Stoichiometric microplastics models in natural and laboratory environments

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A R T I C L E   I N F O

MSC: 92B05, 92D23, 34C60

Keywords: Microplastics Stoichiometric constraints Light intensity Nutrients

A B S T R A C T

Microplastics pose a severe threat to marine ecosystems; however, relevant mathematical modeling and analysis are lacking. This paper formulates two stoichiometric producer-grazer models to investigate the interactive effects of microplastics, nutrients, and light on population dynamics under different settings. One model incorporates optimal microplastic uptake and foraging behavior based on nutrient availability for natural settings, while the other model does not include foraging in laboratory settings. We establish the well-posedness of the models and examine their long-term behaviors. Our results reveal that in natural environments, producers and grazers exhibit higher sensitivity to microplastics, and the system may demonstrate bistability or tristability. Moreover, the influences of microplastics, nutrients, and light intensity are highly intertwined. The presence of microplastics amplifies the constraints on grazer growth related to food quality and quantity imposed by extreme light intensities, while elevated phosphorus input enhances the system’s resistance to intense light conditions. Furthermore, higher environmental microplastic levels do not always imply elevated microplastic body burdens in organisms, as organisms are also influenced by nutrients and light. We also find that grazers are more vulnerable to microplastics, compared to producers. If producers can utilize microplastics for growth, the system displays significantly greater resilience to microplastics.

1. Introduction

Plastic pollution has steadily increased due to widespread plastic use, insufficient disposal practices, and limited waste management capacity over decades. Plastic wastes discharged into terrestrial and aquatic habitats are considered a serious threat to biodiversity, due to their resistance to decomposition (Gall and Thompson, 2015; Carbery et al., 2018). The small plastic particles, ranging from 0.1 μm to 5 mm in size, are called microplastics (EFSA Panel on Contaminants in the Food Chain (CONTAM), 2016). Microplastics are observed almost in all aquatic habitats (Eerkes-Medrano et al., 2015; Peeken et al., 2018). Chronic exposure to microplastics presents several challenges for aquatic organisms (Cui et al., 2017; Duis and Coors, 2016; Nolte et al., 2017; Zhang et al., 2017; Khoironi et al., 2019; Sjollema et al., 2016; Besseling et al., 2014).

Studies have shown that microplastics can be absorbed, concentrated, and transported into various organisms, such as algae. This process significantly hampers algal growth, chlorophyll levels, and photosynthetic activity (Cao et al., 2022; Liu et al., 2019; Chae et al., 2019; Wu et al., 2021; Jiao et al., 2022; Gray and Weinstein, 2017; Zhang et al., 2017; Sjollema et al., 2016; Yang et al., 2020; Ferguson, 2011; Wang et al., 2020; Khoironi et al., 2019; Rodrigues et al., 2019; Salman et al., 2016). This inhibition may occur due to physical factors, such as the blockage of light and airflow (Bhattacharya et al., 2010; Wang et al., 2020; Salman et al., 2016; Bhattacharya et al., 2010), or interactions between microplastics and algae, including adsorption, aggregation (Zhang et al., 2017), and the destruction of algal cell walls through surface absorption (Liu et al., 2019). However, it is worth noting that some laboratory studies suggest that certain algae species may increase in the presence of smaller-sized microplastics, possibly because microplastic particles can be utilized as substrates for the growth of algae (Yokota et al., 2017; Mao et al., 2018; Jiao et al., 2022; Canniff and Hoang, 2018).

Most aquatic animals cannot distinguish plastic from their natural food sources (Sazli et al., 2023). Numerous studies have demonstrated the ingestion of microplastics by a wide range of marine and freshwater species (Lasher, 2015; Scherer et al., 2017), such as cladocerans (Canniff and Hoang, 2018), amphibians (Hu et al., 2016), fish (Lu et al., 2016), and marine mammals (Fossi et al., 2012). These microplastic particles are either selectively ingested, mistaken for prey, or unintentionally consumed during respiration (Gregory, 1996; Derraik, 2002) and may cause long-term accumulation within the digestive tracts. For instance, in filter feeders, microplastics may account for as much as 58% of their stomach content (Goldstein and Goodwin, 2013). Some microplastics are egested but others are internalized and cause a
series of toxic effects, including reductions in body size, reproduction, food uptake, the onset of oxidative stress and inflammation, and even mortality (Blarer and Burkhardt-Holm, 2016; Alomar et al., 2017; Liu et al., 2019; Wang and Wang, 2023; Guilhermino et al., 2021; Simčić and Anton, 1997; Cui et al., 2017; Canniff and Hoang, 2018; Lin et al., 2023; Elizalde-Velázquez et al., 2020; Martínez-Jerónimo et al., 1994; Bertram and Hart, 1979; Hofschoerger et al., 2021; Lu et al., 2016; Besseling et al., 2014). For example, Besseling et al. (2014) found that exposure to high-concentration polystyrene particles led to a decreased clutching rate of up to 68% in young daphnia magna. Additionally, Liu et al. (2019) observed that daphnia pulex experienced delayed first clutch timing and a decreased total number of offspring per female at 21 days with the presence of polystyrene microplastics.

Numerous mathematical models have been done to investigate the interaction between environmental toxics and populations by considering the toxic effect on population growth rates (Hallam et al., 1983b;a; De Luna and Hallam, 1987; Freedman and Shulka, 1991; Thomas et al., 1996; Thieme, 2003). Huang et al. (2013) in 2013 proposed a population model incorporating a dose-dependent mortality rate function, and monitored toxin body burdens within a single species. Subsequently, Huang et al. (2015) developed a fundamental model to describe MeHg’s effect on predator–prey systems:


d\frac{d y}{d t} = \frac{\alpha y}{\beta} - \frac{\sigma_1 y}{\beta} - \frac{\alpha y}{\beta} + \frac{\zeta}{\beta} \frac{\alpha y}{\beta} + e(v) f(y) w - e(v) f(y) w.

(1.1)

Here, $x$ and $y$ (mg C/L) represent prey and predator densities, while $u$ and $v$ (mg M/mg C) represent their respective body toxin burdens. The function $r(u, x)$ characterizes toxin-dependent prey growth, and $d_1(u)$ and $d_2(v)$ are toxicant-related death rates for prey and predator. $f(x)$ and $e(v)$ denote the predator’s ingestion rate and toxin-dependent production efficiency, respectively. The parameters $\alpha_1$ and $\alpha_2$ represent toxicant uptake rates, and $\sigma_1$ and $\sigma_2$ are toxin degradation rates for prey and predator. $\zeta$ indicates predator toxicant assimilation efficiency.

