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ORIGINAL ARTICLE

Life in the fast lane: Revisiting the fast growth—High survival paradigm during the early life stages of fishes

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Abstract

Early life survival is critical to successful replenishment of fish populations, and hypotheses developed under the Growth-Survival Paradigm (GSP) have guided investigations of controlling processes. The GSP postulates that recruitment depends on growth and mortality rates during early life stages, as well as their duration, after which the mortality declines substantially. The GSP predicts a shift in the frequency distribution of growth histories with age towards faster growth rates relative to the initial population because slow-growing individuals are subject to high mortality (via starvation and predation). However, mortality data compiled from 387 cases published in 153 studies (1971–2022) showed that the GSP was only supported in 56% of cases. Selection against slow growth occurred in two-thirds of field studies, leaving a non-negligible fraction of cases showing either an absence of or inverse growth-selective survival, suggesting the growth-survival relationship is more complex than currently considered within the GSP framework. Stochastic simulations allowed us to assess the influence of key intrinsic and extrinsic factors on the characteristics of surviving larvae and identify knowledge gaps on the drivers of variability in growth-selective survival. We suggest caution when interpreting patterns of growth selection because changes in variance and autocorrelation of individual growth rates among cohorts can invalidate fundamental GSP assumptions. We argue that breakthroughs in recruitment research require a comprehensive, population-specific characterization of the role of predation and intrinsic factors in driving variability in the distribution and autocorrelation of larval growth rates, and of the life stage corresponding to the endpoint of pre-recruited life.

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KEYWORDS
critical period, growth-mortality, individual characteristics, larval physiology, predation, recruitment endpoint

1 | INTRODUCTION

Despite more than 100 years of research, many uncertainties still exist with respect to why variability in year-class strength of fishes varies widely. In 1890–1913, Hjort (1914, 1926) benefited from the novel method of ageing fish scales to compile compelling evidence that both the Arcto-Norwegian cod (*Gadus morhua*, Gadidae) and Norwegian spring-spawning herring (*Clupea harengus*, Clupeidae) stocks exhibited poor and strong year-classes. He proposed that this pattern could only be driven by massive and highly variable mortality rates during early life stages (e.g. egg, larval). Hjort (1914) further hypothesized that these changes in mortality rates were due to differences in planktonic prey availability during the larval 'critical period', when individuals make the transition from using yolk reserves to feeding exogenously.

While Hjort's critical period hypothesis has been supported by the results of many laboratory experiments, testing the hypothesis in the field has offered a formidable challenge to fishery scientists. For example, successful laboratory rearing during Hjort's era hinged on the survival of larvae immediately after yolk-sac reserves were exhausted (Fabre-Domergue & Biétrix, 1897, 1898). Likewise, the short period corresponding to the onset of exogenous feeding still remains a critical period characterized by high mortality rates in contemporary studies (Marcus, 2005; Næss et al., 1995; Støttrup & McEvoy, 2003). By contrast, the sequential, synoptic and quantitative sampling needed to estimate mortality during the larval stage has proven logistically difficult (Oozeki et al., 2009), and while several major programs have allowed for substantial gains in our understanding of the role of prey availability on larval survival (Buckley et al., 2006; Kjesbu et al., 2023), the concurrent sampling and characterization of prey fields at the finest spatial scales, needed for further breakthroughs in our understanding of recruitment dynamics (Pepin, 2004), remains challenging (Houde, 2008).

Although limited progress was made in testing the validity of Hjort's hypothesis in the field during the first half of the 20th century, the observation of low survival of reared marine fish larvae at comparable field-observed prey densities (e.g. O'Connell & Raymond, 1970; Werner & Blaxter, 1980) strengthened the focus on follow-up hypotheses. Such hypotheses considered bottom-up processes as the regulator of year-class strength and recruitment variability through its direct and indirect impacts on larval growth and mortality (Anderson, 1988; Cury & Roy, 1989; Cushing & Harris, 1973; Lasker, 1978). More recently, however, the focus has expanded to include predation as a major top-down controlling process of recruitment variation. The addition of predation to the conceptual framework was driven by field observations of predation on marine fish larvae, as well as the high survival rates of marine fish larvae in mesocosms in the absence of fish predators, even when prey levels were low (Hunter, 1984; Möller, 1984; Øiestad, 1985). This evolution from considering starvation to predation as the primary source of mortality signified an important paradigm shift in fisheries science, with their combination forming the foundation of the current 'Growth-Survival Paradigm' (hereafter, GSP).

Cushing (1975) was the first to recognize the close relationship between growth rate and mortality rate and the existence of an integrated process determining recruitment outcome. He proposed that the growth rate to mortality rate ratio could be used as a metric to characterize year-class success. The contemporary GSP (Anderson, 1988; Cushing, 1990; Houde, 1987) that emerged from Cushing's initial concept comprises three functional mechanisms, each encapsulated by an independent hypothesis, which acts to select for fast-growing larvae over their slower-growing counterparts. The 'bigger-is-better' mechanism (Miller et al., 1988) stipulates that larger individuals at any given time experience lower mortality than smaller individuals through their enhanced swim speeds that allow the capture of prey while evading predators. The 'stage-duration' mechanism (Chambers & Leggett, 1987; Houde, 1987, 1989) states that fast growth results in lower cumulative mortality than slow growth by shortening the duration of time that individuals spend in early life stages vulnerable to physical (e.g. storms, temperature reversals) and biological (e.g. variability in predator and prey abundance). Finally, the 'growth-selective predation' mechanism (Takasuka et al., 2003, 2007) predicts higher survival of faster-growing individuals at a given body size as a result of their higher presumed condition status and enhanced ability to escape predators. Given that processes driving mortality operate at the level of the individual, consideration of individual characteristics is essential to refine our understanding of the outcome of selective mortality and eventually, the fluctuations in stock productivity (Brown & Bailey, 1992; Dougherty et al., 2007; Fortier et al., 2006; Fortier & Quiñonez-Velazquez, 1998), which has primarily been acquired through average cohort or population characteristics (Pepin, 1989; Rice et al., 1993).

The ability to test the mechanisms associated with the GSP was aided by the combined discoveries of daily periodicity in otolith formation (Pannella, 1971) and proportionality between the width of daily increments and somatic growth of larval fish (Brothers, 1981). Because the otolith constitutes a permanent record of growth, it became possible to compare the early life characteristics of survivors (i.e. later life stages, such as juveniles) to those of the initial larval cohort (e.g. Hare & Cowen, 1997; Ludsin & DeVries, 1997; Meekan & Fortier, 1996; Robert et al., 2007). The primary approach has been to contrast the frequency distribution of growth rates of a larval cohort to that back-calculated from the otoliths of juvenile survivors originating from the same cohort (Hare & Cowen, 1997; Meekan & Fortier, 1996; Pepin, 1989). The GSP predicts a shift in the growth-rate frequency distribution with larval age towards the fastest growth rates observed within the initial larval population because an increasing proportion of slow-growing individuals succumb to predation mortality relative to their faster-growing counterparts (Pepin, 1989; Rice et al., 1993). The GSP also implies that variability in the level of mortality at the cohort level will result in differences in the magnitude of the shift towards fast growth rates. Hence, in a given larval population, the extent of the shift in growth-rate frequency distributions between the initial larval population and survivors reaching the recruited life stage (hereafter 'endpoint') is predicted to be negatively related to survival and resulting year-class strength (Figure 1a).

A principle of evolutionary ecology is that selection acts on the variability among individuals. Anderson (1988) states the GSP predicts that survival of a cohort is directly related to growth rates during the pre-recruit period. When applied to individual probabilities of survival, the implication is that faster-growing larvae are more likely to survive through a given developmental stage than slower-growing individuals. This should, in turn, result in a positive shift in the distribution of past growth histories when contrasting survivors to the original cohort (Pepin, 1989; Rice et al., 1993), which can affect either the average, variance or skewness in the distribution of growth rates. Therefore, any change in these moments of distribution (sensu Pepin, 1989) can be considered, in the simplest sense, as evidence that the GSP is acting on a population if faster-growing individuals achieve higher overall survival. Importantly, however, it is the strength and consistency of the link between survival and changes in the characteristics of the survivors relative to the initial population, that define the benefit of the GSP to understand what processes are affecting year-class formation. Failure to detect a positive effect in survivorship in the growth distribution of survivors can be indicative of counter-gradient selective processes that negate or act against the benefits of rapid growth (e.g. Kristiansen et al., 2009; Munch & Conover, 2003; Schultz & Conover, 1999; Shropshire et al., 2022), and which could be attributed to growth-dependent losses, or it may reflect our inability to detect changes as a result of sampling errors or limited sample size. However, the GSP is only useful for understanding the underlying processes affecting recruitment success if we understand how the characteristic moments of the distribution can reflect survivorship. Identifying the sensitivity of changes in growth distribution to differences among individuals, or in response to changes in bottom-up (i.e. prey availability translated in the form of growth rates) and top-down (i.e. predation represented as mortality) pressures, therefore, becomes essential to determine what can be inferred from changes in growth distributions and whether observations are consistent with the GSP.

While the GSP and its associated mechanisms are intuitive and appear theoretically sound, the degree to which GSP is supported in the literature remains unquantified and results contrary to it have never been summarized. Towards this end, we conducted a literature review to quantify support for GSP. Finding only inconsistent support for the GSP in this review (see Section 2), we sought to provide the theoretical foundation for a revised framework that integrates results that are contrary to predictions of the current GSP. We identified aspects of larval growth and mortality that may lead to growth-dependent selection. Next, we performed numerical simulations to determine the influence of each aspect in explaining growth-dependent mortality within a cohort (population) of larvae. Finally, we discuss future avenues of research needed to advance our understanding of the factors that drive variability in growth-selective survival and their relation to recruitment, which constitutes a necessary step towards a revised conceptual framework.

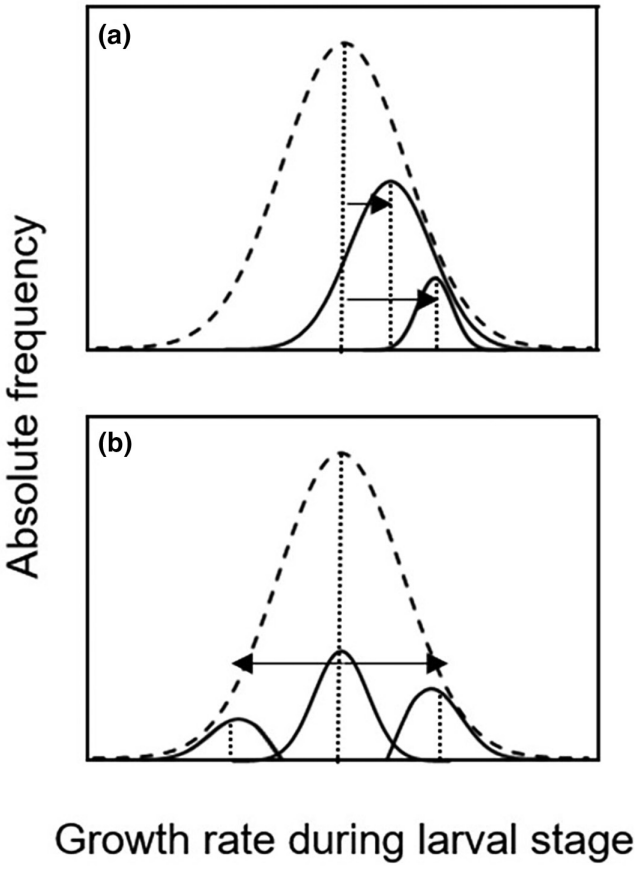

FIGURE 1 (a) Conceptual framework of the Growth-Survival Paradigm (GSP), where the extent of the shift in growth rate-frequency distribution towards higher growth rates from the early larval stage (initial population; dashed line) to the endpoint when year-class strength is set (survivors; solid line) is expected to be proportional to mortality (i.e. stronger size-selective mortality on fast growers leads to smaller year-classes or annual recruitment); (b) Examples illustrating the breadth of results observed in field, mesocosm, laboratory and modelling studies, including expected selection in favour of fast-growing individuals (rightmost distribution), as well as selection in favour of slow-growing individuals (leftmost distribution) and an absence of growth selection (middle distribution). These latter two types of selection do not support the GSP.

