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# MODELISATION ET ETUDE MATHEMATIQUE DE QUELQUES PROBLEMES DE DYNAMIQUE DES POPULATIONS

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*à mes parents*
*à mon frère et mes soeurs*
*à toute ma famille*

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# Introduction générale

## Cadre de l'étude

Dans cette thèse nous nous intéressons à l'élaboration et à l'étude mathématique de modèles provenant de problèmes écologiques.
Les travaux réalisés vont être présentés sous forme d'articles publiés ou soumis, chaque chapitre correspondra à un article. Avant de présenter ces travaux, nous voulons donner un aperçu sur la problématique en écologie dans laquelle nous nous situons.
Comprendre le fonctionnement d'un écosystème (Murray [82]) est un enjeu majeur pour la gestion des ressources et de l'environnement. Cependant ce but reste difficile à atteindre vue la complexité des systèmes naturels, en particulier dans le milieu aquatique où de très nombreux processus de toutes natures interagissent avec des organismes vivants.
Plusieurs questions intéressantes peuvent être posées à propos des écosystèmes (voir par exemple Pielou [86]), notamment :

1) Quels sont les facteurs qui influencent la stabilité d'un écosystème ?
2) Quels sont les facteurs controlant la variabilité des abondances de différentes composantes de l'écosystème et notamment sur sa structure. ?
3) Quel est l'impact de l'hétérogénéité spatiale sur les interactions entre populations et sur le comportement de l'écosystème ?.
   - Quelles sont les variations d'abondance dues aux changements naturels ou anthropiques du milieu ?.

Pour répondre à ces questions, différentes approches méthodologiques existent. On peut citer l'observation directe du milieu, l'expérimentation *in vitro* et *in situ*, la modélisation mathématique et informatique. Mon travail se situe dans cette dernière direction et consiste en l'élaboration et l'étude, essentiellement théorique, de modèles mathématiques.
La modélisation mathématique est avant tout, l'expression d'une démarche visant à expliquer des relations : dans des phénomènes mettant en jeu des relations entre les abondances de plusieurs populations, elle fournit un système théorique capable de combiner ces quantités suivant des mécanismes connus ou supposés.

Elle est en particulier utile pour faire le lien entre les abondances, les distributions, les fluctuations et la production des organismes vivants avec les variations de l'environnement abiotique.
Les modèles mathématiques intègrent pour résumer la dynamique de plusieurs espèces et celle du milieu dans une représentation des processus et de leur interactions (Wroblewski [107]).

Il y a plusieurs approches possibles dans la modélisation mathématique des phénomènes et des systèmes naturels.
L'approche la plus classique est celle qui conduit à ce que l'on appelle aujourd'hui des modèles minimaux. Dans un modèle minimal (Brauer *et col.* [20], Lagues et Lesne [72]) , on cherche à mettre l'accent sur un petit nombre de faits ou propriétés que l'on considère à la fois essentiels et suffisants. Ainsi, un modèle minimal de production du plancton pourrait prendre comme variables d'état les biomasses totales des composants autotrophes et hétérotrophes du plancton à tout instant. Très souvent, un modèle minimal sera de nature phénoménologique : les fonctions introduites dans les équations du modèle ne sont pas déduites de principes mais sont choisies pour leur ressemblance avec le phénomène réel modélisé.
A l'autre extrême, on peut citer les modèles centrés sur l'individu. Ces modèles cherchent à décrire les processus en partant de l'individu et en introduisant des paramètres biologiques, comportementaux propres à chaque individu. Ainsi, ils décrivent les variations des populations comme résultant des variations des individus. Ces modèles sont la plupart du temps si compliqués qu'une étude analytique est impossible et que seule une implémentation numérique peut leur être appliquée.
Il y a en fait une gamme presque continue de modélisations possibles entre les modèles minimaux et le tout -individu. Les modèles représentent un continuum de complexité depuis les courbes simples de réponse jusqu'aux enchaînements marins complexes des chaînes alimentaires aquatiques (Steele [93], Walsh [104], Kremer et Nixon [70]). Les connaissances actuelles sur les populations font que tout modèle comporte nécessairement un mélange de lois observées et quantifiées expérimentalement et de termes phénoménologiques. Ainsi par exemple les relations inter-individuelles sont souvent méconnues et ne peuvent être décrites que du point de vue phénoménologique.

Les modèles peuvent être á buts explicatifs ou prédictifs. Une première étape dans la modélisation est la définition des objectifs de l'étude. Ces objectifs vont déterminer non seulement la portée du modèle mais aussi le type de modèle à utiliser et le type de résultats qui seront recherchés. La construction même du modè le d'un système complexe consiste à identifier les composants "simples" du système et à décrire les interactions entre ces composants et les variables externes du système et entre les composants eux mêmes (Wroblewski [107]). Les modèles qui vont être considérés et étudiés dans ce travail sont explicatifs ; ils servent à tester différents scénarios et progressent dans la confrontation avec les données.

Nous nous plaçons dans la ligne des travaux d'écologie mathématique initiée dans les années 1920 par les travaux de Lotka et Volterra, travaux dans lesquels a été introduite la représentation des interactions entre espèces par des systèmes d'équations différentielles. Les modèles prédateur-proie élaborés par ces deux scientifiques séparément sont les modèles fondateurs de l'écologie moderne. L'objectif poursuivi par l'élaboration du modèle de Volterra était d'expliquer un phénomène très répandu dans les systèmes prédateur-proie réels, à savoir les oscillations avec décalage de phase des abondances des proies et des prédateurs.

# Les systémes prédateur-proie

## Modèle prédateur-proie de Lotka-Volterra

Le premier modèle de prédation fut proposé par les deux auteurs indépendamment : Lotka (1932) et Volterra (1926). Le modèle dit de Lotka-Volterra reçut par la suite des modifications et il est, encore aujourd'hui, à la base de nombreux travaux théoriques et appliqués. $N(t)$ et $P(t)$ désignant respectivement les abondances de proies et de prédateurs, le modèle de Lotka-Volterra est le système ordinaire à deux équations différentielles suivant :

$$\begin{cases} \frac{dN}{dt} = N(r - aP) \\ \frac{dP}{dt} = P(acN - d) \end{cases} \quad r, a, c, d > 0 \tag{1.1}$$

où $r$ est le taux de croissance de la population de proies en l'absence de prédateurs, $a$ est le taux d'attaque des prédateurs (ou nombre de proies consommées par prédateur par unité de temps), $c$ est le taux de conversion de la quantité de proies consommées en effectif de prédateurs, et $d$ est le taux de mortalité des prédateurs en l'abscence de proies.
En dehors des hypothèses d'homogénéité environnementale, de réduction de la population à une variable d'état non structurée en âge ou en taille, et de monophagie absolue du prédateur, ce modèle classique de prédation repose sur un certain nombre d'hypothèses simplificatrices : les processus de natalité et de mortalité des populations proie et prédateur respectivement, sont exponentiels, et la réponse fonctionnelle du prédateur est une fonction linéaire non limitée du nombre de proies (Czaran [32]).
Malgré la simplicité du système (1.1), l'analyse de stabilité de ce système par linéarisation au voisinage du point d'équilibre positif met en évidence l'existence d'oscillations déphasées et indique que les trajectoires dans le plan de phase sont des orbites fermées. Les populations de prédateurs et de proies exhibent des dynamiques périodiques entretenues (Figure 1.1). Les courbes sont obtenues pour différentes valeurs des données initiales $N(0)$ et $P(0)$.

## Formulation générale d'un système prédateur-proie

Depuis le modèle de Lotka-Volterra, de nombreuses études ont contribué à exprimer de différentes manières les taux de croissance des populations et leurs interactions. Les systèmes prédateur-proie ainsi générés exhibent des dynamiques très variées.
Soit la formulation très générale d'un système prédateur-proie (Yodzis [110])

$$\begin{cases} \frac{dN}{dt} = f(N) - PF(N, P) \\ \frac{dP}{dt} = PG(N, P) \end{cases} \tag{1.2}$$

où $N(t)$ et $P(t)$ désignent respectivement les densités de proies et de prédateurs à l'instant $t$.
Dans un système généralisé, trois fonctions sont à spécifier :

- $f(N)$ : le taux de croissance de la population proie en l'absence de prédateurs,
- $F(N, P)$ : la réponse fonctionnelle du prédateur, i.e. le nombre de proies consommées par unité de temps par un prédateur,
- $G(N, P)$ : la réponse numérique du prédateur décrivant la production de prédateurs, i.e. le taux de conversion de la proie en prédateur.

Les formes particulières choisies pour ces trois fonctions contiennent une quantité importante d'informations biologiques et sont déterminantes pour la dynamique du système étudié.

### Taux de croissance de la population proie

L'hypothèse de croissance malthusienne de la population de proies est une hypothèse non réaliste du modèle de Lotka-Volterra. Les modèles subséquents prédateur-proie font plutôt l'hypothèse d'une croissance logistique densité-dépendante (Verhulst [102]) :

$$f(N) = rN(1 - \frac{N}{K})$$

où $K$ représente la capacité de charge de l'habitat par rapport à la proie.

### Différentes formulations de la réponse fonctionnelle du prédateur

Différentes expressions relatives à la réponse fonctionnelle du prédateur sont présentées dans le Tableau 1. Un aspect biologique important de ces systèmes dynamiques est la manière dont les prédateurs interagissent entre eux.
Selon Holling, l'alimentation est composée de deux types d'activité : la recherche de proies et leur capture. L'auteur suppose (i) que le temps total dédié à l'alimentation est la somme du temps de recherche $t_r$ et du temps de capture $t_h$, et (ii) que le temps de capture de chaque proie $t_h$ est une constante. Soit $a$ le taux d'attaques réussies ; c'est le nombre de proies consommées par prédateur et par unité de temps de recherche. On peut écrire

$$F = \frac{\text{Nombre de proies consommées par prédateur}}{\text{Temps total d'alimentation}} = \frac{at_r}{t_r + at_r t_h}$$

La réponse fonctionnelle du prédateur peut donc finalement être exprimée sous la forme suivante (Yodzis [110], Begon *et col.* [18]) :

$$F = \frac{a}{1 + at_h}$$

où $a$ va être exprimé de plusieurs façons en fonction de $N$ mais aussi en fonction de $N$ et $P$.

La situation nommée "Laisser-faire" par Caughley et Lawton ([22]), cité par Yodzis ([110]) correspond à des prédateurs n'interférant pas entre eux dans leurs activités d'alimentation. Dans un tel système, ni la réponse fonctionnelle, ni la réponse numérique du prédateur ne dépend de la densité des prédateurs. Elles sont uniquement fonction de la densité des proies $N$ (modèles classiques). Les réponses fonctionnelles de Holling ([57], [58]) font partie de cette catégorie.

| Hypothèses | Taux d'attaque | Réponse fonctionnelle | Références |
|---|---|---|---|
| Pas d'interactions entre les prédateurs ("laisser-faire") | $a = bN$ | $F = \frac{bN}{1 + bt_hN}$ | type II, Holling 1959 |
| | $a = bN^2$ | $F = \frac{bN^2}{1 + bt_hN^2}$ | type III, Holling 1959 |
| Interférences entre les prédateurs | $a = \frac{QN^n}{P^m}$ | $F = \frac{t_h^{-1}N^n}{(Qt_h)^{-1}P^m + N^n}$ | Hassel et Varley 1969 |
| | $a = Q(\frac{N}{P_0 + P})$ | $F = \frac{t_h^{-1}N}{(Qt_h)^{-1}(P_0 + P) + N}$ | De Angelis *et col.* 1975 |
| | $a = Q(\frac{N}{N + P})$ | $F = \frac{Q(1 + t_h)^{-1}N}{(1 + t_h)^{-1}P + N}$ | Getz 1984 |
| Interférence et ratio dépendance | $a = Q(\frac{N}{P})$ | $F = \frac{QN}{P + Qt_hN}$ | Arditi et Ginzburg 1989 |

**Tableau 1**

L'hypothèse la plus simple pour la formulation du taux d'attaque des proies, et qui correspond à la réponse fonctionnelle de type II de Holling, est une simple proportionalité avec le nombre de proies présentes dans le milieu (Begon *et col.* [18]) : $a = bN$, avec $b$ une constante positive. En milieu marin, les copépodes ont un taux d'ingestion croissant en fonction de la concentration des nutriments (Frost [38]). La réponse fonctionnelle des copépodes a été généralement considédérée sous forme de Holling type II montrant un effet de saturation lorsque la concentration des nutriments est assez élevée (Holling [59], [60]). La forme de Holling type II est justifiée théoriquement comme une fonction de Michaelis-Menten.

Communément, la formulation d'Ivlev ([64]) a été utilisée pour décrire cette réponse fonctionnelle (Steele et Mullin [96]). La formulation d'Ivlev exige deux paramètres : le taux maximal d'ingestion $I_m$ et le taux de saturation $\alpha$. Ainsi, le taux d'ingestion est formulé comme suit

$$I = I_m(1 - e^{-\alpha C})$$

où $C$ est la concentration des nutriments.

La réponse fonctionnelle de type III correspondant à un taux d'attaque proportionnel á $N^2$ tend à représenter le fait que les prédateurs sont moins efficaces dans la capture des proies lorsque celles-ci sont en faible effectif (Yodzis [110]).
Une deuxième famille de modèles suppose l'existence d'une interférence entre les prédateurs, telle que la compétition trophique, la compétition reproductive (recherche de reproducteurs, de sites de ponte), la transmission de maladies, le cannibalisme, l'émigration densité-dépendante, le comportement territorial (Yodzis [110]). Dans cette catégorie, la réponse fonctionnelle la plus fréquente utilisée est peut être celle de Hassel et Varley ([51]). Elle implique que le taux d'attaque décroît lorsque $P$ augmente et que pour une densité donnée de proies $N$, plus le nombre de prédateurs $P$ est élevé, plus le taux de consommation $F$ par prédateur est faible (Tableau 1). Cette formulation très générale

de réponse fonctionnelle avec interférence permet son application à un grand nombre de systèmes prédateurs proie. La théorie de la ratio-dépendance qui a été proposée initialement par Arditi et Ginzburg ([9]) est un cas particulier de la réponse fonctionnelle de Hassel et Varley. Nous donnerons plus de détails sur la ratio-dépendance un peu plus loin dans cette introduction.

### Prise en compte d'autres processus

Plus récemment, d'autres mécanismes autres que la prédation, comme le mutualisme et la compétition, ont été également étudiés, nous citons ici quelques références dans ce contexte (Murray [82], Pielou [87], Yodzis [109]). D'une manière similaire et depuis le travail de Kermack et McKendrick sur le modèle SIRS pour des populations humaines (Kermack et McKendrick [67]), les modèles épidémiques en général ont attiré l'intérêt de la communauté scientifique, intérêt qui a été accentué ces vingt dernières années par l'apparition de nouvelles maladies ou de nouveaux phénomènes tels que l'invasion de certains biotopes par des espèces importées. Un exemple d'un tel phénomème est donné par l'algue verte *Caulerpa taxifolia* partie de Monaco au début des années 80 et qui tend à coloniser la méditerranée et à s'imposer sur d'autres espèces d'algues (Meinesz [79], Olsen *et col.* [84], Jousson *et col.* [65]). Le risque le plus important de cette invasion de la méditerranée est la modification des équilibres écologiques avec une diminution de la biodiversité et de l'écodiversité.
Plusieurs auteurs se sont intéressés aux modèles épidémiologiques, ainsi plusieurs propriétés de base de ce type de modèles sont actuellement bien connues (voir par exemple Thieme [98], Iannelli *et col.* [63], Woolhouse, Venturino, Tapaswi *et col.*, (voir [12])). Parmi les références pertinentes de travaux sur des modèles épidémiologiques nous mentionnons (Bailey [17], Anderson *et al.* [7], Roberts [92]). Cependant, peu de travaux ont été effectués sur les modèles éco-épidémiologiques même si des épidémies récentes ont amené la communauté scientifique à accorder à ces questions un intérêt croissant. Nous citons ici quelques articles dans cette direction (Hadeler, Freedman [45], Venturino [101], Chattopadhyay et Arino [23]).

En général, lorsque des espèces interagissent, la dynamique de chaque espèce est affectée par ces interactions. Il y a principalement trois types d'interactions (voir par exemple Murray [82], Combes, [26]) :

(i) *Compétition :* Lorsque les espèces interagissent et que cette interaction entraîne la décroissance du taux de reproduction ou du taux de croissance de chaque espèce (par exemple, lorsqu'il y a une concurrence sur la nouriture, l'espace, etc), on dit qu'on est en présence de compétition.

(ii) *Mutualisme ou symbiose :* Lorsque les interactions entre espèces entraînent une augmentation du taux de croissance ou de reproduction de chaque population et que chaque espèce tire profit de ces interactions. On peut citer par exemple le mutualisme dans certains cas du parasitisme. Les poissons lumineux de la famille des Gonostomidae sont parés d'un organe "lumineux". Des recherches récentes en biologie moléculaire ont montré que ce sont des bactéries qui émettent la lumière. Ces bactéries sont situées dans de petits organes, sous la peau, entre la nageoire pectorale et la nageoire pelvienne. Ainsi, des bactéries ont colonisé des cellules de l'hôte (poisson) et produisent une lumière dont le poisson tire profit, par exemple, lors de la reconnaissance des partenaires de sa propre espèce (voir l'article de Combes dans [27]).

(iii) *Prédation :* Lorsque le taux de croissance d'une ou de plusieurs espèces décroît tandis que le taux de croissance des autres espèces croît, on dit qu'on est en présence d'un système prédateur-proie.

Dans ce travail, nous étudions ce dernier type d'interaction. En fait, on peut distinguer quatre types de prédation biologique qui sont équivalents lorsque l'on cherche à les formuler mathématiquement (voir Renshaw [90]).

*Le parasitisme* : En fait, les parasites peuvent être symbiontes, neutres ou prédateurs. On peut citer le cas particulier des parasitoïdes. Ce sont des parasites qui pondent leurs oeufs sur ou à coté de leur hôte et ensuite l'hôte est mangé.

*Le cannibalisme* : C'est une forme particulière de prédation, il implique des membres d'une même espèce ; souvent ce sont les les adultes qui mangent les jeunes.

*Prédation des herbivores* : Lorsque les animaux se nourrissent de plantes, de leurs fruits ou de leurs graines. Et bien que les plantes survivent en général à leur consommation par les animaux, elles peuvent être endommagées ou dispersées.

*Prédation des carnivores* : C'est une prédation qui s'applique sur les herbivores ou les carnivores, c'est le comportement le plus commun dans la prédation.

## Le système phytoplancton-zooplancton

Le plancton est un ensemble d'organismes vivants, de nature végétale ou animale, n'ayant pas d'attaches directes avec le sol, et passant leur vie, entièrement ou partiellement, dans le milieu liquide, dans lequel ils flottent plus ou moins passivement.
Le zooplancton est constitué par l'ensemble des organismes hétérotrophes : incapables de synthétiser la matière organique, ils la "récoltent" dans le milieu extérieur à partir de la matière végétale. Le zooplancton est essentiellement représenté par les copépodes (jusqu'à 80%). La taille du zooplancton varie de quelques dizaines de microns (protozoaires) à un peu plus de 2 mm (macrozooplancton). Le zooplancton joue un rôle très important dans les réseaux trophiques non seulement parce qu'il représente une source de nourriture importante pour les poissons et pour les invertébrés prédateurs, mais aussi parce qu'il mange intensivement les algues, les bactéries, les protozoaires et autres invertébrés. Le zooplancton est très sensible aux variations de l'environnement et par conséquent les changements qui se produisent relativement à son abondance, à la diversité des espèces ou à la composition des communautés peuvent apporter des indications importantes sur les changements environnementaux ou sur des perturbations du milieu.
Les modèles du zooplancton sont construits pour au moins trois objectifs principaux :

**a)-** Pour estimer le flux d'énergie et de matière via une entité écologique bien définie. Cette entité peut être un organisme, une population ou la communauté zooplanctonique toute entière.

**b)-** Pour estimer la survie des individus et la persistence des populations dans leurs environnement physique et biologique, et pour la détermination des facteurs et des processus qui réglent

leur variabilité.

**c)-** Pour étudier différents aspects de l'écologie comportementale.

Le phytoplancton regroupe un ensemble d'organismes autotrophes photosynthétiques, capables de produire la matière organique à partir de l'eau, du $CO_2$, des nutriments minéraux et de l'énergie lumineuse.
Il est souvent unicellulaire, primitif et passe sa vie dans la couche de mélange c'est à dire dans la couche située entre la surface et la thermocline niveau où la température de l'eau chute brutalement. Le phytoplancton est à la base de toute la chaîne alimentaire aquatique et sa production détermine en quelque sorte la production entière du milieu marin.

Parmi les espèces phytoplanctoniques il en existe une bonne douzaine qui produisent des toxines. On peut citer quelques unes de ces espèces telles que *Dinoflagellés*, *Gymnodium breve*, *Dinophysis*, *Alexandrium*, *Pseudo-Nitzschia*, etc. Ces toxines peuvent avoir des conséquences néfastes tout le long de la chaine alimentaire passant par les crustacés, les poissons, les mammifères et même les humains. Plusieurs cas d'intoxication ont été observés un peu partout dans le monde et dans plusieurs cas, ce phénomène a coincidé avec les floraisons "*blooms*" de quelques espèces phytoplanctoniques toxiques. Plusieurs auteurs ont essayé d'expliquer le phénomène des *blooms* par différentes approches. On peut citer Edwards et Brindley ([34]) qui ont considéré la remontée des eaux froides (upwelling), Mattews et Brindley ([77]) qui ont travaillé sur la distribution spatiale et Pitchford et Brindley ([88]) qui ont pris en compte la diversité des espèces. Enfin, on peut citer Steele et Henderson ([95]), Edwards et Brindley ([34]) qui ont remarqué que le choix de la fonction de capture ainsi que la mortalité du zooplancton ont une influence majeure sur la dynamique du système. Notre contribution sur ce sujet a porté essentiellement sur le choix des réponses fonctionnelles. Dans les modèles qui vont être présentés dans les chapitres 3 et 4, nous considérons differents choix de la fonction de capture du prédateur, de la mortalité du zooplancton et de l'effet de la toxine. Ainsi la fonction de capture sera considérée sous forme linéaire ou sous forme de la fonction de Michaelis-Menten ou de Holling, type II (Holling [57], [58]), la mortalité sera linéaire ou quadratique et l'effet des substances toxiques sera considéré comme instantané ou retardé. Nous considérons des modèles à retard fini mais aussi des modèles à retard infini, des retards discrets ou distribués. L'intervention du retard dans l'effet des toxines peut s'interpréter de deux manières : dans le cas d'un retard discret, il faut qu'un certain temps se soit écoulé pour que les toxines arrivent à un organe vital, tandis que dans le cas d'un retard distribué, on suppose que l'effet de ces substances est cumulatif. Il faut qu'un certain seuil de consommation soit dépassé pour que l'action se manifeste.

Le retard infini représente dans notre étude l'influence de tout le passé d'une population sur les instants présents. L'introduction du retard infini dans les modèles qui seront présentés repose sur au moins deux faits. D'une part, nous ne connaissons pas la portée des instants passés sur l'évolution de la population et d'autre part, le fait de représenter le terme retard comme une convolution d'une certaine fonction, qu'on appelle noyau retard, et la fonction de consommation, nous permet d'examiner différentes portées du retard. Du point de vue mathématique, les modèles à retard infini conduisent, dans certains cas, à des équations différentielles ordinaires. En effet, ce dernier point est lié au choix de la forme du noyau retard qui est la fonction qui représente la façon avec laquelle influe le retard. Dans le cas de la libération des toxines par des espèces phytoplanctoniques toxiques, la forme de cette

libération demeure encore largement inconnue. Et aussi judicieux que puisse être son choix, il ne sera qu'une approximation de la réalité. Néanmoins, on peut trouver des formes qui possèdent quelques propriétés intéressantes. On peut citer par exemple la fonction gamma qui représente un bon choix dans le sens où une combinaison linéaire des fonctions gamma donne une classe générique de retards distribués (voir MacDonald [75], Busenberg et Travis [21]).