Notably, none of the aforementioned models considered the influence of food nutrients. However, in natural ecosystems, the growth of species is always constrained by limited nutrient availability (Hecky and Kilham, 1988; Sterner and Hessen, 1994). Organisms require specific proportions of chemical elements, such as carbon (C), nitrogen (N), and phosphorus (P), to meet their fundamental nutritional needs (Ghosh and Chattopadhyay, 2005; Jeyasingham et al., 2017; Ji et al., 2023; Hecky and Kilham, 1988). When these nutritional elements are deficient in the food supply, the conventional predator–prey model may not be applicable since it is the deficiency of nutritional substance, rather than energy content, that constrains the predator growth rate. Instead, stoichiometric models have been widely employed to investigate how nutritional elements influence nutrient cycling mechanisms and population dynamics (Loladze et al., 2000; Li et al., 2011; Xie et al., 2018; Rana et al., 2019; Wang et al., 2012, 2018, 2008; Peace et al., 2013, 2014; Peace, 2015; Chen et al., 2017; Peace and Wang, 2019). Peace and Wang (Peace et al., 2016) in 2016 first expanded upon the basic toxin population model to explore the interactive influence of nutrients and MeHg on population dynamics by incorporating stoichiometric constants into growth rates of producers and grazers.

In laboratory experiments, the conditions for organisms are manually controlled. Typically, grazers are provided with food directly in a small confined container and almost do not need to forage. However, in natural environments, grazers must expend significant energy and time on foraging and deal with challenges like predation risk, extreme climates, and human interactions. Their foraging strategies have been shown to depend on the nutritional content of their food (Simpson et al., 2004; Schatz and McCauley, 2007). When food is nutrient-deficient, grazers have to allocate more time to eat more food to meet their nutrient requirements (Raubenheimer and Simpson, 1993).

Although microplastics have been shown to highly threaten aquatic ecosystems and have garnered increasing concern in recent years, there is currently no mathematical model to investigate their influence on population dynamics. Considering the differences between laboratory settings and natural ecosystems, this paper aims to develop two stoichiometric models to explore how microplastics influence producer-grazer population dynamics in different settings: one model includes optimal microplastic uptake and foraging behavior based on nutrient availability for natural settings, while the other model does not consider foraging behavior for laboratory settings.

2. Model formulation

This study focuses on the interaction between producers (e.g., algae) and grazers (e.g., Daphnia) in a closed ecosystem. The overall framework is based on the general toxin model (1.1). First, we decide the growth rate function. Similar to Huang et al. (2015), we assume the influence of microplastics on the growth of producers and grazers follows a linear response:

\[ \max(0, 1 - a_2 u) \]

and

\[ \max(0, 1 - \beta_2 v). \]

(2.2)

where $a_2$ and $\beta_2$ are the influence coefficients of microplastics on the producer growth and grazer reproduction, respectively.

Meanwhile, the growth of producers and predators is also influenced by their internal nutrient element reserves, such as carbon (C) and phosphorus (P). The stoichiometric limitations have a substantial impact on trophic transfer efficiencies (Peace, 2015). We adhere to the classic assumption of a closed phosphorus system, where the P:C ratio varies in producers and remains constant in grazers (Loladze et al., 2000). The total phosphorus content is denoted as $P$. We introduce a variable P:C ratio denoted as $Q$ for producers, with a minimum value of $q$, and a constant P:C ratio represented by $\theta$ for grazers. Then $Q$ is given by

\[ Q = \frac{P - \theta y}{x}. \]

(2.3)

Moreover, light intensity plays a crucial role in producers’ photosynthesis and growth. Therefore, the growth rate of producers and grazers, influenced by light and nutrients, can be represented by:

\[ a_1 \min\left\{1 - \frac{x}{K}, 1 - \frac{q}{Q}\right\}. \]

(2.4)

and

\[ \beta_1 \min\left\{1, \frac{Q}{\theta}\right\}. \]

(2.5)

Here, $a_1$ and $\beta_1$ denote the maximal growth rate and production rate of the producer and grazer, respectively. $K$ represents the maximal producer carrying capacity with respect to light.

Incorporating (2.1), (2.2), (2.4) and (2.5), the growth rate function of producers and the production efficiency function of grazers can be modeled using following minimum functions:

\[ r(u, x) = a_1 \min\left\{\max(0, 1 - a_2 u), 1 - \frac{x}{K}, 1 - \frac{q}{Q}\right\} \]

and

\[ e(v) = \beta_1 \min\left\{\max(0, 1 - \beta_2 v), \frac{Q}{\theta}\right\}. \]
takes the following form: internal nutrient level. The microplastic uptake function for producers
consider the microplastic uptake rate to be a decreasing function of the
et al., 2023), thereby increasing microplastic uptake. Therefore, we
absorbs microplastics through phospholipids. With less phosphorus,
resulting in increased microplastic uptake. On the other hand, plankton
response of producers and grazers to microplastics is strongly influenced
the half-saturation constant.

We now introduce our stoichiometric model incorporating optimal
microplastic uptake and foraging (OMUF model) under a natural
environment as follows:

\[
\frac{dx}{dt} = a_1 \min \left\{ \left(0, 1 - a_2 u \right), 1 - \frac{x}{K}, 1 - \frac{q}{Q} \right\} x - f(x, Q)y, \]

\[
\frac{dy}{dt} = \beta_1 \min \left\{ \left(0, 1 - \beta_2 v \right), \frac{Q}{\theta} \right\} \frac{f(x, Q)y}{x} \]

\[
\frac{du}{dt} = \left( L_1(Q)T - \sigma_1 u \right) \frac{f(x, Q)y}{x} \]

\[
\frac{dv}{dt} = \left( L_2(Q)T - \sigma_2 v + \xi f(x, Q)y \right) \frac{f(x, Q)y}{x} \]

where \( Q, f(x, Q), \eta(Q), L_1(Q), \) and \( L_2(Q) \) are defined in (2.3), (2.6),
(2.7), (2.8), and (2.9), respectively.

In the laboratory setting, the foraging cost for grazers is negligible.
For simplicity, the uptake rate of microplastics by producers is assumed
to be constant. This leads to the non-foraging microplastics model (NM
model):
notation. The rescaled model is given as follows:
\[ \frac{d\bar{u}}{d\tau} = \frac{a_{1}T}{\sigma_{1}u} \text{ uptake from environment} - \sigma_{1}u \]
\[ \frac{d\bar{v}}{d\tau} = b_{1}T - \sigma_{1}v + \xi f(x)u \]
\[ \frac{d\bar{w}}{d\tau} = \beta_{1} \min\{\max\{0, 1 - \beta_{1}v\}, 1 - \frac{\bar{w}}{K} \} \bar{u}, \]
\[ \text{dilution due to growth} \]
\[ \text{uptake from environment} \]
\[ \text{depuration} \]
\[ \text{gain due to predation} \]
\[ \text{dilution due to growth} \]
\[ \text{(2.11)} \]

where
\[ f(x) = \frac{ex}{a + x}. \]

3. Mathematical analysis

Separate mathematical analyses are conducted for the two models. Let us begin with the OMU model.

3.1. The OMU model

The body burden dynamics are assumed to operate on a much faster timescale than population dynamics. Over a long period, the body burden accumulation will reach an equilibrium, allowing the body burden dynamics to attain a quasi-steady state. To simplify the model, we introduce a small parameter, \( \epsilon = a_{1} \sigma_{1} \), for rescaling the model. In our case, \( \epsilon = 0.0061 \). We then apply the quasi-steady state approximation to reduce our model to a two-dimensional form. The well-posedness and long-term behavior of the resulting two-dimensional systems are then explored.