2 | REVIEW OF THE EFFECTS OF GROWTH-SELECTIVE MORTALITY ON THE CHARACTERISTICS OF SURVIVORS

To understand the degree to which the GSP was supported in previous studies, we assembled a list of peer-reviewed articles that investigated growth-selective survival during the larval stage of fish by searching the ISI Web of Science database. The search was inclusive of field, laboratory, mesocosm and modelling studies of marine, estuarine and freshwater species. The initial search was conducted with the following parameters: ALL = (fish*) AND AB = (larva*) AND TI = (growth) AND AB = (surviv*) NOT ALL = (aquaculture). This combination yielded 641 journal articles (1 December 2022). After consideration of the title, abstract, and other sections of each article, and eliminating studies making inferences on growth-dependent survival without consideration of the original larval population, 95 studies were retained. These 95 studies were supplemented by 58 additional studies that were not returned by search results but were identified through cross-referencing of the retained studies. Overall, we considered 153 studies during the period 1971–2022, which contained 387 (ranging from 1 to 36 per study) single tests of the occurrence of growth-selective survival. Single tests comprised within-cohort and among-cohort investigations of growth-dependent survival and were, thus, either based on a single cohort, on multiple cohorts of a given population, on laboratory and mesocosm trials and/or on modelling scenarios (Table S1).

Single tests of the GSP varied in their nature and while some of them targeted individual mechanisms of the GSP (bigger-is-better, stage duration, growth-selective predation), others assessed the integration of all 3 mechanisms. We classified tests that found evidence of selective mortality against slow growers in at least one of the three mechanisms as 'supporting' the GSP over the tested age or size range. Tests that found no evidence of any growth-selective mortality were classified in the 'no selection' category. The 'mixed selection' category included tests that revealed various types of selection in the same cohort over the tested age range. Finally, tests that found evidence for selective mortality against fast growers from the standpoint of at least one mechanism were designated as 'contrary' to the GSP.

Based on the reviewed literature, the GSP was supported in 56% of all tests (total $N = 387$ tests: Figure 2). While examples of growth-dependent and size-dependent mortality confirming the advantage of fast-growing fish larvae pervade the marine (e.g. Hare & Cowen, 1997; Shoji & Tanaka, 2006), estuarine (e.g. Rilling & Houde, 1999; Sirois & Dodson, 2000) and freshwater (e.g. Meekan et al., 1998; Post & Prankevicius, 1987) literature, we found evidence that a non-negligible proportion of tests failed to support the conventional view that fast growth systematically promotes a greater likelihood of survival (Figure 2). In addition to the expected positive relationship between survival and growth, test results included no relationship between growth and mortality, as well as cases where higher survival was recorded in slow-growing individuals (Figure 2).

We looked for publication trends of studies with results that ran counter to the GSP, as well as whether these results emanated from a particular type of investigation (field, laboratory, mesocosm or modelling). We did not observe a strong trend for the publication of non-supportive results over time. Instead, results running contrary to the GSP have been frequently published starting in the early 1990s (Figure 2a). Additionally, tests that did not support the GSP came from the field (e.g. Gleason & Bengtson, 1996; Watanabe & Kuroki, 1997), laboratory (e.g. Litvak & Leggett, 1992; Munch & Conover, 2003), mesocosm (e.g. Fuiman, 1989; Pepin et al., 1992) and modelling (e.g. Cowan & Houde, 1992; Cowan et al., 1996) studies (Figure 2b). However, differences in the proportion of tests that did not support the GSP were observed among study types. The level of support towards the GSP was higher in field and laboratory

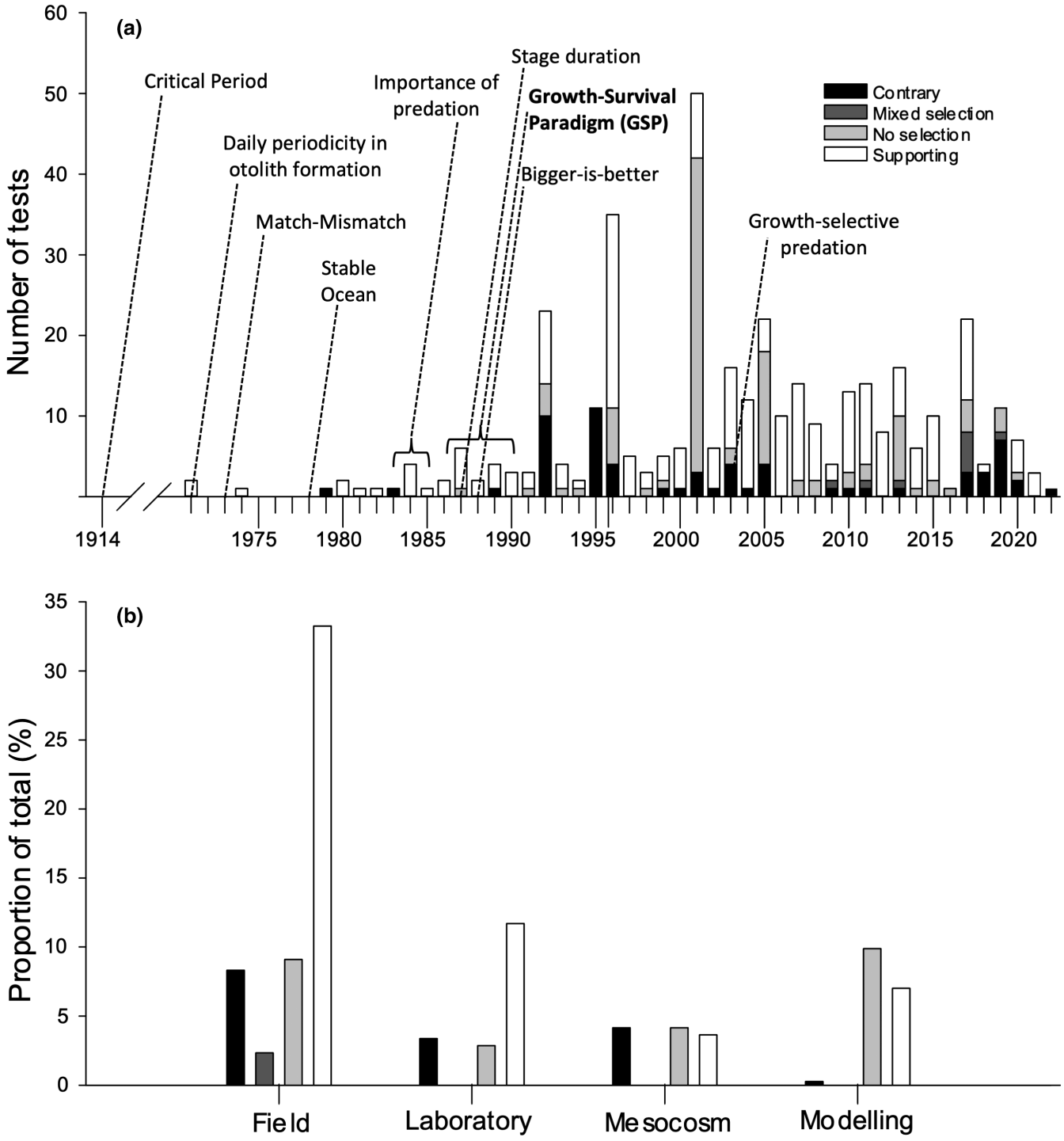

FIGURE 2 (a) Chronology of key recruitment hypotheses and discoveries leading to the current Growth-Survival Paradigm (GSP; Anderson, 1988; Cushing, 1990; Houde, 1987), and the number of tests of the paradigm during 1971–2022. Hypotheses and discoveries consist of the Critical Period hypothesis (Hjort, 1914), the finding of daily periodicity in otolith formation (Pannella, 1971), the Match-Mismatch hypothesis (Cushing & Harris, 1973), the finding of the importance of predation as an agent of mortality (Hunter, 1984; Möller, 1984; Øiestad, 1985), the Stable Ocean hypothesis (Lasker, 1978), the Stage Duration hypothesis (Chambers & Leggett, 1987; Houde, 1987), the Bigger-is-better hypothesis (Miller et al., 1988) and the Growth-selective predation hypothesis (Takasuka et al., 2003). Tests are represented with different levels of support for the GSP: (1) selective mortality against slow growers (Supporting); (2) absence of growth-selective mortality (No selection); (3) mixed selective mortality (Mixed selection); and (4) selective mortality against fast growers (Contrary). (b) Proportion of test results in field, laboratory, mesocosm and modelling studies. See Table S1 for study details.

investigations, for which more than two-thirds of cases reviewed supported the GSP, while the level of support dropped below 50% in mesocosm studies and numerical simulations (Figure 2b).

Because field-based studies integrate all sources of mortality that affected the dynamics of a given cohort between sequentially sampled life stages, in contrast to the majority of laboratory,

mesocosm and modelling studies which instead focus on a limited number of mortality components, field-based studies should be considered as 'gold standard' in terms of test quality (Houde, 1997a). Results from field-based investigations suggested that even though the GSP was supported in two-thirds of all tests, fast growth is not always necessary or even advantageous for larval fish survival. Some field-based studies have demonstrated that higher mortality rate of larger or faster-growing pre-recruits may occur due to higher predation risk (e.g. Takasuka et al., 2017; Takasuka, Aoki, & Mitani, 2004). Studies that compared the dynamics of multiple cohorts of a population in the field also highlighted that growth-selective processes can vary through time and space within a given ecosystem (Baumann et al., 2003; Grorud-Colvert & Sponaugle, 2011; May et al., 2020; Rankin & Sponaugle, 2011; Robert et al., 2007).

Even though laboratory, mesocosm or modelling may depict a partial representation of growth-selective mortality sources relative to those encountered by individuals in the field (Houde, 1997a), they can be useful to precisely identify mechanisms from which results that do not support the GSP originate. For example, some of those studies have precisely described predator–prey interactions that can lead to higher predation mortality in larger or faster-growing individuals (e.g. Fuiman, 1989; Litvak & Leggett, 1992; Pepin et al., 1992).

When considering tests that did not support the GSP across study types, the most common case was an absence of growth-selective mortality (26% of all tests). Cases, where slow-growing individuals exhibited a survival advantage over fast growers, represented 16% of all tests. Finally, 2% of the tests revealed mixed growth-selective mortality over the course of the larval stage.

Overall, our literature review indicated that even though selective mortality against slow-growing individuals remains the most common observation, a non-negligible fraction of the test cases (studied cohorts) was characterized by the absence of growth-selective survival or by selective mortality directed *against* fast-growing individuals. This suggests that the relationship between larval growth and survival is more complex than currently considered within the current framework of the GSP. Given this perspective, we aim to identify aspects of larval growth and mortality that may lead to growth-dependent selection and to test the relative importance of each aspect in driving growth-dependent mortality within a cohort (population) of larvae. By doing so, we hope to stimulate further progress towards understanding processes regulating fish recruitment variability.

3 | THEORETICAL BASIS OF THE GROWTH-SURVIVAL PARADIGM

The GSP assumes that the annual recruitment (i.e. year-class strength) within a population depends on the growth rate of individuals, the rate of mortality imposed by predators, and the duration of the interval between hatching and the 'endpoint' of pre-recruited life. This endpoint is generally considered as the life stage at which year-class strength is set and the mortality rate has stabilized near adult levels such that predictions of recruitment to adulthood or the fishery can be reliably predicted (Houde, 2016; Ludsin et al., 2014). The endpoint is often characterized by the occurrence of changes in trophic status or habitats (Houde, 1997a), which Benoît et al. (2000) suggested was principally determined by length, although there is also increased variability in length among individuals with increasing length at transition. Per the stage-duration mechanism, faster-growing individuals are expected to reach this endpoint earlier than their slower-growing counterparts, thereby reducing time spent in life stages during which mortality is high (Chambers & Leggett, 1987; Houde, 1987, 1989). This mechanism also implies that, under a scenario in which mortality rate is independent of growth rate, moderate selection favouring fast-growing individuals will occur based on their lower cumulative mortality rates due to shorter larval stage, as shown in simulations from Pepin (1989). This shift in growth-rate frequency distribution towards faster growth rates in survivors relative to the initial population should be accentuated under the expected scenario of growth-dependent mortality causing increased losses of slow-growing individuals. In addition to the increase in mean growth rate as the population experiences growth-selective survival, a parallel decrease of variance in growth rates is expected (Rice et al., 1993).

Despite these expectations, which have been supported by numerous studies (Hare & Cowen, 1997; Meekan et al., 1998; Sirois & Dodson, 2000), other studies have documented a shift in the growth-rate frequency distribution towards slower growth rates, suggesting that fast growth can be disadvantageous. While catastrophic mortality could potentially lead to losses of fast-growing cohorts, selection against fast-growing individuals has been most common when predators are large relative to larval prey or are not gape-limited (Litvak & Leggett, 1992; Pepin et al., 1992; Robert et al., 2010; Takasuka, Aoki, & Mitani, 2004; von Westernhagen et al., 1979). Thus, as shown previously (see Figure 2), positive selection for fast growth does not always occur, begging the question of why this is the case.