## Introduction des équations à retard dans la modélisation des phénomènes naturels

De manière générale, les équations à retard apparaîssent de plus en plus fréquemment dans la modélisation de l'évolution des populations. Les phénomènes "retard" s'observent également en physique, en mécanique des matériaux, dans la modélisation des chocs de particules, etc. On pourra consulter le livre de Kolmanovskii et Nosov ([68]) pour des exemples de modèles comportant des termes à retard. Dans la plupart des situations de la physique ou de la mécanique, le retard est considéré comme petit et sans effet qualitatif : il est donc le plus souvent ignoré. Dans l'étude des populations vivantes, la nécessité de tenir compte du retard s'est imposée progressivement : introduite en écologie par V. Volterra, elle a été étendue dans les années 70 à la biologie (prolifération de certains constituants du sang, Glass et Mackey [42], production cellulaire, Arino et Kimmel [15]), à la théorie des épidémies (York [111] et Cooke [28]) et a, peu à peu, fait son chemin dans ces domaines. Dans le contexte des dynamiques de populations et de la biologie mathématique en général, nous citons le livre de Bellman et Cooke ([19]) dans lequel nous pouvons trouver des motivations pour étudier les systèmes à retard. Des motivations biologiques, pour modéliser et étudier théoriquement les systèmes à retard, peuvent être trouvées aussi dans les livres de May ([78]), de Smith ([93]), et de Pielou ([87]). Des exemples concrets de modèles à retard en biologie mathématique sont contenus dans les livres de Cushing ([30]), MacDonald ([76]) et Gopalsamy ([43]). Nous citons aussi le livre de Hale ([47]) qui a poussé l'étude des équations à retard à un niveau très avancé.
La prise en compte du retard répond d'une part au problème posé par les données recueillies dans de nombreux exemples, données qui mettent en évidence des propriétés oscillatoires qui manifestent un rôle significatif des termes à retard, et d'autre part, il semble que l'enchaînement de nombreux processus dans les populations "macroscopiques" nécessite souvent un délai (voir Renshaw [90], Kuang [71]).
En fait, les modèles sans retard qui ont pu être étudiés préalablement ont souvent le défaut d'être sans oscillations, contrairement à ce que fournissent les observations. Or, l'introduction du retard dans un système ordinaire s'accompagne très généralement d'oscillations, les positions d'équilibre du système dynamique ordinaire ayant tendance à se destabiliser sous l'effet du retard (Minorsky [80], Minorsky [81], Wright [106], Hutchinson [62], Cushing [30], May [78]). Parmi les exemples les plus connus à cet égard est celui de l'équation logistique introduite par Verhulst en 1838 et que nous exposons dans ce qui suit. Nous allons donné l'équation sans retard, avec retard discret et enfin avec retard distribué. Nous résumerons quelques résultats significatifs et nous montrerons à travers ces résultats l'effet du retard sur le comportement qualitatif du système.
En fait, le premier modèle de développement démographique est celui introduit par Malthus

$$\frac{dx}{dt}(t) = \lambda x(t), \lambda > 0$$

Il traduit l'accroissement exponentiel d'une population au cours du temps. Dans sa simplicité il oublie en particulier que de nombreuses populations ont un plafond démographique imposé par des contraintes extérieures comme l'espace, les ressources, etc. Pour remédier à ce problème, un des moyens les plus simples est d'évaluer la capacité maximum $M$ de la population et de remplacer le taux de croissance $\lambda$ par une quantité qui sera d'autant plus petite que l'on s'approche de $M$. Encore une fois, le souci de simplification amène à proposer comme deuxième modèle l'équation suivante

$$\frac{dx}{dt}(t) = rx(t)(1 - \frac{x(t)}{K}) \tag{1.3}$$

Même si ce modèle paraît plus réaliste, il présente encore un défaut de principe :
Il n'est pas évident d'admettre que les populations animales -voire même humaines- ont une connaissance claire de leur capacité démographique et, une possibilité instantanée de recensement et par suite de contrôle.
Ainsi, il se peut que la capacité maximale soit atteinte et même dépassée (situation que le modèle ne peut pas prévoir). Cette situation ne se manifeste qu'à travers l'apparition de problèmes, tels que l'apparition de maladies, le manque d'espace, le manque de ressources, etc. On peut donc déduire au moins deux remarques intéressantes :

a)- La courbe d'évolution de la population présente certainement des oscillations autour de la capacité maximale $M$.
b)- Le taux de croissance de la population à l'instant $t$ dans l'équation logistique ne fait pas intervenir la taille de la population à cet instant mais il est fonction de la taille de la population précédente.

Une traduction mathématique simple de ce qui précéde est l'équation logistique retardée, connue aussi sous le nom de l'équation de Hutchinson suivante (Hutchinson [62]) :

$$\frac{dx}{dt}(t) = rx(t)(1 - \frac{x(t-\tau)}{K}) \tag{1.4}$$

où $r$ est le taux de croissance de la population, $K$ est la capacité de charge du milieu et $\tau > 0$ est le retard. Cette dernière a été étudiée par plusieurs auteurs, on peut citer par exemple Wright ([106]), Nussbaum ([83]), Walther ([105]), Hadeler ([44]), Kaplan et York ([66]), etc.

L'équation de Hutchinson suppose que l'effet de régulation dépend de la population au temps $t - \tau$ plutôt qu'à l'instant $t$. Dans un modèle plus réaliste, l'effet du retard devrait être une moyenne distribuée sur tout le passé de la population ou sur une partie de ce passé (voir Kuang [71]). Ceci a comme conséquence une équation à retard distribué ou à retard infini. Le premier travail utilisant une équation à retard distribué est dû à Volterra ([103]). Ce travail fut ensuite étendu par Kostitzin ([69]). Dans les années 30, plusieurs expériences ont été réalisées sur des populations de laboratoire ayant un temps de génération court. Les tentatives pour appliquer les modèles logistiques à ces populations échouèrent par extinction de la population. Parmi les causes de cette extinction, on peut citer le fait que dans un environnement clos, les déchets se révélent nuisibles aux organismes. Volterra ([103]) a utilisé un terme intégral où il a distribué le retard pour examiner l'effet cumulatif du taux de mortalité d'une espèce.
Le modèle qui a été considéré est une équation intégro-différentielle

$$\frac{dx}{dt} = rx(t)\left(1 - \frac{1}{K}\int_{-\infty}^{t} F(t-s)x(s)ds\right) \tag{1.5}$$

où $F$ représente le noyau retard, et correspond à une pondération du retard. Habituellement, le noyau retard est normalisé de sorte que

$$\int_0^{+\infty} F(s)ds = 1$$

De cette façon, nous assurons que l'équilibre de l'équation sans retard demeure aussi un équilibre de l'équation avec retard. La fonction $F$ peut prendre différentes formes suivant le phénomène réel à modéliser. Dans le cas où $F$ est une distribution de Dirac avec masse en un point $\tau$, l'équation (1.5) prend la forme de l'équation (1.4).
Dans le cas général, le retard moyen est défini comme suit

$$T = \int_0^{+\infty} sF(s)ds$$

Un noyau retard largement utilisé en modélisation de phénomènes biologiques (voir par exemple MacDonald, [70] ; Cushing [30]) est celui associé aux fonctions gamma

$$F(s) = \alpha^{n+1}\frac{s^n}{(n+1)!}e^{-\alpha s} \tag{1.6}$$

où $\alpha > 0$ et $n \in IN$. Le retard moyen dans ce cas est $\frac{n}{\alpha}$.
La condition initiale de l'équation intégro-différentielle (1.5) est

$$x(\theta) = \phi(\theta), \quad -\infty < \theta \leq 0$$

où $\phi(\theta)$ est une fonction continue sur $(-\infty, 0]$.
L'équation (1.5) est une équation à retard infini, sa résolution doit se faire dans un espace de Banach adéquat et exige la construction d'un espace de phase convenable. A cette étape, nous nous contentons de donner quelques résultats qualitatifs qui montrent l'influence de l'introduction du retard dans l'équation logistique (1.3) et nous discuterons dans la suite la nature de l'espace de phase des équations du type (1.5) dans un cadre plus général.

**Théorème 1.** *L'équilibre positif $x^* = K$ de l'équation logistique (1.3) est globalement stable ; c'est à dire que $\lim_{t\to+\infty} x(t) = K$, où $x(t)$ est la solution de (1.3) partant d'une condition initiale quelconque $x(0) = x_0$.*

**Théorème 2.** *Les assertions suivantes sont vraies :*

*(i) Si $0 \leq \tau < \frac{\pi}{2r}$, alors l'équilibre positif $x^* = K$ de l'équation à retard discret (1.4) est asymptotiquement stable.*

*(ii) Si $\tau > \frac{\pi}{2r}$, alors $x^* = K$ est instable.*

*(ii) Lorsque $\tau = \frac{\pi}{2r}$, une bifurcation de Hopf apparaît au voisinage de $x^* = K$. Les solutions périodiques existent pour $\tau > \frac{\pi}{2r}$ et sont stables.*

Afin de formuler un résultat analogue aux précédents pour l'équation (1.5), nous allons nous restreindre au cas d'un noyau retard dont la forme est celle d'une fonction gamma (1.6) avec $n = 1$, ce type de noyau est aussi appelé un noyau fort. Ainsi nous avons le rèsultat suivant :

**Théorème 3.** *Les assertions suivantes sont vraies :*
*(i) Si le retard moyen $T = \frac{2}{\alpha} < \frac{4}{r}$, alors l'équilibre positif $x^* = K$ est asymptotiquement stable.*
*(ii) Si le retard moyen $T = \frac{2}{\alpha} > \frac{4}{r}$, alors l'équilibre positif $x^* = K$ est instable.*
*(ii) Si $T = \frac{4}{r}$, une bifurcation de Hopf apparaît au voisinage de $x^* = K$. Les solutions périodiques existent pour $T > \frac{4}{r}$ et sont stables.*

Ainsi, nous concluons à travers ces exemples un phénomène de bifurcation typique : Lorsque le retard (ou le retard moyen) dépasse une valeur critique (ou une valeur seuil), alors l'équilibre positif passe de la stabilité à l'instabilité et il y a naissance et stabilité de solutions périodiques à droite de la valeur critique. Ce type de résultat constitue l'un des principaux comportements qualitatifs que nous allons rencontrer le long du travail présenter dans cette thèse.

Dans le cadre général, considérons l'équation différentielle fonctionnelle à retard suivante :

$$\begin{cases} \dot{x}(t) = f(t, x_t) \\ x_0 = \varphi \in \mathcal{B}_0 \end{cases} \tag{1.7}$$

où $\mathcal{B}_0$ est l'espace des fonctions définies de $[-r, 0]$ à valeurs dans $IR^n$, où $0 < r \leq +\infty$ et où $x_t \in \mathcal{B}_0$ et est défine par $x_t(\theta) = x(t + \theta)$ pour tout $-r \leq \theta \leq 0$. On remarque ici que, contrairement aux équations ordinaires, la connaissance de la solution à l'instant $t$ nécessite la connaissance de la solution dans tous les instants passés $t + \theta, \theta \in [-r, 0[$ et non pas seulement à l'instant initial. C'est pour cette raison que même si la solution est à valeurs dans $IR^n$, l'espace de phase $\mathcal{B}_0$ est de dimension infinie. Ce type d'équations a été étudiées par plusieurs autheurs. Nous citons quelques uns tels que Hale *et al.* ([49]), , Hino, Murakami et Yoshizawa ([56]). Dans le cas où l'espace $\mathcal{B}_0$ est réduit à l'espace des fonctions continues sur $[-r, 0]$ à valeurs dans un espace de Banach $X$, muni de la norme de la convergence uniforme, on parle d'équations différentielles à retard fini. Ces dernières ont fait l'objet d'étude de plusieurs travaux : Wu ([108]), Travis et Webb ([100]), Arino et Sanchez ([16]), etc.
Afin de montrer l'existence et l'unicité de la solution de l'équation (1.7) dans le cas du retard infini, Hale et Kato ([48]) ont introduit une définition axiomatique de l'espace $\mathcal{B}$. Ainsi, $\mathcal{B}$ a été considéré comme l'espace de fonctions définies sur $(-\infty, 0]$ à valeurs dans $X$, muni d'une semi norme $\|.\|_{\mathcal{B}}$ et satisfaisant les axiomes suivants :

**(A)** Si $x : (-\infty, a] \to X$ est continue sur $[0, a)$ et $x_0 \in \mathcal{B}$ alors pour tout $t \in [0, a)$ les conditions suivantes sont satisfaites :
(i) $x_t \in \mathcal{B}$;
(ii) $\|x(t)\| \leq H \|x_t\|_{\mathcal{B}}$ ;
(ii) $\|x_t\|_{\mathcal{B}} \leq Kt \sup\{\|x(s)\| : 0 \leq s \leq t\} + Mt \|x_0\|_{\mathcal{B}}$ ,
où $H \geq 0$ est une constante, $K, M : [0, \infty) \to [0, \infty)$, $K$ est continue et $M$ est localement bornée et $H, K$ et $M$ sont indépendantes de $x(.)$.
**(B)** Pour la fonction $x(.)$ définie dans (A), la fonction $t \to x_t$ est continue de $[0, a)$ vers $\mathcal{B}$.

Quelques exemples d'espaces vérifiant **(A)** et **(B)** peuvent être trouvés dans le livre de Hale et Lunel, ([49]).

Dans le cas de dimension infinie, on s'intéresse aux équations différentielles à retard du type suivant

$$\begin{cases} \dot{x}(t) = Ax(t) + F(t, x_t), & t \geq 0 \\ x_0 = \varphi \in \mathcal{B} \end{cases} \tag{1.8}$$

définie dans un espace de Banach $X$, muni d'une norme $\|.\|$. On suppose que $A : D(A) \subset X \to X$ est le générateur infinitésimal d'un $C_0$ semi groupe d'opérateurs linéaires bornés $T(t)$ défini sur $X$ (voir chapitre 7). La fonction $F(0, a] \times \mathcal{B} \to X$, $a > 0$, est une fonction continue ; $x_t : (-\infty, 0] \to X$ définie par $x_t(\theta) = x(t + \theta)$, $-\infty < \theta \leq 0$ et $\mathcal{B}$ est un espace de phase abstrait. Pour plus de détails sur la théorie des semi groupes (voir Pazy ([85]), Engel et Nagel [37], Henry [54]).
L'équation (1.8) a fait l'objet de plusieurs publications ces dernières années. Nous citons ici les travaux de Henriquez ([53]), Hino, Murakami et Naito ([55]), Arino, Burton et Haddock ([13]), Adimy *et col* ([3], [4]), etc.
Ainsi en 1985, Arino, Burton et Haddock ont introduit l'espace de phase suivant et qu'on rencontre souvent dans des travaux sur cette classe d'équations

$$C_g = \{\phi \in C(]-\infty, 0], X); \sup_{-\infty < \theta \leq 0} \frac{\phi(\theta)}{g(\theta)} < \infty\} \tag{1.9}$$

où $g$ est une fonction continue, decroissante et strictement positive sur $(-\infty, 0]$ et vérifiant $g(0) = 1$. Ceci a rendu l'espace $C_g$ comme espace de phase pour étudier l'équation (1.8).
Un cas spécial de (1.9) est lorsque $g(\theta) = \exp(-\gamma\theta)$, où $\gamma > 0$. Dans ce cas l'espace $C_g$ est noté $C_\gamma$ et c'est ce type d'espace que nous considérons dans le chapitre 6 lorsque nous étudions le mouvement nycthéméral du plancton.

## Description de la thèse

Dans ce travail, nous considérons, dans une première partie, la prédation dans un cadre général dans le sens où on ne précise pas le type de prédation modélisée. Dans une seconde partie nous nous intéressons à la prédation herbivore du zooplancton sur le phytoplancton. Notre contribution porte principalement sur l'étude de propriétés qualitatives de modèles par des techniques mathématiques, propriétés qui ont des interprétations en termes écologiques.

La thèse est donc scindée en deux parties. Dans la première, la question traitée est celle d'un système à deux espèces, un prédateur et sa proie, avec une maladie qui affecte la proie : cette maladie peut être une maladie transmissible- mais qui reste limitée à la proie- ou un état physiologique diminué, les individus présentant cet état constituent des proies plus faciles pour le prédateur. Ce problème peut plus généralement être formulé comme celui de l'action "sanitaire" du prédateur dans un troupeau, qui s'attaque principalement aux animaux malades ou moins forts.

Deux modèles de type prédateur-proie vont être exposés. Le chapitre 2 présente notre travail publié dans le Journal *Mathematical Methods in the Applied Sciences* (Chattopadhyay *et col.* [24]). Nous considérons un modèle classique composé de trois équations différentielles ordinaires scalaires quadratiques, dont le terme de prédation obéit à la loi d'action de masse et ne dépend que de la

disponibilité de la proie. Le système est constitué de trois groupes de populations : la proie saine, la proie malade, qui peut transmettre la maladie et enfin le prédateur. Dans ce système, nous étudions le comportement qualitatif en fonction du taux de transmission de la maladie $\lambda$. Nos résultats principaux dans ce chapitre sont deux théorèmes.
Dans le théorème 1, nous montrons, en construisant une fonction de Lyapounov, que la stabilité locale du système étudié au voisinage de la solution stationnaire positive entraîne sa stabilité globale.
Dans le théorème 2, nous mettons en évidence l'existence d'une branche de bifurcation au voisinage de l'équilibre positif et nous montrons que la bifurcation est supercritique.
Nous développons une méthode de réduction à la forme normale et à la variété centre dans le cas des équations différentielles ordinaires. Son implémentation en dimension trois, nous a permis non seulement de déterminer la stabilité du point d'équilibre de notre système mais aussi de montrer que les oscillations sont supercritiques.

Le chapitre 3 présente un deuxième modèle qui diffère du premier par la réponse fonctionnelle du prédateur. Cette réponse fonctionnelle dépend cette fois, non pas de la disponibilité de la proie, mais du rapport proie par prédateur. Ce travail a été accepté pour publication au journal *Nonlinearity* (Arino *et col.* [14]). Il s'agit d'un modèle ratio-dépendant, suivant la terminologie adoptée après les travaux d'Arditi et Ginzburg ([9]). L'attention apportée à l'hypothèse de la ratio-dépendance ne cesse de croître (Arditi *et col.* [10], [11], Akacakaya *et col.* [5], Cosner *et col.* [29]). Spécialement dans les situations où le prédateur doit chercher sa part de nourriture et donc est en compétition pour cette nourriture, l'hypothèse de ratio-dépendance, pour laquelle la réponse fonctionnelle est fonction du rapport de l'abondance de la proie sur l'abondance du prédateur, s'avère pertinente. La ratio-dépendance fait appel à l'idée intuitive que le taux de consommation décroît proportionnellement à l'abondance des prédateurs ; le partage d'une même ressource devant se faire entre un plus grand nombre de consommateurs. L'approche ratio-dépendante procède du concept de distribution idéale libre, suivant lequel quand plusieurs prédateurs se partagent une ressource (ou proie), chaque prédateur reçoit en moyenne une fraction de la ressource égale au rapport du nombre de proies sur le nombre de prédateurs. R. Arditi a pu mettre au point des dispositifs expérimentaux qui corroborent cette hypothèse (voir [11]).
Les modèles classiques prédateur-proie qui ne dépendent que de la disponibilité de la proie présentent quelques défauts. On peut citer par exemple le paradoxe de l'enrichissement (Hairston *et col.* [46], Rosenzweig [91]) : si on enrichit un système prédateur - proie (en augmentant sa capacité de charge par exemple), alors à l'équilibre, seule la densité du prédateur augmente et non pas la densité de la proie. Il entraîne aussi la déstabilisation de l'équilibre positif, ce qui contredit différentes observations faites sur le système (Abrams et Walter [2]). Le paradoxe du contrôle biologique (Luck [73]) : le modèle classique ne permet pas d'avoir un équilibre de faible densité et qui soit stable. Cependant, il existe de nombreux exemples dans lesquels la proie est maintenue à une densité très faible comparée avec sa capacité de charge (Arditi et Berryman [8]). Un autre défaut des modèles classiques, c'est qu'ils ne permettent pas l'extinction mutuelle du prédateur et de la proie, ce qui contredit l'observation classique faite par Gause sur l'extinction mutuelle dans le système prédateur-proie *Didinium-Paramecium* (Gause [40], Luckinbill [74], Abrams et Ginzburg [1]) et l'observation expérimentale réalisée par Huffaker ([61]).
Le modèle ratio-dépendant que nous étudions dans le chapitre 2 présente une difficulté, car le champ qui détermine les équations n'est pas régulier à l'origine et par suite sa linéarisation au voisinage de zéro n'est pas possible. Pour étudier le comportement qualitatif du système au voisinage de l'ori-

gine : Nous avons développé une méthode de réduction d'un système de $N$ équations ordinaires à un système de $N-1$ équations. Ceci a permis dans notre cas de ramener l'étude du système en trois dimensions à celle d'un système plan, puis d'appliquer le théorème de Poincaré-Bendixson pour prouver la non existence de solutions périodiques.
Notre résultat principal tiré de l'étude de ce modèle est le théorème 8 dans lequel nous avons donné des conditions suffisantes pour que le système soit persistant.
Nous avons montré que la présence de la maladie dans la population de la proie peut être bénéfique à l'écosystème proie-prédateur, peut entraîner la coexistence des espèces et peut donc agir comme un contrôle biologique.

Dans la deuxième partie, nous étudions le système phytoplancton-zooplancton en milieu marin, en supposant la présence de phytoplancton toxique empoisonnant à la longue le zooplancton. Nous envisageons l'effet négatif que peut avoir la prédation du zooplacton sur le phytoplancton toxique en augmentant son taux de mortalité et en diminuant son taux de prédation.

Dans le chapitre 4 nous présentons notre travail publié dans *IMA Journal of Math. Appl. in Med. Biol. (*Chattopadhyay *et col.* [25]*)*. Dans cet article, différentes formes de mortalité par toxicité sont considérées ; dans un premier temps, on considère l'effet instantané de la toxine et dans un deuxième temps on considère un effet cumulatif qui se traduit dans notre cas par l'introduction d'un retard distribué avec un noyau retard modélisé par une fonction gamma. Nous montrons dans le théorème 3.1 que l'équilibre positif, quand il existe, est globalement asymptotiquement stable dans le premier modèle et localement asymptotiquement stable dans le deuxième modèle. Nous présentons aussi un troisième modèle à retard discret, dont l'étude qualitative s'avère riche et intéressante. Quelques résultats intéressants obtenus par l'étude de ce modèle sont énoncés dans le théorème 5.2 et le lemme 6.2. Ainsi, nous montrons que l'équilibre non trivial peut être stable ou bien instable selon les valeurs des paramètres et qu'il existe une valeur critique du retard, lorsqu'il est considéré en tant que paramètre de bifurcation, au voisinage de laquelle une bifurcation de Hopf apparaît. Nous montrons, en utilisant l'approche de Hassard et Kazarinoff (B.D. Hassard, N.D. Kazarinoff et Y-H.Wan [50]), que ces oscillations peuvent être sous-critiques ou super-critiques suivant les valeurs des paramètres du modèle. Les résultats qualitatifs de l'analyse de stabilité ont été comparés aux données récoltées et ont montré une grande ressemblance. Le modèle à retard discret est une approximation assez satisfaisante du phénomène observé.

Dans le chapitre 5 nous présentons un modèle homogène plus général que ceux qui ont été considérés dans le chapitre 4 dans le sens où des propriétés sont établies en introduisant, dans le système, des fonctions plus générales. Ce travail a fait l'objet d'une publication dans *Journal Math. Mod. Method. Appl. Sc*, (El Abdllaoui *et col.* [35]). Nous étudions le modèle en fonction du paramètre d'efficacité de la toxine $\theta$. Nous montrons que la forme de la fonction de capture est très importante. En effet, lorsque nous considérons une forme linéaire de la réponse fonctionnelle, nous montrons que le système reste stable autour de sa position d'équilibre même si nous considérons différentes formes de la mortalité du zooplancton et différentes formes de la distribution de la toxine (voir théorème 2). Lorsque la fonction de capture est de type Holling II, nous avons pu mettre en évidence le fait que le système pouvait être stable ou instable selon la valeur de $\theta$. Nous avons montré l'existence d'un seuil $\theta^*$ au voisinage duquel une bifurcation de Hopf apparaît. Nous avons pu monter, en utilisant l'opérateur de Poincaré, que cette bifurcation est supercritique.

Dans le chapitre 6, nous présentons une extension des modèles étudiés dans les chapitre 4 et 5. C'est une extension dans le sens où les modèles des chapitres précédents sont globaux exprimés en fonction de la population totale sans structuration, tandis que le modèle qui est présenté dans le chapitre 5 est un modèle structuré. La variable de structure est l'âge des individus zooplanctoniques. Toutefois, nous commencons par une formulation en termes de date de naissance des individus zooplanctoniques. L'objectif de la structuration est d'abord une bonne formulation du problème et ensuite la comparaison des résultats qualitatifs à ceux obtenus dans le cas du modèle sans structuration. Le modèle est exprimé en termes d'équations aux dérivées partielles. Différents résultats de stabilité, d'instabilité et de bifurcation ont été mis en évidence. Leur comparaison à ceux du modèle global étudié dans le chapitre 4 montre clairement quelques différences qui sont surtout des différences quantitatives. Nous renvoyons le lecteur à la discussion détaillée du chapitre 6. Ce travail a été soumis pour publication au journal *Ecological Complexity*, A. El abdllaoui *et al.* ([36]).