3.1.1. Rescaling and quasi-steady-state approximation

Rescale the model (2.10) as follows:
\[ \bar{u} = a_{1}u, \quad \bar{v} = \frac{a_{1}}{\sigma_{1}}v, \quad \bar{t} = a_{1}t, \quad \bar{a}_{1} = a_{1}, \quad \bar{a}_{2} = a_{2}, \quad \bar{\sigma}_{1} = \sigma_{1}, \quad \bar{\sigma}_{2} = \sigma_{2}, \quad \bar{y} = \frac{a_{1}}{\sigma_{1}}y, \quad \bar{h}_{1} = \frac{a_{1}}{\sigma_{1}}h_{1}, \quad \bar{\gamma} = \frac{a_{1}}{\sigma_{1}}\gamma, \quad \bar{Q} = \frac{a_{1}}{\sigma_{1}}Q, \quad \bar{L}_{1} = \bar{a}_{1}Q_{0}, \quad \bar{L}_{2} = \bar{a}_{1}Q_{0}. \]

For simplicity, we still use the original notation instead of the tilde notation. The rescaled model is given as follows:
\[ \frac{dx}{dt} = a_{1} \min\{\max\{0, 1 - u\}, 1 - \frac{x}{K} - \frac{y}{Q} \} - \frac{\eta(Q)xy}{a + \eta(Q)x}, \]
\[ \frac{dy}{dt} = \beta_{1} \min\{\max\{0, 1 - v\}, 1 - \frac{\eta(Q)xy}{a + \eta(Q)x} - \eta(Q)xy - (\bar{h}_{1}v + \bar{m}_{2})y \}, \]
\[ \frac{dz}{dt} = \bar{L}_{1}(Q)\bar{T} - \sigma_{1}w - \epsilon \min\{\max\{0, 1 - u\}, 1 - \frac{x}{K} - \frac{y}{Q} \}u, \]
\[ \frac{dw}{dt} = \bar{L}_{2}(\bar{T})\bar{T} - \sigma_{1}v + \beta_{1} \min\{\max\{0, 1 - v\}, 1 - \frac{\eta(Q)xy}{a + \eta(Q)x} \} \frac{\eta(Q)xy}{a + \eta(Q)x}. \]
\[ \text{(3.1)} \]

Let \( \epsilon \to 0 \), the system (3.1) reaches a quasi-steady-state system with
\[ u = \frac{L_{1}(\bar{T})}{\sigma_{1}}, \quad v = \frac{L_{2}(\bar{T})}{\sigma_{2}} + \frac{\beta_{1}L_{1}(\bar{T})}{\sigma_{1}\sigma_{2}\bar{T}} \frac{\eta(Q)xy}{a + \eta(Q)x}. \]
\[ \text{(3.2)} \]

Let
\[ A_{1} = 1 - \frac{L_{1}(\bar{T})}{\sigma_{1}}, \]
\[ A_{2} = \frac{\beta_{1}L_{1}(\bar{T})}{\sigma_{1}\sigma_{2}} > 0. \]
\[ \text{(3.3)} \]

Substituting (3.2) into (3.1), we have
\[ \frac{dx}{dt} = a_{1} \min\{\max\{0, 1 - \frac{L_{1}(\bar{T})}{\sigma_{1}}\}, 1 - \frac{x}{K} - \frac{y}{Q} \} - \frac{\eta(Q)xy}{a + \eta(Q)x} \]
\[ \frac{dy}{dt} = \beta_{1} \min\{\max\{0, A_{1} - A_{2}L_{1}(\bar{T})\frac{\eta(Q)xy}{a + \eta(Q)x} + \frac{\sigma_{1}}{\sigma_{2}}\bar{m}_{2} \}, \frac{\eta(Q)xy}{a + \eta(Q)x} \} - \frac{\eta(Q)xy}{a + \eta(Q)x}, \]
\[ \text{(3.5)} \]

3.1.2. Positivity and boundedness

Let
\[ \Omega = \{(x, y) : 0 \leq x \leq k, y \geq 0, Qx + \theta y < P \}. \]
\[ \text{(3.6)} \]

where \( k = \min\{\frac{P}{Q}, K \} \). The following theorem guarantees that system (3.5) is biologically well defined.

Theorem 3.1. Solutions of (3.5) with initial conditions in the \( \Omega \) remain there for all forward time.

Proof. Assume \( S(t) = (x(t), y(t)) \) is a solution of system (3.5) with \( S(0) \in \Omega \) and \( t_{1} \) is the first time that \( S(t) \) touches or crosses the boundary of \( \Omega \). We will prove the theorem by contradiction arguments from five cases.

Case 1. \( x(t_{1}) = 0 \). Let \( y = \max_{Q(0,1)}(x) < \frac{P}{Q}, \hat{y} = \max_{Q(0,1)}\eta(Q)(t) \). Then \( \forall \in [0, t_{1}] \),
\[ \frac{dx}{dt} = a_{1} \min\{\max\{0, 1 - \frac{L_{1}(T)}{\sigma_{1}}\}, 1 - \frac{x}{K} - \frac{y}{Q} \} - \frac{\eta(Q)xy}{a + \eta(Q)x} \]
\[ \geq - \frac{\eta(Q)xy}{a + \eta(Q)x} \]
\[ \geq - \frac{\hat{y}}{a} x \equiv q_{1}x, \]

where \( q_{1} \) is a constant. Thus, \( x(t_{1}) \geq x(0)e^{q_{1}t_{1}} > 0 \) holds, which contradicts with \( x(t_{1}) = 0 \). Therefore, \( S(t) \) cannot reach this boundary.

Case 2. \( y(t_{1}) = 0 \). Let \( \hat{x} = \max_{Q(0,1)}(x) \leq k = \min\{K, \frac{P}{Q} \} \).
\[ \forall \in [0, t_{1}] \],
\[ \frac{dy}{dt} = -\eta(Q)xy - \left( h_{2} \left( 1 - A_{1} + A_{2}L_{1}(\bar{T})\frac{\eta(Q)xy}{a + \eta(Q)x} \right) + m_{2} \right) y \]
\[ \geq - \left( h_{2} \left( 1 - A_{1} + A_{2}(a_{1} + a_{2})\frac{\eta(Q)xy}{a + \eta(Q)x} \right) + m_{2} \right) y \equiv q_{2}y, \]

where \( q_{2} \) is a constant. Thus, \( y(t_{1}) \geq y(0)e^{q_{2}t_{1}} > 0 \) holds, which contradicts with \( y(t_{1}) = 0 \). Therefore, \( S(t) \) cannot reach this boundary.

Case 3. \( x(t_{1}) = k \). Let
\[ \frac{dx}{dt} = a_{1} \min\{\max\{0, 1 - \frac{L_{1}(T)}{\sigma_{1}}\}, 1 - \frac{x}{K} - \frac{y}{Q} \} - \frac{\eta(Q)xy}{a + \eta(Q)x} \]
\[ \text{(3.7)} \]

Therefore, \( S(t) \) cannot cross this boundary.