When using frequency distributions of growth histories to explore positive or negative selection for growth rate, as has been commonly done for tests of the GSP, the difference in mean larval growth rates between survivors and the initial population can range from positive values (expected selection for fast growth) to negative values (selection for slow growth), with no selection also being a possibility (see Figure 1b). As such, when slow-growing individuals are favoured, the cumulative mortality experienced by the population across pre-recruited life stages should be higher than when equivalent selective forces favour fast-growing individuals because slow-growing larvae will require a longer period to reach the endpoint relative to their fast-growing counterparts (stage-duration mechanism). As a result, annual cohorts characterized by selection for slow growth are predicted to be associated with weaker recruitment strength than those associated with selection for fast growth (e.g. May et al., 2020).

4 | SIMULATION MODELLING

To test predictions of the GSP, including how the demographic characteristics of a population (i.e. growth and mortality rates) influence the traits of survivors from hatching to recruitment (i.e. their endpoint), we performed a series of computer simulations. The aim was to provide a foundation against which patterns of growth-dependent survivorship could be interpreted among cohorts of larval fish, we assessed how the mean and variance (measured as the standard deviation, SD) in growth rates of the survivors were affected by five important aspects of growth and mortality rates: (1) autocorrelation in growth; (2) mean growth rate; (3) variance in growth rate; (4) mean mortality rate; (5) length-dependent mortality rates; and (6) the degree of growth-dependent mortality. In essence, our simulations are intended as a sensitivity analysis of how the growth characteristics of survival respond to the underlying processes that determine the probability of survival and evaluate how the patterns of change are linked with the GSP. This contrasts with most individual-based models which aim to identify the time, location and environmental conditions that yield patterns consistent with successful growth and/or survival rather than focussing on evaluating how the distribution of growth histories responded to the underlying distribution of vital rates (e.g. Hufnagl et al., 2015; Langangen et al., 2014; Peck et al., 2013; Rose et al., 1999; Xu et al., 2015).

4.1 | Autocorrelation in growth

Recent analyses have demonstrated that daily somatic growth rates of larval fish during any given period of time are not random but, rather, tend to be autocorrelated from a certain time in life (Dower et al., 2009; Murphy et al., 2018; Pepin et al., 2015; Robert et al., 2014). Thus, individuals growing fast on 1 day would be expected to be growing fast on previous and subsequent days, suggesting possible 'carry-over effects' of fast (or slow) growth. Such growth autocorrelation can cause higher variance in growth rates and body size within the initial population, increasing the potential for growth-dependent mortality, as well as survivorship (Rice et al., 1993). Therefore, the degree to which an individual maintains a consistent growth rate, whether high or low relative to the average of other individuals in a population, may be critical to determining the cumulative effect of sources of mortality on its probability of survival. Herein, we considered several scenarios of growth autocorrelation based on the range of observations reported by Pepin et al. (2015). Results from this analysis provided the foundation for parameterization of all subsequent simulations.

4.2 | Mean growth rate

Variability in environmental conditions has been shown to regulate the mean growth rate across individuals within a given cohort (Anderson, 1988; Cushing, 1990). In turn, mean growth rate has been shown to be a key driver of overall survival and recruitment by determining the duration of time spent in vulnerable life stages during which the mortality rate exceeds the population growth rate (Stage-duration hypothesis; Chambers & Leggett, 1987; Houde, 1987). Given that both interspecific and interannual variability in somatic growth can influence the magnitude of survival and recruitment through effects on life-stage duration (Houde, 1987), in our simulations, we tested a range of mean growth rates based on observations reported by Pepin et al. (2015) for species whose mean growth rate varied from slow (e.g. Arctic cod *Boreogadus saida*, Gadidae, ice cod *Arctogadus glacialis*, Gadidae) to fast (e.g. sailfish *Istiophorus platypterus*, Istiophoridae, blue marlin *Makaira nigricans*, Istiophoridae).

4.3 | Variance in growth rates within the initial population

Previous modelling exercises have demonstrated that, for a given mean growth rate, increasing variance in growth rates within the initial population results in higher survival rates and a higher mean growth rate in survivors (Pepin, 1989; Rice et al., 1993). Thus, in our simulations, we also assessed the relative importance of variance in growth rates in driving growth and survival patterns measured at the endpoint of our simulations.

4.4 | Mean mortality rate imposed on the population

Along with mean growth rate, mortality rate is a fundamental component of the GSP (Anderson, 1988; Cushing, 1990; Houde, 1987). Because daily mortality rates can be both high and variable during early larval stages, mortality is often considered the single most important driver of recruitment variability (Houde, 1997b, 2016). Thus, we considered a range of daily mortality rates representative of values reported by Pepin (2016), to understand its importance relative to growth rate variation in driving recruitment.

4.5 | Length-dependent mortality

We also considered the potential effect of length-dependent mortality rates (Peterson & Wroblewski, 1984) to assess whether the bigger-is-better concept that larger individuals are subject to lower overall mortality (Miller et al., 1988) provides an advantage in terms of variability in growth rates relative to that achieved when mortality rates are constant.

4.6 | Growth-dependent mortality

Compelling evidence exists from field, laboratory, and modelling studies to suggest that mortality is often size- or

growth-dependent (Bochdansky et al., 2005; Cowan et al., 1996; Ludsin & DeVries, 1997; Robert et al., 2007; Shoji et al., 2005; Thanassekos et al., 2012). While several studies have indicated that gape-limited predators selectively remove smaller or slower-growing individuals from populations (e.g. Juanes et al., 2002; Juanes & Conover, 1994), other investigations have reported that some larger, raptorial predators target larger or fast-growing individuals (e.g. Robert et al., 2010; Takasuka et al., 2017). To account for this important component of mortality, we performed simulations in which mortality rates differ between slow- and fast-growing individuals in a given cohort.

5 | SIMULATION SETUP

We performed stochastic simulations to track growth in total length (TL) of individual larvae from hatch to an endpoint informative of future recruitment. Although the life stage and timing of this endpoint can be expected to vary among species (e.g. Benoît et al., 2000; Houde, 1994, 1997b; Sogard, 1997), for our simulations, we considered individuals to have recruited once they achieved a size of 12.5 mm TL. The choice of this particular invariant endpoint was done to avoid introducing uncontrolled random variation in the outcome of the simulations, to avoid confounding the effect of growth with the age or length at transition (Benoît et al., 2000), and to allow a sufficient number of survivors at the end of the runs. In each simulation, we followed a population originally composed of 100,000 individuals with a mean (±1 SD) hatch size of 3.5 ± 0.15 mm TL, based on average values reported by Pepin et al. (2015). We chose length rather than weight as our focal measure because otolith radius and larval length are typically highly correlated (Campana, 1990), although we would expect our assumptions and findings to hold for weight as well, as long as individuals do not experience bouts of starvation that cause marked changes in weight-at-length (Peck et al., 2015). Our baseline simulations assumed a mean growth rate of 0.4 ± 0.1 mm d^{-1} (Pepin et al., 2015) and a mean mortality rate of 0.25 ± 0.2 d^{-1} (Pepin, 2016). We investigated the effects of changing growth and mortality rates by ±0.1 and altering the SD in growth rates by ±0.05. Length-dependency in mortality rates was applied using $0.914 \cdot TL^{-0.68}$ from Pepin (1991), and a weaker length-dependency of $0.483 \cdot TL^{-0.34}$, both of which were adjusted to yield an average mortality rate of ~0.25 d^{-1} from hatch to 12.5 mm. All simulations were carried out in R (Version 3.4) and RStudio (Version 1.1.453).

Autocorrelation in growth was introduced at the ages of 9, 5, and 2 d post-hatch (dph), by changing, for each individual, the original mean growth rate from 0.4 mm d^{-1} to the mean achieved during the previous 8, 4, and 1 day(s) of life ±0.1 mm d^{-1} for all subsequent days. These different timings for the onset of growth autocorrelation reflect the findings of Pepin et al. (2015) for a wide variety of species. Finding similar results among these trials, we primarily present results from the day 5 autocorrelation because it best represented a majority of species investigated (Pepin et al., 2015).

To investigate the potential effect of growth-dependent mortality, we conducted a series of simulations in which we imposed contrasting mortality rates for larvae with growth rates above and below our baseline growth rate of 0.4 mm d^{-1}. To achieve a high level of contrast centred on our baseline mortality rate of 0.25 d^{-1}, we applied three levels of contrasting mortality rates (0.20 vs. 0.30 d^{-1}; 0.15 vs. 0.35 d^{-1}; and 0.05 vs. 0.45 d^{-1}) either in favour or against fast-growing individuals.

Stochastic patterns of variation for both growth and mortality rates were drawn from a Gamma distribution to reflect underlying distributions (Pepin, 2016; Pepin et al., 2015). Data from these studies allowed us to estimate the shape and rate parameters, which remained constant for all simulations, in which we varied the mean and SD. Stochastic realizations for each individual larva and day, based on the specified mean and SD in vital rates, were drawn from a Gamma distribution with rate, shape and offset parameters of 2.65, 7.31 and 2.74 for growth, and 1.51, 2.26 and 1.5 for mortality. Each individual was assigned a daily growth and mortality rate based on a draw from each of the Gamma distributions. The mortality rate was used to determine the probability of survival, and a draw from a uniform distribution (0,1) served to determine if the individual died on that day when the draw yielded a value greater than that probability. Growth rate characteristics (mean and SD) of the initial larval population were estimated for all individuals that had survived to an age of 6 d in a given simulation. These characteristics were then contrasted with those from the surviving population that reached the simulation endpoint, thus allowing us to assess the role of the different aspects of growth and mortality in driving growth-selective survivorship.

6 | SIMULATION RESULTS

6.1 | Effect of serial correlation on growth

As expected from Rice et al. (1993), an early onset of growth autocorrelation had the effect of increasing the mean and variance in growth rate of survivors, as well as survivorship, relative to a situation where daily growth was determined randomly (Table 1, Figure 3). In simulations where growth of individual larvae was determined randomly from day to day, the influence of early growth (to day 6) on the duration of the larval stage was small, and positive selection for individuals with faster relative early growth was limited (Figure 3a). As a result, the mean early growth rate of survivors to our simulation endpoint (12.5 mm TL) increased by only 1.4% relative to that of the initial population, although the SD of the growth rate of survivors decreased by nearly 67% (Table 1). This reduction in SD indicates that under random growth, fast-growing and slow-growing individuals are almost equally likely to not recruit because mortality of slow growers is high and the likelihood of maintaining high growth rates is very low.

The survival advantage of faster early growth was increased by introducing serial correlation. For example, when serial autocorrelation

TABLE 1 Results from the stochastic simulations used to examine how daily growth rate (*G*) and mortality rate (*Z*) during the larval stage affect eventual growth rate distributions and survival (i.e. recruitment to 12.5 mm in total length, TL).