Dans le chapitre 7, nous introduisons un modèle spatialisé représentant la migration nycthémérale du plancton. En effet, le phytoplancton qui se trouve dans la couche euphotique (ou couche du mélange) se reproduit au cours de la journée en synthétisant la matière organique, tandis que le zooplancton migre vers les couches les plus profondes pour se mettre à l'abri des prédateurs. Pendant la nuit, le zooplancton monte vers la surface pour se nourrir et consomme le phytoplancton qui a été produit pendant le jour (Anderson *et col.* [6]).
Dans ce travail, nous mettons l'accent sur la modélisation du mouvement nycthéméral du zooplancton. Ainsi, nous prenons en considération plusieurs processus physiques et biologiques, tels que la diffusion, le transport, la croissance, la prédation, la mortalité naturelle et par prédation et la mortalité par toxicité.
Le système fait intervenir des équations aux dérivées partielles représentant l'évolution de la biomasse planctonique. Afin de mettre en évidence l'effet cumulatif de la toxine, nous tenons compte de l'histoire du zooplancton en considérant ses trajectoires parcourues dans le passé depuis sa naissance jusqu'à l'instant $t$. Ce dernier point entraîne l'introduction du retard infini qui représente l'accumulation des substances toxiques depuis le passé jusqu'à l'instant $t$. Notre but est d'examiner l'impact du processus spatial associé à la migration nycthémérale du zooplancton sur la dynamique du système, tels que la stabilité, l'instabilté, la bifurcation, etc .... Les résultats qui sont présentés dans la thèse représentent une première étape de l'étude et sont surtout des résultats d'existence et d'unicité de la solution de ce problème.

## Problèmes mathématiques étudiés

Etude du comportement asymptotique d'équations différentielles à retard, dans le cas de retards discrets ou distribués, éventuellement infinis.
Puisque nous partons d'un système à trois variables d'état (Phytoplancton, Phytoplancton toxique, Zooplancton), les résultats mathématiques élaborés pour des systèmes prédateur-proie ne s'appliquent pas directement. Nous avons travaillé sur la réduction de la dimension dans le but d'utiliser quand c'était possible la théorie des systèmes plans. Nous avons fait l'étude de la stabilité linéarisée par diverses méthodes, suivant la forme de l'équation caractérisque obtenue, notamment, la méthode de Sturm, les critères de Routh-Hurwitz (Gantmacher [39]), le critère de Nyquist (voir Thingstad et

Langland [99]) ou encore la théorie développée par Chebotarev et ensuite par Pontriaguine pour la localisation des zéros des fonctions quasi-polynomiales de la forme $P(z, e^z)$, où $P(x, y)$ est un polynôme en $x$ et $y$ (voir par exemple Pontriaguine [89], Bellman et Cooke [19] ou Stépan [97]).
Dans l'étude de l'effet du mouvement nycthéméral, nous avons affaire à des équations aux dérivées partielles avec des variables spatiales représentant la migration du zooplancton dans la colonne d'eau. Il s'agit en fait des équations d'évolution semi-linéaires (les non linéarités sont contenues dans les termes d'ordre zéro) qui sont résolues par la méthode de variation de la constante et par la théorie des semi-groupe (voir Pazy, [85], Engel et Nagel [37]). La partie non linéaire est traîtée comme la partie non homogène d'une équation d'évolution linéaire définissant un semi-groupe fortement continu d'opérateurs linéaires bornés.

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FIG. 1.1 – Lotka-Volterra model

# Première Partie

## Système Prédateur - Proie en présence d'infection

# Introduction au chapitre 2

Dans ce chapitre nous nous intéressons au système prédateur-proie en présence d'une infection ou d'une maladie dans la proie. Nous proposons et analysons un modèle classique, c'est à dire un modèle dont la réponse fonctionnelle est une fonction de la densité de la proie seulement. Le modèle est constitué de trois groupes de populations : la proie saine, la proie infectée et le prédateur. Nous supposons que la maladie est transmissible par contact avec un taux de transmission $\lambda$.
Nous étudions le comportement asymptotique au voisinage de tous les points d'équilibre en fonction du paramètre $\lambda$. En supposant que la proie infectée a le même effet que celui de la proie saine sur la croissance du prédateur, nous avons pu montrer, en utilisant le critère de Routh-Hurwitz, que le système est localement asymptotiquement stable au voisinage de l'équilibre positif. Nous avons montré aussi que sous cette même hypothèse, et en construisant une fonction de Liapunov, que la stabilité locale du point d'équilibre non trivial, lorsque ce dernier existe, entraîne sa stabilité globale. Nous mettons en évidence l'existence d'un seuil $\lambda^*$ en dessous duquel l'équilibre positif est localement asymptotiquement stable (état endémique), c'est à dire que toutes les espèces coexistent et au dessus duquel cet équilibre est instable, c'est à dire qu'il est épidémique. Nous montrons aussi qu'au voisinage de la valeur critique $\lambda^*$, une bifurcation de Hopf apparaît, que cette bifurcation existe à droite de la valeur critique du paramètre et qu'elle est supercritique. En d'autres termes, lorsque le paramètre $\lambda$ croit et traverse la valeur $\lambda^*$, il y a naissance de solutions périodiques. L'équilibre positif perd sa stabilité qui est récupérée par la branche périodique bifurquée. Pour déterminer la nature de la branche bifurquée, nous avons développé une méthode de réduction à la forme normale et à la variété centre pour les systèmes ordinaires en dimension $N$. Pour l'implémentation de la méthode, nous nous sommes restreint à la dimension trois. Ceci nous a permis d'appliquer notre approche directement sur le système étudié. Ainsi, nous avons construit l'opérateur de Poincaré à partir du système réduit. Le code de cette approche a été écrit en langage de programmation formelle MATHEMATICA.
Des simulations numériques ont été réalisées pour illustrer les résultats analytiques obtenus. Des valeurs hypothétiques des paramètres hormis $\lambda$ ont été considérées.

# Chapitre 2

# Classical predator-prey system with infection on prey population a mathematical model [1]

**Abstract :** The present paper deals with the problem of a classical predator-prey system with infection on prey population. A classical predator-prey system is splitted into three groups, namely susceptible prey, infected prey and predator. The relative removal rate of the susceptible prey due to infection is worked out. We observe the dynamical behaviour of this system around each of the equilibrium and point out the "exchange of stability". It is shown that local asymptotic stability of the system around the positive interior equilibrium ensures its global asymptotic stability. We prove that there is always a Hopf bifurcation for increasing transmission rate. To substantiate the analytical findings, numerical experiments have been carried out for hypothetical set of parameter values. Our analysis shows that there is threshold level of infection below which all the three species will persist and above which the disease will be epidemic.

**Key words :** Susceptible and infected prey, predator, global stability, Hopf-bifurcation.

[1] This chapter represents our paper published in the journal Mathematical Methods in the Applied Sciences (M2AS), (2002). It is realized in collaboration with J. Chattopadhyay and S. Pal.

## 2.1 Introduction

Ecology and epidemiology are major fields of study in their own right. Lotka (1924) and Volterra (1926) established their original works on the expression of predator-prey and competition species relations in terms of simultaneous nonlinear differential equations, making the first breakthough in modern mathematical ecology. Similarly most models for the transmission of infectious diseases descend from the classical SIR model of Kermack and McKendrick (1927). Eco-epidemiology study is becoming important as it involves persistence-extinction threshold of each population in systems of two or more interacting species subjected to parasitism (for example, see Hadeler and Freedman, 1989; Chattopadhyay and Arino, 1999; Venturino, 1995; Beltrami and Carroll, 1994). In this paper, we consider a prey-predator system where the prey is subjected to an epidemic disease with possibly different predation rates on infected and uninfected prey. This is in accordance with the fact that the infected individuals can be caught more easily. For example; Peterson and Page (1987) indicated that wolf attacks on moose are more often successful if the moose is heavily infected by " Echinococcus granucosus". We have two species ecological system :

1. The prey, whose total population density is denoted by N.
2. The predator, whose population density is denoted by F.

We make the following assumptions

(A1) : In the absence of diseases the prey population follows the law of logistic growth with carrying capacity K ($\in R+$), with an intrinsic birth rate constant $a(a \in R+)$ :

$$\frac{dN}{dt} = aN(1 - \frac{N}{K}) \tag{2.1}$$

(A2) : In the presence of diseases the total prey population is divided into two classes, namely, susceptible prey (R) and infected prey (U).

(A3) : We assume only the susceptible prey are capable of reproducing with logistic law (equation (2.1)). In this case equation (2.1) becomes :

$$\frac{dR}{dt} = aR(1 - \frac{R}{K}) \tag{2.2}$$

(A4) : We also assume that the infected prey do not grow, recover and reproduce. Experiment on dinoflagellate Noctiluca scintillans (miliaris) in the German Bight by Uhlig and Sahling (1992) indicated that the cells become damaged and they do not feed anymore nor reproduce. The model of Hamilton et al. (1990) showed that no infected individuals contributes in the reproduction process, it rather reduces the remaining capacity due to inability to compete for resources.
In this case, it can be argued that the infected prey do not contribute to the carrying capacity and equation (2.2) becomes :

$$\frac{dR}{dt} = aR - bR^2$$

where $b = \frac{a}{K}$.

(A5) : We assume that the disease is spreading among the prey only and the predator population is not affected due to predation of infected prey.

(A6) : A susceptible prey R becomes infected under the attack of many parasities. The attacking rate as well as the predation rates follow the law of mass action. The contact process is admittedly debatable. Some researchers argue that a proportional mixing rate is more appropriate than that of simple mass action. But the data of greenwood experiment suggests that there is no change of qualitative properties upon the contact process whether it follows the law of mass action or proportional mixing rate (see, De Jong et al., 1994).

Considering the above basic assumptions we can now write the following dynamical system :

$$\begin{cases} \frac{dR}{dt} = R(a - bR - cF - \lambda U) \\ \frac{dU}{dt} = U(\lambda R - kF - \gamma) \\ \frac{dF}{dt} = F(-d + eR - fF + hU) \end{cases} \tag{2.3}$$

as our model. Here $R = R(t)$ = concentration of the susceptible prey population at time t ; $U = U(t)$ = concentration of the infected prey population at time t ; $F = F(t)$ = concentration of the predator population at time t ; a denotes the rate of increase of susceptible prey in the absence of predators and d denotes the death rate of predators in the absence of prey ; b and f denote the rate of crowding effects of the susceptible prey and predator respectively ; $c$ and $k$ are the capturing rates of susceptible prey and infected prey respectively by the predator ; $e$ ( $\leq c$) and $h$ ( $\leq k$) are the growth rates of predators due to predation of susceptible prey and infected prey respectively. $\lambda$ is the force of infection between susceptible and infected prey populations ; $\gamma$ is the death rate of infected prey.
System (2.3) needs to be analysed with the following initial conditions :

$$R(0) \geq 0, U(0) \geq 0, F(0) \geq 0. \tag{2.4}$$

Our model is similar to Venturino (1995). However, in Venturino's predator-prey system, the predator can survive in the abscence of the prey and both predator and prey grow logistically. Venturino also considered that portion of infected prey will become susceptible again after recovery. Venturino's analysis includes SI and SIS models, mass action and standard incidence. The author showed that under suitable assumptions the disease can act as a control of the system but did not discuss the nature of the solutions of the system arising from Hopf bifurcation.
The main objective of this paper is not only discuss the nature of Hopf-bifurcating solutions arising from the system but also find out the threshold level of infection below which all the three species co-exist and above which the dynamics of the system represent the epidemic situation.

## 2.2 Preliminaries

It is easy to verify that the existence, uniqueness and continuous dependence of initial conditions are evidently satisfied.

### 2.2.1 Boundedness of the system

**Lemma 1.** *All the solutions of (2.3) which initiate in $R^3_+$ are uniformly bounded.*

**Proof :** We define a function.

$$W = R + U + F. \tag{2.5}$$

The time derivative of (2.5) along the solutions of (2.3) is

$$\frac{dW}{dt} = R(a - bR) + (e - c)FR - \gamma U - F(d + fF) + FU(h - k).$$

As $e \leq c$ and also $h \leq k$, then for each $\mu > 0$, the following inequality holds :

$$\frac{dW}{dt} + \mu W \leq R(a - bR + \mu) + U(\mu - \gamma) + F(\mu - d - fF)$$

$$\leq \frac{(\mu + a)^2}{4b} + U(\mu - \gamma) + \frac{(\mu - d)^2}{4f}.$$

If we take $\mu \leq \gamma$, the above expression becomes :

$$\frac{dW}{dt} + \mu W \leq \frac{(\mu + a)^2}{4b} + \frac{(\mu - d)^2}{4f}. \tag{2.6}$$

It is clear that the right hand side of (2.6) is bounded.
Then we can find a constant $m > 0$ such that

$$\frac{dW}{dt} + \mu W < m.$$

Applying the theory of differential inequalities [Birkhoff and Rota (1982)], we obtain

$$0 < W(R, U, F) \leq \frac{m}{\mu}(1 - e^{-\mu t}) + W(R(0), U(0), F(0))e^{-\mu t}$$

and for $t \longrightarrow \infty$, we have

$$0 < W < \frac{m}{\mu}. \tag{2.7}$$

Hence all the solutions of (2.3) which initiate in $R_3^+$ are eventually confined in the region :

$$B = \{(R, U, F) \in R_+^3 : R + U + F = \frac{m}{\mu} + \epsilon, \forall \epsilon > 0\}$$

### 2.2.2 Equilibria

System (2.3) possesses the following equilibria : $E_0(0, 0, 0)$, $E_1(\frac{a}{b}, 0, 0)$, $E_2(\frac{af+cd}{ec+bf}, 0, \frac{ae-bd}{ec+bf})$, $E_3(\frac{\gamma}{\lambda}, \frac{a\lambda-b\gamma}{\lambda^2}, 0)$ and the positive equilibrium $E^*(R^*, U^*, F^*)$, where

$$R^* = \frac{\lambda\gamma f - \lambda kd + ahk + c\gamma h}{A_2} \tag{2.8}$$

$$U^* = \frac{a\lambda f - aek - b\gamma f + c\lambda d - c\gamma e + bkd}{A_2} \tag{2.9}$$

$$F^* = \frac{-\lambda^2 d + \lambda e\gamma + a\lambda h - bh\gamma}{A_2}, \tag{2.10}$$

where

$$A_2 = \lambda^2 f + c\lambda h - \lambda ke + bkh. \tag{2.11}$$

The equilibrium $E_2$ exists only if $\frac{a}{d} > \frac{b}{e}$. The steady state $E_3$ exists only if $\frac{\gamma}{\lambda} < \frac{a}{b}$.
We find that $\frac{dU}{dt}|_{t=0} < 0$ if $R(0) < \frac{\gamma}{\lambda}$ and since $R \leq R(0)$ at any time t, we find that in this case $R \leq R(0) < \frac{\gamma}{\lambda}$, so that $\lambda R - \gamma < 0$. Hence $\frac{dU}{dt} < 0$ for all $t$ when $R(0) < \rho$ where $\rho = \frac{\gamma}{\lambda}$, is the relative removal rate of the susceptible prey due to infection. Infection in the susceptible prey population can not spread unless $R(0) > \rho$. The initial concentration of the susceptible prey population must exceed the threshold value $\rho$ in order to spread the infection. Thus the existence of $E_3$ implies that the relative removal rate due to infection must be lower than that of the ratio of the birth rate of susceptible prey and its crowding coefficient.

## 2.3 Stability Analysis

In this section, we investigate the local behaviour of system (2.3) around each of the equilibria. Let $\widetilde{E} = (\widetilde{R}, \widetilde{U}, \widetilde{F})$ be any one of the equilibria. Local stability analysis (LAS) of the equilibria can be studied by computing the variational matrix corresponding to each $\widetilde{E}$.

It is easy to verify that $E_0$ is always unstable . LAS of $E_1$ implies non-existence of $E_2$ and $E_3$. Existence of positive interior equilibrium $E^*$ ensures that $E_2$ and $E_3$ are unstable.
Now, if we project our system on the plane, we can prove by constructing suitable Liapunov functions that $E_2$ and $E_3$ are globaly asymptotically stable.
A graph of $IR^3$ (see figure 1) and insight into two-dimensional subsystems should tell us the following facts :

(1) There will be an uninfected prey-predator equilibrium $E_2$ (if prey capacity is sufficiently high and/or predator mortality is low). This equilibrium is a local attractor for low transmission rate and a 2-1 saddle point for high transmission rate.
(2) There will be epidemic equilibrium $E_3$ without predator which is an attractor for poorly performing predators and becomes a 2-1 saddle point for effective predators.

Hence, with respect to the three equilibria $E_2$, $E_3$ and $E^*$ we should have the four standard cases.

$$E_2 \rightarrow E_3$$

$$E_2 \leftarrow E_3$$

$$E_2 \leftarrow E^* \rightarrow E_3$$

$$E_2 \rightarrow E^* \leftarrow E_3$$

The observation (1) is confirmed by the roots $\mu_1$, $\mu_2$ and $\mu_3$ of the characteristic equation associated with $E_2$

$$\mu_1 = \frac{\lambda(af + dc) - k(ae - bd) - \gamma(ec + bf)}{ec + bf}$$

$\mu_2$ and $\mu_3$ are the roots of the following equation :

$$\mu^2 - \mu(K_1 + K_2) + K_1K_2(1 + \frac{ec}{bf}) = 0$$

where $K_1 = \frac{f(ae-bd)}{ec+bf} > 0$, $K_2 = \frac{b(af+dc)}{ec+bf} > 0$.
Observation (2) is also confirmed by the following roots $\eta_1$, $\eta_2$ and $\eta_3$ of the characteristic equation associated with $E_3$

$$\eta_1 = \frac{-d\lambda^2 + e\gamma\lambda + h(a\lambda - b\gamma)}{\lambda^2}$$

and the other eigenvalues are the roots of the following equation :

$$\eta^2 + \eta l + l' = 0$$

where $l = \frac{b\gamma}{\lambda} > 0$, $l' = \frac{\gamma(a\lambda-b\gamma)}{\lambda} > 0$.

Now, we are in a position to see the dynamical behaviour of the system around the positive equilibrium $E^*$. We make the following hypothesis which will be required for local and global asymptotic stability of the positive equilibrium.

Hypothesis 1 :

Let us assume that $\frac{e}{c} = \frac{h}{k} = m$ (say) $(0 \leq m \leq 1)$, which is biologically realistic in the sense that the infected prey have the same effects as that of susceptible prey on the growth of predator.
The characteristic equation is given by

$$\mu^3 + d_1\mu^2 + d_2\mu + d_3 = 0 \tag{2.12}$$

where

$$\begin{aligned} d_1 &= bR^* + fF^* \\ d_2 &= bfF^*R^*khU^*F^* + \lambda^2U^*R^* + ceR^*F^* \\ d_3 &= khbR^*U^*F^* + \lambda^2fR^*U^*F^* - ke\lambda R^*U^*F^* + c\lambda kR^*U^*F^* \end{aligned} \tag{2.13}$$

A set of necessary and sufficient conditions for all the roots of (2.15) to have negative real part is $d_1 > 0$, $d_3 > 0$ and $d_1d_2 - d_3 > 0$. In our case $d_1$ is always greater than zero and the last two conditions are evidently satisfied for the hypothesis. Hence the system around $E^*$ is LAS.
Now we shall study the global asymptotic stability of the system around $E^*$ by constructing a suitable Liapunov function.

**Theorem 1.** *Local asymptotic stability of the positive interior equilibrium of (2.3) $E^*(R^*, U^*, F^*)$ ensures its global asymptotic stability.*

**Proof.** Let us consider the positive definite Liapunov function :

$$M = (R - R^* - R^*log(\frac{R}{R^*})) + (U - U^* - U^*log(\frac{U}{U^*})) + \frac{c}{e}(F - F^* - F^*log(\frac{F}{F^*}))$$

The time derivative of $M$ along the solutions of (2.3) is

$$\frac{dM}{dt} = (R - R^*)(a - bR - cF - \lambda U) + (U - U^*)(\lambda R - kF - \gamma) + \frac{c}{e}(F - F^*)(-d + eR - fF + hU)$$

$$= -b(R-R^*)^2 - \frac{(ke-ch)}{e}(U-U^*)(F-F^*) - \frac{cf}{e}(F-F^*)^2.$$

Now, using hypothesis 1 in the above expression, we have

$$\frac{dM}{dt} = -b(R-R^*)^2 - \frac{cf}{e}(F-F^*)^2 < 0.$$

We can therefore apply Lasalle 's theorem (Khalil, 1992) and deduce that any trajectory goes towards the maximal invariant set $Q$ included in the set :

$$S = \{(R,U,F) \in B; R = R^*, F = F^*\}$$

On this straight line S, in the positive space, the only invariant set is the equilibrium $E^*$. Thus the trajectory converges towards $E^*$ and hence positive interior equilibrium of (2.3) is globally asymptotically stable.
Now, we shall find out the conditions for which the strictly positive interior equilibrium enters into Hopf-bifurcation.

**Theorem 2.** *If the positive equilibrium $E^*$ of system (2.3) exists then system (2.3) around $E^*$ enters Hopf-bifurcation when $\lambda$ passes through $\lambda^*$ ($\lambda^*$ is given by (2.14).*

**Proof.** Necessary and sufficient conditions for Hopf bifurcation to occur is that there exists a $\lambda = \lambda^*$, such that

$$(i) g(\lambda^*) \equiv d_1(\lambda^*)d_2(\lambda^*) - d_3(\lambda^*) = 0$$

$$(ii) \frac{d}{d\lambda} Re(\mu(\lambda))_{\lambda=\lambda^*} \neq 0$$

The condition $d_1 d_2 - d_3 = 0$ is given by

$$H \equiv fceF^{*2}R^* + b\lambda^2 U^* R^{*2} + ke\lambda F^* R^* U^* - c\lambda h R^* U^* F^* + bf^2 F^{*2} R^*$$
$$+b^2 f F^* R^{*2} + hfkU^* F^{*2} + bceF^* R^{*2} = 0. \tag{2.14}$$

For $\lambda = \lambda^*$, we have

$$\mu^3 + d_1\mu^2 + d_2\mu + d_3 = 0. \tag{2.15}$$

that is

$$(\mu^2 + d_2)(\mu + d_1) = 0 \tag{2.16}$$

which has three roots
$\mu_1 = +i\sqrt{d_2}, \mu_2 = -i\sqrt{d_2}, \mu_3 = -d_1$.
For all $\lambda$, the roots are in general of the form

$$\mu_1(\lambda) = \beta_1(\lambda) + i\beta_2(\lambda)$$

$$\mu_2(\lambda) = \beta_1(\lambda) - i\beta_2(\lambda)$$

$$\mu_3(\lambda) = -d_1(\lambda).$$

Now, we shall verify the transversality condition

$$\frac{d}{d\lambda}(Re(\mu_j(\lambda))_{\lambda=\lambda^*} \neq 0, j = 1, 2. \tag{2.17}$$

Substituting $\mu_j(\lambda) = \beta_1(\lambda) + i\beta_2(\lambda)$ into (2.16) and calculating the derivative, we have

$$\begin{array}{l} K(\lambda)\beta_1'(\lambda) - L(\lambda)\beta_2'(\lambda) + M(\lambda) = 0 \\ L(\lambda)\beta_1'(\lambda) + K(\lambda)\beta_2'(\lambda) + N(\lambda) = 0 \end{array} \tag{2.18}$$

where

$$K(\lambda) = 3{\beta_1}^2(\lambda) + 2d_1(\lambda)\beta_1(\lambda) + d_2(\lambda) - 3{\beta_2}^2(\lambda)$$

$$L(\lambda) = 6\beta_1(\lambda)\beta_2(\lambda) + 2d_1(\lambda)\beta_2(\lambda)$$

$$M(\lambda) = {\beta_1}^2(\lambda){d_1}'(\lambda) + {d_2}'(\lambda)\beta_1(\lambda) + {d_3}'(\lambda) - {d_1}'(\lambda){\beta_2}^2(\lambda)$$

$$N(\lambda) = 2\beta_1(\lambda)\beta_2(\lambda){d_1}'(\lambda) + {d_2}'(\lambda)\beta_2(\lambda).$$

Since $L(\lambda^*)N(\lambda^*) + K(\lambda^*)M(\lambda^*) \neq 0$,
we have

$$\frac{d}{d\lambda}Re(\mu_j(\lambda))_{\lambda=\lambda^*} = \frac{LN + KM}{K^2 + L^2}|_{\lambda=\lambda^*} \neq 0,$$

and

$$\mu_3(\lambda^*) = -d_1(\lambda^*) \neq 0.$$

Therefore the transversality condition holds. This implies that a Hopf-bifurcation occurs at $\lambda = \lambda^*$ and is non-degenerate. This completes the proof.