Case 4. \( qx(t_{1}) + \theta y(t_{1}) = P \), \( \frac{Q(t)}{\theta} = q \). It follows that
\[ a_{1} \min\{\max\{0, 1 - \frac{L_{1}(T)}{\sigma_{1}}\}, 1 - \frac{x}{K} - \frac{y}{Q} \} = 0. \]

Since \( \beta_{1} < 1 \),
\[ \frac{dz}{dt} = \left. \frac{dx(t_{1})}{dt} + \frac{dy(t_{1})}{dt} \right|_{t=t_{1}} \]
\[ \leq q_{2}x_{1} \equiv q_{3}x_{1}, \]
\[ \leq - \frac{\eta(Q)xy}{a + \eta(Q)x} + \frac{\beta_{1}Q(t)}{\theta} \eta(Q)xy \]
\[ \leq - \frac{\eta(Q)xy}{a + \eta(Q)x} + \frac{\eta(Q)xy}{a + \eta(Q)x} < 0. \]

This implies that \( S(t) \) cannot cross this boundary.

In summary, the solution \( S(t) \) of system (3.5) starting from \( \Omega \) will stay in \( \Omega \) for all forward time. This completes the proof. \( \square \)
3.1.3. Equilibrium analysis

In this section, we analyze the stability of equilibria to investigate the long-term behavior of the system. To simplify the analysis, we rewrite (3.5) as follows:

\[
\frac{dx}{dt} = xF_1(x, y), \\
\frac{dy}{dt} = yG_1(x, y),
\]

where

\[
F_1(x, y) = a_1 \min \left\{ \max \left\{ 0, 1 - \frac{L_1(Q)T}{a_1^2} \right\}, 1 - \frac{x}{K}, 1 - \frac{q}{Q} \right\} - \frac{\eta(Q)y}{a + \eta(Q)x},
\]

\[
G_1(x, y) = b_1 \min \left\{ \max \left\{ 0, A_1 - A_2 L_1(Q) \right\}, \frac{\eta(Q)y}{a + \eta(Q)x} \right\} - \left( \frac{h_2}{1 - A_1 + A_2 L_1(Q)} \right) \left( \frac{\eta(Q)x}{a + \eta(Q)x} + m_2 \right).
\]

For the local stability of equilibria, we apply the method of the Jacobian matrix (Loladze et al., 2000), where

\[
J(x, y) = \begin{pmatrix}
F_1(x, y) & x \cdot F_1(x, y) \\
G_1(x, y) & G_1(x, y)
\end{pmatrix}.
\]

Theorem 3.2. The extinction equilibrium \( E_0(0, 0) \) is unstable.

Proof. At \( E_0(0, 0) \), the Jacobian matrix is given by

\[
J(E_0) = \begin{pmatrix}
F_1(0, 0) & 0 \\
0 & \begin{pmatrix} h_2 & 0 \\
0 & m_2 \end{pmatrix}
\end{pmatrix},
\]

where

\[
F_1(0, 0) = \begin{pmatrix}
a_1 \max \left\{ 0, 1 - \frac{a_1 T}{a_1^2} \right\} & a_2 = 0, \\
a_1 > 0, & a_2 \neq 0.
\end{pmatrix}
\]

Since the eigenvalues have different signs, \( E_0 \) is unstable.

Biologically, this implies that this ecosystem will never collapse completely.

Theorem 3.3. The producer-only equilibrium \( E_1(k, 0) \) is locally asymptotically stable (LAS) if \( G_1(k, 0) < 0 \); otherwise, it is unstable.

Proof. At \( E_1(k, 0) \), the Jacobian matrix is given by

\[
J(E_1) = \begin{pmatrix}
kF_1(k, 0) & kF_1(k, 0) \\
0 & G_1(k, 0)
\end{pmatrix}.
\]

It is easy to calculate that \( F_1(k, 0) < 0 \), therefore, if \( G_1(k, 0) > 0 \), then \( E_1 \) is unstable; otherwise, \( E_1 \) is LAS.

This indicates that when the growth rate of grazers is less than their death rate, grazers will die out and producers will stabilize at \( k = \min[K, P/q] \) eventually.

3.2. The NM model

Through similar rescaling and letting \( \varepsilon \to 0 \), we obtain a quasi-steady system with

\[
u = \frac{a_1 T}{a_1^2}, \quad \nu = \frac{T}{a_1^2} \left( \frac{h_1 + a_1 h_2}{a_2} \right) \frac{x}{a + x}.
\]

(3.7)

Let \( B_2 = \max \{0, 1 - \frac{a_1 T}{a_1^2} \} \), \( B_3 = A_3 a_1 \), where \( A_3 \) is given by (3.4). The degenerated two-dimensional equation is then given by

\[
\frac{dx}{dt} = a_1 \min \left\{ B_1, 1 - \frac{x}{K}, 1 - \frac{q}{Q} \right\} - \frac{\nu y}{a + \nu x},
\]

\[
\frac{dy}{dt} = b_1 \min \left\{ \max \left\{ 0, A_1 - B_3 \right\}, \frac{\nu y}{a + \nu x} \right\} - \left( \frac{h_2}{1 - A_1 + B_3} \right) \left( \frac{\nu x}{a + \nu x} + m_2 \right).
\]

Using a similar argument, it can be shown that \( \Omega \) remains the invariant set of (3.8), and the stability analysis of boundary equilibria for the system (3.8) can also be determined by Theorems 3.2 and 3.3. Detailed proofs will not be provided here. We now proceed to determine the stability of internal equilibria.

Rewrite (3.8) as

\[
\frac{dx}{dt} = xF_2(x, y), \\
\frac{dy}{dt} = yG_2(x, y).
\]

Here,

\[
F_2(x, y) = a_1 \min \left\{ B_1, 1 - \frac{x}{K}, 1 - \frac{q}{Q} \right\} - \frac{\nu y}{a + \nu x},
\]

\[
G_2(x, y) = b_1 \min \left\{ \max \left\{ 0, A_1 - B_3 \right\}, \frac{\nu y}{a + \nu x} \right\} - \left( \frac{h_2}{1 - A_1 + B_3} \right) \left( \frac{\nu x}{a + \nu x} + m_2 \right).
\]

If \( A_1 < B_2 \),

\[
\begin{align*}
& x < \frac{\nu y}{a + \nu x}, \\
& y > \frac{\nu x}{a + \nu x}.
\end{align*}
\]

If \( A_1 \geq B_2 \),

\[
\begin{align*}
& \left( \frac{h_2}{1 - A_1 + B_3} \right) \left( \frac{\nu x}{a + \nu x} + m_2 \right) \leq \frac{\nu y}{a + \nu x}, \\
& x > \frac{\nu y}{a + \nu x}.
\end{align*}
\]

The derivatives of \( F_2(x, y) \) and \( G_2(x, y) \) are given as follows:

\[
F_2(x, y) = \begin{cases}
\frac{\nu y}{a + \nu x} > 0, & x < K(1 - B_1), y > (1 - B_1)P, \\
\frac{\nu y}{a + \nu x} < 0, & x > K(1 - B_1), \frac{\nu y}{a + \nu x} \leq \frac{\nu x}{a + \nu x}, \\
\frac{\nu y}{a + \nu x} \geq 0, & \frac{\nu y}{a + \nu x} \leq \frac{\nu x}{a + \nu x},
\end{cases}
\]

\[
G_2(x, y) = \begin{cases}
\frac{\nu y}{a + \nu x} > 0, & x < K(1 - B_1), y > (1 - B_1)P, \\
\frac{\nu y}{a + \nu x} < 0, & x > K(1 - B_1), \frac{\nu y}{a + \nu x} \leq \frac{\nu x}{a + \nu x}, \\
\frac{\nu y}{a + \nu x} \leq 0, & \frac{\nu y}{a + \nu x} \leq \frac{\nu x}{a + \nu x},
\end{cases}
\]