	Growth (*G*)	SD	Mortality (*Z*)	SD	Serial	Sample	Sample growth rate		N	Survivors/all 6 d old larvae		
Treatment	(mm d^{-1})		(d^{-1})		Correlation		Mean	SD		Mean G	SD (G)	Survival
Autocorrelation in *G*	0.4	0.1	0.25	0.2	*Nil*	Survivors	0.408	0.022	519	1.014	0.332	0.0052
						All 6 d larvae	0.402	0.065	30,790			
	0.4	0.1	0.25	0.2	*Day 9*	Survivors	0.421	0.037	555	1.047	0.555	0.0056
						All 6 d larvae	0.402	0.066	30,864			
	0.4	0.1	0.25	0.2	*Day 5*	Survivors	0.432	0.049	572	1.074	0.717	0.0057
						All 6 d larvae	0.402	0.068	30,804			
	0.4	0.1	0.25	0.2	*Day 2*	Survivors	0.466	0.078	624	**1.159**	**0.936**	**0.0062**
						All 6 d larvae	0.402	0.083	31,073			
Growth rate (*G*)	*0.3*	0.1	0.25	0.2	Day 5	Survivors	0.352	0.052	142	**1.163**	**0.759**	0.0014
						All 6 d larvae	0.302	0.068	31,267			
	0.4	0.1	0.25	0.2	Day 5	Survivors	0.432	0.049	572	1.074	0.717	0.0057
						All 6 d larvae	0.402	0.068	30,804			
	0.5	0.1	0.25	0.2	Day 5	Survivors	0.523	0.051	1480	1.042	0.740	**0.0148**
						All 6 d larvae	0.502	0.068	29,976			
Variance in *G*	0.4	*0.05*	0.25	0.2	Day 5	Survivors	0.408	0.025	496	1.018	**0.743**	0.0050
						All 6 d larvae	0.401	0.034	30,904			
	0.4	*0.10*	0.25	0.2	Day 5	Survivors	0.432	0.049	572	1.074	0.717	0.0057
						All 6 d larvae	0.402	0.068	30,804			
	0.4	*0.15*	0.25	0.2	Day 5	Survivors	0.468	0.072	662	**1.160**	0.707	**0.0066**
						All 6 d larvae	0.403	0.102	30,777			
Mortality rate (*Z*)	0.4	0.1	*0.15*	0.12	Day 5	Survivors	0.424	0.050	3942	1.055	**0.784**	**0.0394**
						All 6 d larvae	0.402	0.063	45,106			
	0.4	0.1	*0.25*	0.20	Day 5	Survivors	0.432	0.049	572	1.074	0.717	0.0057
						All 6 d larvae	0.402	0.068	30,804			
	0.4	0.1	*0.35*	0.28	Day 5	Survivors	0.440	0.048	90	**1.094**	0.665	0.0009
						All 6 d larvae	0.402	0.073	20,481			
Length-dependent mortality	0.4	0.1	$0.485 \cdot TL^{-0.34}$ *Weak dependency*	0.2	Day 5	Survivors	0.432	0.053	560	1.073	**1.053**	**0.0056**
						All 6 d larvae	0.402	0.050	24,945			
	0.4	0.1	$0.914 \cdot TL^{-0.68}$ *Strong dependency*	0.2	Day 5	Survivors	0.436	0.052	382	**1.082**	1.032	0.0038
						All 6 d larvae	0.403	0.050	16,766			

(Continues)

TABLE 1 (Continued)

Treatment	Growth (*G*) (mm d^{-1})	SD	Mortality (*Z*) (d^{-1})	SD	Serial Correlation	Sample	Sample growth rate Mean	Sample growth rate SD	N	Survivors/all 6 d old larvae Mean G	Survivors/all 6 d old larvae SD (G)	Survivors/all 6 d old larvae Survival
Contrasting Z between slow/fast growing larvae	0.4	0.1	*0.45/0.05*	0.8 * *Z*	Day 5	Survivors	0.444	0.037	7073	**1.113**	0.535	**0.0707**
						All 6 d larvae	0.399	0.070	24,280			
	0.4	0.1	*0.35/0.15*	0.8 * *Z*	Day 5	Survivors	0.447	0.040	1599	**1.114**	0.586	0.0160
						All 6 d larvae	0.402	0.069	29,754			
	0.4	0.1	*0.30/0.05*	0.8 * *Z*	Day 5	Survivors	0.446	0.044	839	1.109	**0.632**	0.0084
						All 6 d larvae	0.402	0.068	30,526			
	0.4	0.1	*0.20/0.30*	0.8 * *Z*	Day 5	Survivors	0.408	0.051	589	1.013	**0.738**	0.0059
						All 6 d larvae	0.402	0.069	30,816			
	0.4	0.1	*0.15/0.35*	0.8 * *Z*	Day 5	Survivors	0.386	0.041	1021	0.961	0.594	0.0102
						All 6 d larvae	0.402	0.068	30,444			
	0.4	0.1	*0.05/0.45*	0.8 * *Z*	Day 5	Survivors	0.372	0.031	5983	0.920	0.445	**0.0598**
						All 6 d larvae	0.404	0.069	25,303			

Note: The left column identifies the six aspects of growth and mortality investigated in each set of simulations within that block. Variables altered in each treatment simulation are in italic. The mean *G* and *Z*, their associated standard deviation (SD) and the day of onset of serial correlation in individual growth rates (beginning at 9, 5 or 2 d post-hatch) are provided for each treatment. Statistics (mean and SD in *G*) are provided along with the number of individuals (*N*) reaching 6 d of age (All 6 d larvae) and our simulation endpoint (Survivors; those reaching 12.5 mm TL). The last three columns provide ratios between the mean *G* and SD of *G* between larvae reaching the simulation's endpoint (Survivors) and that of all larvae at 6 d of age, as well as the overall survival relative to the initial number of simulated larvae ($N = 100{,}000$). Here the highest values for each treatment are in bold (except in the last case where the two largest are in bold).

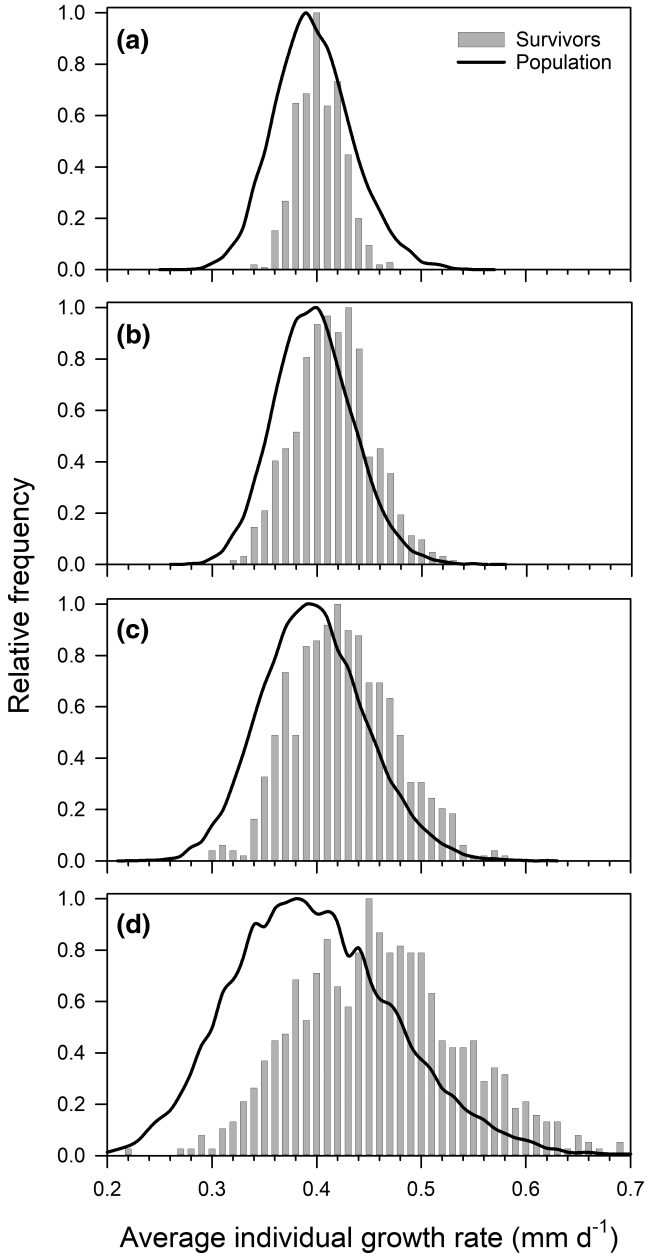

FIGURE 3 Effect of the timing of the onset of growth autocorrelation (beginning at day 9 vs. day 5 vs. day 2) on the distribution of mean individual growth rates (G) between the initial larval population (all larvae 6 d of age) and survivors to 12.5 mm total length (G = 0.4 mm d^{-1}, standard deviation, SD = 0.1). (a) Simulation with random individual growth (i.e. no autocorrelation in growth). Autocorrelated growth with the mean individual G from: (b) day 9 onward set as the average of growth achieved in days 1–8 (SD = 0.1); (c) day 5 set as the average of growth of days 1–4 (SD = 0.1); and (d) day 2 set as growth achieved on day 1 (SD = 0.1). All simulations used a mortality rate of 0.25 ± 0.2 d^{-1}, with the initial larval population size for all simulations being 100,000.

in growth was introduced 9 dph, the mean early-life (through day 6 of life) growth rate of survivors was 4.7% greater than the growth rate of the initial population (individuals 6 d of age) with a milder (45%) decrease in the variance in growth rates relative to the random growth scenario (Table 1, Figure 3b). Introducing serial correlation in growth at earlier ages (5 and 2 dph), respectively accentuated the shift of the growth distribution of survivors towards fast growth rates by 7.4% and 15.9% relative to the initial population (Table 1, Figure 3c,d). The decline of SD in growth rates of survivors relative to the random growth scenario also was progressively reduced to 28.3% and 6.4%, respectively (Table 1). Overall, changes in mean early growth rates between the initial population and survivors were considerably smaller than the differences in the SD of survivors. Increased autocorrelation, thus, primarily preserved population-level variability in early growth rates. Moving from random growth to growth autocorrelation from the age of 2 d resulted in a ~20% increase in survivorship.

6.2 | Effect of mean growth rate

Increasing mean growth rate generally increased survival, while reducing the relative survival disadvantage of slow-growing compared to fast-growing individuals (Table 1, Figure 4a–c). Increasing mean growth rate from 0.3 to 0.5 mm d^{-1} reduced the difference in mean growth rate between survivors and the initial population from 16.3% to 4.2%, but had little effect on variance (as measured by SD) in the growth rate of survivors (Table 1, Figure 4a–c). Parallel to the decrease in the difference in the mean growth rates of survivors and the original population, survival increased from 0.14% to 1.48% (Table 1). This 10.5-fold increase in survival corresponded to a substantial decrease in time needed to reach the endpoint for all larvae, resulting in the higher apparent survival of slow-growing larvae. The change in the variance between the initial population and survivors was modest and within the margin of error for measuring changes in the variance.

6.3 | Effect of variance in growth rate

Increasing variance in growth rates within the initial population resulted in an increased mean growth rate of survivors, an increased difference in mean growth rate between survivors and the initial population, and an increase in total survival (Table 1, Figure 4d–f). In particular, increasing SD in growth rates from 0.05 to 0.15 mm d^{-1} increased the mean growth rate of survivors by 15% relative to that of the initial population (Table 1, Figure 4d–f) and resulted in a 33% increase in absolute survival (Table 1). We identified a notable interaction between the average and variance in growth rate. The impacts of variance in growth rate diminished with increasing mean growth rate (Figure 5). Independent of mean growth rate, increasing the SD resulted in greater-than-linear increases in selective survival (Figure 5), which is more complex than would have been expected based on the simple relationship defined in the GSP.

6.4 | Effect of mean mortality rate

Increasing the mean mortality rate had the opposite effect on the characteristics of the survivors relative to an increase in growth rate.

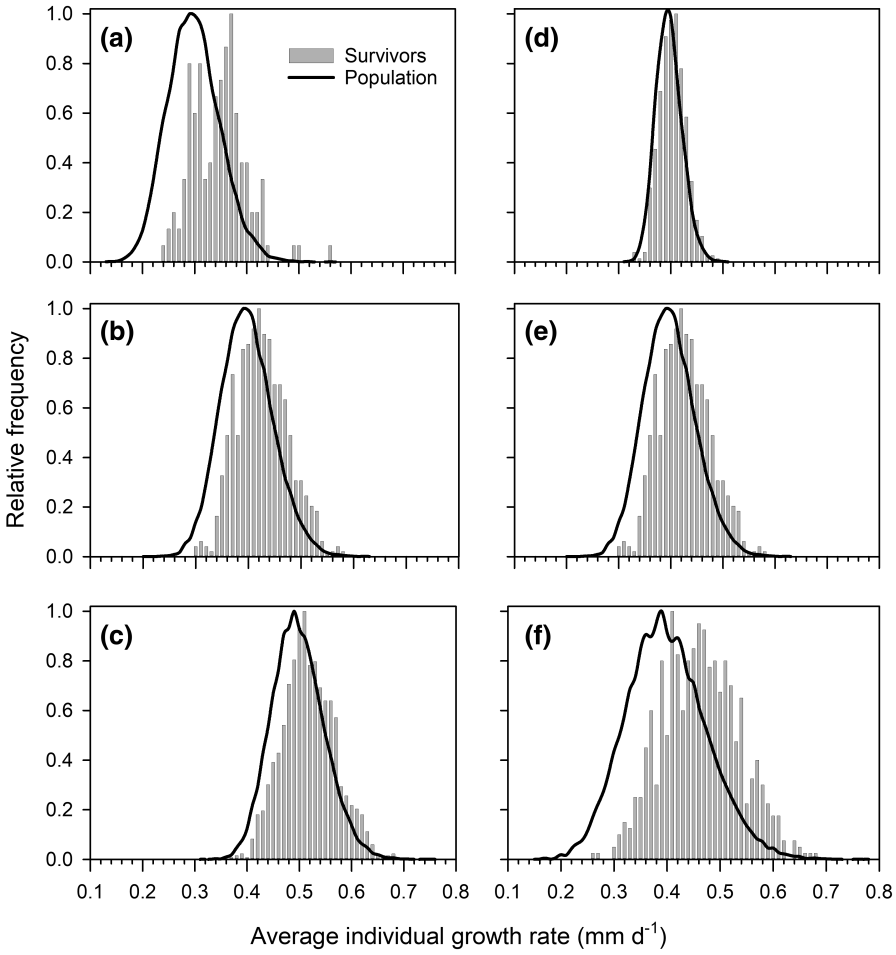

FIGURE 4 Effect of variations in the mean (a–c) and variance (standard deviation, SD; d–f) in growth rate (G) on the change in growth-rate frequency distribution between the initial larval population (all 6 d old larvae) and survivors (to 12.5 mm in total length) in our simulations. (a) $G = 0.30 \pm 0.10$ mm d^{-1}; (b) $G = 0.40 \pm 0.10$ mm d^{-1}; (c) $G = 0.50 \pm 0.10$ mm d^{-1}; (d) $G = 0.40 \pm 0.05$ mm d^{-1}; (e) $G = 0.40 \pm 0.10$ mm d^{-1} (same as b); (f) $G = 0.40 \pm 0.15$ mm d^{-1}. All simulations used a mortality rate of 0.25 ± 0.2 d^{-1}, with the initial larval population size for all simulations being 100,000.