## 2.4 Stability of bifurcating branches

In this section, we construct a method of reduction to a center manifold and normal form. Our objective is to apply this method to determin the stability of the bifurcation branch mentioned in theorem 2. We focus on a system of ordinary differential equations of dimension $N$. Let us consider the system :

$$\begin{cases} \frac{dx}{dt}(t) = Ax + \Phi(x, y) \\ \\ \frac{dy}{dt}(t) = By + \Psi(x, y) \end{cases} \tag{2.19}$$

with $x \in R^m$ and $y \in R^{n-m}(n > m > 0)$, $\Phi$ and $\Psi$ are two regular functions such that

$$\begin{aligned} \Phi(0,0) &= 0, \Psi(0,0) = 0 \\ D\Phi(0,0) &= 0, D\Psi(0,0) = 0. \end{aligned}$$

Expanding $\Phi$ and $\Psi$ into Taylor's series up to order $p(p \geq 2)$ near the origin $(0,0)$ we obtain

$$\Phi = \Phi_2 + \Phi_3 + ..$$

$$\Psi = \Psi_2 + \Psi_3 + ..$$

where $\Phi_i$ and $\Psi_i$ are the terms of degree $i$ of $\Phi$ and $\Psi$ respectively.
We suppose that

$$\sigma(A) = \sigma_c(A)$$

and

$$\sigma(B) = \sigma_s(B) \cup \sigma_u(B)$$

where $\sigma(A)$ is the spectrum of A, $\sigma_c(A)$ is the center spectrum of A, $\sigma_s(B)$ is the stable spectrum of B and $\sigma_u(B)$ is the unstable spectrum of $B$ respectively.
Now in this step, our aim is to transform each term of degree i of $\Phi$ and $\Psi$ into a normal form, and it will be better if we can do this by vanishing these terms. We assume that all terms of degree $i < k$ are in normal form and we will present our strategy from step $k$. Let us consider the following change of variables

$$(x, y) = (X, Y) + (\eta_k^1, \eta_k^2)(X) \tag{2.20}$$

Here $\eta_k^i$, $i = 1, 2$ is an homogeneous polynomial of degree $k$ in the variable $X$. We will show here that by the change of variable given in (2.20), the second equation of system (2.19) has no influence on the dynamics of the whole system (2.19).
So, we have,

$$\frac{dx}{dt} = [I + D\eta_k^1(X)]\frac{dX}{dt}. \tag{2.21}$$

As $X$ is small enough, $[I + D\eta_k^1(X)]$ is invertible and its inverse is given by

$$[I + D\eta_k^1(X)]^{-1} = I - D\eta_k^1(X) + O(|X|^{2(k-1)}).$$

Then

$$\frac{dX}{dt} = [I - D\eta_k^1 + O(|X|^{2(k-1)})]\frac{dx}{dt}$$

From (refm2as20) we have

$$\frac{dY}{dt} = \frac{dy}{dt} - D\eta_k^2(X)\frac{dX}{dt} \tag{2.22}$$

Substituting (2.21) and (2.22) in system (2.19), we obtain

$$\begin{cases} \frac{dX}{dt} = AX + \Phi_2 + .. + \Phi_{k-1} + [\Phi_k - (D\eta_k^1(X)AX - A\eta_k^1(X))] + o(|X|^k + |Y|^k) \\ \frac{dY}{dt} = BY + \Psi_2 + .. + \Psi_{k-1} + [\Psi_k - (D\eta_k^2(X)AX - B\eta_k^2(X))] + o(|X|^k + |Y|^k) \end{cases} \tag{2.23}$$

It is to be noted here that the terms up to $\Phi_{k-1}$ and also $\Psi_{k-1}$ are the same as before the application of the change of variables (given by (2.20)). Now we consider the following two operators $M_1$ and $M_2$ defined in $C^\infty$

$$\begin{cases} M_1(p)(X) = Dp(X)AX - Ap(X) \\ M_2(q)(X) = Dq(X)AX - Bq(X) \end{cases}$$

We put

$$M = (M_1, M_2)$$

such that

$$M(p,q) = (M_1 p, M_2 q), \forall (p,q) \in (\mathcal{C}^\infty)^2$$

We denote by

$$\mathcal{P}_k^m(R^l) = \{ \sum_{\sigma=|k|} a_\sigma X^\sigma, \sigma = (\sigma_1, .., \sigma_m), a_\sigma \in R^l \}$$

the set of all homogeneous polynomials of degree k in the variable X, with values in $R^l$, where $X^\sigma = X_1^{\sigma_1}..X_m^{\sigma_m}$.
We consider

$$M_1^k : \mathcal{P}_k^m(R^m) \to \mathcal{P}_k^m(R^m)$$

and

$$M_2^k : \mathcal{P}_k^m(R^{n-m}) \to \mathcal{P}_k^m(R^{n-m})$$

where $M_1^k$ and $M_2^k$ are restrictions of $M_1$ and $M_2$ to $\mathcal{P}_k^m(R^m)$ and $\mathcal{P}_k^m(R^{n-m})$ respectively.
We put $M_k = (M_1^k, M_2^k)$, with

$$M_k(p,q) = (M_1^k p, M_2^k q), \forall (p,q) \in \mathcal{P}_k^m(R^m) \times \mathcal{P}_k^m(R^{n-m})$$

After applying the change of variables (given in (2.20)) in system (2.19), the term of degree $k$ now becomes,

$$(\widetilde{\Phi_k}(X,Y), \widetilde{\Psi_k}(X,Y)) = (\Phi_k(X,Y), \Psi_k(X,Y)) - M_k(\eta_k^1, \eta_k^2)(X)$$

or

$$\begin{cases} \widetilde{\Phi_k}(X,Y) = \Phi_k(X,Y) - M_1^k(\eta_k^1)(X) \\ \widetilde{\Psi_k}(X,Y) = \Psi_k(X,Y) - M_2^k(\eta_k^2)(X) \end{cases}$$

To normalise $(\widetilde{\Phi_k}(X,Y), \widetilde{\Psi_k}(X,Y))$ we shall have to search $\eta_k^1$ and $\eta_k^2$ in such a way that we can eliminate the parts of $\Phi_k$ and $\Psi_k$ which are in the range $R(M_1^k)$ and $R(M_2^k)$ respectively. This elimination depends on the operator $M_k$. Note that here $M_k(\eta_k^1, \eta_k^2)$ depends on the variable $X$ only, our aim is to simplify only the term $(\Phi_i(.,0), \Psi_i(.,0))$.
Now, the problem reduces to a linear algebraic one, i.e : we shall have to solve the following problem

$$M_k(\eta_k^1, \eta_k^2) = (\Phi_k(.,0), \Psi_k(.,0)). \tag{2.24}$$

with respect to $\eta_k^1$ and $\eta_k^2$, where $\Phi_k(.,0)$ and $\Psi_k(.,0)$ are known and $\Phi_k(.,0) \in \mathcal{P}_k^m(R^m)$ and $\Psi_k(.,0) \in \mathcal{P}_k^m(R^{n-m})$.
To solve equation (refm2as24), we study the linear operator

$$M_k : \mathcal{P}_k^m(R^m) \times \mathcal{P}_k^m(R^{n-m}) \to \mathcal{P}_k^m(R^m) \times \mathcal{P}_k^m(R^{n-m})$$

If $(\Phi_k(.,0),\Psi_k(.,0)) \in R(M_k)$, then equation (2.24) is completely solvable. But if $(\Phi_k(.,0),\Psi_k(.,0)) \notin R(M_k)$, we shall follow the classical method, i.e, we decompose the space $\mathcal{P}_k^m(R^m) \times \mathcal{P}_k^m(R^{n-m})$. Let us consider $\mathcal{C}_k$ as a complementary subspace of $R(M_k)$ in $\mathcal{P}_k(R^m) \times \mathcal{P}_k(R^{n-m})$

$$\mathcal{P}_k(R^m) \times \mathcal{P}_k(IR^{n-m}) = R(M_k) \oplus \mathcal{C}_k$$

Different choices of $\mathcal{C}_k$ are possible. We present some of these
(a)-If $M_k$ is a projector ; i.e $M_k o M_k = M_k$, then we can choose $\mathcal{C}_k = Ker(M_k)$.
(b)-We can choose also $\mathcal{C}_k = \mathrm{Ker}(M_k^T)$, where $M_k^T$ represents the adjoint of the operator $M_k$ with respect to some adequate inner product.
Consider $\pi_k^1$ and $\pi_k^2$ the projections of $\mathcal{P}_k^m(R^m)$ and $\mathcal{P}_k^m(R^{n-m})$ onto $R(M_1^k)$ and $R(M_2^k)$ respectively and let $\pi_k = (\pi_1^k, \pi_2^k)$ such that

$$\pi_k(p,q) = (\pi_1^k p, \pi_2^k q)$$

Now, we shall have to solve the following equation

$$M_k(\eta_k^1, \eta_k^2) = \pi_k(\Phi_k(.,0), \Psi_k(.,0))$$

or

$$\begin{cases} M_1^k(\eta_k^1) = \pi_1^k \Phi_k(.,0) \\ M_2^k(\eta_k^2) = \pi_2^k \Psi_k(.,0) \end{cases}$$

We have

$$\sigma(M_1^k) = \{ \sum_{|\sigma|=k} \sigma_i \lambda_i - \lambda_j, \lambda_i \in \sigma(A); \sigma = (\sigma_1, .., \sigma_m)\}$$

$$\sigma(M_2^k) = \{ \sum_{|\sigma|=k} \sigma_i \lambda_i - \mu_j, \lambda_i \in \sigma(A), \mu_j \in \sigma(B), \sigma = (\sigma_1, .., \sigma_m)\}$$

The second equation of (2.24) is completely solvable in $\mathcal{P}_k^m(R^{n-m})$ as $0 \notin \sigma(M_2^k)$ i.e. $M_2^k$ is invertible. Then the term of degree $k$ in the second equation of system (2.19) becomes

$$\widetilde{\Psi}_k(X,Y) = \Psi_k(X,Y) - \Psi_k(X,0)$$

Hence

$$\widetilde{\Psi}_k(.,0) \equiv 0$$

And the term of degree $k$ in the first equation of sytem (2.19) becomes

$$\widetilde{\Phi}_k(X,Y) = \Phi_k(X,Y) - \pi_1^k \Phi_k(X,0)$$

In other words

$$\widetilde{\Phi}_k(.,0) = (I - \pi_1^k)\Phi_k(.,0) \in \mathcal{C}_k$$

If we repeat the same procedure for all $2 \leq k \leq p,$, we obtain the following system

$$\begin{cases} \frac{dX}{dt} = AX + \sum_{j=2}^p \widetilde{\Phi}_j(X,Y) + o(|X|^p + |Y|^p) \\ \frac{dY}{dt} = BY + \sum_{j=2}^p \widetilde{\Psi}_j(X,Y) + o(|X|^p + |Y|^p) \end{cases} \tag{2.25}$$

with

$$\begin{cases} (\widetilde{\Phi}_j(.,0), \widetilde{\Psi}_j(.,0)) \in \mathcal{C}_j \\ \widetilde{\Psi}_j(.,0) \equiv 0 \end{cases} , \forall 2 \leq j \leq p$$

We can prove that Y = 0 is an approximation up to order $p$ of a center manifold of system (2.25). Then the reduced system in this center manifold is

$$\frac{dX}{dt} = AX + \sum_{j=2}^{p} \widetilde{\Phi}_j(X,0) + o(|X|^p)$$

For the present system given by equations (2.3), we find that the variational matrix $V^*$ [see equation (2.16)] has a real negative eigenvalue and a pair of purely imaginary eigenvalues at Hopf bifurcation. We may therefore analyze this system with the above method.

We first translate the origin of the coordinate system to the equilibrium (R*, U*, F*) by writing

$$R = R - R^*, U = U - U^*, F = F - F^* \tag{2.26}$$

Then equations (2.3) can be written in the form

$$\frac{d}{dt}\begin{pmatrix} \overline{R} \\ \overline{U} \\ \overline{F} \end{pmatrix} = V^* \begin{pmatrix} \overline{R} \\ \overline{U} \\ \overline{F} \end{pmatrix} + \begin{pmatrix} h_1 \\ h_2 \\ h_3 \end{pmatrix} \tag{2.27}$$

Here $V^*$ is the variational matrix computed at $\lambda^*$, while the nonlinear terms are

$$\begin{aligned} h_1 &= -b\overline{R}^2 - c\overline{FR} - \lambda\overline{UR} \\ h_2 &= \lambda\overline{RU} - k\overline{FU} \\ h_3 &= -f\overline{F}^2 + e\overline{FR} - h\overline{UF}. \end{aligned}$$

At Hopf bifurcation, equation (2.16) holds and the eigenvalues of $V^*$ are $\mu_1 = \alpha$ and $\mu_{2,3} = \pm i\omega$, where $\alpha = -(bR^* + fF^*)$, and

$$\omega = \sqrt{bfF^*R^* + khF^*U^* + \lambda^2 U^*R^* + ceF^*R^*}. \tag{2.28}$$

If the eigenvector of $V^*$ associated with $\mu_1$ is denoted by $W_1$ and the eigenvectors corresponding to $\mu_{2,3}$ are denoted by $W_2 \pm iW_3$ ($W_1$, $W_2$ and $W_3$ are real), then it can be shown that the matrix $P = (W_3, W_2, W_1)$ is non singular. Furthermore

$$P^{-1}V^*P = \begin{pmatrix} 0 & -\omega & 0 \\ \omega & 0 & 0 \\ 0 & 0 & \alpha \end{pmatrix} \tag{2.29}$$

We find that

$$P = \begin{pmatrix} \omega cR^* & -\lambda kR^*U^* & -\lambda kR^*U^* + c\alpha R^* \\ \omega kU^* & \lambda cR^*U^* + bkR^*U^* & c\lambda R^*U^* + kU^*(bR^* + \alpha) \\ -\omega cR^* & -\omega^2 - \lambda^2 R^*U^* & -\alpha(bR^* + \alpha) - \lambda^2 R^*U^* \end{pmatrix} \tag{2.30}$$

and

$$Q = P^{-1} = \frac{1}{A_1}\begin{pmatrix} q_{11} & q_{12} & q_{13} \\ q_{21} & q_{22} & q_{23} \\ q_{31} & q_{32} & q_{33} \end{pmatrix} \tag{2.31}$$

where

$$q_{11} = U^*[abc\lambda {R^*}^2 + ab^2k{R^*}^2 + \alpha^2c\lambda R^* + \alpha^2bkR^* + \omega^2(c\lambda R^* + kbR^* + \alpha k) - \lambda^2\alpha kR^*U^*]$$

$$q_{12} = \lambda R^*U^*(\alpha bkR^* + \alpha^2k + \alpha\lambda cR^* + k\omega^2) - c\alpha\omega^2R^*$$

$$q_{13} = \alpha kR^*U^*(k\lambda U^* + c^2\lambda R^* + bcR^*)$$

$$q_{21} = -k\omega U^*(\alpha^2 + \lambda^2R^*U^*) + b\omega {R^*}^2U^*(c\lambda + bk)$$

$$q_{22} = \omega cR^*(\alpha^2 + \lambda^2R^*U^*) + \omega b\lambda kR^*U^*$$

$$q_{23} = \omega c{R^*}^2U^*(c\lambda + bk) + \lambda\omega k^2R^*U^*$$

$$q_{31} = \omega kU^*(\lambda^2R^*U^* - \omega^2) - \omega b{R^*}^2U^*(c\lambda + bk)$$

$$q_{32} = \omega cR^*(\omega^2 - \lambda^2R^*U^*) - \lambda k^2\omega R^*{U^*}^2$$

$$q_{33} = -\omega c{R^*}^2U^*(c\lambda + bk) - \lambda k^2\omega R^*U^*$$

and

$$A_1 = \omega(\alpha^2 + \omega^2)R^*U^*(\lambda c^2R^* + bckR^* + \lambda k^2U^*)$$

Now, we use the linear transformation

$$\begin{pmatrix} \overline{R} \\ \overline{U} \\ \overline{F} \end{pmatrix} = P \begin{pmatrix} \overline{U_1} \\ \overline{U_2} \\ \overline{U_3} \end{pmatrix} \tag{2.32}$$

which can be written as

$$\overline{\overline{Z}} = PW, W = P^{-1}\overline{\overline{Z}}. \tag{2.33}$$

where $\overline{\overline{Z}} = (\overline{R}, \overline{U}, \overline{F})^T$ and P is given by (2.30).
Substituting (2.33) in (2.27) gives

$$\frac{d}{dt}(PW) = V^*PW + F_1(PW)$$

where, $F(\overline{\overline{Z}}) = (h_1, h_2, h_3)^T$ which implies that

$$\frac{dW}{dt} = (P^{-1}V^*P)W + P^{-1}F_1(PW). \tag{2.34}$$

where $W = (U_1, U_2, U_3)^T$ and $P^{-1}V^*P$ is a constant matrix given by (2.29). Now we can write (2.34) in the following manner :

$$\begin{cases} \frac{dx}{dt}(t) = Ax + F_1(x, y) \\ \frac{dy}{dt}(t) = By + G_{12}(x, y) \end{cases} \tag{2.35}$$

where $x = (U_1, U_2)^T$, $y = (U_3)$, A and B are the constant matrices

$$A = \begin{pmatrix} 0 & -\omega \\ \omega & 0 \end{pmatrix}, B = (\alpha)$$

and $F_1$ and $G_1$ are both $C^2$ functions. System (2.34) can now be written as

$$\frac{d}{dt}\begin{pmatrix} U_1 \\ U_2 \\ U_3 \end{pmatrix} = \begin{pmatrix} 0 & -\omega & 0 \\ \omega & 0 & 0 \\ 0 & 0 & \alpha \end{pmatrix}\begin{pmatrix} U_1 \\ U_2 \\ U_3 \end{pmatrix} + Q\begin{pmatrix} h_1(P(U_1,U_2,U_3)) \\ h_2(P(U_1,U_2,U_3)) \\ h_3(P(U_1,U_2,U_3)) \end{pmatrix} \tag{2.36}$$

Now, by applying the above method to our system (2.36), we obtain the following reduced system

$$\begin{cases} \frac{dU}{dt} = -\omega V + a_1(U^3 + UV^2) + o(|U|^4 + |V|^4) \\ \frac{dV}{dt} = \omega U + b_1(U^2V + V^3) + o(|U|^4 + |V|^4) \end{cases} \tag{2.37}$$

where $a_1$ and $b_1$ are the coefficients of the normal form and are functions of the parameters. We have,

$$\begin{cases} U\dot{U} + V\dot{V} = a_1U^4 + (a_1 + b_1)U^2V^2 + b_1V^4 + o(|U|^5 + |V|^5) \\ U\dot{V} - V\dot{U} = \omega(U^2 + V^2) + o(|U|^3 + |V|^3) \end{cases} \tag{2.38}$$

Now, using polar coordinates

$$U = rcos(\theta), V = rsin(\theta) \ . \tag{2.39}$$

in system (2.38), we have

$$\begin{cases} r\,\dot{r} = r^4(a_1\cos^4(\theta) + b_1\sin^4(\theta) + (a_1 + b_1)\cos^2(\theta)\sin^2(\theta) + o(r^5) \\ \dot{\theta} = \omega + o(r) \end{cases} \tag{2.40}$$

System (2.40) is equivalent to

$$\begin{cases} \frac{dr}{dt} = r^3(a_1\cos^4(\theta) + b_1\sin^4(\theta) + (a_1 + b_1)\cos^2(\theta)\sin^2(\theta) + o(r^4) \\ \frac{d\theta}{dt} = \omega + o(r) \end{cases} \tag{2.41}$$

Eliminating $t$ from equation (2.41), we obtain

$$\frac{1}{r^3}\frac{dr}{d\theta} = \frac{a_1\cos^4(\theta) + b_1\sin^4(\theta) + (a_1 + b_1)\cos^2(\theta)\sin^2(\theta)}{\omega} + o(r). \tag{2.42}$$

To discuss the stability of the periodic solution, we shall now construct a Poincaré map. Integrating equation (2.42) between 0 and $2\pi$, we obtain

$$r^2(2\pi) = r^2(0)(1 - \frac{9\pi}{8}\frac{(a_1 + b_1)}{\omega}r^2(0) + o(r^3)). \tag{2.43}$$

We consider now the Poincaré map associated to equation (2.43)

$$P(r) = r(1 - \frac{9\pi}{16}\frac{(a_1 + b_1)}{\omega}r^2)$$

Now we are in a position to state the main result of this section.

**Lemma 2.** *If $a_1 + b_1 > 0$, the bifurcating branches are supercritical. If $a_1 + b_1 < 0$, the bifurcating branches are subcritical. In the case when $a_1 + b_1 = 0$, to conclude the nature of bifurcating branches we shall have to compute higher order terms of the normal form.*

## 2.5 Conclusion

In this paper we proposed and analysed a predator-prey system in which some members of the prey population are infected by some transmissible disease and thus form a new group, namely, the infected prey. We have subdivided the prey into two classes, namely, susceptible and infected. We have modeled the above situation by means of three ordinary differential equations. The behaviour of the system near each of the equilibria has been studied. Threshold value of the relative removal rate which determines the spread of infection has been worked out and is closely related to the basic reproductive ratio, $R_0$, of epidemic theory. Local and global asymptotic stability analysis of the system near the positive interior equilibrium has been performed. We also proved that there is always a Hopf bifurcation for increasing transmission rate. Computing the coefficients of the normal form, we found that $a_1 + b_1 > 0$ which implies that the bifurcation is supercritical.

To substantiate analytical findings we performed some numerical experiments based on the formulas of section 3 and are obtained for the hypothetical values a = 4, b = 0.006, c = 0.09, d = 0.25, e = 0.01, f = 0.005, h = 0.09, k = 0.01, $\gamma = 0.3$ and for different values of $\lambda$. Using these sets of parameter values in (2.14) we obtain the results for which system 2.3 about $E^*$ is stable (if $H > 0$ ) or unstable (if $H < 0$ ). Table 2.1 summarizes the results. For the above set of parameter values and for $\lambda = 0.02$, we observe that the system settles down to steady state solutions, depicting stable situation (figure 2). Now if we increase the force of infection from $\lambda = 0.02$ to $\lambda = 0.025$, the system settles down to steady state solution through decaying oscillations (figure 3). If we further increase $\lambda = 0.025$ to $\lambda = 0.026$, system is unstable through growing oscillations (figure 4) and limit cycle oscillations occurs at $\lambda = 0.0255$ (figure 5). Hence we may conclude that there is a threshold level of force of infection, below which all the three species will persist and above which the disease will be epidemic.

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| $\lambda$ | $H$ |
|---|---|
| 0.01599 | 0.792416 |
| 0.016 | 0.7909747 |
| 0.017 | 0.6560341 |
| 0.018 | 0.537175 |
| 0.019 | 0.431783 |
| 0.02 | 0.3377763 |
| 0.023 | 0.1087768 |
| 0.024 | 0.0463195 |
| 0.0248 | 0.0350319 |
| 0.02481 | - 0.0203458 |
| 0.02482 | - 0.0007568 |
| 0.02483 | - 0.0013097 |
| 0.02484 | - 0.0018619 |

TAB. 2.1 – Values of the function $H(\lambda)$ for different values of the parameter $\lambda$. All other parameters are fixed.

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FIG. 2.1 – A schematic diagram representing the exchange of stability for different equilibra of system 2.3.

FIG. 2.2 – Stable solution of system 2.3 for $\lambda = 0.02$.

FIG. 2.3 – Decaying oscillations of system 2.3 for $\lambda = 0.025$.

FIG. 2.4 – Growing oscillations of system 2.3 for $\lambda = 0.026$.

Fig. 2.5 – Limit cycles of system 2.3 for $\lambda = 0.0255$.

# Introduction au chapitre 3

Dans le chapitre précédent, nous avons émis l'hypothèse que la réponse fonctionnelle était uniquement conditionnée par l'abondance de la proie. Dans ce chapitre est présentée une autre approche qui consiste à considérer non plus uniquement l'abondance de proies mais le ratio proie/prédateur. On considère ainsi que les prédateurs ont d'autant plus de difficultés à trouver des proies quand ils sont en abondance par rapport à celles-ci. Nous proposons et analysons un modèle prédateur-proie ratio-dépendent avec une infection dans la population des proies. Notre objectif est d'étudier le comportement qualitatif du système en fonction du paramètre de transmission de l'infection $\lambda$.

Le résultat principal de l'étude de ce modèle est la persistence du système. Nous montrons, en utilisant le critère de Li-Muldowney en dimension trois, la non existence de solutions périodiques. Nous montrons aussi, sous certaines conditions sur les paramètres, que tous les points d'équilibres axiaux sont répulsifs et que les trajectoires ne peuvent pas atteindre l'origine ni suivant des directions fixes ni en spirale. En fait, l'étude au voisinage de l'origine est très importante dans la mesure où elle nous permet de conclure à propos de l'extinction des espèces.
Cette étude présente cependant une difficulté due au fait que le champ de vecteurs définissant les équations du système n'est pas défini à l'origine et par conséquent la méthode de linéarisation n'est plus possible. Pour mener l'analyse à son terme en surmontant ces difficultés, nous avons développé, dans un contexte général, une méthode de réduction d'un système ordinaire de dimension $N$ à un système de dimension $N-1$. La méthode de réduction concerne les systèmes ordinaires de la forme

$$\begin{cases} \frac{dX}{dt}(t) = H(X(t)) + Q(X(t)) \\ X(0) = X_0 \end{cases}$$

où $H : IR^N \to IR^N$ est une fonction de classe $C^1$ et homogène de degré 1, c'est à dire : $H(sX) = sH(X)$, $\forall s \in IR, \forall X \in IR^N$.
La fonction $Q$ est supposée assimilable à une petite perturbation : $Q(X) = o(X)$.
Cette méthode nous a permis, dans notre cas, de réduire le système en dimension trois à un système plan et d'appliquer le théorème de Poincaré-Bendixson sur ce dernier.