If \( A_1 < B_2 \), we have

\[
\begin{align*}
& -h_2 B_3 \frac{\frac{\nu y}{a + \nu x}}{a + \nu x} < 0, \\
& x > \frac{\nu y}{a + \nu x},
\end{align*}
\]

\[
\begin{align*}
& -B_3 \frac{\nu y}{a + \nu x} + \left( \frac{h_3}{1 - A_1 + B_3} \right) \frac{\nu y}{a + \nu x} \leq \frac{\nu x}{a + \nu x}, \\
& x < \frac{\nu y}{a + \nu x},
\end{align*}
\]

\[
\begin{align*}
& -h_2 B_3 \frac{\nu y}{a + \nu x} \leq \frac{\nu x}{a + \nu x} < 0, \\
& x < \frac{\nu y}{a + \nu x}.
\end{align*}
\]
Consider the case when
\[ \text{If } A_1 \geq B_2, \text{ it follows} \]
\[ G_{x_2} = \begin{cases} 
-\frac{\beta B_2 x_1^2}{a(x+y)^2} + (\beta A_1 - h_2 B_2) - a \frac{a}{(a+x)^3} \bigg( A_1 - B_2 \frac{x}{a+x} \bigg) x + y < \frac{p}{q} \bigg) \quad x + y > \frac{p}{q} 
\end{cases} \]

\[ G_{y_2} = \begin{cases} 
-\frac{\beta B_2 y_1^2}{a(x+y)^2} + (\beta A_1 - h_2 B_2) - a \frac{a}{(a+y)^3} \bigg( A_1 - B_2 \frac{x}{a+x} \bigg) x + y < \frac{p}{q} \bigg) \quad x + y > \frac{p}{q} 
\end{cases} \]

Consider the case when
\[ G_{x_1} = \begin{cases} 
-\frac{\beta B_2 x_1^2}{a(x+y)^2} + (\beta A_1 - h_2 B_2) - a \frac{a}{(a+x)^3} \bigg( A_1 - B_2 \frac{x}{a+x} \bigg) x + y < \frac{p}{q} \bigg) \quad x + y > \frac{p}{q} 
\end{cases} \]

Let \( \hat{\beta} = \left( \frac{\beta A_1 - h_2 B_2}{a} \right) \).

If \( \hat{\beta} A_1 < h_2 B_2 \), then \( A_1 - 2 \hat{\beta} B_2 - h_2 B_2 < 0 \) and \( G_{x_2} < 0, \forall (x,y) \in \Omega \).

If \( \hat{\beta} A_1 > 2 \hat{\beta} B_2 + h_2 B_2 \), then \( G_{y_2} > 0, \forall (x,y) \in \Omega \).

If \( h_2 B_2 < \hat{\beta} A_1 < 2 \hat{\beta} B_2 + h_2 B_2 \), then \( G_{y_2} > 0, \forall 0 < x < \hat{x}, \) and \( G_{x_2} < 0, \forall y > \hat{x} \).

Theorem 3.4. The stability of internal equilibrium \( E^* (x^*, y^*) \) follows that

I When \( x^* > \frac{aA_1}{B_1 - A_1} \) with \( A_1 < B_2 \), \( E^* \) is a saddle.

II When \( x^* < \frac{aA_1}{B_1 - A_1} \) and \( (A_1 - B_2 \frac{x^*}{a+x})x + y^* < \frac{p}{q} \) with \( A_1 < B_2 \); or \( (A_1 - B_2 \frac{x^*}{a+x})x + y^* > \frac{p}{q} \) with \( A_1 > B_2 \).

i If \( \beta A_1 < h_2 B_2 \), \( E^* \) is a saddle.

ii If \( \beta A_1 \gtrless 2 \hat{\beta} B_2 + h_2 B_2 \), \( E^* \) is LAS if the producer nullcline is decreasing at \( E^* \); otherwise, \( E^* \) is unstable.

iii If \( h_2 B_2 < \beta A_1 < 2 \hat{\beta} B_2 + h_2 B_2 \), \( \forall 0 < x^* < \hat{x} \), \( E^* \) is LAS if the producer nullcline is at \( E^* \); otherwise, \( E^* \) is unstable.

Therefore, at \( E^* \), if the slope of the grazer nullcline is less than the slope of the producer nullcline, \( i.e., \frac{G_{x_1}}{G_{y_1}} < \frac{G_{x_2}}{G_{y_2}} \), then \( \text{Det}(J_{E^*}) < 0 \) and \( E^* \) is a saddle. Otherwise, the slope of grazers is higher, \( i.e., \frac{G_{x_1}}{G_{y_1}} > \frac{G_{x_2}}{G_{y_2}} \), then \( \text{Det}(J_{E^*}) > 0 \), \( F_{x_2} < 0 \), and \( \text{Tr}(J_{E^*}) < 0 \). Hence, \( E^* \) is LAS.

Two examples are provided to show the application of Theorem 3.4.

For the first example, we choose \( \beta_1 = 9.8, \beta_2 = 20, h_1 = 0.4, m_1 = 0.25, a_2 = 0.192, \sigma_1 = 0.001, \sigma_2 = 0.4, a = 0.25, T = 0.01, P = 0.05, \) and \( K = 1.85 \). Rest parameters are shown in Table 2.1. It is easy to calculate that \( A_1 = 0.9985 < B_2 = 1.7285 \). There are two internal equilibria denoted as \( E_1 \) and \( E_2 \), as shown in Fig. 1(a). \( E_2 \) satisfies that \( x_2 < \frac{aA_1}{B_1 - A_1} \) and \( (A_1 - B_2 \frac{x^*}{a+x})x + y^* < \frac{p}{q} \). Furthermore, \( \beta_1 A_1 = 6.6149 > 2 \hat{\beta} B_2 + h_2 B_2 = 0.2292 \). Therefore, as producer nullcline is increasing, based on Theorem 3.4 Case II, \( E_2 \) is unstable. \( E_2 \) satisfies \( x_2 < \frac{aA_1}{B_1 - A_1} \). Based on Theorem 3.4 case I, \( E_1 \) is a saddle. These conclusions are further approved by phase portrait in Fig. 1(b).

Another example is given as \( h_1 = 0.4, m_1 = 0.25, \sigma_1 = 0.5, \sigma_2 = 0.4, a = 0.25, T = 0.01, P = 0.05 \) and \( K = 1.85 \). In this case, there are three internal equilibria denoted as \( E_3, E_1 \), and \( E_2 \), as shown in Fig. 2(a). \( E_2 \) satisfies that \( (A_1 - B_2 \frac{x^*}{a+x})x + y^* < \frac{p}{q} \). Meanwhile, \( A_1 = 0.6229 > B_2 = 0.0017 \) and \( \beta_1 A_1 = 3.3633 > 2 \hat{\beta} B_2 + h_2 B_2 = 0.0019 \) hold. The producer nullcline is increasing at \( E_2 \). According to Theorem 3.4 case II, \( E_2 \) is unstable. On the other hand, \( E_1 \) and \( E_2 \) satisfies \( (A_1 - B_2 \frac{x^*}{a+x})x + y^* > \frac{p}{q} \). At \( E_1 \) the slope of the producer nullcline is higher, while at \( E_2 \), the slope of the grazer is higher. By Theorem 3.4 case III, \( E_1 \) is a saddle and \( E_2 \) is LAS. These results align with simulation results in Fig. 2(b).