A change in mortality rate from 0.15 to 0.35 d^{-1} resulted in a moderate increase of the mean growth rate of survivors (3.8%) towards faster average growth rates relative to the initial population (Table 1, Figure 6). While changes in mean growth were limited, increasing mortality had a large negative effect on variance (as measured by SD) in growth of survivors. For example, the 12% reduction in the SD in growth rate of survivors after increasing the mortality rate from 0.15 to 0.35 d^{-1} was explained by a smaller proportion of slower-growing individuals reaching the endpoint of our simulations (Figure 6). Survivorship decreased drastically (by 97.7%) as mortality rate increased from 0.15 to 0.35 d^{-1}.

6.5 | Effect of length-dependent mortality

Length-dependent mortality varied from 0.47 d^{-1} for the smallest larvae and 0.18 d^{-1} for the largest. Length-dependent mortality resulted in a substantial increase in the mean growth rate of survivors, comparable to the increase associated with a mean mortality rate of 0.35 d^{-1} (Table 1, Figure 7). However, variability in growth of the survivors increased relative to that of the cohort on day 6, which differs from the outcome of simulations based on length-independent mortality rate, although the absolute difference in variance was considerably smaller than in other simulations. Overall survival was also lower than the simulation with a length-independent 0.25 d^{-1} mortality rate, but the survival to day 6, which is used to define the initial population, was lower than that in all other simulations. The small increase in the variability in growth rates of survivors is associated with the low number of larvae making it to day 6, relative to all other simulations, which limited the variance in growth rates among individuals on which selective loss could act during the remainder of the simulation. Simulations with weaker length-dependent mortality yielded overall results that were similar to that achieved with a length-independent 0.25 d^{-1} mortality rate, indicative that pronounced length-dependency is required to achieve notable differences in the growth distribution of survivors. This result illustrates that the impact of 'bigger-is-better' was most important during the very early stages of development when small individuals had a lower likelihood of survival than their slightly larger conspecifics, and compounded with the stage-duration concept. The degree of length- or stage-dependency in mortality rates may therefore affect the variability in growth rates, over which evaluation of the linkage GSP to cohort survival occurs.

6.6 | Effect of growth-dependent mortality

Changing growth-dependent mortality had a profound effect on the growth characteristics of survivors (Figure 8). In our first

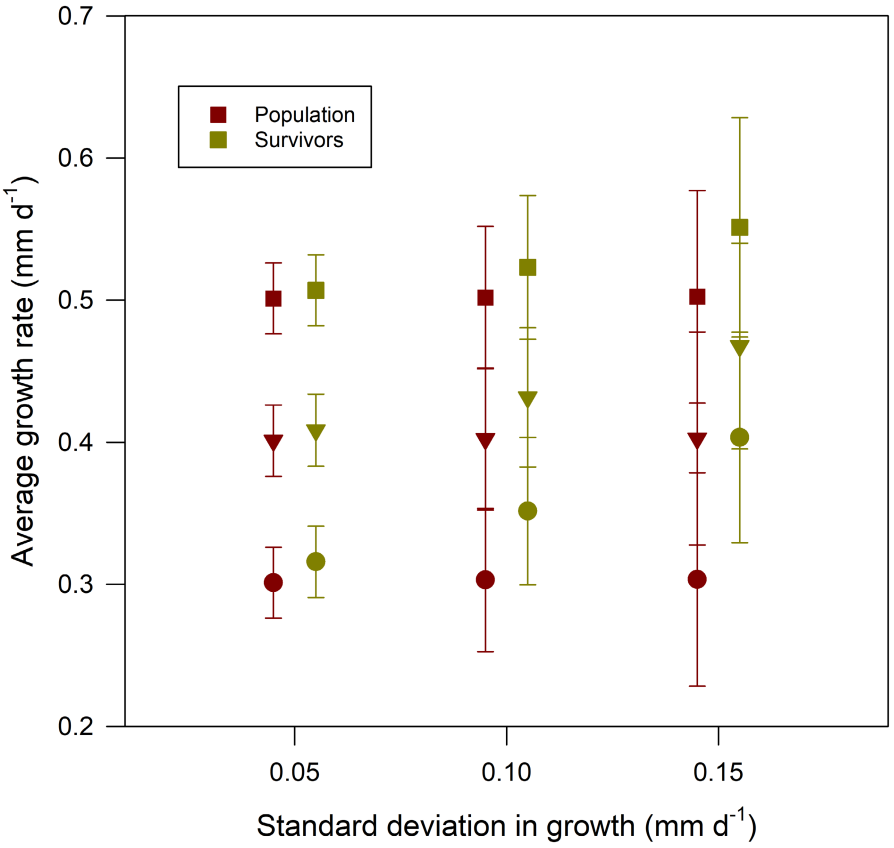

FIGURE 5 Difference in mean growth (±1 standard deviation, SD) of larvae in simulated cohorts between the initial population (all 6 d old larvae) and survivors (larvae reaching 12.5 mm in total length) for three initial conditions of SD in growth (0.05, 0.10 and 0.15 mm d^{-1}; initial population and survivor groups were slightly offset to improve visibility). Initial growth rates in the simulations were set to 0.3 (○), 0.4 (▽) or 0.5 (□) mm d^{-1}. The mean individual growth rate from day 5 onward was set as that individual's average of growth achieved during days 1–4, varying with the population's SD.

set of simulations, where a higher mortality rate was applied to slow-growing (<0.4 mm d^{-1}) larvae, the mean growth rate of survivors was ~11% higher and its SD ranged from 35% to 47% lower than that of the initial population in all three simulations (Table 1, Figure 8a–c). While the difference in mean growth rate between survivors and the initial population remained relatively stable across these simulations, the SD of growth rates of survivors decreased by ~18% and survival rate increased by ~840% (from 0.8% to 7.1%) with decreasing mortality rate against fast-growing individuals (Table 1). Overall, this set of conditions supports the GSP.

In our second set of simulations, in which higher mortality rates were applied to faster-growing individuals, the patterns of change in the characteristics of survivors proved more complex. At the lowest contrast (0.20 vs. 0.30), the mean growth rate of survivors was still greater (by 1.3%) than that of the initial population, whereas the SD of growth showed a milder decline (~26%) compared to when higher mortality was applied to slow-growing individuals, which was consistent with the GSP. At higher contrasts in mortality rates, the mean growth rate of survivors declined by 3.9% (0.15 vs. 0.35) and 8.0% (0.05 vs. 0.45) relative to that of the initial population (Table 1, Figure 8d–f), in a manner contrary to expectations from the GSP. These two scenarios also resulted in a dramatic decrease in the variance in the growth rate of survivors, with declines exceeding 40% and 55%, respectively. Survival rates in this second set of simulations were lower than for corresponding mortality rate contrasts in the first set of simulations, ranging from 0.6% to 6.0% (Table 1).

7 | DISCUSSION

7.1 | Selection for fast growth and the growth-survival paradigm

Since Hjort's (Hjort, 1914) seminal demonstration that fluctuations in the abundance of fish stocks were attributable to tremendous variability in recruitment success, 'solving the recruitment problem' (sensu Sissenwine, 1984) became the Holy Grail of fisheries science and management (Houde, 2008). Attempts to solve the recruitment problem resulted in major advances in the late 20th century, which linked larval survival potential to growth and larval mortality to predation, and have led to the development of the Growth-Survival Paradigm (GSP). The GSP predicts that fast growth during the larval stage will result in high survival and subsequent strong recruitment by reducing cumulative predation mortality (Anderson, 1988; Cushing, 1990; Houde, 1987). However, given the unequivocal support of studies aimed at validating the GSP, the main research direction in the early 21st century shifted from solving to 'understanding the recruitment problem' (sensu Houde, 2008). Our review of the large body of research that has tested the GSP during the past three decades, using field, laboratory, mesocosm and modelling approaches revealed that, although the GSP was supported in the majority of cases, a substantial portion of all tests (44%) including a third of field-based investigations did not support the paradigm. Moreover, a non-negligible proportion of reviewed studies demonstrated the occurrence of selection for slow-growing

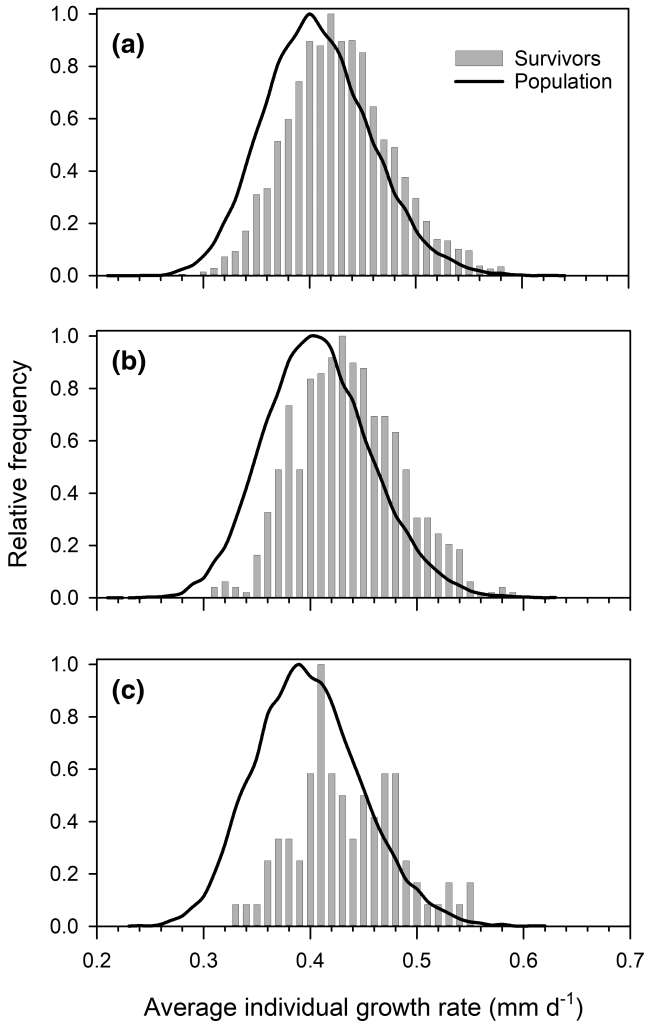

FIGURE 6 Effect of variations in mortality rate (±1 standard deviation, SD) on the frequency distribution of larval growth rate for the initial larval population (all 6 d old larvae) and survivors (larvae reaching 12.5 mm in total length) in our simulations. (a) Mortality = 0.15 ± 0.12 d^{-1}; (b) mortality = 0.25 ± 0.20 d^{-1}; and (c) mortality = 0.35 ± 0.28 d^{-1}. All simulations were based on initial individual random growth (G = 0.4 ± 0.1 mm d^{-1}) with the mean growth rate of each individual from day 5 onward equal to that individual's average growth rate achieved during days 1–4, with a SD of 0.1.