Les simulations entreprises ont été réalisées dans le but de montrer qu'il existe des valeurs des paramètres pour lesquelles l'équilibre positif est localement asymptotiquement stable. Les valeurs hypothétiques ont été choisies les plus proches de ce qui existe en littérature (voir par exemple Edwards et Brindley, 1999).

# Chapitre 3

# Infection on prey population may act as a biological control in ratio-dependent predator-prey model [1]

**Abstract :** A ratio-dependent predator-prey model with infection on prey population is proposed and analyzed. The behaviour of the system near the biological feasible equilibria is observed. The conditions for which no trajectory can reach the origin following any fixed direction or spirally are worked out. We investigate the criteria for which the system will persist. It is observed that introduction of infected population in the classical ratio-dependent predator-prey model may act as a system saver. To substantiate our analytical findings, numerical simulations are also performed for a hypothetical set of parameter values.

**Key words :** Infection on prey population, Ratio-dependency, Predator-prey model.

[1] This chapter represents our work accepted for a publication in the journal Nonlinearity (2003). It is a work realized in collaboration with O. Arino, J. Mikram and J. Chattopadhyay.

## 3.1 Introduction

After the pioneering works of Alfred James Lotka and Vito Volterra in the middle of 1920 for predator-prey interactions, prey-dependent predator prey models were studied extensively (see, for example, Murray 1989, Freedman 1980 and the references therein). Similarly, epidemiological models have also received much attention after the seminal model of Kermack-McKendrick on SIRS (susceptible-infective-removed-susceptible) systems. There are so many references in this context, we have just cited some of the many references in this context (see for example, Anderson 1991, Bailey 1975, and the references therein).

Ecology and epidemiology are major fields of study in their own right, but there are some common features between these systems. It is interesting and important from biological viewpoints to study ecological systems under the influence of the epidemiological factors. Quite a good number of studies have already been performed in eco-epidemiological systems (see for example, Hadeler and Freedman, 1989, Venturino, 1995, Beltrami and Carroll, 1995, Chattopadhyay and Arino, 1997, Chattopadhyay and Bairagi, 2001, Chattopadhyay and Pal, 2002, etc.). Most of the works on such eco-epidemiological systems are based on prey-dependent models. As far as our knowledge goes, no work has been carried out on such systems with ratio-dependent functional response. The present paper deals with an eco-epidemiological model with disease in the prey population and functional responses following the law of ratio-dependent theory.
Before introducing the model, we would like to present a brief historical account of the biological relevance of the classical prey-dependent model and the controversial (Abrams (1994), Abrams and Ginzburg (2000)) ratio-dependent model.

The classical prey-dependent predator-prey model exhibits not only the well known "paradox of enrichment" formulated by Hairston et al. (1960) and Rosenzweig (1969) but also the so-called "biological control paradox", which was recently brought into discussion by Luck (1990).
The "paradox of enrichment" states that enriching a predator-prey system (increasing the carrying capacity) will cause an increase in the equilibrium density of the predator but not in that of the prey, and will finally destabilize the positive equilibrium. As a result it increases the possibility of stochastic extinction of the predator. But what is observed in nature is that enriching the system increases the prey-density, does not destabilize a stable steady state and fails to increase the amplitude of oscillations in systems that already cycle (Abrams and Walter, (1996)).
The so-called "biological control paradox" states that we cannot have a low and stable prey equilibrium density, which is in contradiction with many examples of successful biological controls where the prey is maintained at low densities compared with its carrying capacity (Arditi and Berryman (1991)).
Recently, models with such a prey-dependent response function have been facing challenges both from biological and physiological researchers (see, for example, Arditi and Ginzburg, 1989, Arditi et al., 1991, Akcakaya, 1992, Gutierrez, 1992). Based on biological and physiological evidences, some researchers argue that the functional response in a predator-prey model should be based on the ratio-dependent theory, especially when predators have to search for food and the per capita predator growth rate should be a function of the ratio of prey to predator abundance. Arditi and Ginzburg (1989) first proposed the following Michaelis-Menten type, ratio-dependent predator-prey system which was then studied extensively by a number of authors (see for example Kuang and Beretta,

1998, Jost et al., 1999, Hsu et al., 2001)

$$\begin{cases} \dfrac{dx}{dt} = rx(1-\dfrac{x}{K}) - \alpha\dfrac{xP}{k_1P+x} \\ \dfrac{dP}{dt} = P(-\delta_2 + \dfrac{fx}{k_1P+x}) \\ x(0) > 0, P(0) > 0 \end{cases} \tag{3.1}$$

The analysis of the ratio-dependent predator-prey model shows that it will produce neither the paradox of enrichment nor the biological control paradox (see Hsu, Hwang and Kuang (2001)). It also allows mutual extinction as a possible outcome of a predator-prey interaction (Kuang and Beretta (1998), Jost et al. (1999)).
In spite of these merits, there are still some controversial aspects of such ratio-dependent models (see Abrams and Ginzburg (2000)). The main point is that ratio-dependent models require high population densities for both prey and predator while the most interesting dynamics is near the origin (see for example, Xiao and Ruan (2001)).

Disease in ecological systems can not be ignored. From this fact, we now modify the above model (3.1) by introducing a transmissible disease in the prey population. We make the following assumptions :

**(A1)** In the absence of disease the prey population grows in logistic fashion with carrying capacity $K > 0$ and an intrinsic birth rate constant $r > 0$.
**(A2)** In the presence of the disease the prey population is divided into two parts, the susceptible prey ($S(t)$) and the infected prey ($I(t)$). Therefore, at time $t$ the total prey population is

$$x(t) = S(t) + I(t)$$

**(A3)** We assume that only the susceptible prey population is capable of reproducing and contributing to its carrying capacity.
**(A4)** We also assume that the disease transmission follows the simple law of mass-action.

With the above assumptions, model (3.1) leads to the following set of ordinary differential equations

$$\begin{cases} \dfrac{dS}{dt} = rS(1-\dfrac{S}{K}) - \alpha_s\dfrac{SP}{k_1P+S+I} - \lambda SI \\ \dfrac{dI}{dt} = \lambda SI - \alpha_I\dfrac{IP}{k_1P+S+I} - \delta_1 I \\ \dfrac{dP}{dt} = \dfrac{k(\alpha_s S+\alpha_I I)P}{k_1P+S+I} - \delta_2 P \\ S(0) > 0, I(0) > 0, P(0) > 0 \end{cases} \tag{3.2}$$

Here $\alpha_S$ and $\alpha_I$ are the searching efficiency constants or the predation rate on the susceptible and infective prey respectively. It is observed in nature that $\alpha_S$ can be less or, on the contrary, bigger than $\alpha_I$ depending on the type of parasitism. We just mention here some books (see for example Dawkins "The Extended Phenotype, (1982)", Combes "Les associations du vivant, (2001)" and Combes "Parasitism, The Ecology and Evolution of Intimate Interactions, University of Chicago Press, (2001)").

$\frac{\alpha_S}{k_1}$ and $\frac{\alpha_I}{k_1}$ are the maximum per capita capturing rate for predator on susceptible prey and infected prey respectively. $\lambda$ is the force of infection. $\delta_1$ and $\delta_2$ are the death rates of infected prey and predator respectively. $k$ is a conversion rate.

This work can be seen as a continuation of the work done by two of the authors (Chattopadhyay and Arino 1997) on a predator-prey system with disease in the prey. The main modification here is that we take into account the ratio-dependent theory, which has some important features on the dynamics of the system.

Kuang and Beretta (1998), Jost et al. (1999), Xiao and Ruan (2001), observed that the dynamics of system (3.1) near the origin is more complicated since the vector field is not well defined at that point and can not be linearized around this point. There exist numerous kinds of topological structures in the vicinity of the origin (see for example Xiao and Ruan (2001), Berezovskaya et al. (2001)). This is the main reason for ratio-dependent models possibly to have complicated rich dynamics. Kuang and Beretta (1998) proved that total extinction is also possible. Jost et al. (1999) proved that the origin can be a saddle-point or an attractor. Xiao and Ruan (2001) analyzed a situation where solutions reach the origin following a fixed direction. The results obtained in the three above mentioned papers are roughly complementary to each other. We like to mention that the diseased population can not be ignored in such models and has some influence on the dynamics. We study the dynamics of the zero equilibrium starting from the first positive quadrant. We give a complete study near the origin in the sense that we have studied the possibilities to reach this critical point following any fixed direction of the domain of interest. In this paper, we use a reduction principle which allows us to reduce the system to a two-dimensional system where the Poincaré-Bendixson result can be applied. We find suitable conditions on the parameters such that we can not reach the origin spirally. We have also pointed out that total extinction can be controlled by the diseased prey population and that under some conditions, the model is persistent.

The paper is organized as follows : Section 2 gives preliminary results, behaviour of the system around $E_0(0,0,0)$ is given in section 3. Persistence results are presented in section 4. In section 5 we show that the infected prey may act as a system saver. Section 6 deals with the problem of finding some suitable conditions for which there is no periodic solution around the positive equilibrium and the paper ends with a conclusion.

## 3.2 Preliminary results

We first observe that the right hand side of system (3.2) is a smooth function of the variables $(S, I, P)$ and the parameters in the positive octant, as long as the sum of these quantities is non zero. So, local existence and uniqueness properties hold in the positive octant.
From system (3.2), it follows that $S = 0$ (resp $I = 0, P = 0$) is an invariant subset, that is, $S \equiv 0$ (resp $I \equiv 0, P \equiv 0$) if and only if $S(t) = 0$ (resp $I(t) = 0, P(t) = 0$) for some $t$. Thus $S(t) > 0$ (resp $I(t) > 0, P(t) > 0$) for all $t$ if $S(0) > 0$ (resp $I(0) > 0, P(0) > 0$).
So, if $I = 0$, which corresponds to a system without disease, then the system reduces to a two dimensional one which has been studied extensively by several authors (see for example, Kuang and Beretta (1998), Jost et al. (1999), Xiao and Ruan (2001)).
We first prove the boundedness of system (3.2).

**Lemma 1.** *All the solutions of system (3.2) which initiate in* $\mathbb{R}^3_+$ *are bounded, with ultimate bound.*

**Proof :** We define a function

$$W(t) = kS(t) + kI(t) + P(t) \tag{3.3}$$

Taking the time derivative of $W$ along the solutions of (3.2), we have

$$\frac{dW}{dt}(t) = rkS(t)(1 - \frac{S(t)}{K}) - k\delta_1 I(t) - \delta_2 P(t)$$

For any positive constant $\mu(> 0)$, we have

$$\frac{dW}{dt} + \mu W = S\{rk + \mu - rk\frac{S}{K}\} + (\mu - k\delta_1)I + (\mu - \delta_2)P$$

If we take $\mu$ such that $0 < \mu < \min(k\delta_1, \delta_2)$ then we obtain

$$\frac{dW}{dt} + \mu W \leq \frac{K}{rk}(\frac{rk + \mu}{2})^2 = M \tag{3.4}$$

where $M$ is the maximum value of the function $S\{rk + \mu - rk\frac{S}{K}\}$.
From (3.4) we have

$$\frac{dW}{dt} \leq -\mu W + M$$

which implies that

$$\begin{aligned} W(t) &\leq e^{-\mu t}W(0) + \frac{M}{\mu}(1 - e^{-\mu t}) \\ &\leq \max(W(0), \frac{M}{\mu}) \end{aligned}$$

Moreover, we have

$$\limsup_{t \to \infty} W(t) \leq \frac{M}{\mu}$$

which is independent on the initial condition.□

## 3.3 Equilibria

System (3.2) has the following equilibria
$E_0(0, 0, 0)$, $E_1(K, 0, 0)$,
$E_2(S_2, 0, P_2)$ with

$$S_2 = K(1 - \frac{k\alpha_s - \delta_2}{kk_1 r}) \text{ and } P_2 = r(1 - \frac{S_2}{K})\frac{kS_2}{\delta_2}.$$

$E_3(S_3, I_3, 0)$ with

$$S_3 = \frac{\delta_1}{\lambda} \text{ and } I_3 = \frac{r}{\lambda}(1 - \frac{\delta_1}{\lambda K}).$$

and an interior equilibrium $E^*(S^*, I^*, P^*)$ where

$$\begin{aligned} I^* &= -(\frac{r}{\lambda K} + \frac{\alpha_s}{\alpha_I})S^* + (\frac{r}{\lambda} + \frac{\alpha_s \delta_1}{\lambda \alpha_I}) \\ P^* &= \frac{(k\alpha_s - \delta_2)S^* + (k\alpha_I - \delta_2)I^*}{\delta_2 K} \end{aligned}$$

and $S^*$ satisfies the following equation

$$AS^2 + BS + C = 0$$

with

$$\begin{aligned} A &= kk_1 r\alpha_I \lambda \\ B &= -k\left\{r\alpha_I(\alpha_I + k_1\delta_1) + k_1 K(r\alpha_I + \alpha_S\delta_1)\lambda + \delta_2(-r\alpha_I + k\lambda(\alpha_I - \alpha_S))\right\} \\ C &= K(r\alpha_I + \alpha_S\delta_1)\left\{k(\alpha_I + k_1\delta_1) - \delta_2\right\} \end{aligned}$$

It is easy to see that a necessary and sufficient condition for the existence of $E_2$ is

$$0 < k\alpha_S + \delta_2 < kk_1 r.$$

From the expression of $I_3$, it is clear that a necessary and sufficient condition for the existence of $E_3$ is

$$\lambda K - \delta_1 > 0.$$

It can be easily shown that $E^*$ exists and is unique if the following set of inequalities hold simultaneously

$$\begin{aligned} k &< 1,\ \delta_1 < K\lambda < \frac{r\alpha_I(1-k)}{k(\alpha_I + \alpha_S)} \\ \delta_2 &< \min(k\alpha_I, k\alpha_S, \frac{r(k\alpha_I + \alpha_S)(K\lambda - \delta_1)}{r\alpha_I + \alpha_S\lambda K}) \\ r &> \frac{k\alpha_S\delta_1 + \alpha_I(\delta_2 + k\delta_1)}{\alpha_I(1-k)} \end{aligned}$$

## 3.4 Behaviour of the system around $E_0(0,0,0)$

At the trivial equilibrium $E_0$, the jacobian matrix is not defined. Let us now, for a moment, consider the problem in a general context, that is to say, we consider a system in $\mathbb{R}^N$,

$$\frac{dX}{dt} = H(X(t)) + Q(X(t)) \tag{3.5}$$

in which, $H$ is $C^1$ outside the origin , is continous and homogenous of degree 1

$$H(sX) = sH(X)$$

for all $s \geq 0, X \in \mathbb{R}^N$, and $Q$ is a $C^1$ function such that

$$Q(X) = o(X)$$

in the vicinity of the origin.
Throughout the section, $\|.\|$ denotes the Euclidian norm on $\mathbb{R}^N$ and $(.,.)$ the associated inner product. In the case of our model, $N = 3$,

$$\begin{aligned} X &= (x_1, x_2, x_3) = (S, I, P) \\ H(X) &= (H_1(X), H_2(X), H_3(X)) \\ Q(X) &= (Q_1(X), Q_2(X), Q_3(X)) \end{aligned}$$

The functions $H_i$ and $Q_i$ $(i = 1, 2, 3)$ are given by

$$\begin{aligned} H_1(X) &= rx_1 - \alpha_s \frac{x_1 x_3}{k_1 x_3 + x_1 + x_2},\ H_2(X) = -\alpha_I \frac{x_2 x_3}{k_1 x_3 + x_1 + x_2} - \delta_1 x_2 \\ H_3(X) &= \frac{k(\alpha_s x_1 + \alpha_I x_2) x_3}{k_1 x_3 + x_1 + x_2} - \delta_2 x_3 \\ Q_1(X) &= -r\frac{x_1^2}{K} - \lambda x_1 x_2,\ Q_2(X) = \lambda x_1 x_2,\ Q_3(X) = 0 \end{aligned}$$

Let $X(t)$ be a solution of system (3.5). Assume that $\liminf_{t\to\infty} \|X(t)\| = 0$, and $X$ is bounded. One can extract from the family $(X(t+.))_{t\geq 0}$ sequences $X(t_n + .), t_n \to \infty$, such that $X(t_n + .) \to 0$ locally uniformly on $s \in \mathbb{R}$.
Define

$$y_n(s) = \frac{X(t_n + s)}{\|X(t_n + s)\|}$$

We have

$$\begin{aligned} \frac{dy_n}{ds} &= [H(y_n(s)) - (y_n(s), H(y_n(s)))y_n(s)] \\ &\quad + \|X(t_n + s)\| \, [Q(y_n(s)) - (y_n(s), Q(y_n(s)))y_n(s)] \end{aligned}$$

Clearly, $y_n$ is bounded, $\|y_n(s)\| = 1, \forall s$, and $\frac{dy_n}{ds}$ is bounded too. So, applying the Ascoli-Arzela theorem (see for example Brezis [12]), one can extract from $y_n$ a subsequence - still denoted $y_n$- which converges locally uniformly on $\mathbb{R}$- towards some function $y$, which satisfies the equation

$$\begin{cases} \dfrac{dy}{dt} = H(y(t)) - (y(t), H(y(t)))y(t) \\ \|y(t)\| = 1, \forall t \end{cases} \tag{3.6}$$

Equation (3.6) is defined for all $t \in \mathbb{R}$.
Let us, for a moment, focus on the study on equation (3.6).
The steady states of $H$ are vectors $V$ verifying :

$$H(V) = (V, H(V))V$$

This is a so-called nonlinear eigenvalue problem. Note that the equation can be alternatively written as

$$H(V) = \mu V, \tag{3.7}$$

with $\|V\| = 1$, it then holds that $\mu = (V, H(V))$.
These stationary solutions correspond to fixed directions that the trajectories of equation (3.6) may reach asymptotically.
Equation (3.7) can be written as

$$[(\mu - r)v_1 + (\mu - r)v_2 + (\alpha_S + k_1\mu - k_1 r)v_3]\, v_1 = 0 \quad (3.8)$$

$$[(\mu + \delta_1)v_1 + (\mu + \delta_1)v_2 + (\alpha_I + k_1\mu + k_1\delta_1)v_3]\, v_2 = 0 \quad (3.9)$$

$$[(\mu + \delta_2 - k\alpha_S)v_1 + (\mu + \delta_2 - k\alpha_I)v_2 + k_1(\mu + \delta_3)v_3]\, v_3 = 0 \quad (3.10)$$

Now we are in a position to discuss in detail the possibility to reach the origin following fixed directions.

**Case1-** $v_1 = 0$

**a)** $v_2 = 0$ and $v_3 \neq 0$ In this case, there is a possibility to reach the origin following the $P-$axis, with $\mu = -\delta_3$.

**b)** $v_2 \neq 0$ and $v_3 = 0$ In this case also, there is a possibility to reach zero following the $I$-axis, with $\mu = -\delta_1$.

**c)** $v_2 \neq 0$ and $v_3 \neq 0$ In this case, we obtain different results depending on the parameters

**Subcase1-** If $kk_1 < 1$ then

(i) We reach the origin if $kk_1\delta_1 + k\alpha_I - \delta_2 < 0$.

(ii) We can not reach the origin if $kk_1\delta_1 + k\alpha_I - \delta_2 > 0$.

**Subcase2-** If $kk_1 > 1$ then the conclusion is reverse of subcase 1.

**Case2-** $v_1 \neq 0$

**a)** $v_2 = 0$ and $v_3 = 0$ In this case, we can not reach the origin following the $S$-axis, that is to say that the $S$-axis is not a fixed direction that the trajectories can follow to reach zero.

**b)** $v_2 = 0$ and $v_3 \neq 0$ In this case, we have two possibilities

(i) If $E_2$ exists and $kk_1 < 1$ then the $SP$-plane can be followed by the trajectories to reach the origin.

(ii) If $kk_1 > 1$ then there is no possibility of reaching the origin following the $SP$-plane.

**c)** $v_2 \neq 0$ and $v_3 = 0$. In this case, there is no possibility to reach the origin following the $SI$ -plane.

**d)** $v_2 \neq 0$ and $v_3 \neq 0$. In this case, there are two subcases

(i) $\alpha_S \leq \alpha_I$. In this case, there is no possibility to go to the origin following a fixed direction which is contained in the positive octant.

(ii) $\alpha_S > \alpha_I$. Under this condition, the trajectories may follow a fixed direction which is contained in the positive octant.

Now, we want to show that under some suitable conditions, no orbit of sytem (3.2) tends to the critical point spirally. To do that, we return to equation (3.6).

**Proposition 2.** *If we assume that :*

$$H_i(X) = X_i\overline{H}_i(X), \forall i$$

*then equation (3.6) preserves positiveness. So, every non zero solution of equation (3.6) with closure at a positive distance from at least one of the* $(N-1)$*- coordinate hyperplanes can in fact be seen as a solution of an o.d.e in* $\mathbb{R}^{N-1}$.

**Proof.** Let $y$ be a non zero solution of

$$\frac{dy}{dt} = H(y(t)) - (H(y(t)), y(t))y(t)$$

With no loss of generality, assume that : $y_N(t) \geq m > 0$, we may write

$$y_N = \sqrt{1 - y_1^2 - .. - y_{N-1}^2}$$

then, $\widetilde{y} = (y_1, .., y_{N-1})$ satisfies a system of o.d.e of theform

$$\frac{d\widetilde{y}}{dt}(t) = \widetilde{G}(\widetilde{y}(t))$$

in an open subset of $\mathbb{R}_+^{N-1}$ in which $\widetilde{G}$ is $C^1$. □

**Corollary 3.** *In the case $N = 3$, the reduced system is a planar o.d.e to which the Poincaré-Bendixson theorem applies.*

We now return to the equation under study with $y$ being the limit of a sequence $\frac{X(s+t_n)}{\|X(s+t_n)\|}$. Let us investigate the asymptotic behavior of $y(t)$. Either there exists $j \in \{1,2,3\}$ such that $y_j(t) \geq m > 0\ \forall t$. In this case, the equation can be reduced to its planar projection onto $(y_i, y_k)$, $i, k \neq j$, or $\liminf_{t\to\infty} y_i(t) = 0$, for $i = 1, 2, 3$.
We have

$$\begin{cases} \frac{dy}{dt}(t) = H(y(t)) - (y(t), H(y(t)))y(t) \\ \|y(t)\| = 1 \end{cases}$$

If we put $y = (y_1, y_2, y_3)$ and $\widetilde{y} = (y_1, y_2)$ then we can write $y_3 = \sqrt{1 - y_1^2 - y_2^2}$ and reduce the system to the following two dimensional system

$$\begin{cases} \widetilde{y}'(t) = \overline{H}(\widetilde{y}(t)) - \widetilde{y}(t)[(\widetilde{y}(t), \overline{H}(\widetilde{y}(t))) + \sqrt{1 - \widetilde{y}^2(t)}\overline{H}_3(\widetilde{y}(t))] \\ \|\widetilde{y}(t)\| < 1 \end{cases} \tag{3.11}$$

with

$$\begin{aligned} \overline{H}((y_1, y_2)) &= (H_1, H_2)(y_1, y_2, \sqrt{1 - y_1^2 - y_2^2}) \\ \overline{H}_3((y_1, y_2)) &= H_3(y_1, y_2, \sqrt{1 - y_1^2 - y_2^2}) \end{aligned}$$

Now, we will use the Poincaré-Bendixson criteria to show that under some suitable conditions, if a solution of (3.11) tends to the origin then it must tend to it along a fixed direction.
We define

$$\begin{aligned} f_1((y_1, y_2)) &= H_1(y_1, y_2, \sqrt{1 - y_1^2 - y_2^2}) - y_1 g((y_1, y_2)) \\ f_2((y_1, y_2)) &= H_2(y_1, y_2, \sqrt{1 - y_1^2 - y_2^2}) - y_2 g((y_1, y_2)) \\ g((y_1, y_2)) &= ((y_1, y_2), \overline{H}((y_1, y_2))) + \sqrt{1 - \widetilde{y}^2(t)}\overline{H}_3(\widetilde{y}(t)) \end{aligned}$$

We have

$$y_1(t)\frac{dy_2}{dt}(t) - y_2(t)\frac{dy_1}{dt}(t) = y_1(t)H_2(\widetilde{y}(t), \sqrt{1-\widetilde{y}^2(t)}) - y_2(t)H_1(\widetilde{y}(t), \sqrt{1-\widetilde{y}^2(t)})$$

using the expression of $H_1$ and $H_2$, we find that

$$\frac{d}{dt}[\ln(\frac{y_2(t)}{y_1(t)})] = (\alpha_s - \alpha_I)\frac{\sqrt{1-\widetilde{y}^2(t)}}{y_1 + y_2 + k\sqrt{1-\widetilde{y}^2(t)}} - (\delta_1 + r) \tag{3.12}$$

From equation (3.12), we can conclude that if $\alpha_s \leq \alpha_I$ then we can not have a periodic solution because $\ln(\frac{y_2(t)}{y_1(t)})$ can not change signs. In the other case, when $\alpha_s > \alpha_I$, suppose that $\widetilde{y}(t)$ is a periodic solution, then from equation (3.12), we can see that a necessary condition is that on the minimum point, the quantity $\ln(\frac{y_2(t)}{y_1(t)})$ vanishes and is less than the maximum value of the right hand side of equation (3.12), which implies that

$$0 \leq \frac{(\alpha_s - \alpha_I)}{k} - (\delta_1 + r) \tag{3.13}$$

We conclude that if $\alpha_I > \alpha_s - k(\delta_1 + r)$, then we can not reach the origin spirally.
Now we are in a position to summarize the above results in the following two theorems.

