based theory.

Kane, A. S., C. B. Stine, L. Hungerford, M. Matsche, C. Driscoll, and A. Baya. 2007. Mycobacteria as environmental portent in Chesapeake Bay fish species. Emerging Infectious Diseases 13:329–331.

Kohlenstein, L. C. 1981. On the Proportion of the Chesapeake Bay Stock of Striped Bass That Migrates into the Coastal Fishery. Fisheries Society 110:168–179.

Kraus, R. T., D. H. Secor, and R. L. Wingate. 2015. Testing the thermal-niche oxygen-squeeze hypothesis for estuarine striped bass. Environmental Biology of

Fishes 98:2083–2092.

Lafferty, K. D., C. D. Harvell, J. M. Conrad, C. S. Friedman, M. L. Kent, A. M. Kuris, E. N. Powell, D. Rondeau, and S. M. Saksida. 2015. Infectious Diseases Affect Marine Fisheries and Aquaculture Economics. Annual Review of Marine Science 7:471–496.

Lapointe, D., W. K. Vogelbein, M. C. Fabrizio, D. T. Gauthier, and R. W. Brill. 2014. Temperature, hypoxia, and mycobacteriosis: Effects on adult striped bass Morone saxatilis metabolic performance. Diseases of Aquatic Organisms 108:113–127.

Lapointe, M. F., R. M. Peterman, and D. Maccall. 1989. Trends in Fishing Mortality Rate along with Errors in Natural Mortality Rate can cause Spurious Time Trends in Fish Stock Abundances Estimated by Virtual Population Analysis (VPA). Canadian Journal of Fisheries and Aquatic Sciences 46:2129–2139.

Lapointe, M. F., R. M. Peterman, and B. J. Rothschild. 1992. Variable Natural Mortality Rates Inflate Variance of Recruitments Estimated from Virtual Population Analysis (VPA). Canadian Journal of Fisheries and Aquatic Sciences 49:2020–2027.

Latour, R. J., D. T. Gauthier, J. Gartland, C. F. Bonzek, K. A. McNamee, W. K. Vogelbein, and D. MacLatchey. 2012. Impacts of mycobacteriosis on the growth of striped bass (Morone saxatilis) in Chesapeake Bay. Canadian Journal of Fisheries and Aquatic Sciences 69:247–258.

Legault, C. M., and M. C. Palmer. 2015. In what direction should the fishing mortality target change when natural mortality increases within an assessment? Canadian Journal of Fisheries and Aquatic Sciences 73:349–357.

Li, M., Y. J. Lee, J. M. Testa, Y. Li, W. Ni, W. M. Kemp, and D. M. Di Toro. 2016. What drives interannual variability of hypoxia in Chesapeake Bay: Climate forcing versus nutrient loading? Geophysical Research Letters 43:2127–2134.

Mansueti, R. J. 1961. Age, growth, and movements of the striped bass, Roccus saxatilis, taken in size selective fishing gear in Maryland. Chesapeake Science 2:9–36.

Marty, G. D., P. J. F. Hulson, S. E. Miller, T. J. Quinn, S. D. Moffitt, and R. A. Merizon. 2010. Failure of population recovery in relation to disease in Pacific herring. Diseases of Aquatic Organisms 90:1–14.

Marty, G. D., T. J. Quinn, G. Carpenter, T. R. Meyers, and N. H. Willits. 2003. Role of disease in abundance of a Pacific herring (Clupea pallasi) population. Canadian Journal of Fisheries and Aquatic Sciences 60:1258–1265.

McCullagh, P., and J. A. Nedler. 1989. Generalized Linear Models. second edi. Chapman and Hall/ CRC.

Morgan, M. J., P. A. Shelton, and R. M. Rideout. 2014. Varying components of productivity and their impact on fishing mortality reference points for Grand Bank Atlantic cod and American plaice. Fisheries Research 155:64–73.

Muhling, B. A., C. F. Gaitán, C. A. Stock, V. S. Saba, D. Tommasi, and K. W. Dixon. 2018. Potential Salinity and Temperature Futures for the Chesapeake Bay Using a Statistical Downscaling Spatial Disaggregation Framework. Estuaries and Coasts 41:349–372.

Najjar, R. G., C. R. Pyke, M. B. Adams, D. Breitburg, C. Hershner, M. Kemp, R.

Howarth, M. R. Mulholland, M. Paolisso, D. Secor, K. Sellner, D. Wardrop, and R. Wood. 2010. Potential climate-change impacts on the Chesapeake Bay. Estuarine, Coastal and Shelf Science 86:1–20.

Najjar, R., L. Patterson, and S. Graham. 2009. Climate simulations of major estuarine watersheds in the Mid-Atlantic region of the US. Climatic Change 95:139–168.

Nash, R. D. M., A. H. Valencia, and A. J. Geffen. 2006. The origin of Fulton's condition factor - Setting the record straight. Fisheries 31:236–238.

National Marine Fisheries Service. 2018. Fisheries of the United States 2017.

National Marine Fisheries Service. 2020. Fisheries of the United States, 2018.

Ni, W., M. Li, A. C. Ross, and R. G. Najjar. 2019. Large Projected Decline in Dissolved Oxygen in a Eutrophic Estuary Due to Climate Change. Journal of Geophysical Research: Oceans 124:8271–8289.

Northeast Fisheries Science Center. 2009. 48th Northeast Regional Stock Assessment Workshop (48th SAW) Assessment Report.

Northeast Fisheries Science Center. 2018. 65th Northeast Regional Stock Assessment Workshop (65th SAW) Assessment Report.