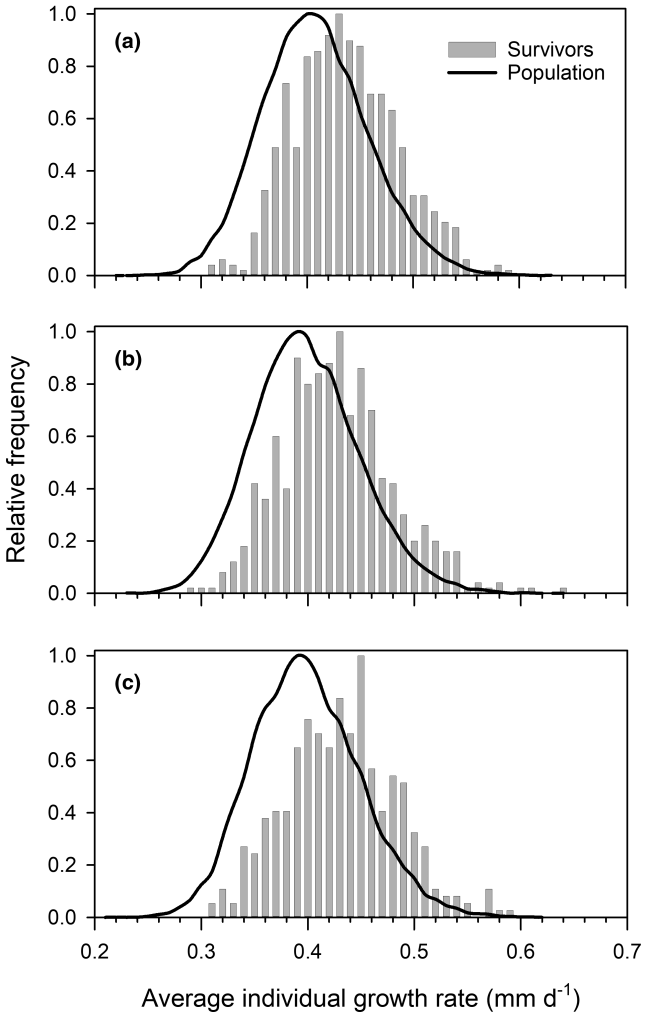

FIGURE 7 Effect of variations in mortality rate (±1 standard deviation, SD) on the frequency distribution of larval growth rate for the initial larval population (all 6 d old larvae) and survivors (larvae reaching 12.5 mm in total length) in our simulations. (a) No length-dependent mortality = 0.25 ± 0.20 d^{-1}, to provide a reference for comparison; (b) weak length-dependent mortality = 0.485·$TL^{-0.34}$ ± 0.2 d^{-1}; and (c) strong length-dependent mortality = 0.914·$TL^{-0.68}$ ± 0.2 d^{-1}. All simulations were based on initial individual random growth (G = 0.4 ± 0.1 mm d^{-1}) with the mean growth rate of each individual from day 5 onward equal to that individual's average growth rate achieved during days 1–4, with a SD of 0.1.

individuals in the field, a possibility generally disregarded in the current GSP. The wide breadth of possible situations that occur in the field might have led some authors to conclude that investigations focussed on the current GSP framework may have limited progress towards better understanding the recruitment processes (Leggett & Frank, 2008). In this context, we sought to develop a revised framework that would explain the differential support (or lack thereof) for the GSP in previous investigations by identifying the contextual factors causing disparities among studies. Such an ability would offer a way forward by allowing researchers to assess under what conditions the tenets of the GSP might hold and should be investigated to understand and/or solve the recruitment problem at the level of the stock.

A key finding originating from our simulations is that the original conceptualization of the GSP, and the way of testing it through the comparison of growth rate frequency distributions between the initial larval population and survivors, is only valid when among-cohort variance in growth and mortality rates is constant (Figure 9a). As predicted by the GSP, the effects of growth and mortality were opposite, with growth having a larger influence on the differences in mean growth achieved by survivors relative to the original population. Changes in mortality rates had more influence on the variance in growth rates of survivors relative to the original population, primarily because mortality acts on the contrast of growth rate among

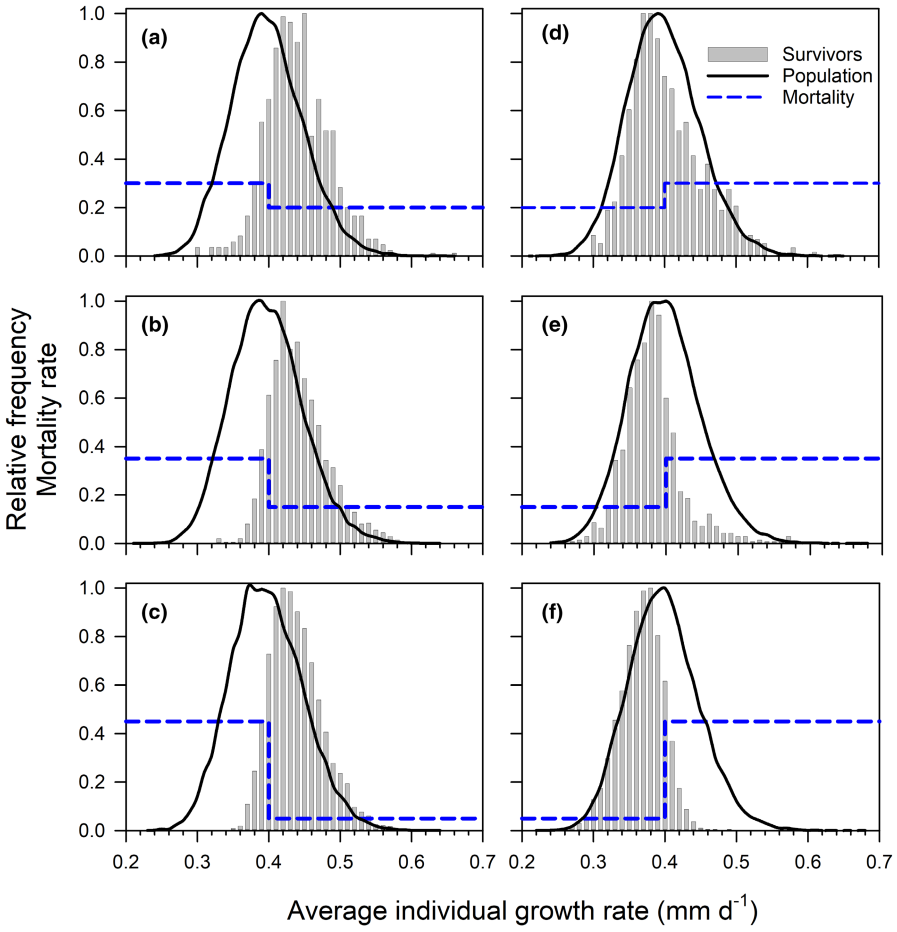

FIGURE 8 Effect of variations in growth-dependent mortality rate on the change in the frequency distribution between the initial larval population (all 6 d old larvae) and survivors (larvae reaching 12.5 mm in total length) in simulations where mortality rate selected against slow-growing individuals (a–c) or against fast-growing individuals (d–f). The blue line represents the growth-dependent mortality rate applied in the simulations. All simulations were based on initial individual random growth ($G = 0.4 \pm 0.1$ mm d^{-1}) but with the mean individual growth rate from day 5 onward set as that individual's average of growth achieved during days 1–4, with a SD of 0.1.

individuals whereas changes in average growth rates affected all individuals to a similar degree.

Our results, however, also indicated that changes in growth autocorrelation, variance in growth among cohorts or changes in growth-dependent mortality rates, can lead to errors when interpreting the meaning of apparent selection for fast growth rates relative to survival and recruitment. Variance in growth rate within the initial population, as well as differences in the timing of the onset of growth autocorrelation, had subtle but important effects on the growth characteristics of survivors, with an outcome that did not support predictions from the GSP because increased survival was achieved with increasing differences in the average growth rates of survivors. More specifically, variability in both of these aspects of growth resulted in situations where an increase in the difference in mean growth rate between survivors and the initial population (selection for fast growth) was only weakly linked to an increase in survival rate. Growth-dependent mortality scenarios also did not entirely support predictions of the GSP. In particular, increasing mortality rates directed towards slow-growing individuals had no major effect on the difference in mean growth between survivors and the initial population. In the case of increasing mortality against fast-growing individuals, we observed a shift in mean growth rate of survivors to slower growth rates relative to the initial population (selection against fast growth).

As previously suggested by Rice et al. (1993) and Houde (1997a), our results indicate that drivers of variance in the growth rate of a larval population could result in selection for fast growth over a broad range of mortality rates with the degree of selection increasing with mortality rates (Figure 9a,b). This outcome is possible given that higher variance in growth rates around the same mean value implies a relatively larger number of individuals within the population that have high growth rates and are characterized by higher survival probabilities to the endpoint resulting from a shorter larval stage duration. Our results also suggest that growth- or size-selective predation targeted at fast-growing or larger-at-age individuals could account for field-based observations of higher survival in slow-growing individuals (Figure 9 c). These situations constitute a reversal of the prediction of the GSP that increasingly high survival should be linked to decreasing selection for fast growth.

Importantly, testing the conceptual framework of the GSP has mostly relied on the comparison of growth-rate frequency distributions between survivors and the larval population from which they originate (e.g. Hare & Cowen, 1997; Meekan & Fortier, 1996; Pepin, 1989). While being useful, this approach extends beyond the notion of mean growth and mortality. It integrates both the direct and indirect influences of these rates and largely ignores the role that variance plays in driving selection and survival outcomes, a key component revealed by our simulations. Because growth and mortality rates and their direct and indirect effects are likely to be unique for a given population, effort should be spent identifying the mechanisms responsible for observed growth selection in each system and determining how they vary.