**Theorem 4.** *If the predation rate on the infected prey is higher than or equal to that of susceptible prey, then any trajectory can reach the origin from the interior following a fixed direction.*

**Theorem 5.** *If the difference of the predation rate has an upper threshold value, given by*

$$\alpha_S - \alpha_I < k(\delta_2 + r)$$

*then any trajectory can reach the origin spirally starting from the interior.*

**Remark 6.** *In the paper of Jost et al. (1999), $(0,0,0)$ is a global attractor if $Q + R < S$ (see proposition 4.1, p.p.27), which in terms of our model parameter corresponds to $\alpha_S > k_1(r + \delta_2)$. We find that if $\alpha_S \leq \alpha_I$ then no trajectory can reach $E_0(0,0,0)$. So, if we set*

$$k_1(r + \delta_2) < \alpha_S \leq \alpha_I$$

*then total extinction is not possible with our model while it is possible with Jost et al. model.*
*Xiao and Ruan (2000) studied the behavior of zero equilibrium as $t \to +\infty$ or $t \to -\infty$ depending on the parameters in the interior of the first octant, but they did not discuss the behavior of this equilibrium starting from the rest of the domain. Our analysis is taking this into account. So, our*

*results include the whole topological structure near* $(0,0,0)$ *starting from any position of the domain of interest. (In Ruan's paper, the parameters* $\frac{b}{a}$, *resp.* $c, m, d$ *and* $f$ *correspond to* $K$, *resp.* $\alpha_S, k_1, \delta_2$ *and* $k$*).*

*Kuang and Beretta (see Th.2.6) proved that under some conditions, the origin is globally asymptotically stable ; that is to say that the system goes to total extinction. Their conditions can be formulated in terms of our system parameters as follows*

$$k \geq \frac{\delta}{\alpha_S - k_1 r} \text{ and } \alpha_S > k_1 r$$

## 3.5 System saver

In the remark of section 3 we find that the predator-prey system considered by Jost et al. (1999) and Kuang and Beretta (1998) goes to total extinction under some parametric conditions. It is to be noted here that our system (3.1) is equivalent to their system, considered in the above mentioned papers, for $I = 0$.
In the following theroem we show that introduction of infected prey to the predator-prey system prevent the total extinction and may act as a biological control.

**Theorem 7.** *Assume that* $\lambda < \lambda^*, kk_1 > 1$ *and*

$$\delta_2 > \frac{k\alpha_S}{kk_1 - 1}$$

*then the boundary steady state* $E_2$ *is locally asymptotically stable (LAS), where* $\lambda^*$ *is given by (3.14).*

**Proof.** Local stability of $E_2$ depends on the sign of $a_{22}$ and the eigenvalues of the following matrix

$$V_2^0 = \begin{pmatrix} a_{11} & a_{13} \\ a_{31} & a_{33} \end{pmatrix},$$

where $(a_{ij})$ are the coefficients of the jacobian matrix $V_2$ computed on $E_2$ and are given in the proof of theorem 11.
We have

$$\begin{aligned} \det(V_2) &= a_{22}\left[\frac{\delta(k\alpha_S - \delta_2)(kk_1 r - k\alpha_S + \delta_2)}{k_1 k^2 \alpha_S}\right] \\ tr(V_2) &= a_{22} - r - \frac{(k\alpha_S - \delta_2)\left[(kk_1 - 1)\delta_2 - k\alpha_S\right]}{k_1 k^2 \alpha_S} \end{aligned}$$

Existence of $E_2$ implies that $\det(V_2)$ has the same sign as that of $a_{22}$.
Now if $\lambda < \lambda^*$ then $a_{22} < 0$. In this case, the stability of $E_2$ depends on the eigenvalues of $V_2^0$.
We have

$$\begin{aligned} \det(V_2^0) &= \frac{1}{a_{22}} \det(V_2) \\ tr(V_2^0) &= \frac{1}{a_{22}} Tr(V_2) \end{aligned}$$

We know that $\det(V_2^0) > 0$. Sufficient conditions to have $Tr(V_2^0) < 0$ are

$$\begin{aligned} kk_1 &> 1 \\ \delta_2 &> \frac{k\alpha_S}{kk_1 - 1}.\square \end{aligned}$$

## 3.6 Persistence results

We have already proved that system (3.2) is uniformly bounded. Now in order to prove the persistence of the system, first we will show that all the boundary equilibria are repellers.

**Theorem 8.** *If the following conditions hold*

$$\begin{aligned} (i)\alpha_I &\geq \alpha_S \\ (ii)0 &< k\alpha_S - \delta_2 < kk_1 r \\ (iii)\lambda &> \lambda^* \end{aligned}$$

*where $\lambda^*$ is given in (3.14), then the system is persistent.*

**Proof :** After computing the variational matrix associated with $E_1(K,0,0)$, we find the following eigenvalues : $\mu_1 = -r, \mu_2 = \lambda K - \delta_1$ and $\mu_3 = k\alpha_s - \delta_2$.
We conclude that existence of $E_2$ or $E_3$ implies that $E_1$ is unstable.
For the equilibrium point $E_2$, the entries of the Jacobian matrix $V_2$ computed at $E_2$ are as follows :

$$\begin{aligned} a_{11} &= \alpha_s \frac{S_2P_2}{(k_1P_2+S_2)^2} - \frac{rS_2}{K},\ a_{12} = \alpha_s S_2P_2 \frac{1}{(k_1P_2+S_2)^2} - \lambda S_2, \\ a_{13} &= -\frac{\alpha_s S_2^2}{(k_1P_2+S_2)^2},\ a_{21} = 0,\ a_{22} = \lambda S_2 - \frac{\alpha_I P_2}{(k_1P_2+S_2)} - \delta_1,\ a_{23} = 0, \\ a_{31} &= \frac{kk_1\alpha_s P_2^2}{(k_1P_2+S_2)^2},\ a_{32} = \frac{kP_2\{\alpha_I k_1 P_2 + (\alpha_I - \alpha_s)S_2\}}{(k_1P_2+S_2)^2},\ a_{33} = \frac{k\alpha_s S_2^2}{(k_1P_2+S_2)^2} - \delta_2 \end{aligned}$$

Since $a_{21} = a_{23} = 0$, we see that $a_{22}$ is an eigenvalue of $V_2$ and can be written as

$$a_{22} = \frac{K\lambda\alpha_S(kk_1r - k\alpha_S + \delta_2) - r\alpha_I(k\alpha_S - \delta_2) - kk_1r\delta_1\alpha_S}{kk_1r\alpha_S}$$

Let

$$\lambda^* = \frac{r\alpha_I(k\alpha_S - \delta_2) + kk_1r\delta_1\alpha_S}{K\alpha_S(kk_1r - k\alpha_S + \delta_2)} \tag{3.14}$$

It is easy to see that if $\lambda > \lambda^*$ then $E_2$ is unstable (as $a_{22} > 0$).
From the variational matrix computed at $E_3$ we conclude that if the sufficient condition

$$k\min(\alpha_I, \alpha_S) - \delta_2 > 0$$

is true then $E_3$ is unstable (because one of the eigenvalues is $\frac{k(\alpha_S S_3 + \alpha_I I_3)}{(S_3+I_3)} - \delta_2 > 0$).
Thus we see that all the boundary equilibria of system (3.1) are repellers if the conditions stated in theorem 8 hold. $\square$

## 3.7 Conditions for non existence of periodic solutions around $E^*$

In this section we want to prove that under some suitable conditions, there is no periodic solution of system (3.2) around the positive equilibrium $E^*$.
To prove this, the following criteria by Li and Muldowney can be applied.
Consider the general autonomous ordinary differential equation

$$\frac{dX}{dt} = F(X(t)) \tag{3.15}$$

where $F$ is a $C^1$ function in some open subset of $\mathbb{R}^N$ with values in $\mathbb{R}^N$. Denote by $J = (\frac{\partial F}{\partial X})$ the jacobian matrix associated with (3.15). Denote by $J^{[2]}$ the $\binom{N}{2} \times \binom{N}{2}$ matrix which is the second additive compound matrix associated to the jacobian matrix $J$ (see appendix for more details) and remind that if $X \in R^N$ then the corresponding logarithmic norm of $J^{[2]}$ (that we denote by $\mu_\infty(J^{[2]})$) endowed by the vector norm $|X|_\infty = \sup_i |X_i|$ is

$$\mu_\infty(J^{[2]}) = \sup\left\{\frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} + \sum_{j\neq r,s}\left(\left|\frac{\partial F_r}{\partial x_j}\right| + \left|\frac{\partial F_s}{\partial x_j}\right|\right) : 1 \leq r < s \leq N\right\} \tag{3.16}$$

where $\mu_\infty(J^{[2]}) < 0$ implies the diagonal dominance by row matrix $J^{[2]}$. Then the following holds :

**Theorem 9.** *A simple closed rectifiable curve which is invariant with respect to system (3.2) cannot exist if* $\mu_\infty(J^{[2]}) < 0$.

Before we find conditions under which there is no periodic solution, we perform a change of coordinate to lower the number of parameters of system (3.2).
We put

$$\begin{aligned} U &= \frac{S}{K},\ V = \frac{I}{K},\ W = \frac{P}{K} \text{ and } \tau = rt \\ &\text{and} \\ \alpha'_S &= \frac{\alpha_S}{r},\ \alpha'_I = \frac{\alpha_I}{r},\ \delta'_1 = \frac{\delta_1}{r},\ \delta'_2 = \frac{\delta_2}{r},\ \lambda' = \frac{\lambda K}{r} \end{aligned} \tag{3.17}$$

then system (3.2) preserves the same form but with $r = 1$ and $K = 1$ and the new parameters mentioned in (3.17) for which we omit the prime to simplify the notation.

Let us now apply Li-Muldowney's criteria (see [27]) in the new coordinates for the non existence of periodic solutions of system (3.2).The logarithmic norm $\mu_\infty$ endowed by the norm $|X|_\infty$ of the second additive compound matrix $J^{[2]}$ associated with the jacobian matrix $J$ computed on $E^*$ is negative if and only if the supremums of the following functions satisfy the conditions

$$1 - (2-\lambda)S - \lambda I - \delta_1 - \frac{\alpha_S P(2k_1P + S + I)}{(k_1P + S + I)^2} \tag{3.18}$$

$$+\frac{kP\,|I(\alpha_S-\alpha_I)+k_1\alpha_S P|+kPS(\alpha_I-\alpha_S)+k_1P}{(k_1P+S+I)^2}<0$$

$$1-2S-\lambda I-\delta+\lambda S+\frac{(S+I)k(\alpha_S S+\alpha_I I)-\alpha_S(k_1P+I)}{(k_1P+S+I)^2} \tag{3.19}$$

$$+\frac{\alpha_S PS}{(k_1P+S+I)^2}+\frac{\alpha_I kP(k_1P+(\frac{\alpha_S-\alpha_I}{\alpha_I})S)}{(k_1P+S+I)^2}<0$$

$$2\lambda S-\alpha_I P\frac{(k_1P+S)}{(k_1P+S+I)^2}-\delta_1-\delta_2 \tag{3.20}$$

$$+\frac{k(\alpha_S S+\alpha_I I)(S+I)+\alpha_S PS+\alpha_S S(S+I)}{(k_1P+S+I)^2}<0$$

Sufficient conditions to satisfy (3.18), (3.19) and (3.20) are respectivelly

$$0<\lambda<2 \text{ and } \delta_1-1-\alpha_S>0, \tag{3.21}$$

$$0<\lambda<2 \text{ and } \delta_2-1-\alpha_S(k+\frac{1}{2k_1}+\frac{k}{k_1})\geq 0, \tag{3.22}$$

$$0<2\lambda<\delta_1+\delta_2+\frac{\alpha_I}{2k_1}-\alpha_S(k+\frac{1}{2k_1}) \text{ and } \delta_1+\delta_2+\frac{\alpha_I}{2k_1}>\alpha_S(k+\frac{1}{2k_1}). \tag{3.23}$$

A direct application of Li-Muldowney's method shows that under conditions (3.21)-(3.23), there is no periodic solution for system (3.2).

## Conclusion

The classical prey-dependent and ratio-dependent models are well studied. In this paper we have investigated the dynamical behaviour of a ratio-dependent predator-prey model with infection on prey population. The proposed model is a modification of the model proposed by Arditi and Ginzburg (1989). The behaviour of the system near the biologically feasible equilibria has been studied. The parametric conditions for which the solutions of the system can not reach the origin following fixed direction or in a spiral fashion have been work out. All the topological structures near the origin starting from any position of the domain of interest have been taken care of. Moreover, Jost et al. (1999), Kuang and Beretta (1998) also studied the model of Arditi and Ginzburg; they obtained the conditions for which the whole population may go to extinction. In contrast, we have observed that introduction of diseased prey into the system may save the population from the extinction. Thus, we may conclude that infected prey population in a classical ratio-dependent predator-prey system may act as a biological control. Moreover, using Muldowney's criteria, persistence conditions have been worked out. To substantiate analytical findings, numerical simulations have been carried out for a hypothetical set of parameter values.

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## Appendix

The definition of the second additive compound matrix can be found in the paper of Li and Muldowney (1993).

Let $A = (a_{ij})$ be an $n \times n$ matrix. The *second additive compound* $A^{[2]}$ is the $\binom{n}{2} \times \binom{n}{2}$ matrix defined as follows.

For any integer $i = 1, .., \binom{n}{2}$, let $(i) = (i_1, i_2)$ be the $i$*th* member in the lexicographic ordering of integer pairs $(i_1, i_2)$ such that $1 \leq i_1 < i_2 \leq n$. Then the element in the i-row and the j-column of $A^{[2]}$ is

$$a_{i_1 i_1} + a_{i_2 i_2}, \text{ if } (j) = (i)$$
$$(-1)^{r+s} a_{i_r j_s}, \text{ if exactly one entry } i_r \text{ of } (i) \text{ does not occur in } (j) \text{ and } j_s \text{ does not occur in } (i)$$
$$0, \text{ if neither entry from } (i) \text{ occurs in } (j).$$

For $n = 3$

$$A = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix}$$

its second additive compound matrix is

$$A^{[2]} = \begin{pmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{pmatrix}$$

In this case, $(1) = (1,2)$, $(2) = (1,3)$ and $(3) = (2,3)$.

**Theorem : (Bendixson's Criterion in $R^n$).** *A simple closed rectifiable curve which is invariant with respect to (3.15) cannot exist if any one of the following conditions is satisfied on $R^n$ :*

(i) $\sup \left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} + \sum_{q \neq r,s} \left( \left| \frac{\partial F_q}{\partial x_r} \right| + \left| \frac{\partial F_q}{\partial x_s} \right| \right) : 1 \leq r < s \leq n \right\} < 0,$

(ii) $\sup \left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} + \sum_{q \neq r,s} \left( \left| \frac{\partial F_r}{\partial x_q} \right| + \left| \frac{\partial F_s}{\partial x_q} \right| \right) : 1 \leq r < s \leq n \right\} < 0,$

(iii) $\lambda_1 + \lambda_2 < 0,$

(iv) $\inf \left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} - \sum_{q \neq r,s} \left( \left| \frac{\partial F_q}{\partial x_r} \right| + \left| \frac{\partial F_q}{\partial x_s} \right| \right) : 1 \leq r < s \leq n \right\} > 0,$

(v) $\inf \left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} - \sum_{q \neq r,s} \left( \left| \frac{\partial F_r}{\partial x_q} \right| + \left| \frac{\partial F_s}{\partial x_q} \right| \right) : 1 \leq r < s \leq n \right\} > 0,$

(vi) $\lambda_{n-1} + \lambda_n > 0.$

where $\lambda_1 \geq \lambda_2 \geq .. \geq \lambda_n$ are the eigenvalues of $\frac{1}{2}((\frac{\partial F}{\partial x})^* + \frac{\partial F}{\partial x})$ and where $\frac{\partial F}{\partial x}$ is the Jacobian matrix of $F$ and the asterisk denotes transposition.

FIG. 3.1 – Numerical simulations

# Deuxième Partie

## Système Phytoplancton - Zooplankton en présence de toxine

# Introduction au chapitre 4

Dans ce chapitre nous nous intéressons au système phytoplancton-zooplancton dans un environement toxique. Notre objectif est l'élaboration et l'étude mathématique d'un modèle d'interactions entre le phytoplancton et le zooplancton en tenant compte de quelques mécanismes de base du système. Les modèles que nous présentons dans ce travail sont explicatifs. Ils permettent de mieux comprendre le fonctionnement du système en reproduisant quelques phénomènes observés, tel que les efflorescences nuisibles ou ce qu'on appelle aussi les *Harmful algal blooms (HAB)*. En effet, la nature périodique des floraisons du plancton est une caractéristique principale du fonctionnement des écosystèmes marins. La libération des substances toxiques par des espèces phytoplanctoniques toxiques réduit la croissance du zooplancton en réduisant sa capacité de prédation et son taux d'attaque. Ce phénomène de toxicité joue un rôle très important dans les blooms du plancton.

Dans ce travail, nous proposons quelques modèles mathématiques simples en tenant compte des considérations précédantes. Nous étudions les comportements qualitatifs de ces modèles au voisinage de leurs points d'équilibre en fonction des paramètres et nous mettons l'accent sur le paramètre retard $\tau$. Sachant que le processus de libération ou d'absorption des toxines reste jusqu'à présent inconnu, nous consiérons différentes formes de l'impact de la toxicité allant d'un effet instantané dans le cas des équations ordinaires jusqu'à son effet cumulatif représenté par un retard distribué en passant par un effet retardé représenté par un retard discret.

Nous cherchons à reproduire la nature cyclique des blooms, nature qui semble avoir été observée fréquemment au cours de ces dernières décennies un peu partout dans le monde. Ainsi par exemple, nous avons pu analyser les données concernant la dynamique du phytoplancton et du zooplancton dans la baie du Bengale (voir figure 1). La figure 2 représentant ces données, montre clairement une périodicité de l'ordre de 10 mois. L'analyse mathématique des modéles proposés dans ce chapitre a montré que le modèle sans retard et le modèle à retard distribué sont stables autour de leur position d'équilibre. Par conséquent, les oscillations ne peuvent être générées par ces modèles. Dans le cas du modéle à retard discret, nous avons pu par contre mettre en évidence l'existence d'oscillations et avons pu montré que ces oscillations sont entretenues.

L'analyse mathématique des modèles a nécessité l'application de quelques méthodes telles que le critère de Routh-Hurwitz, le critère de Bendixson-Dulac, la transformée de Laplace, l'approche de Hassard et Kazarinoff (voir [21]).
Les simulations que nous avions effectuées à partir de notre modèle confirme l'existence de ces phénomènes cycliques avec une périodicité du même ordre de grandeur que celle des observations.

# Chapitre 4

# A Delay differential equation model on harmful algal blooms in the presence of toxic substances [1]

**Abstract :** The periodic nature of blooms is the main characteristic in marine plankton ecology. Release of toxic substances by phytoplankton species or toxic phytoplankton reduce the growth of zooplankton by decreasing grazing pressure and have an important role in planktonic blooms. A simple mathematical model of phytoplankton-zooplankton systems with such characteristics is proposed and analysed. As the process of liberation of toxic substances by phytoplankton species is still not clear, we try to describe a suitable mechanism to explain the cyclic nature of bloom dynamics by using different forms of toxin liberation process. To substantiate our analytical findings numerical simulations are performed and these adequately resemble the results obtained in our field study.

**Key words :** Phytoplankton ; zooplankton ; toxin ; bloom ; Hopf bifurcation.

[1] This chapter represents our paper published in IMA journal of Mathematics Applied to Medicine and Biology, (2002). This work is realized in collaboration with J. Chattopadhyay and R. Sarkar.

## 4.1 Introduction

Plankton are the basis of all aquatic food chains and phytoplankton in particular occupy the first trophic level. Phytoplankton do huge services for our earth : they provide food for marine life, oxygen for human life and also absorb half of the carbon dioxide which may be contributing to global warming (Duinker & Wefer, 1994). The dynamics of rapid (massive) increase or almost equally decrease of phytoplankton populations is a common feature in marine plankton ecology and known as bloom. This phenomenon can occur in a matter of days and can disappear just as rapidly. In recent years there has been considerable scientific attention towards harmful algal blooms (HABs) (e.g. Blaxter & Southward, 1997; Stoerner & Smol, 1999). Several authors have argued that there has been a global increase in harmful phytoplankton blooms in recent decades (e.g. Anderson *et al.* 1990; Smayda, 1990; Hallegraeff,1993). In a broad sense planktonic blooms may be categorized into two types, 'spring blooms' and 'red tides'. Spring blooms occur seasonally as a result of changes in temperature or nutrient availability which are connected with seasonal changes in thermocline depth and strength, and consequent mixing. Red tides are the result of localized outbreaks associated with water temperature (see Truscott and Brindley, 1994). They are also associated with greater stability of the water column and higher growth rates.

Blooms of blue-green algae have been linked to health problems ranging from skin irritation to liver damage depending on time and duration of exposure. The livelihood of many fish and shellfish have also been endangered due to toxin. Blooms of red tide produce chemical toxins, a type of paralytic poison which can be harmful to zooplankton, finfish, shellfish, fish, birds, marine mammals and humans also. Only a few dozen of the many thousands of species of microscopic or macroscopic algae are repeatedly associated with toxic or harmful blooms. Some species, such as the dinoflagellate *Alexandrium tamarense* and the diatom *Pseudo-nitzschia australis* (Work et al., 1993) produce potent toxins which are liberated into the water before they are eaten and they may well affect zooplankton when they are in water. It is now well established that a significant number of phytoplankton species produce toxin, such as *Pseudo-nitzschia sp, Gambierdiscus toxicus, Prorocentrum sp, Ostrepsis sp, Coolia monotis, Thecadinium sp, Amphidinium cartera, Dinophysis sp, Gymnodinium breve, Alexandrium sp, Gymodinium catenatum, Pyrodinium bahamense, Pfiesteria piscicida, Chrysochromulina polylepis, Prymnesium patelliferum, P. parvum* (see Steidinger *et al.*, 1996; Nielsen *et al.*, 1990; Aure & Rey, 1992; Hallegraeff, 1993).

Reduction of grazing pressure of zooplankton due tu release of toxic substances by phytoplankton is one of the most vital parameters in this context (see Keating, 1976; Lefevre *et al.*, 1952; Kirk & Gilbert, 1992; Fay, 1983). There is also some evidence that herbivore (zooplankton, see Odum, 1971) grazing plays a crucial role in the initial stages of a red tide outbreak (Wyatt & Horwood, 1973; Levin & Segel, 1976; Uye, 1986). Areas rich in some phytoplankton organisms, e.g. *Phaeocystis, Coscinodiscus, Rhizosolenia* are unaccepted/avoided by zooplankton due to dense concentration of phytoplankton or the production of toxic as well as unpleasant factors by them and this phenomena can well be explained by the 'exclusion' principle (see Odum, 1971; Boney, 1976). Buskey & Stockwell (1993) have demonstrated in their field studies that micro and meso zooplankton populations are reduced during the blooms of a chrysophyte *Aureococcus anophagefferens* of the southern Texas coast. Toxicity may be a strong mediator of zooplankton feeding rate, as shown in both field studies (Estep *et al.*, 1990; Nielsen *et al.*, 1990; Hansen, 1995) and laboratory studies (Huntley *et al.*, 1986;

Ives, 1987; Buskey & Hyatt, 1995; Nejstgaard & Solberg, 1996). These observations indicate that the toxic substance as well as toxic phytoplankton plays an important role in the growth of the zooplankton population and has a greater impact on phytoplankton-zooplankton interactions.