4. Numerical analysis

In this section, we employ numerical simulations to visually explore the impact of microplastics on aquatic population dynamics in both natural and laboratory settings separately.

4.1. The OMUF model

Here, we investigate the influence of microplastics using the OMUF model (3.5) and compare the differences between them under natural and laboratory settings. We set \( a = 0.0012, m_1 = 0.025, P = 0.03, \) and \( K = 2.5 \) for all simulations. Bifurcation analysis is employed. Fig. 3(a) illustrates the bifurcation diagram over \( T \) for the system (3.5), where
Fig. 1. (a) Nullclines of producers ($x$) and grazers ($y$) for system (3.8) for example 1. The red parabola-shaped curve is $x$-nullcline; the blue straight lines are $y$-nullclines. Two unstable equilibria $E_2$ and $E_3$ are highlighted as circles. (b) Corresponding phase portrait of system (3.8). $E_2$ is unstable and $E_3$ is a saddle.

Fig. 2. (a) Nullclines of producers ($x$) and grazers ($y$) in system (3.8) for example 2. The $x$-nullcline is denoted by a red parabolic curve, and the $y$-nullclines are denoted by blue peaked lines. Unstable equilibria are marked with circles, and the stable equilibria are denoted by dots. (b) The corresponding phase portrait of system (3.8). $E_2$ is unstable, $E_3$ is a saddle point, and $E_4$ is stable.

Fig. 3. Bifurcation diagram over varying microplastic concentration $T$. (a) OMUF model (3.5). (b) NM model (2.11).
Fig. 4. Nullclines for system (3.5) under different microplastic concentration. (a) $T = 0.0001$, (b) $T = 0.0005$, (c) $T = 0.001$, (d) $T = 0.0025$. Red dashed curves represent the x-nullcline, while blue solid lines represent the y-nullclines. Unstable equilibria are marked with circles, and the stable equilibria are denoted by dots. Here we take $P = 0.03$, $K = 2.5$.

$T$ represents the total microplastic concentration in the environment. Additionally, Fig. 4 displays the nullclines of producers and grazers.

The system (3.5) exhibits complicated dynamics. It admits a critical threshold at $T = 0.00165$, as illustrated in Fig. 3(a). When the microplastic concentration is higher than 0.000165, grazers go to extinction while producers stabilize at maximum carrying capacity $K$ eventually, e.g., $T = 0.0025$ in Fig. 5(d). This aligns with that the producer-only equilibrium is the only stable equilibrium, as shown in Fig. 4(d).

However, when the microplastic concentration is below this threshold, the system displays diverse stability patterns. Specifically, when $T < 0.00015$, the system shows bistability. As demonstrated in 4(a) and Fig. 5(a), when $T = 0.0001$, there are five intersection points of the x-nullclines and y-nullclines, two of which are stable (denoted as black dots), while the remaining three are unstable (denoted as circles). In this case, the state of the system is highly dependent on the initial state. When $0.00015 < T < 0.00081$, the system (3.5) displays three stable states simultaneously. This is further supported by Figs. 4(b) and 5(b), when $T = 0.0005$, eight equilibria are present, with three of them being stable. Further increasing the microplastic concentration in the environment, the system still exhibits three stable states, while one of them is the producer-only state. An example is given when $T = 0.001$ in Figs. 4(c) and 5(c). This implies that if the initial grazer density is too low, although plenty of food is provided, grazers will still go to extinction. When $0.0015 < T < 0.00165$, the system again shows bistability with one stable boundary equilibrium.

The tolerance of the producer-grazer system described in (3.5) to microplastics is significantly lower, compared to that of the system defined in (2.11), as demonstrated in Fig. 3. This implies that producers and grazers are more sensitive to microplastics in natural environments. This heightened sensitivity can be attributed to various challenges such as food scarcity, predation risk, exposure to multiple toxins, and human disruptions. All of these challenges increase the predation difficulty and vulnerability of grazers. Interestingly, under natural conditions for grazers, species tend to exhibit multiple stability, while at most one stable state is observed for the NM model.

4.2. The NM model

In this section, we try to investigate the effect of light, microplastics, and nutrients on the population dynamics respectively through NM model (2.11). For a simple Holling type II functional response, we take $c = 0.81$, $a = 0.25$, $m_2 = 0.25$ in this section (Anderson et al., 2015). Fig. 7 shows the comparison between two different total phosphorus $P = 0.023$ and $P = 0.05$. $K$ implies the light intensity, which influences the quality and quantity of food for grazers. Fig. 7(a) and 7(b) illustrate how densities of producers and grazers vary when the light intensity and microplastic concentration vary. Figs. 7(c) and 7(d) show the corresponding two parameters bifurcation diagrams. In Region II (black), the system admits a limit cycle; in Region I (red), the system admits a stable coexistence state; in Region III (blue), the system admits a producer-only state. Figs. 7(e) and 7(f) show the bifurcation diagrams for grazer production efficiency function, i.e. $e(v)$, illustrating the main limitation for their growth. Red dots indicate the nutrient is the main limitation; Blue dots indicate the microplastics are the main limitation for grazers. Figs. 7(g) and 7(h) shows the body burdens for $P = 0.023$ and $P = 0.05$, respectively.
4.2.1. Influence of light intensity

Referring to Figs. 7(c) and 7(d), we observe that grazers cannot survive when the light intensity is either too low or too high. Optimal survival conditions occur within a moderate range of light intensity, benefiting both producers and grazers. To gain deeper insights into how light intensity influences population dynamics, we employ bifurcation analysis over the parameter $K$. Fig. 6 provides an example when $P = 0.023$, $T = 0.05$. The corresponding time series of the solution is presented in Fig. 8.

When the light intensity is extremely low ($K < 0.268$), producer growth is severely constrained due to insufficient photosynthesis, resulting in low producer density. Consequently, grazers face extinction due to food scarcity. An example with $K = 0.1$ is illustrated in Fig. 8(a). Within the moderate range of light intensity ($0.268 < K < 1.296$), producers and grazers find a balance for coexistence. Specifically, when $0.268 < K < 0.596$, the system maintains a stable coexistence state, exemplified by $K = 0.4$ in Fig. 8(b). At $K = 0.596$, a Hopf bifurcation occurs. With further increases in light intensity, the densities of producers and grazers oscillate periodically, as demonstrated by $K = 0.8$.
Fig. 7. (a)(b) 3D phase portraits of (2.11). (c)(d) Bifurcation diagrams for light intensity $K$ and microplastic concentration $T$. (e)(f) Main limitation on grazer growth rate. (g)(h) Microplastic body burdens for producers and grazers. For the panels on the left, we choose $P = 0.023$, and for the panels on the right, we choose $P = 0.05$. 
in Fig. 8(c). At $K = 0.856$, another Hopf bifurcation happens, and the system transitions to a stable state again, e.g., $K = 1$ in Fig. 8(d). Higher light intensity typically indicates stronger photosynthesis, which promotes producer growth. However, with an abundance of carbon, the P:C ratio in producers decreases, indicating a decline in food quality for grazers. When light intensity becomes excessively strong, the low-quality food becomes insufficient to support the survival of grazers, ultimately leading to their extinction. This scenario is depicted in Fig. 8(e) with $K = 2$.