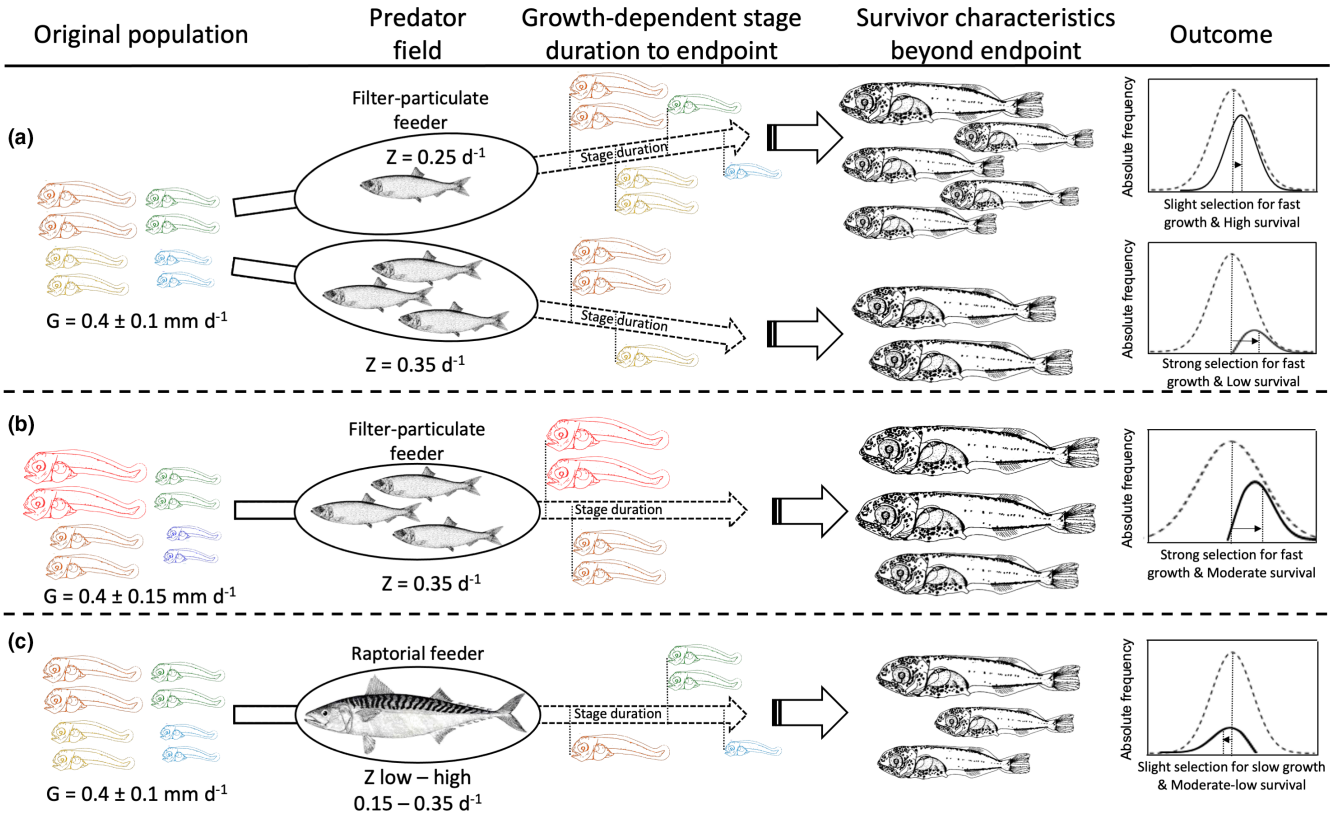

FIGURE 9 Growth-Survival Paradigm (GSP) revisited after simulation results from the present study. The different scenarios show how characteristics of survivors (to the endpoint when year-class or recruitment success is established), as well as growth rate frequency distributions of the initial (dashed line) and surviving (solid line) populations (on the right), relate to various factors and processes (indicated to the left), including differences in: (i) characteristics of the original population—the variance of larval growth rates, with different growth rates represented by larvae of different sizes and colours ranging from red (highest growth rate) to blue (lowest growth rate); (ii) the predator field—the type and amount of daily predation mortality (Z) experienced; and (iii) growth-dependent stage duration to the recruitment endpoint—the amount of time individuals suffer high rates of mortality due to predators. Larvae of a given growth rate category that have disappeared along the stage duration line have been eaten by predators. The figure depicts two types of predators: a filter/particulate feeder (e.g. juvenile herring) and a raptorial feeder (e.g. adult mackerel). (a) Representation of the original conceptualization of the GSP, where predation mortality favours fast-growing individuals through growth-selective mortality against slow-growers that experience a longer amount of time in stages vulnerable to predators. Under that conceptualization, high survival (low mortality) to the endpoint is associated with slight selection for fast growth, whereas low survival (high mortality) is associated with strong selection for fast growth. (b) Results from the present study show that, if the original population is characterized by a high variance in growth rate, caused by either differences in the characteristics of spawners or an early onset of growth autocorrelation, strong selection for fast growth can result, which is not coupled with low survival. (c) Simulations also showed that when predation mortality is primarily directed against larger, fast-growing larvae (mortality rate = 0.35 d^{-1}) compared to smaller, slower-growing larvae (mortality rate = 0.15 d^{-1}), the stage-duration advantage of fast-growing individuals can be offset by predation, resulting in selection for slow-growing larvae and low survival. The drawings of larvae and juveniles are from Fahay (2007).

To this end, we discuss context-dependency in the recruitment process below, including other important knowledge gaps that have been hampering progress in GSP research. We focus primarily on predation, which has been considered the most important source of growth-dependent mortality (Anderson, 1988; Houde, 2016), and on the nature and role of intrinsic factors that modulate predation mortality (leading to context-dependent outcomes). In addition to discussing how extrinsic and intrinsic factors can determine the duration of the critical period that drives recruitment variability in a population or cohort of interest, we also consider how variability in defining the pre-recruitment endpoint can affect perceived changes in survival and the value of the GSP in predicting recruitment variability and stock productivity.

7.2 | Predation

Predation mortality is generally considered the main direct driver of recruitment variability and population dynamics of fish during early life stages (Bailey & Houde, 1989; Cowan & Houde, 1993; Heath, 1992; Hunter, 1984). The conventional view within fisheries science is that predation is a size-selective process, with smaller individuals more likely to be preyed upon than their larger conspecifics (Anderson, 1988; Cowan & Houde, 1992; Miller et al., 1988; Pepin, 1991; Peterson & Wroblewski, 1984; Rice et al., 1993). This notion is embodied by the bigger-is-better (Miller et al., 1988), and growth-selective predation (Takasuka et al., 2003, 2007) mechanisms, which form key tenets of the GSP.

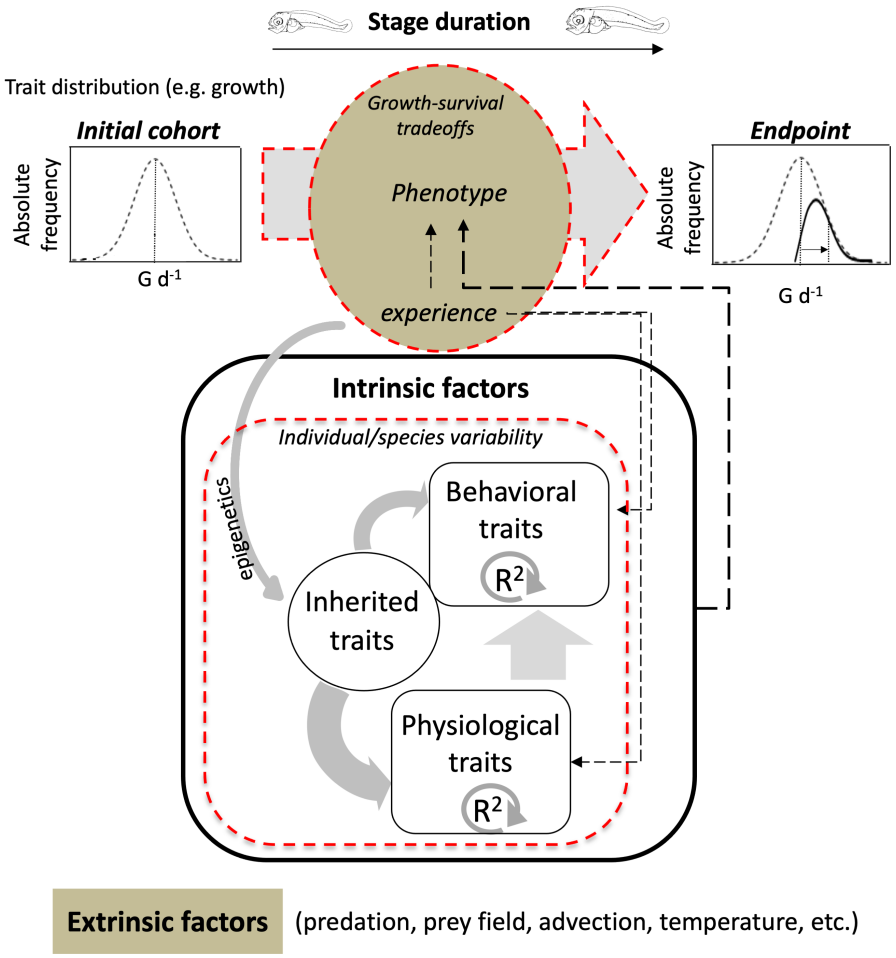

FIGURE 10 Conceptual diagram showing how intrinsic factors (lower part) and extrinsic factors (shaded circle, listed at the top) act on the larval phenotype, resulting in growth-survival trade-offs (sensu Jørgensen et al., 2013) that influence the outcome of cohort growth selection until the endpoint when recruitment is set. Variability (dashed red lines) in intrinsic and extrinsic factors influence the variability in stage duration (upper part, from left to right), which also depends on genetic factors. Extrinsic factors can affect individuals by triggering intrinsic responses that can range from behavioural to physiological manifestations, including epigenetics. Some traits can be autocorrelated (indicated by R^2). The width of arrows in the intrinsic factor panel reflects the authors' interpretation of the relative importance of the different factors based on a qualitative assessment.

Despite the assumption that predation is the main mortality agent in the GSP (Anderson, 1988), most field studies have not quantified or defined the predator field (e.g. Hare & Cowen, 1997; Meekan & Fortier, 1996; Robert et al., 2007). Predation has often been treated as a constant among cohorts, leading to the assumption that selective mortality primarily depends on larval growth performance (such as depicted in Figure 4a–c). Little attention has been given to the idea that the nature, timing, and intensity of predation mortality could represent a major source of variability in growth-dependent survival that remains unexplained by current GSP research. This gap includes spatially explicit individual-based biophysical models of fish early life stages designed to generate and/or test recruitment hypotheses (Peck & Hufnagl, 2012). In most models, for lack of better in situ estimates or process knowledge, mortality rates are assumed to monotonically decline with increasing body size. Moreover, the potential effect of other environmental drivers acting on both mortality and growth rates (e.g. temperature influences on predator appetite and larval growth capacity, behavioural responses to particular predator/prey fields) are rarely incorporated (Akimova et al., 2016; Jørgensen et al., 2013) (Figure 10).

Documenting shifts in predator fields can be challenging and doing so simultaneously with sampling larval fish prey is even more difficult (Houde, 1997a). Yet, changes in both mean predator abundance and patchiness across time and space have been shown to influence observed growth of larval fish prey (Gleiber et al., 2020; Swieca et al., 2023). In the rare cases where temporal and spatial variation in predator fields have been incorporated, simulations suggested high inter-annual differences in the strength of different sources of mortality (e.g. losses due to poor retention or increased starvation and predation) at specific spawning grounds (Akimova et al., 2019). Such a gap in knowledge of interacting factors and processes contributing to mortality and year-class success represents an important limitation for interpreting the various types of selectivity patterns that appeared in peer-reviewed studies and our ability to build process models advancing predictive capacity.

A notable exception that has identified in situ evidence of the impact of predators on larval population characteristics comes from work from Takasuka et al. (2003, 2007, 2017), Takasuka, Aoki, et al. (2004); Takasuka, Oozeki, et al. (2004) on larval Japanese anchovy (*Engraulis japonicus*, Engraulidae), which revealed different patterns and levels of growth-selective mortality among various fish predators. Based on the comparison of otolith growth trajectories between ichthyoplankton consumed and not consumed by predators, these authors demonstrated that the nature of growth-selective predation depended on the type of predator. Small, filter-feeding pelagic fish selected slow-growing larvae whereas raptorial piscivorous fish ingested larvae independent of larval growth rate. McCormick and Hoey (2004) also reported that different size classes

of predators induced differences in mortality patterns dependent on growth history and size at settlement. These results are consistent with laboratory studies, which showed that size differences between a predator and its prey, as well as the foraging strategy of the predator, result in different prey vulnerabilities owing to differences in encounter, attack and capture probabilities (Bailey & Houde, 1989; Paradis et al., 1996). These differences in predator attributes can result in situations where fast growth and large body size during the larval stage may or may not be beneficial for survival (Figure 8). For example, most filter-feeding predators are gape-limited, with gape width proportional to body size. In this context, fast larval growth can be advantageous by allowing larvae to minimize the time spent in the 'window of vulnerability' to these predators. Importantly, however, predators of different sizes would be expected to select for larval fish prey of different ranges in size, with consumed prey sizes determined by optimal foraging rules (i.e. predators are expected to maximize net energy gain; Townsend & Winfield, 1985; Werner & Hall, 1974). In this way, fast growth that leads to a larger size could cause larvae to grow into a window of vulnerability to predation (Cowan et al., 1996; McCormick & Hoey, 2004). In the field, the changes in species within a given predator assemblage may inflict a varying mixture of growth-selective pressure on a given larval fish population (McCormick & Hoey, 2004; Takasuka et al., 2003, 2007, 2017). Therefore, knowledge of the characteristics of the main larval fish predators is essential for understanding processes driving differences in growth-selective survival among cohorts.

7.3 | Intrinsic factors

Even though interactions between intrinsic factors and agents of mortality have previously been reviewed (e.g. Govoni, 2005; Houde, 2008, 2016; Peck et al., 2012), they have rarely been considered in the context of the GSP. In fact, the relative importance of factors accounting for variability in the growth-rate frequency distribution of a given larval fish cohort relative to others and, thus, providing the raw material for growth-dependent mortality to operate, remains poorly known. While the respective effects of intrinsic factors in driving growth-dependent mortality can be intermingled (Conover & Baumann, 2009), factors that are considered important in shaping growth characteristics of survivors could be divided into physiological, inherited and behavioural traits (Figure 10).

Physiological processes may mediate how individual traits vary in response to exposure to different environmental factors (Angilletta et al., 2003). The most important factor affecting larval physiology and leading to individual differences in growth rate is temperature. The thermal history of larvae has been shown to determine survival potential during the juvenile stage through physiology-related differences in development (e.g. Catalán et al., 2004; Johnston et al., 2001; Moyano et al., 2014). In turn, these differences in development can induce variability in growth, metabolism and swimming capacity (Batty et al., 1991; Johnston, 1993) that can affect survival probability through their influence on the ability of a given individual to successfully feed and escape predators.

At the cohort level, physiologically determined traits likely constitute a particularly important source of variability in larval development and growth of species characterized by protracted spawning, covering a wide temperature range (e.g. Temple et al., 2001). In temperate environments, late spawning and hatching occur at higher temperatures relative to early hatching in the spring, often resulting in faster larval growth (Fortier et al., 2006; Ludsin & DeVries, 1997). However, late-spawned individuals do not always reach the minimum size necessary for them to survive their first winter. In such a situation, size-based selection may occur against individuals that were characterized by fast growth during the larval stage (e.g. Ludsin & DeVries, 1997), which has important implications for endpoint selection used to test growth-survival relationships. Because the effects of physiologically determined traits on the ontogeny of development and growth are highly species- and system-specific (Balon, 1979; Blaxter, 1986; Govoni, 1980; Job & Bellwood, 2000; Peck et al., 2013; Webb & Weihs, 1986), we highlight the importance of conducting population-specific investigations to quantify the effects of these traits on growth-selective survival.