The process of production of toxic substances by phytoplankton species is still not clear. Modelling on plankton communities in HABs is very rare in the literature. Franks (1997) reviewed different models which describe the phenomenon of red tide outbreak. To our knowledge, in describing blomm phenomena HAB models do not take into account the effect of toxin which causes the grazing pressure of zooplankton to decrease. The release of toxic substances by phytoplankton may terminate the planktonic blooms-something which is not yet well recognized but cannot be ignored; naturally, interdisciplinary involvement of experimental ecologists and mathematical ecologists is necessary. This study is devoted to establishing the role of toxin in the reduction of grazing pressure of zooplankton with the help of both field study and mathematical modelling. We believe that it is the first model in this direction. Monitoring of plankton population was carried out throught the year 2000 off the north west coast of the Bay of Bengal. As we are interested to report the effect of toxic phytoplankton on zooplankton, we chose *Noctiluca scintillans* (phytoplankton) and *Paracalanus* (zooplankton) for this study. Motivated by our field observations, a mathematical model of the phytoplankton-zooplankton system in which the grazing pressure of zooplankton decreases due to toxic phytoplankton species is proposed and analysed. As the process of toxin liberation is still not clear, we shall try to explain the bloom dynamics by assuming various forms of toxin libeartion process and also by the cyclic nature of the system through periodicity.

## 4.2 Formulation of the model

### 4.2.1 Observational background

In this study we concentrate our observations on the effects of harmful phytoplankton on planktonic blooms, and on what follows. The study area extends from Talsari (Orissa, India) to Digha Mohana (West Bengal, India) on the north west coast of the bay of Bengal (geographically the area is situated between 2137' Northern Latitude, 8725' Eastern Longitude and 2142' Northern Latitude, 8731' Eastern Longitude, see Fig. 1). The study was carried out during the period Jan-Dec 2000. Sampling were done aboard a 10 m fishing vessel hired from the Talsari fish landing centre. Frequency of sampling was every fortnight excepet for the months of September and October when, because of the roughness of the sea, the sampling programme had to be suspended. Plankton samples were collected both from the surface and subsurface water (1-2 m depth) by a horizontal plankton tow with a 20 $\mu$m mesh net $0.3m$ in diameter. The collected samples were preserved in 3% formaldehyde in seawater. Counting of phytoplankton was done under microscope using a Sedgewick-Rafter counting cell and counts are expressed in no/litre. Identification of the plankton community was done following Davis (1955); Newell & Newell (1979) and Tomas (1997). There were altogether 16 sampling days in the year 2000. Numbers of samples (surface collection) analysed were 112. The study reveals the presence of altogether 115 phytoplanktonic species of which 65 are from the diatoms following by 19 of green algae (Chlorophyceae), 9 of blue-greens (Cyanophyceae) and 22 of Dinoflagellates from the surface waters. In each group there were some unidentified species. Out of the total 22 species of Di-

noflagellates identified both from surface and subsurface water, only three species (*Dinophysis acuta, Noctiluca scintillans and Prorocentrum sp.*) were noted as harmful (Richardson, 1997). Six species of the diatoms examined in both the surface and subsurface water (*Chaetoceros spp., Skeletonema costatum, Cerataulina spp., Leptocylindricus spp., Nitzschia spp.* and *Phaeocystis spp.*) are believed to be harmful alga (Sournia, 1995).

Our tested phytoplankton species is *Noctiluca scintillans* belonging to the group *Dinoflagellates*, which is also capable of producing toxin that are released into the seawater. Among zooplankton species we chose *Paracalanus* belonging to the group *Copepoda* which dominates the zooplankton in community in all the word oceans, and is the major herbivore which determines the form of the phytoplankton curve. The blooms of *Noctiluca scintillans* occur in January and December. *Paracalanus* bloom also coexists with *Noctiluca scintillans*. Figure 2 shows that after the bloom of both the species (see the high peak obtained on the sampling date 20/01/2000), *Noctiluca scintillans* decreases and simultaneously the *Paracalanus* also decreases. The population of *Noctiluca scintillans* then remains very low up to the sampling date 29/11/2000 and during this period the *Paracalanus* does not show any large change in population. On the sampling date 13/12/2000 we again observe that both species attain another high peak and then slowly decrease. This observation indicates that *Noctiluca scintillans* attaining the first peak in January (with *Paracalanus* also present in high abundance) starts to release toxic substance, and as a result it controls the bloom of *Paracalanus* population and also its own bloom. This phenomenon persists for a long time (probably due to the effect of toxin concentration) until there is again a low concentration of toxin, both populations again blooms and the process continues. Our experimental result is similar to the observation at Vasilev Bay, where *Paracalanus sp.* decreased drastically after 1987 due to increase in biomass of *Noctiluca scintillans* (see Kideys et al., 2001). Although the chemical toxin released by phytoplankton is not yet tested, the results of our field observations as well as what is already known motivaties us to formulate a mathematical model on the phytoplankton-zooplankton system in which the grazing pressure of zooplankton decreases due to release of toxic substances by the phytoplankton species. It may be noted that the reduction of grazing pressure of zooplankton due to release of toxic substances will have an important role in the termination of planktonic bloom-our analysis bears out this fact very nicely.

FIG. 4.1 – Map of coastal region of West Bengal and part of Orissa, India. (Source : CIFRI, Barrackpore, India.)

### 4.2.2 Mathematical Model

In the formulation of the model we assume that the growth of phytoplankton population follows the logistic law (see Murray, 1989; Odum, 1971) with intrinsic growth rate $r$ and environmental carrying capacity $K$. It is already confirmed by our field observation and the literature that toxic substances released by phytoplankton reduce the grazing pressure of its predator, zooplankton. As the fractional changes in the phytoplankton population per unit time effectively illustrate the impact of predation on the population at any particular time, it is interesting to examine the specific predation rate for the system as the outbreak advances (see Truscott & Brindley, 1994). It is well known that at certain times, conditions (adequate temperature, proper light intensity, warmer water and minimal predation pressure) are adequate for planktonic growth. The phytoplankton will continue to bloom until one or more of the key factors prompting phytoplankton growth is no longer available. Keeping the above-mentioned facts of phytoplankton-zooplankton population in mind, we assume two predational forms for describing the dynamics. When phytoplankton populations do not produce toxin, we assume that the predation rate will follow the simple law of mass action. But as libeartion of toxin reduces the growth of zooplankton, it causes substantial mortality of zooplankton and in

FIG. 4.2 – Field observation.

this period phytoplankton population is not easily accessible, hence a more common and intuitively obvious choice is of the Holling type II functional form to describe the grazing phenomenon. Moreover, saturation of grazing function allows the phytoplankton population to escape from the grazing pressure of the zooplankton and form a tide. This supression of grazing is usually associated with active hunting behaviour on the part of the predator, as opposed to passively waiting to encounter food, and there is a maximum rate of consumption per individual however large the phytoplankton population becomes. Holling type II or type III predational form (Ludwig et al., 1978) is an obvious choice to present the hunting behaviour of predator.
In reality, the raptorial behaviour of Copepods is highly complex and exhibits a hunting behaviour (Uye, 1986), and hence type II or type III is an appropriate choice.

From the above assumptions we can write down the following differential equations :

$$\begin{cases} \frac{dP}{dt} = rP(1 - \frac{P}{K}) - \alpha PZ \\ \frac{dZ}{dt} = \beta PZ - \mu Z - \frac{\theta P}{\gamma + P} Z \end{cases} \tag{4.1}$$

Here $P$ and $Z$ represent the density of phytoplankton and zooplankton population respectively, $\alpha(> 0)$ is the specific predation rate and $\beta(> 0)$ represents the ratio of biomass consumed per zooplankton for the production of new zooplankton. $\mu(> 0)$ is the mortality rate of zooplankton. $\theta(> 0)$ is the rate of toxin production per phytoplankton species and $\gamma(> 0)$ is the half saturation constant.

System (4.1) has to be analysed with the following initial conditions

$$P(0) > 0, Z(0) > 0. \tag{4.2}$$

System (4.1) has the following non-negative equilibria, namely, a trivial equilibrium $E_0(0,0)$, an axial equilibrium $E_1(K,0)$ and the interior equilibrium $E^*(P^*, Z^*)$, where

$$\begin{aligned} P^* &= \frac{-(\beta\gamma - \mu - \theta) + \sqrt{(\beta\gamma - \mu - \theta)^2 + 4\beta\gamma\mu}}{2\beta} \\ Z^* &= \frac{r}{\alpha}(1 - \frac{P^*}{K}). \end{aligned} \tag{4.3}$$

A simple algebraic calculation shows that a necessary and sufficient condition for the existence of positive equilibrium $E^*$ is

$$\theta < (\beta K + \beta\gamma - \mu) - \frac{\gamma\mu}{K}. \tag{4.4}$$

We first observe that the right hand side of system (4.1) is a smooth function of the variables $(P, Z)$ and the paramaters, as long as these quantities are non-negative, so local existence and uniquness properties hold in the positive quadrant.
From the first equation of system (4.1), it follows that $P = 0$ is an invariant subset, that is $P = 0$ if and only if $P(t) = 0$ for some $t$. Thus, $P(t) > 0$ for all $t$ if $P(0) > 0$. A Similar argument follows for $Z = 0$ from the second equation of system (4.1).
Now, we consider the boundedness of solutions of system (4.1).

**Lemma 1.** All the solutions which initiate in $\{R^2_+ \backslash 0\}$ are uniformly bounded.

**Proof.** We define a function

$$W = \beta P + \alpha Z. \tag{4.5}$$

The time derivative of (4.5) along the solutions of (4.1) is

$$\begin{aligned} \frac{dW}{dt} &= r\beta P(1 - \frac{P}{K}) - \alpha\mu Z - \frac{\alpha\theta P}{\gamma + P} Z \\ &\leq r\beta P(1 - \frac{P}{K}) - \alpha\mu Z \\ &= r\beta P(1 - \frac{P}{K}) + \mu\beta P - \mu W. \end{aligned}$$

The term $[r\beta P(1 - \frac{P}{K}) + \mu\beta P]$ has a maximum value, so the above expression reduces to

$$\begin{aligned} \frac{dW}{dt} + \mu W &\leq C \\ \text{where } C &= \frac{\beta K(\mu + r)^2}{4r}. \end{aligned}$$

Applying the theorem of differential inequality (Birkhoff and Rota, 1982), we obtain

$$0 < W(P, Z) \leq \frac{C}{\mu}(1 - e^{-\mu t}) + W(P(0), Z(0))e^{-\mu t}$$

and for $t \to \infty$, we have

$$0 < W \leq \frac{\beta K \mu (1 + \frac{\gamma}{\mu})^2}{4r}.$$

Hence all the solutions of (4.1) that initiate in $\{R_+^2 \backslash 0\}$ are confined in the region

$$B = \{(P, Z) \in R_+^2 : W = \frac{C}{\mu} + \epsilon\},$$

for any $\epsilon > 0$ and for $t$ large enough.

*Note.* The upper bound of $W$ implies that the linear combination of phytoplankton-zooplankton population is less than a finite quantity which is determined by the ratio of the effective growth rate of zooplankton to the net growth rate of phytoplankton.

Before analysing the model system, we would like to mention the meaning of the periodic nature of blooms. It is well established that the occurrence of more than one bloom in a season suggets that the features influencing a red tide event are cyclic (e.g. see Satora & Laws, 1989). The periodic nature of blooms, in the sense of the rapid onset and disappearance of oscillations under supposedly favourable environmental condition, is one of the main characteristics in plankton ecosystems. This may happen in two ways : namely multistability, in which the system tends to one of the coexisting stable equilibria, and periodicity (Hopf bifurcation), in which the system oscillates around an unstable equilibrium. At this point it may be mentioned that an external forcing agent in a proper measure can also bring out the essential physicalities of the system under study. But we feel that such an addition only supresses a proper understanding of the system. This is because, given the extent of the regulatory behaviour shown by the system, the external forcing agent remains to a large extent arbitrary and needs very fine tuning for which there may not be any adequate explanation.

Hence we are trying to explore a suitable mechanism for planktonic blooms which is present within the system.

## 4.3 Stability Analysis

Local stability analysis (LAS) of system (4.1) around the equilibria can be studied by computing variational matrix.

It is easy to see that the trivial equilibrium $E_0$ is an unstable saddle point. Existence of a positive interior equilibrium implies that the axial equilibrium $E_1$ is also an unstable saddle in character. Violation of positive equilibrium ensures that $E_1$ is locally asymptotically stable. The characteristic equation of system (4.1) around the positive interior equilibrium $E^*$ is given by

$$\lambda^2 - M\lambda + N = 0$$

where

$$\begin{aligned} M &= -\frac{rP^*}{K}(< 0) \\ N &= \frac{\alpha P^* Z^*}{(\gamma + P^*)^2}[\beta(\gamma + P^*)^2 - \theta\gamma]. \end{aligned}$$

It can be easily verified from the second equation of system (4.1) that $\theta \geq \frac{\beta(\gamma+P^*)^2}{\gamma}$ can never be a solution of the system, hence simple bifurcation is also not possible in this case.
Now since $M < 0$, the system (4.1) around $E^*(P^*, Z^*)$ is locally asymptotically stable.
To investigate the global behaviour of system (4.1) we first prove that system (4.1) around $E^*$ has no nontrivial periodic solutions. The proof is based on an application of a divergence criterion (Hale, 1993).
Let $h(P,Z) = \frac{1}{PZ}$.
Obviously $h(P,Z) > 0$ if $P > 0, Z > 0$.
We define :

$$\begin{aligned} g_1(P,Z) &= rP(1-\frac{P}{K}) - \alpha PZ \\ g_2(P,Z) &= \beta PZ - \mu Z - \frac{\theta PZ}{\gamma+P} \\ \Delta(P,Z) &= \frac{\partial}{\partial P}(g_1 h) + \frac{\partial}{\partial Z}(g_2 h), \end{aligned}$$

and find that

$$\Delta(P,Z) = -\frac{r}{KZ}$$

which is less than zero for all $P > 0, Z > 0$. Therefore by the Bendixson-Dulac criterion, there will be no limit cycle in the first quadrant.
Now, we are in a position to prove the following theorem.

**Theorem 1.** *Existence of a positive interior equilibrium ensures that system (4.1) around $E^*(P^*, Z^*)$ is globally asymptotically stable.*

**Proof.** The proof is based on the following arguments :
(a)-System (4.1) is bounded and positively invariant in the first quadrant if $\theta < (\beta K + \beta\gamma - \mu) - \frac{\gamma\mu}{K}$
(b)-Trivial equilibrium $E_0$ is always an unstable saddle point and existence of positive equilibrium confirms that the axial equilibrium $E_1$ is also an unstable saddle point.
(c)-Positive equilibrium $E^*$ is LAS.
(d)-System (4.1) around $E^*$ has no non-trivial periodic solutions.

From the above observation we find that there is no chance of exchange of stability. Hence the cyclic nature of the bloom phenomenon which is very common in marine phytoplankton-zooplankton systems can not be explained by the above mechanism. At this stage we wish to mention that various combinations of predational functional response and toxin liberation process give rise to exchange of stability through Hopf bifurcation or multistability of the positive equilibrium but in this study we are mainly interested in presenting a mechanism for planktonic blooms in which the liberation of toxic substance or the effect of toxic phytoplankton is not an instantaneous process but is mediated by some time lag.

## 4.4 Model with distributed delay

We assume that the liberation of toxic substances by phytoplankton species is not an instantaneous process but is mediated by some time lag required for maturity of the species. There are also several reports that the zooplankton mortality due to the toxic phytoplankton bloom occurs after some time lapse (see http ://www.mote.org, http ://www.mdsg.umd.edu). Our field observation also suggests that the abundance of *Paracalanus* (zooplankton) population reduces after some time lapse of the bloom of toxic phytoplankton *Noctiluca scintillans* (see Fig. 2) and this allows us some considerable freedom for considering the delay factor in the model construction.
It is not usually possible to know the past history of the release of toxic substances by phytoplankton or the actual form of the delay kernel. So a particular member of the family of kernels is at best an approximation. To search for excitability (and/or) a cyclic nature of blooms in the system we now assume that the release of toxic substances by the phytoplankton population follows a gamma distribution . This form of distributed delay kernel has been widely used in biological modelling (see Cushing, 1997; MacDonald, 1978, and references therein) and seems to be the most useful family of reducible kernels (i.e. delay kernels that allow a distributed delay model to be converted to an equivalent system of ordinary differential equations). These kernels are not only mathematically convenient, but also linear combinations of them represent a generic class of distributed delay kernels (see Busenberg & Travis, 1982). In this case system (4.1) can be represented as

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha PZ \\ \frac{dZ}{dt} = \beta PZ - \mu Z - \theta \left[ \int_{-\infty}^{t} \sigma^{k+1} \frac{(t-s)^k}{k!} e^{-\sigma(t-s)} \frac{P(s)}{\gamma + P(s)} ds \right] Z \end{cases} \tag{4.6}$$

Here $k$, a non negative integer, is the order of the delay kernel and $\sigma$, is real non negative. These are linked to the mean time lag by $T = \frac{k+1}{\sigma}$. It is interesting to note that when the value of $k$ increases then the phytoplankton consumed in the past by zooplankton becomes more important compared to the case when $k$ is small. In particular, when $k = 1$ we have a strong kernel and when $k = 0$ we have a weak kernel in the memory function.
This system also possesses the same equilibria as in system (4.1).
Stability analysis of each equilibrium can be performed by using a variational matrix. The behaviour of this system around $E_0$ and $E_1$ is the same as we observed in the previous case.

The characteristic equation of system (4.6) around $E^*(P^*, Z^*)$ is

$$\lambda(\lambda + \frac{rP^*}{K}) - \frac{\theta\alpha\gamma P^* Z^*}{(\gamma + P^*)^2} G_k(\lambda) + \alpha\beta P^* Z^* = 0$$

where

$$G_k(\lambda) = \int_{-\infty}^{t} \sigma^{k+1} \frac{(t-s)^k}{k!} e^{-(\sigma+\lambda)(t-s)} ds.$$

We shall study system (4.6) with $k = 1$.

In this case $G_1(\lambda) = \left(\frac{\sigma}{\lambda+\sigma}\right)^2$ and the characteristic equation becomes

$$\lambda(\lambda + \frac{rP^*}{K})(\lambda+\sigma)^2 - \frac{\theta\alpha\gamma P^*Z^*}{(\gamma+P^*)^2}\sigma^2 + \alpha\beta P^*Z^*(\lambda+\sigma)^2 = 0. \tag{4.7}$$

Equation (4.7) can be written in the form

$$\lambda^4 + f_1(\sigma)\lambda^3 + f_2(\sigma)\lambda^2 + f_3(\sigma)\lambda + f_4(\sigma) = 0 \tag{4.8}$$

where

$$\begin{aligned} f_1(\sigma) &= 2\sigma + \frac{rP^*}{K} \\ f_2(\sigma) &= \sigma^2 + \frac{2\sigma rP^*}{K} + \alpha\beta P^*Z^* \\ f_3(\sigma) &= \frac{\sigma^2 rP^*}{K} + 2\alpha\beta\sigma P^*Z^* \\ f_4(\sigma) &= \alpha\beta\sigma^2 P^*Z^* - \frac{\theta\alpha\gamma\sigma^2 P^*Z^*}{(\gamma+P^*)^2}. \end{aligned}$$

By using the Routh-Hurwitz criterion, we find that the real part of all roots are negative. So in this case also there is no possibility for exchange of stability. Hence the cyclic nature of blooms cannot be explained by this type of distribution of toxic substance or toxic phytoplankton. The prediction based on the system involving distributed delay illustrates that concentration of toxic substances or toxic phytoplankton eventually approaches equilibrium concentration and hence no periodic solutions are possible. It is also worth noting that if the order $(k)$ of the delay kernel, goes to infinity while keeping the mean delay, $T = \frac{k+1}{\sigma}$ fixed, then the distributed delay can be viewed as a discrete delay (for details see Wolkowicz et al., 1997). Now, to explain the periodic nature of bloom phenomena we shall assume the process of toxic liberation as a break-even point by discrete delay.

## 4.5 Model with discrete delay

We now assume that the process of toxic liberation follows a discrete time variation.

System (4.1) now takes the form

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha PZ \\ \frac{dZ}{dt} = \beta PZ - \mu Z - \frac{\theta P(t-\tau)}{\gamma+P(t-\tau)}Z \end{cases} \tag{4.9}$$

where $\tau$ is the discrete time delay.

As in the previous two cases system (4.9) has the same equilibria. System (4.9) around $E_0$ is an unstable saddle. Existence of $E^*$ implies that $E_1$ is also an unstable saddle. Non existence of $E^*$ implies that $E_1$ is stable in nature.

To investigate local asymptotic stability of system (4.9) around $E^*$ we perturb the system (4.9) around $E^*(P^*, Z^*)$ and obtain the following system of differential equations

$$\begin{cases} \frac{dx}{dt} = Ax + By + a_{11}xy + a_{20}x^2 \\ \frac{dy}{dt} = Cx + Ex(t-\tau) + b_{11}xy + b'_{11}\frac{x(t-\tau)y}{\gamma+x(t-\tau)} + b'_{12}\frac{x(t-\tau)}{\gamma+x(t-\tau)} \end{cases} \tag{4.10}$$

where

$$\begin{gathered} x = P - P^*,\ y = Z - Z^*,\ A = -\frac{rP^*}{K}, \\ B = -\alpha P^*,\ C = \beta Z^*,\ E = -\frac{\theta\gamma Z^*}{(\gamma+P^*)^2}, \\ a_{11} = -\alpha,\ a_{20} = \frac{-r}{K},\ b_{11} = \beta, \\ b'_{11} = \theta,\ b'_{12} = -\theta Z^*. \end{gathered} \tag{4.11}$$

Retaining only the linear terms in (4.10), the linearized system becomes

$$\begin{cases} \frac{dx}{dt} = Ax + By \\ \frac{dy}{dt} = Cx + Ex(t-\tau). \end{cases} \tag{4.12}$$

System (4.12) can be written as

$$\frac{d^2x}{dt^2} - A\frac{dx}{dt} - BCx - BEx(t-\tau) = 0. \tag{4.13}$$

We assume a solution of the form $x(t) = e^{\lambda t}$, and we have the corresponding characteristic equation as

$$\Delta(\lambda, \tau) = \lambda^2 - A\lambda - BC - BEe^{-\lambda\tau} = 0. \tag{4.14}$$

Now substituting $\lambda = \alpha_1 + i\omega$ in (4.14) and separating the real and imaginary parts we obtain the system of transcendental equations

$$\begin{gathered} {\alpha_1}^2 - \omega^2 - A\alpha_1 - BC - BEe^{-\alpha_1\tau}\cos\omega\tau = 0 \\ 2\alpha_1\omega - A\omega + BEe^{-\alpha_1\tau}\sin\omega\tau = 0. \end{gathered} \tag{4.15}$$

The stability or instability of the system is determined by the sign of those $\lambda$ satisfying (4.14) if $\lambda$ is real or the sign of $\alpha_1$ satisfying (4.15) if $\lambda$ is complex.

**Theorem 2.**
*The following are necessary and sufficient conditions for $E^*$ to be asymptotically stable for every $\tau \geq 0$ :*
*(1) The real part of every root of $\Delta(\lambda, 0) = 0$ is negative.*
*(2) For all real $\omega_0$ and $\tau \geq 0$, $\Delta(i\omega_0, \tau) \neq 0$.*

**Theorem 3.**
*As $A < 0$ and $B < 0$, then in the parametric region $-E < C$ the interior equilibrium $E^*$ of system (4.9) is locally asymptotically stable for $0 < \tau < \frac{\pi}{\omega_0}$.*

**Proof.** From (4.14) it is clear that $E^*$ is asymptotically stable for $\tau = 0$ if $-E < C$.
Proving the second condition of theorem 2 requires the Nyquist criterion and its consequences.

Consider (4.12) and the space of all real-valued continuous functions defined on $[-\tau, \infty)$ satisfying the initial condition $x(t) = 0$ for $-\tau \leq t < 0$, $x(0^+) = P_1 > 0$ and $\dot{x}(0^+) = P_2 > 0$. After taking the Laplace transform of (4.13) and simplifying, we have

$$L(x(s)) \equiv L(s) = \frac{P_1 s + P_2 - AP_1}{s^2 - As - BC - BEe^{-\tau s}}. \tag{4.16}$$

The inverse Laplace transform of $L(s)$ will have terms which increase exponentially with $t$ if $L(s)$ has poles with positive real part. Thus it is clear that a condition for stability of $E^*$ is that all poles of $L(s)$ have negative real parts. We apply the Nyquist criterion (see Thingstad and Langeland, 1974) to assess whether $L(s)$ has any pole in the right half-plane.