### 4.2.3. Influence of nutrient

From Fig. 7(a)–7(d), it is evident that when the concentration of microplastics in the environment becomes excessively high ($T > 1.8$), grazers are unable to survive. Fig. 8(f) provides an example when $T = 2.5$ for the case $P = 0.23$. However, when the density of microplastics remains within a reasonable range, both producers and grazers have an opportunity to coexist. We conduct a bifurcation analysis over $T$, using moderate light intensity ($K = 0.7$) as an example, as illustrated in Fig. 3(b). When microplastic concentration is low ($T < 0.57$), the system (2.11) admits a limit cycle. Further increasing microplastic concentration, producers and grazers can coexist stably.

Low light intensity and a low nutrient level naturally limit the growth of producers and grazers, even without the presence of microplastics. However, the existence of microplastics amplifies this limitation imposed by light intensity and nutrient availability. The upper boundary of the top region II decreases, and the lower boundary of the bottom region II increases when parameter $T$ rises, as shown in Figs. 7(c) and 7(d). This indicates that as the concentration of microplastics in the environment increases, the system exhibits reduced resistance to low light intensity and nutrient-poor conditions. For example, consider the case of $P = 0.023$. When $T = 0$ (no microplastics), a relatively low light intensity of $K = 0.185$ is enough to support the survival of grazers, as shown in Fig. 7(c). However, when $T = 0.5$, the producer-grazer system requires a higher minimum light intensity of $K = 0.26$ to maintain coexistence, as depicted in Fig. 6(b).

High levels of microplastics in the environment do not always correspond to high body burdens in organisms. As seen in Fig. 7(g) and 7(h), in general, an increase in the toxin concentration in the environment leads to higher body burdens in both producers and grazers. However, when food nutrients are abundant, and light intensity is moderate, the body burdens remain at a low level even with increasing levels of microplastics in the environment. This observation potentially explains why, in most experiments, the inhibition effects on grazer growth show a positive correlation with the total abundance of microplastics in the environment, whereas in some experiments, their relationship is not strictly positive (Canniff and Hoang, 2018).

Grazers exhibit greater sensitivity to microplastics, compared to producers. As depicted in Figs. 7(g) and 7(h), the body burdens in producers consistently remain at relatively low levels when light intensity and microplastic concentration vary, whereas the body burdens in grazers can reach significantly higher values. When the body burdens in grazers become excessively high, they face the risk of extinction, as illustrated in Fig. 8(a), Fig. 8(e), Fig. 8(f), Fig. 9(a), Fig. 9(e), and Fig. 9(f). Moreover, the stability of producers and grazers is significantly influenced by their body burdens. For instance, the densities of producers and grazers remain stable when their body burdens exhibit stability, as observed in Fig. 8(b), Fig. 8(d), Fig. 9(b), and Fig. 9(d). Conversely, when their body burdens oscillate periodically, both producers and grazers experience oscillations in their populations, as exemplified in Figs. 8(c) and 9(c).

In the case where producers can utilize microplastics for their growth, we set $a_2 = -0.09$, $P = 0.05$, and $a_1 = 0.6$. This results in a coexistence area that is more than three times larger than that shown in Fig. 7(d), as illustrated in Fig. 10. This implies that the producer-grazer system shows higher resistance to microplastics. In particular, the maximum microplastic concentration that allows the survival of both producers and grazers is 4.5, which is significantly higher than that shown in Fig. 7(d).

### 4.2.4. Influence of nutrient

As the total phosphorus concentration in the environment (i.e., $P$) increases, the producer-grazer system exhibits greater resistance to intense light conditions. For instance, when $P = 0.023$, the maximum light intensity that can be utilized to sustain the coexistence of both
Fig. 9. Time series of microplastic body burdens of producers and grazers in system (2.11) with $P = 0.023$.

Fig. 10. Two-parameter bifurcation diagram for varying light level $K$ and microplastics level $T$ for system (2.11). Here we take $\sigma_1 = 0.6$, $\alpha_2 = -0.09$.

producers and grazers is 1.66, as illustrated in Fig. 7(c). However, when $P = 0.05$, this maximum light intensity increases to 4.05, as shown in Fig. 7(d). This phenomenon occurs because higher light intensity typically leads to increased producer density. With more carbon available, the producer’s P:C ratio decreases, meaning that the nutrient level in producers decreases. When the nutrient level falls below the grazers’ demand threshold, grazers go extinct. When the total phosphorus concentration in the environment is low, even moderate light intensity can lead to low nutrient levels in producers, making it difficult for grazers to survive. However, as the total phosphorus concentration in the environment increases, high light intensity can still ensure that the nutrient levels in producers remain at a moderate level, which is sufficient to support the survival of grazers.

Grazers can survive only when their body burdens of microplastics are relatively low as discussed in Section 4.2.2. However, the main limitation for the growth of grazers is not always the same. When the light intensity is low, the nutrient level in producers is sufficient, their microplastic body burdens become the primary limiting factor. As light intensity increases, the nutrient level in producers decreases. Consequently, the primary limitation to grazer growth transitions to inadequate nutrients gradually, as shown in Figs. 7(e) and 7(f).

5. Discussion

Microplastics can be mistakenly ingested or adhere to the surfaces of marine organisms, resulting in significant adverse effects (Zhang et al., 2017; Liu et al., 2019; Sazli et al., 2023; Gregory, 1996; Derraik, 2002; Blarer and Burkhardt-Holm, 2016; Alomar et al., 2017). This paper presents two stoichiometric models to investigate population dynamics in the presence of microplastics in both field and laboratory settings. The interactive effects of light, nutrients, and microplastics on population dynamics have been rigorously studied.

For natural ecosystems, the OMUF model reveals complex dynamics. When the microplastic concentration is relatively low, the behavior of the system is strongly influenced by the initial conditions and can exhibit bistability or tristability. Conversely, when microplastics in the environment become excessively abundant, the only outcome is the extinction of grazers. Furthermore, the tolerance to microplastics of the producer-grazer system described in OMUF model (3.5) is significantly lower, compared to that of the system defined in NM model (2.11). This discrepancy potentially arises from the fact that in the natural ecosystem, grazers have to face more challenges and pay significant feeding costs, such as food scarcity, nutrient deficiencies, predation risk, multiple toxins, and human impact, making them more susceptible to microplastics.

Extreme light condition, either too low or too high, restricts the growth of grazers. When light intensity is excessively low, producers can only survive at low densities due to weakened photosynthesis, and grazers go extinct because of insufficient food. When light intensity is excessively high, the P:C ratio in producers becomes very low, and grazers go extinct due to nutrient deficiency. However, when light intensity falls within a moderate range, coexistence of both species may occur, and the system demonstrates a greater ability to withstand microplastics.