Inherited traits can also account for individual phenotypic differences leading to variability in survival potential. Variability in larval size and condition attributable to parental effects likely constitutes an important source of differences in the variance of growth rates among cohorts. Such variation then has potential implications for the extent of observed differences in growth-rate frequency distribution between survivors and the initial population (Chambers & Leggett, 1992), and on the significance of these differences to survival (see Figure 4d–f). Parental effects on variability in larval size and condition could affect underlying variability in growth characteristics of the initial population and, therefore, influence growth-selective survivorship within the context of the GSP.

Variability in larval behaviour can influence survival probability at the individual level (Jørgensen et al., 2013). Larval behaviour is highly dependent on the physical and sensory capabilities at a given developmental stage (Blaxter, 1986; Houde & Schekter, 1980; Skajaa et al., 2003). Developmental priorities of vision (Loew & Wahl, 2008), swimming speed (Leis, 2006) and feeding tactics (Lewis & Bala, 2008; O'Brien et al., 1990) partly depend on body size and its relation to water viscosity (Osse & van den Boogaart, 1999), but are also driven by growth-dependent predation risk under a given feeding environment (Jørgensen et al., 2013). Under limited prey availability, maintaining high growth rate implies increased foraging activity (Vollset et al., 2013), which also results in an increased encounter rate with predators and ultimately increased mortality rate until reaching the endpoint, as observed in previous studies (Fiksen & Jørgensen, 2011; Fuiman, 1989; Lankford et al., 2001) (see Figure 8d–f).

Although larval behaviour is not considered in the current conceptualization of the GSP, the theory of behavioural trade-offs between growth and survival (e.g. Biro & Stamps, 2010) needs to be explored in the context of the GSP. Jørgensen et al. (2013) presented

a conceptual framework that predicted that behavioural trade-offs between growth and predation risk are particularly important during the late larval stage and that individuals will display rapid adaptations to optimize their growth-to-mortality ratio under variable food supply, predation pressure or other environmental conditions. As an example, theoretical modelling, laboratory experimentation and field observations have shown that larval fish will adjust their foraging rate on zooplankton (i.e. potential for growth) depending on water clarity (water-column turbidity), which can influence susceptibility to predation risk (Pangle et al., 2012). Long-term, population-level changes in larval behaviour have also been documented such as the loss of diel vertical migration in response to changes in the prey community (Hinrichsen et al., 2010), which will alter the exposure of larvae to specific predators. Larval behaviour, thus, may have important, dynamic and even counter-intuitive effects on the observed differences in growth-frequency distribution between survivors and the initial population. Given that behavioural responses to varying prey supply and predation pressure vary widely among taxa, measuring the relative importance of behavioural traits in driving growth-selective patterns constitutes an important and almost unexplored research avenue.

Under specific circumstances, intrinsic factors can interact with predation to modify growth-dependent mortality patterns, including selection against large or fast-growing larvae over small or slow-growing individuals. For example, high feeding activity displayed by fast-growing individuals to meet their higher energetic demands may result in higher encounter rates with predators (Fuiman, 1989; Lankford et al., 2001). Temperature may also play an important role in driving growth-dependent vulnerability to predation if the change in temperature-mediated metabolic processes is different for predators (their feeding rates on larvae) and larvae (their growth rates) (Akimova et al., 2016). Also, differences in growth rates may reflect differences in individual condition, affecting the ability of larvae to avoid an attack by a predator (Lankford et al., 2001; Takasuka et al., 2003). Therefore, understanding the pathways of effects that intrinsic factors may have on an individual larva's probability of encountering, being attacked and escaping a predator and how these intrinsic and extrinsic factors interact, is essential to determine how they may affect the outcome of growth-selective processes (Figure 10).

7.4 | Endpoints

Studies focussed on inferences from the application of the GSP generally consider processes affecting the larval stage, but it is important to recognize that the relative rank of a year-class may be determined during any developmental stages prior to fish entering the fishery or the reproductive population (e.g. Sissenwine, 1984). Variability in the nature, timing and intensity of predation, combined with species-specific intrinsic factors, will act in concert to determine the duration of the critical phase during which mortality rate is high and variable. The end of this period of vulnerability for early survival corresponds to the 'endpoint' at which year-class strength can be established. In many cases, tests of the GSP have consisted of comparing the growth-rate frequency distribution of a cohort between two life stages without an a priori validation that the endpoint had been reached within the tested interval. Conclusions drawn from such studies may be misleading and, for a number of reasons, assumptions should be avoided on the timing (or body size) of this biological endpoint. The endpoint at which recruitment (year-class strength) is set differs widely among species, but also can vary among cohorts within a given population (Benoît et al., 2000; Chambers & Leggett, 1987; Houde, 1997b, 2008; Sissenwine, 1984). For example, two consecutive annual cohorts of striped bass (*Morone saxatilis*, Moronidae) from the Nanticoke River population were characterized by a transition stage when cohort biomass started increasing at 34.9 and 113.5 d, suggesting wide interannual variability in recruitment endpoint (Houde, 1997b). In the population of striped bass in the St. Lawrence River, the endpoint was detected after the end of the first winter, but the strength of overwinter mortality differed between years (Peres et al., 2022). These examples stress the importance of measuring and considering endpoint variability within a given population of interest instead of relying on an arbitrary reference point such as the metamorphosis (age 35–45 d in striped bass) that may not fully cover the cumulative forces shaping growth characteristics of survivors. The arbitrary selection of an endpoint is unlikely to capture the full impact of processes that govern the determination of year-class strength or to provide a robust test of the GSP.

The endpoint for a given cohort can be estimated from the sequential sampling of successive early life stages. Samples of both the initial and surviving life stages must always accurately represent the cohort of interest so that the successful estimation of the endpoint directly depends upon our capacity to obtain representative samples of the successive early life stage (Khamassi et al., 2020). In many cases, broad spatial and temporal sampling of an area is necessary to adequately capture representatives of the initial and surviving populations. By indicating transitions in biomass dynamics in a developing cohort, the ratio of mortality (M) to growth (G) (M/G) can be useful for identifying the life stage from which the endpoint is likely to occur (Houde, 1997a, 1997b). Following hatching, mortality rate is generally much higher than population growth, resulting in a net loss in biomass for the cohort. The age from which $M/G \geq 1$, implying net growth of the cohort, corresponds to the minimum life stage from which the endpoint could be reached. From that life stage, the precise timing of the endpoint can then be investigated by comparing the otolith-derived growth rate-frequency distributions between the initial population and multiple subsequent life stages. Examples of species that are known to experience drastic changes in growth-selective survival prior to the endpoint include flatfishes and coral reef fishes, groups characterized by a settlement stage that involves a major change in habitat and predation pressure (e.g. Gagliano et al., 2007; Geffen et al., 2007; Hoey & McCormick, 2004). Potential consequences of the interplay between habitat shifts and selective mortality were well described by D'Alessandro et al. (2013), who successfully sampled several cohorts of different

snapper species (Lutjanidae) and great barracuda (*Sphyraena barracuda*, Sphyraenidae) at different life stages in the Straits of Florida. Notably, their study found a reversal in growth selection for one of the species during the settlement period, highlighting how species-specific ontogenetic shifts in selection pressure may constitute a challenge when testing the GSP.

Otolith microstructure analysis represents an ideal tool to assess endpoint considerations. In addition to representing a permanent record of early individual growth, the otoliths of species that undergo a major transition from pelagic to benthic life are often characterized by a distinct 'metamorphic mark' (Geffen et al., 2011; Joh et al., 2013), allowing the investigation of potential shifts in growth-dependent mortality following that key life stage transition. The interpretation of the otolith microstructure, however, may be difficult during the early larval stage when the deposition of the first otolith increment is delayed (e.g. Fox et al., 2003; Ivarjord et al., 2008) and during the juvenile stage as a result of the formation of secondary primordia (e.g. Joh et al., 2015; Narimatsu et al., 2007). In cases where using otoliths to estimate age is not possible, verifying whether individuals sampled at the endpoint stage were part of the original larval cohort being compared may be difficult, except for species characterized by a punctual spawning season (e.g. Robert et al., 2007). Overall, in cases where the otolith growth trajectory is truncated (e.g. May et al., 2020), one must consider that a proportion of the variability in growth-selective survival relevant to the GSP may be missing from the analysis.

8 | CONCLUSIONS

Assessment of the validity of the GSP has been based on the analysis of the characteristics of survivors relative to those of individuals at earlier stages of development within the same cohort. Our review of the evidence and simulations has demonstrated that caution should be applied when interpreting patterns in growth selection and that rapid growth of survivors relative to the initial population does not always reflect an overall high mortality rate. In previous studies, contrasts have typically been made between the back-calculated mean growth rates of survivors and the initial population. However, our simulations indicate that a more comprehensive evaluation is necessary, including analyses of the variance of individual growth rates and autocorrelation in growth. Moreover, assessing growth-selective mortality by contrasting the initial population to a life stage located beyond the range of possible cohort endpoints for a given stock, at which individual growth characteristics will represent those of recruits, appears to be crucial. However, such an approach does not provide information on the proximate causes of mortality. Several examples illustrate how sampling among cohorts (Khamassi et al., 2020; Robert et al., 2007), or at different spatial (Leclerc, 2010) or temporal (Sponaugle et al., 2011) scales can be used to assess variability in growth-selective survival, confirm or refute assumptions of the GSP and inform about processes across ecosystems, species and populations.

The paucity of research that identifies and/or quantifies the role of predation mortality, and how rates of predatory losses are potentially related to intrinsic larval traits, represents the most critical bottleneck in our ability to further refine the GSP and make robust forecasts of changes in fish stock productivity, whether because of changes in year-class strength or ecosystem state. Enhanced metrics to contrast differences among individuals and quantification of prey–predator dynamics will generally require observational programs that are difficult to sustain through time. They do, however, represent basic knowledge to be collected prior to comprehensive efforts aimed at investigating the drivers that govern the dynamics of fish populations, and should be the foundation for further studies into the GSP.

Despite the potential limitations of the GSP, relative changes between the growth rate distribution of survivors and that of earlier life stages from the same cohort can provide valuable information on rates of mortality. This information is important when conducting retrospective analyses using, for example, field-based or mechanistic models depicting how bottom-up and top-down processes impact the survival of specific species (Akimova et al., 2019; Reichert et al., 2010) or, at a much broader level, marine food web dynamics (Lynam et al., 2017). One must be cautious, however, in the attribution of the dominant processes underlying changes in patterns of growth rate distributions. Various factors (apart from selective predation) can alter the underlying variation in growth among individuals within a given cohort and cause misleading evidence of factors and processes ultimately controlling the survivorship and recruitment success of different cohorts. Our work demonstrates that a comprehensive characterization of differences in the distributions of larval growth rates between the initial population and individuals that have survived to the endpoint is essential in testing and referencing the GSP as an explanation of selective loss during the early life history of fish.

AUTHOR CONTRIBUTIONS

The manuscript is the outcome of a unique symposium and workshop. Authors contributed to the discussions and manuscript in the following manner: Dominique Robert, Jun Shoji, Pascal Sirois, Akinori Takasuka and Pierre Pepin conceptualization and organization of the symposium and workshop and primary authors. Ignacio A. Catalán, Arild Folkvord, Stuart A. Ludsin, Myron A. Peck and Su Sponaugle were meeting participants and key contributing authors. Patricia M. Ayón, Richard D. Brodeur, Emily Y. Campbell, Evan K. D'Alessandro, John F. Dower, Louis Fortier, Alberto G. García, Klaus B. Huebert, Marc Hufnagl, Shin-ichi Ito, Mikimasa Joh, Francis Juanes, Mitsuo Nyuji, Yoshioki Oozeki, Guido Plaza, Motomitsu Takahashi, Yosuke Tanaka, Naoki Tojo, Shingo Watari and Naotaka Yasue were meeting participants and supporting authors.

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CONFLICT OF INTEREST STATEMENT

The authors do not have any interest or relationship, financial or otherwise, that might be perceived as influencing an author's objectivity and that might be considered a potential source of conflict of interest.

DATA AVAILABILITY STATEMENT

Data sources are provided in the Supporting Information. Simulation code can be obtained from PP.

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