Northeast Fisheries Science Center. 2019. 66st Northeast Regional Stock Assessment Workshop (66th SAW) Assessment Report. Page US Dept Commer, Northeast Fish Sci Cent Ref Doc. 19-08.

Overton, A. S., F. J. Margraf, C. A. Weedon, L. H. Pieper, and E. B. May. 2003. The prevalence of mycobacterial infections in striped bass in Chesapeake Bay. Fisheries Management and Ecology 10:301–308.

Pauly, D. 1980. On the interrelationships between natural mortality, growth parameters, and mean environmental temperature in 175 fish stocks. ICES Journal of Marine Science 39:175–192.

Quinn, T. J., and R. B. Deriso. 1999. Quantitative Fish Dynamics. Oxford University Press.

Richards, R. A., and P. J. Rago. 1999. A Case History of Effective Fishery Management: Chesapeake Bay Striped Bass. North American Journal of Fisheries Management 19:356–375.

Ricker, W. E. 1975. Computation and Interpretation of Biological Statistics of Fish Popuations. Bulletin of the Fisheries Research Board of Canada 191.

Roessig, J. M., C. M. Woodley, J. J. Cech, and L. J. Hansen. 2004. Effects of global climate change on marine and estuarine fishes and fisheries. Reviews in Fish Biology and Fisheries 14:251–275.

Secor, D. H., M. H. P. O. Brien, B. I. Gahagan, J. C. Watterson, and D. A. Fox. 2020. Differential migration in Chesapeake Bay striped bass. PloS ONE 15.

Secor, D. H., and P. M. Piccoli. 2007. Oceanic migration rates of Upper Chesapeake Bay striped bass (Morone saxatilis), determined by otolith microchemical analysis. Fishery Bulletin 105:62–73.

SEDAR. 2020. Atlantic Menhaden Benchmark Stock Assessment Report. North Charleston, SC.

Seekell, D. A., and M. L. Pace. 2011. Climate change drives warming in the Hudson River Estuary, New York (USA). Journal of Environmental Monitoring 13:2321–2327.

Sharp, J. H. 2010. Estuarine oxygen dynamics: What can we learn about hypoxia

from long-time records in the Delaware Estuary? Limnology and Oceanography 55:535–548.

Shen, C., J. M. Testa, M. Li, and W. J. Cai. 2020. Understanding Anthropogenic Impacts on pH and Aragonite Saturation State in Chesapeake Bay: Insights From a 30-Year Model Study. Journal of Geophysical Research: Biogeosciences 125.

Shepherd, J. G. 1982. A Versatile New Stock-Recruitment Relationship for Fisheries, and the Construction of Sustainable Yield Curves. ICES Journal of Marine Science 40:67–75.

Shepherd, J. G., and J. G. Pope. 2008. Dynamic Pool Models II: Short-Term and Long-Term Forecasts of Catch and Biomass. Pages 164–188 Handbook of Fish Biology and Fisheries, Volume 2. Blackwell Science Ltd, Oxford, UK.

Southwick Associates. 2005. The Economics of Recreational & Commercial Striped Bass Fishing in Maryland.

Stine, C. B., J. M. Jacobs, M. R. Rhodes, A. Overton, M. Fast, and A. Baya. 2009. Expanded range and new host species of Mycobacterium shottsii and M. pseudoshottsii. Journal of Aquatic Animal Health 21:179–183.

Stine, C. B., A. S. Kane, and A. Baya. 2010. Mycobacteria isolated from Chesapeake Bay fish. Journal of Fish Diseases 33:39–46.

Testa, J. M., J. Blake Clark, W. C. Dennison, E. Caroline Donovan, A. W. Fisher, W. Ni, M. Parker, D. Scavia, S. E. Spitzer, A. M. Waldrop, V. M. D. Vargas, and G. Ziegler. 2017. Ecological Forecasting and the Science of Hypoxia in Chesapeake Bay. BioScience 67:614–626.

Testa, J. M., Y. Li, Y. J. Lee, M. Li, D. C. Brady, and D. M. Di. 2014. Quantifying the effects of nutrient loading and carbon production in dissolved O2 in Chesapeake Bay using a coupled hydrodynamic-biogeochemical model Quantifying the effects of nutrient loading on dissolved O 2 cycling and hypoxia in Chesapeake Bay using. Journal of Marine Systems 139.

Uphoff, J. H. 2003. Predator-prey analysis of striped bass and Atlantic menhaden in upper Chesapeake Bay. Fisheries Management and Ecology 10:313–322.

Vetter, E. F. 1988. Estimation of Natural Mortality in Fish Stocks: a Review. Fishery Bulletin 86:25–43.

Wagner, D., and L. S. Young. 2004. Nontuberculous mycobacterial infections: A clinical review. Infection 32:257–270.

Ward, J. R., and K. D. Lafferty. 2004. The elusive baseline of marine disease: are diseases in ocean ecosystems increasing? PLoS biology 2:e120.

Wilberg, M. J., M. E. Livings, J. S. Barkman, B. T. Morris, and J. M. Robinson. 2011. Overfishing, disease, habitat loss, and potential extirpation of oysters in upper Chesapeake Bay. Marine Ecology Progress Series 436:131–144.

Wilberg, M. J., J. R. Wiedenmann, and J. M. Robinson. 2013. Sustainable exploitation and management of autogenic ecosystem engineers : application to oysters in Chesapeake Bay 23:766–776.

Wingate, R. L., D. H. Secor, and R. T. Kraus. 2011. Seasonal Patterns of Movement and Residency by Striped Bass within a Subestuary of the Chesapeake Bay. Transactions of the American Fisheries Society 140:1441–1450.