The populations of producers and grazers are highly impacted by the concentration of microplastics. Grazers prove to be more vulnerable to microplastics compared to producers. High microplastic concentrations lead to the extinction of grazers, while producers can persist even in...
the presence of extremely high microplastic levels. This aligns with the stability analysis in Theorem 3.2 that the system will never fully collapse. In cases where producers can utilize microplastics for growth, the system shows much greater resilience to microplastics.

Intuitively, high environmental microplastic levels imply elevated microplastic body burdens in organisms, but this is not always true. In most situations, higher environmental microplastic concentrations result in increased body burdens for producers and grazers. However, when nutrients are plentiful and light is moderate, their body burdens remain low despite increasing environmental microplastics. This may explain why, in some experiments, the mortality rate or growth inhibition is not positively correlated with microplastic concentration (Canniff and Hoang, 2018).

The influence of light intensity, nutrients, and microplastics on population dynamics is highly intertwined. When light intensity is relatively low, the primary limitation on the growth of grazers is microplastic body burdens, while with high light intensity, their growth is primarily constrained by food quality. Increasing phosphorus concentration in the environment enhances the resistance of the producer-grazer system to intense light conditions. Conversely, the presence of microplastics amplifies the constraints on grazer growth related to food quality and quantity imposed by excessively low or high light intensities. Moreover, as the concentration of microplastics in the environment rises, the system demonstrates decreased resilience to food and nutrient discrepancy conditions.

The models developed here primarily focus on producer-grazer dynamics. However, the transfer of microplastics through multiple trophic interactions can significantly affect the trophic cascade strength and stability of plankton ecosystems (Pan et al., 2022; Lu et al., 2016). Considering a higher-dimensional food chain model in the future could provide further insights. Additionally, beyond their impact on population growth, microplastics can also influence the behavior and personality of individual organisms (Bhuyan, 2022; Chen et al., 2022), such as the boldness and shyness of fish. Investigating these effects on the personality of organisms could be an intriguing avenue for future research. Furthermore, the impact of microplastics is closely related to particle size (Wu et al., 2021; Gray and Weinstein, 2017). Organisms may show opposite responses to various sizes of microplastics, and the underlying mechanisms are complicated (Yokota et al., 2017; Mao et al., 2018; Jiao et al., 2022; Canniff and Hoang, 2018; Cui et al., 2017; Duis and Coors, 2016; Nolte et al., 2017; Zhang et al., 2017; Khoirioni et al., 2019; Sjollema et al., 2016; Besseling et al., 2014). Therefore, incorporating different microplastic sizes into future models is expected to be helpful for better understanding the mechanism of how microplastics influence aquatic ecosystem dynamics.

CRediT authorship contribution statement

Tianxu Wang: Formal analysis, Software, Validation, Visualization, Writing – original draft. Hao Wang: Conceptualization, Data curation, Funding acquisition, Investigation, Methodology, Project administration, Supervision, Validation, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

The research of Hao Wang was partially supported by the Natural Sciences and Engineering Research Council of Canada (Individual Discovery Grant RGPIN-2020-03911 and Discovery Accelerator Supplement Award RGPAS-2020-00090) and the Canada Research Chairs program (Tier 1 Canada Research Chair Award).

Appendix. Parameters

(a) \( a_1 \) is estimated by

\[
\begin{align*}
\alpha_1 (\text{mg C/mg M}) & = \text{Decrease of algae density (mg C/L)} \\
& \text{Microplastic concentration (mg M/L)}
\end{align*}
\]

The reduction in algae optical density (\( OD_{680} \)) ranges from 0.05 to 0.48 across various categories of 100 mg/L microplastics (Yang et al., 2020). The relationship between (mg C/L) and (\( OD_{680} \)) is expressed as follows (Lavrinič et al., 2021): (mg C/L) = 1000(0.4076 \times (OD_{680}) – 0.0052).

Therefore,

\[
\begin{align*}
\sigma_{\text{max}} &= \frac{1000(0.4076 \times 0.48 – 0.0052)}{100} = 1.9 \\
\sigma_{\text{min}} &= \frac{1000(0.4076 \times 0.05 – 0.0052)}{100} = 0.152
\end{align*}
\]

The parameter \( a_2 \) can also be deduced using the four-day algae density data in Zhang et al. (2017). Considering a cell density of skeletonema costatum at \( 10^{-8} \) mg/cell, \( a_2 \) is estimated as \( -0.0989 \) as in Fig. 11(a). Consequently, we consider \( a_2 \) to fall within the range of \(-0.0989 \) to \( 1.9 \).

(b) \( \beta_2 \) is estimated as

\[
\beta_2 (\text{mg C/mg M}) = \frac{\text{Decrease of neonate density (mg C/L)}}{\text{Microplastic concentration (mg M/L)}}
\]

The reduction in the count of neonates varies from 3 to 73 under diverse light conditions and microplastic concentrations (Guilhermino et al., 2021). The dry weight of the Daphnia neonate is estimated as 0.002 mg/individual. Then by fitting data, we obtain \( 8 < \beta_2 < 15.78 \), as shown in Fig. 11(b).

(c) \( h_2 \) is estimated as

\[
\frac{\text{Density of death neonate (mg C/L)}}{\text{Microplastic concentration (mg M/L)}} = \frac{\text{Experiment time (day)}}{\text{Density of death neonate (mg C/L)}} = \frac{\text{Microplastic concentration (mg M/L)}}{\text{Experiment time (day)}}
\]

Based on data in Guilhermino et al. (2021), \( 0.11 < h_2 < 0.29 \).

(d) \( a_1 \) is estimated by

\[
\frac{\text{Uptake microplastic concentration (mg C/L)}}{\text{Uptake microplastic concentration (mg C/L)}} = \frac{\text{Algae density (mg C/L)}}{\text{Experiment time (day)}}
\]

According to Bhattacharya et al. (2010), the uptake rate (adsorption and absorption capacities) for positively and negatively charged nanoparticles are approximately 7/8 and 3/8, respectively. Algae density is considered equivalent to microplastic concentration. This leads to a range of 0.0056 < \( a_1 < 0.0131 \).

(e) Assume the dry weight of one daphnia is 0.179 mg (Šimčič and Anton, 1997), then the density of 10 daphnia in 50 ml media is given by

\[
\frac{10 \times 0.178}{50 \times 10^{-3}} = 35.6 \text{ (mg C/L)}.
\]

\( b_1 \) is estimated as

\[
\frac{\text{Mean number particles counted inside Daphnia per day}}{\text{Mean number particles counted in Water per day}} = \frac{1}{\text{Density of Daphnia(mg C/L)}}.
\]

Utilizing the data from Elizalde-Velázquez et al. (2020), the values for \( b_1 \) are determined as follows:

For high concentration exposure: \( b_1 = 0.034 \times 10^{-6} = 0.00096 \).

For low concentration exposure: \( b_1 = 0.026 \times 10^{-6} = 0.00073 \).

Therefore, the range of \( b_1 \) is set to be 0.00073 to 0.00096.