This criterion leads us to the conditions

$$Im\ \psi(i\omega_0) > 0. \tag{4.17}$$

$$Re\ \psi(i\omega_0) = 0. \tag{4.18}$$

where

$$\psi(s) = s^2 - As - BC - BEe^{-\tau s}, \tag{4.19}$$

with $\omega_0$ the smallest positive value of $\omega$ for which equation (4.18) holds. Now,

$$\psi(i\omega_0) = -{\omega_0}^2 - iA\omega_0 BC - BE(\cos\omega_0\tau - i\sin\omega_0\tau). \tag{4.20}$$

$$Im\ \psi(i\omega_0) = -A\omega_0 + BE\sin\omega_0\tau \tag{4.21}$$

and

$$Re\ \psi(i\omega_0) = -{\omega_0}^2 - BC - BE\cos\omega_0\tau. \tag{4.22}$$

Writing conditions (4.17) and (4.18) using the expressions (4.21) and (4.22) and taking account of $B < 0$ and $E < 0$ we obtain $\frac{A}{BE\tau} < \frac{\sin\omega_0\tau}{\omega_0\tau}$ and ${\omega_0}^2 = -BC - BE\cos\omega_0\tau$.

Since $A < 0, -(BE) < 0$, condition (4.17) is satisfied for $0 < \tau < \frac{\pi}{\omega_0}$.
Further since $B < 0,\ E < 0$ we have

$$-BC - BE < -BC - BE\cos\omega_0\tau < -BC + BE.$$

Hence $z = {\omega_0}^2$ and $z = -BC - BE\cos\omega_0\tau$ intersect on $0 < \omega_0 < \frac{\pi}{\tau}$. From (4.22) we also have (in the parametric region $-E < C$)

$$0 < -BC - BE < {\omega_0}^2 < -BC + BE,\ for\ 0 < \omega_0 < \tfrac{\pi}{\tau} \tag{4.23}$$

so we have an upper bound $\omega_+$ of $\omega_0$ given by

$$\omega_+ = \sqrt{BE - BC}. \tag{4.24}$$

Hence we can conclude that in our case the Nyquist criterion holds and the interior equilibrium $E^*$ of the system (4.9) is locally asymptotically stable for all values of $\tau$ satisfying $0 < \tau < \frac{\pi}{\omega_0}$.

## 4.6 Bifurcation of the solutions

In this section we state a condition under which the system goes through a point where a Hopf bifurcation occurs. We show the existence of such a $\tau$ $(= \tau_0)$ and $\omega$ $(= \omega_0)$.

**Lemma 2.** *If $A^2 + 2BC < 0$ and $0 \leq C < -E$ then there exists a unique pair $(\omega_0,\ \tau_0)$ with $\omega_0,\ \tau_0 \geq 0$, $\omega_0\tau_0 < 2\pi$ such that $\Delta(i\omega_0, \tau_0) = 0$, where $\omega_0$ and $\tau_0$ are given by (4.28) and (4.33), respectively.*

**Proof.** From $\Delta(i\omega_0, \tau_0) = 0$ and from (4.21) and (4.22) we have

$$-A\omega_0 + BE\sin\omega_0\tau_0 = 0 \tag{4.25}$$

and

$$-{\omega_0}^2 - BC - BE\cos\omega_0\tau_0 = 0. \tag{4.26}$$

Squaring and adding together (4.25) and (4.26) we arrive at

$${\omega_0}^4 + (A^2 + 2BC){\omega_0}^2 + (B^2C^2 - B^2E^2) = 0. \tag{4.27}$$

We see from (4.27) that $\lambda$ has a pair of purely imaginary roots of the form $\pm i\omega_0$ provided $A^2 + 2BC < 0$ and $0 \leq C \leq -E$.
The corresponding roots of (4.27) in this case are

$${\omega_0}^2 = \tfrac{1}{2}[-(A^2 + 2BC) + \sqrt{(A^2 + 2BC)^2 - 4(B^2C^2 - B^2E^2)}\ ] \tag{4.28}$$

Using (4.25) in (4.26), we obtain

$$-[\frac{(BE)^2(1 - \cos^2\omega_0\tau_0)}{A^2}] - BC - BE\cos\omega_0\tau_0 = 0$$

or

$$(BE)^2\cos^2\omega_0\tau_0 - BEA^2\cos\omega_0\tau_0 - BCA^2 - (BE)^2 = 0. \tag{4.29}$$

Set

$$f(z) = (BE)^2z^2 - BEA^2z - BCA^2 - (BE)^2 = 0. \tag{4.30}$$

We have

$$f(1) = -BE^2(C + E) < 0 \tag{4.31}$$

and

$$f(-1) = -BE^2(C - E) > 0 \tag{4.32}$$

Hence $f(z)$ has a real solution in (-1,1) of the form $\cos\omega_0\tau_0 = k$, where $|k| < 1$.
From (4.25),

$$\tau_0 = \tfrac{1}{\omega_0}\arcsin(\tfrac{-A\omega_0}{BE}) + \tfrac{2n\pi}{\omega_0},\ n = 0,\ 1,\ 2, .... \tag{4.33}$$

In (4.28) we assume $A^2+2BC<0$, so that there is only one imaginary solution $\lambda=i\omega_0$ ($\omega_0>0$) and therefore the only crossing of imaginary axis is from left to right as $\tau$ increases and the stability of the trivial solution can only be lost and not regained.
Obviously in this case n=0.

**Lemma 3.** *Let $A^2+2BC<0, 0\le C<-E$. Then the real parts of the solutions of (4.14) are negative for $\tau<\tau_0$, where $\tau_0>0$ is the smallest value for which there is a solution to (4.14) with real part zero. For $\tau>\tau_0$, $E^*$ is unstable. Further as $\tau$ increases through $\tau_0$, $E^*$ bifurcates into small amplitude of periodic solutions.*

**Proof.** For $\tau=0$, it is obvious that $E^*$ is stable. Hence by Butlers' lemma (see Freedman & Rao, 1983), $E^*$ remains stable for $\tau<\tau_0$. We have now to show that $\frac{d\alpha_1}{d\tau}|_{\tau=\tau_0}>0$ where $\omega=\omega_0$ (for n = 0, 1, 2,...). This will signify that there exists at least one eigenvalue with positive real part for $\tau>\tau_0$, and hence $E^*$ is unstable for $\tau>\tau_0$. Moreover, the condition for Hopf bifurcation (see Hale, 1993) are then satisfied yielding the required periodic solutions. Now differentiating (4.15) with respect to $\tau$, we get

$$\begin{aligned}\{2\alpha_1-A+BE\tau e^{-\alpha_1\tau}\cos\omega\tau\}\tfrac{d\alpha_1}{d\tau}+\{-2\omega+BE\tau e^{-\alpha_1\tau}\sin\omega\tau\}\tfrac{d\omega}{d\tau}\\ =BEe^{-\alpha_1\tau}\{-\alpha_1\cos\omega\tau-\omega\sin\omega\tau\}\end{aligned}\tag{4.34}$$

and

$$\begin{aligned}\{2\omega-BE\tau e^{-\alpha_1\tau}\sin\omega\tau\}\tfrac{d\alpha_1}{d\tau}+\{2\alpha_1-A+BE\tau e^{-\alpha_1\tau}\cos\omega\tau\}\tfrac{d\omega}{d\tau}\\ =BEe^{-\alpha_1\tau}\{\alpha_1\sin\omega\tau-\omega\cos\omega\tau\}\end{aligned}\tag{4.35}$$

Therefore

$$[\{2\alpha_1-A+BE\tau e^{-\alpha_1\tau}\cos\omega\tau\}^2-(2\omega-BE\tau e^{-\alpha_1\tau}\sin\omega\tau)\{-2\omega+BE\tau e^{-\alpha_1\tau}\sin\omega\tau\}]\frac{d\alpha_1}{d\tau}$$

$$=BE\tau e^{-\alpha_1\tau}[(-\alpha_1\cos\omega\tau-\omega\sin\omega\tau)\{2\alpha_1-A+BE\tau e^{-\alpha_1\tau}\cos\omega\tau\}$$

$$-(\alpha_1\sin\omega\tau-\omega\cos\omega\tau)(-2\omega+BE\tau e^{-\alpha_1\tau}\sin\omega\tau)]$$

Now at $\alpha_1=0,\ \tau=\tau_0,\ \omega=\omega_0$, we have

$$\begin{aligned}&[\{-A+BE\tau_0\cos\omega_0\tau_0\}^2+\{2\omega_0-BE\tau_0\sin\omega_0\tau_0\}^2]\tfrac{d\alpha_1}{d\tau}|_{(\alpha_1 0,\ \tau=\tau_0,\ \omega=\omega_0)}\\ &=BE[-\omega_0\sin\omega_0\tau_0\{-A+BE\tau_0\cos\omega_0\tau_0\}+\omega_0\cos\omega_0\tau_0(-2\omega_0+BE\tau_0\sin\omega_0\tau_0)]\\ &=BE\omega_0[A\sin\omega_0\tau_0-2\omega_0\cos\omega_0\tau_0]\\ &=BE\omega_0 p_1\cos(\omega_0\tau_0-\theta_1)>0\end{aligned}\tag{4.36}$$

where ${p_1}^2=A^2+4{\omega_0}^2$, $\tan\theta_1=(\frac{-A}{2\omega_0})$.
Hence $\frac{d\alpha_1}{d\tau}|_{(\alpha_1 0,\ \tau=\tau_0,\ \omega=\omega_0)}>0$. Therefore the transversality condition holds and hence a Hopf bifurcation occurs at $\omega=\omega_0,\ \tau=\tau_0$.
The stability of the bifurcating branches is given in the appendix.

## 4.7 Discussion

The dynamics of planktonic bloom is very complex and the role of algal toxin in the complex ecology of HABs is still not clear. Researchers are trying to find a suitable mechanism for this. Apart from some noticeable poisoning by phytoplankton, the ecological consequences of algal toxins are also not well elaborated. This allows us some considerable freedom to formulate a mathematical model. A simple mathematical model of phytoplankton-zooplankton (prey-predator) system in which the grazing pressure of zooplankton reduce due to release of toxic chemical by phytoplankton or due to toxic phytoplankton being eaten by zooplankton has been proposed and analysed. In our study we have tried to establish the following three major processes :
(i) the cyclic nature of the phytoplankton-zooplankton system around the positive equilibrium,
(ii) that phytoplankton start to release toxic chemical or become toxic very quickly in the presence of dense zooplankton population ; as the result the grazing pressure decreases, and
(iii) the toxic effect on zooplankton will help in the termination of blooms.

It was stated clearly in the introduction that toxic phytoplankton or toxic chemicals reduce the growth of zooplankton populations and as the process of toxic liberation is still not clear, we have investigated the model under three types of distribution of toxic substances. We have observed that the cyclic nature of blooms which are a very common feature in the planktonic world can not be explained by our model formulation if the distribution of toxic substances is of Holling type II or if it follows the gamma type distribution, whereas if the distribution of toxin substances is of discrete type, we have observed that the system around the positive equilibrium enters a Hopf-type bifurcation and exhibits the cyclic nature of blooms for a certain amount of time delay. To ascertain this local behaviour we have performed the stability analysis of bifurcating periodic solutions (see the appendix) and obtained the conditions for supercritical and subcritical bifurcations. In most situations, the oscillations phenomena of ecological systems are generally described by distributed delay models. The point is that reduction of grazing pressure on phytoplankton due to release of toxin is not continous but follows a discrete fashion. The research by JoAnn Burkholder and others at North Carolina State University also reflects our observation. They suggest that *Pfiesteria piscicida* assumes more than 20 different forms during its lifetime, including a difficult-to-detect cyst stage, an amoeboid stage and a toxic vegetative stage, in which, propelled by its flagella, it can kill its predator (see http ://www.mdsg.umd.edu/MarineNotes/Jul-Aug97).
To substantiate the analytical findings we have used the parameter values which are presented and discussed elaborately in Edwards & Brindley (1999). Abbreviations, default values (which we have used) and the ranges of the parameter values are given in Table 1. For these sets of values and for $\tau = 18h$, $K = 400l^{-1}$ and $\theta = 0.9h^{-1}$, we have obtained the values of $\beta_1 = 0.0223115 - 6.337212i$, $\overline{\nu_1} = -0.0007769111 + 0.07877564i$, $\overline{\nu_2} = 0.4992216 + 0.004632169i$, $g_{20} = 0.5868627 + 3.29125i$, $g_{02} = 2.349286 + 3.496705i$, $g_{11} = -7.952592 - 0.08011834i$, $g_{21} = 33.582 - 13.22366i$, $Re\ C_1(0) = -13.89942$, $\mu_2 = 53499.1$ and $\tau_2 = 621.9235$.
For these sets of parameter values we have obtained $\mu_2 > 0$, the bifurcation is supercritical and the system exhibits a stable limit cycle. Further since $\tau_2 > 0$, the period of the oscillations increases with $\tau$. Numerical solutions of (9) were carried out using the modified fourth-order-Runge-Kutta method. The results indicate that the equilibrium solution is stable (by decaying oscillations) for $0 \leq \tau < 18$ and unstable (by growing oscillations) for $\tau > 18$ (see figure 3 and figure 4). The system exhibits a stable limit cycle periodic solution at the bifurcation value $\tau_0 = 18h$ (see figure 5, which is quite

reasonable for the life span of phytoplankton). This observation indicates that there is a threshold limit $\tau$, below which the system shows no excitability and above which the system enters into excitable range. These findings demonstrate the delayed effect of toxic phytoplankton and the cyclic nature of blooms in this phytoplankton-zooplankton system. We would like to mention here that in our field study we observed that the blooms reappear after 10 months whereas our model simulation shows that the blooms reappear after 6 months. The above findings show that the toxin producing planktons may act as biological control for the termination of planktonic blooms. Although these results give only quantitative agreement (this may be due to sampling process, environmental factors, etc.) this fact cannot be ignored. We believe that biologists might be interested in this idea and will perform more explicit studies in the laboratory in this direction.

We further observed that when the ratio of initial phytoplankton-zooplankton population was 5 :1, the system around positive equilibrium exhibits a stable limit cycle for $\tau = 18h$, but when there is a dense concentration of zooplankton (we chose initial phytoplankton-zooplankton population ratio as 3 :1) the time lag decreases and the periodicity (through the stable limit cycle) occurs at $\tau = 6h$ (see Fig.6). This result shows the sensitivity of toxic phytoplankton in the presence of dense zooplankton populations. Diminution of time lag also implies that toxin acts as a controling agent in the presence of dense zooplankton. The results obtained by our field observation also suggest that toxic substances or toxic phytoplankton may serve as a key factor in the termination of planktonic blooms. Thus, we may conclude that the above observations establish the role of toxin in the reduction of grazing pressure of zooplankton. It may also be noted that the experimental and mathematical observations of Chattopadhyay *et al.* (2002) and the experimental research of Buskey & Stockwell (1993) support our conclusion.

Finally, we would like to mention that the dynamics of the planktonic community, specifically the understanding of the role of HABs in the planktonic world, is still in a state of infancy and hence interdisciplinary involvement is necessary. For example, the life stage of an individual (larva, juvenile or adult) will also greatly affect the response to a toxic substance. In general, larvae and juveniles are more vulnerable to injury or death from exposure to these substances. Studies of the effects of toxic substances must consider both the age an species of specimens to fully access the chemical toxicity. Also, to study the dynamics under the presence of external force may be another interesting problem in this context, as massive phytoplankton blooms were observed in Seto Inland Sea, Japan (Prakash, 1987) and in Hong Kong Harbour (Lam & Ho, 1989) which were due to artificial eutrophication, although we feel such an approach may be viewed as very artificial and hence at present we have avoided it. So, all the possible mechanisms existing in the planktonic world may not be captured in a single mathematical model. However, the present simple model with its outcome may give some insight to researchers of this very complex and important issue.

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## Appendix. Stability of the bifurcation.

Here we determine a formula that establishes the stability of bifurcating periodic orbits. The calculation is based on Hassard *et al.* (1981). We assume the case where Hopf bifurcation occurs (at $\tau = \tau_0$ and $\omega = \omega_0$) and using the standard notation as in Hassard *et al.* (1981) we rewrite (4.10) in the form

$$\dot{x}_t = A_{\alpha'} x_t + R x_t \tag{4.37}$$

where $x_t \in C([-\tau, 0], \Re)$ is given by $x_t(\theta') = x(t + \theta')$; $\alpha'$ represents the parameter values at $\tau = \tau_0$, $\omega = \omega_0$.

$$A_{\alpha'}\phi(\theta') \begin{cases} \frac{d\phi}{d\theta'} & -\tau \leq \theta' < 0 \\ \int_{-\tau}^{0} d\eta(\rho, \alpha')\phi(\rho) & \theta' = 0. \end{cases} \tag{4.38}$$

$$R\phi(\theta') = \begin{cases} \begin{pmatrix} 0 \\ 0 \end{pmatrix} & -\tau \leq \theta' < 0 \\ \begin{pmatrix} a_{11}\phi_1(\theta')\phi_2(\theta') + a_{20}\phi_1^2(\theta') \\ \{b_{11}\phi_1(\theta')\phi_2(\theta') + b_{11}'\frac{\phi_1(\theta'-\tau)}{\gamma+\phi_1(\theta'-\tau)}\phi_2(\theta') \\ +b_{12}'\frac{\phi_1(\theta'-\tau)}{\gamma+\phi_1(\theta'-\tau)}\} \end{pmatrix} & \theta' = 0 \end{cases}$$

$$d\eta(\theta';\alpha') = \begin{pmatrix} A\ \delta(\theta') & B\ \delta(\theta') \\ C\ \delta(\theta') + \alpha'\delta(\theta'+\tau) & D\ \delta(\theta') \end{pmatrix} d\theta'$$

An eigenfunction of the problem corresponding to the eigenvalue $i\omega_0$

$$q(\theta') = \begin{pmatrix} \beta_1 \\ 1 \end{pmatrix} e^{i\omega_0\theta'}$$

where,

$$\beta_1 \frac{B}{\lambda - A}$$

At $\lambda = i\omega_0$

$$\beta_1 = \frac{AB + iB\omega_0}{A^2 + \omega_0^2}$$

Now we define the following bilinear form as

$$\langle \psi, \phi \rangle \overline{\psi(0)}\phi(0) - \int_{\theta'-\tau}^{0} \int_{\xi=0}^{\theta'} \overline{\psi(\xi - \theta')}[d\eta(\theta')]\phi(\xi)d\xi$$

So, to obtain the corresponding adjoint eigenfunction $\acute{q}(\theta')$, we use the standard result $\langle \acute{q}, q \rangle = 1$ and $\langle \acute{q}, \bar{q} \rangle = 0$, letting $\acute{q}e^{i\omega_0\theta'}(\nu_1, \nu_2)$, then we have

$$\langle \acute{q}, q \rangle = \overline{\acute{q}(0)}q(0) - \int_{-\tau}^{0} \int_{0}^{\theta'} \overline{\acute{q}(\xi - \theta')}d\eta(\theta')q(\xi)d\xi$$

$$\begin{aligned} &= \beta\bar{\nu_1} + \bar{\nu_2} - \int_{-\tau}^{0} \int_{0}^{\theta'} e^{-i\omega_0(\xi-\theta')}(\bar{\nu_1}\bar{\nu_2}) \begin{pmatrix} 0 \\ \alpha'\delta(\theta'+\tau) \end{pmatrix} \begin{pmatrix} \beta_1 \\ 1 \end{pmatrix} e^{i\omega_0\xi}d\theta' d\xi \\ &= \beta\bar{\nu_1} + \bar{\nu_2} - \int_{-\tau}^{0} \int_{0}^{\theta'} e^{i\omega_0\theta'}\alpha'\bar{\nu_2}\delta(\theta'+\tau)d\theta' d\xi \\ &= \beta\bar{\nu_1} + \bar{\nu_2} - \alpha'\bar{\nu_2}(\tau\cos\omega_0\tau - i\tau\sin\omega_0\tau) \end{aligned}$$

Therefore

$$\beta_1\bar{\nu_1} + \bar{\nu_2}(1 - \alpha'\tau\ \cos\omega_0\tau + i\alpha'\tau\ \sin\omega_0\tau) = 1$$

$$\begin{aligned}\langle \acute{q}, \bar{q} \rangle &= \bar{\beta}_1\bar{\nu}_1 + \bar{\nu}_2 \\ &\quad - \int_{-\tau}^{0}\int_{0}^{\theta'} e^{-i\omega_0(\xi-\theta')}(\bar{\nu}_1 \;\; \bar{\nu}_2)\begin{pmatrix} 0 \\ \alpha'\delta(\theta'+\tau)\end{pmatrix}\begin{pmatrix}\bar{\beta}_1 \\ 1\end{pmatrix} e^{-i\omega_0\xi}d\theta' d\xi \\ &= \bar{\beta}_1\bar{\nu}_1 + \bar{\nu}_2 - \int_{-\tau}^{0}\int_{0}^{\theta'} e^{-i\omega_0(\xi-\theta')}\alpha'\bar{\nu}_2\delta(\theta'+\tau)e^{-i\omega_0\xi}d\theta' d\xi\end{aligned}$$

Therefore

$$\bar{\beta}_1\bar{\nu}_1 + \bar{\nu}_2 + i\alpha'\frac{\bar{\nu}_2}{\omega_0}\sin\omega_0\tau = 0$$

So, the required equations for $\bar{\nu}_1$ and $\bar{\nu}_2$ are

$$\left.\begin{aligned}\beta_1\bar{\nu}_1 + e_1\bar{\nu}_2 &= 1 \\ \bar{\beta}_1\bar{\nu}_1 + e_2\bar{\nu}_2 &= 0\end{aligned}\right\}$$

where

$$\left.\begin{aligned}e_1 &= 1 - \alpha'\tau\cos\omega_0\tau + i\alpha'\tau\ \sin\omega_0\tau \\ e_2 &= 1 + i\tfrac{\alpha'}{\omega_0}\sin\omega_0\tau\end{aligned}\right\}$$

$$\left.\begin{aligned}\bar{\nu}_1 &= \tfrac{e_2}{e_2\beta_1 - e_1\bar{\beta}_1} \\ \bar{\nu}_2 &= \tfrac{-\bar{\beta}_1}{e_2\beta_1 - e_1\bar{\beta}_1}\end{aligned}\right\} \tag{4.39}$$

Finally, we have the values of $\nu_1$ and $\nu_2$ by taking the complex conjugate of (4.39). Using the notation as in Hassard et al., (1981), we write

$$\begin{pmatrix} x \\ y\end{pmatrix} = zq + \bar{z}\bar{q} + W$$

$$z\ \langle\ \acute{q}\ \begin{pmatrix} x \\ y\end{pmatrix}\ \rangle$$

$$\begin{aligned}\dot{z}(t) &= i\omega_0 z(t) + \bar{\acute{q}}(0).f(w(z,\bar{z},\theta') + \mathbf{Re}\{z(t)q(\theta')\}) \\ &= i\omega_0 z(t) + \bar{\acute{q}}(0).f_0(z,\bar{z})\end{aligned}$$

where

$$f_0 = \begin{pmatrix} f_0^1 \\ f_0^2\end{pmatrix} \qquad (86)$$

$$\left.\begin{aligned}f_0^1 &= \{W^1(0) + 2\mathbf{Re}(z(t)\beta_1)\}[a_{11}(W^2(0) + 2\mathbf{Re}\ z(t)) \\ &\quad + a_{20}(W^1(0) + 2\mathbf{Re}\ (z(t)\beta_1))], \\ f_0^2 &= \{W^2(0) + 2\mathbf{Re}\ z(t)\}[b_{11}(W^1(0) + 2\mathbf{Re}\ (z(t)\beta_1)) \\ &\quad + b_{11}'(W^1(-\tau) + 2\mathbf{Re}\ (z(t)e^{-i\omega_0\tau}\beta_1))] \\ &\quad + b_{12}'(W^1(-\tau) + 2\mathbf{Re}\ (z(t)e^{-i\omega_0\tau}\beta_1))^2\end{aligned}\right\}$$

Using the result $\Delta(i\omega,\tau)=0$, for $\omega=\omega_0,\tau=\tau_0$, and letting $\Omega_0=e^{-i\omega_0\tau_0}$ also we have assumed that $W=O(|z|^2)$. We have retained only the terms necessary to compute $C_1(0)$. Therefore

$$\left.\begin{aligned} f_0^1 &= a_{11}(W^1(0)+\beta_1 z+\bar{\beta}_1\bar{z})(W^2(0)+z+\bar{z}) \\ &\quad +a_{20}(W^1(0)+\beta_1 z+\bar{\beta}_1\bar{z})^2 \\ &= (a_{11}\beta_1+a_{20}{\beta_1}^2)z^2+(a_{11}\bar{\beta}_1+a_{20}{\bar{\beta}_1}^2)\bar{z}^2 \\ &\quad +(a_{11}(\beta_1+\bar{\beta}_1)+2a_{20}\beta_1\bar{\beta}_1)z\bar{z}+O(|z|^4) \end{aligned}\right\}$$

$$\left.\begin{aligned} f_0^2 &= b_{11}(W^1(0)+\beta_1 z+\bar{\beta}_1\bar{z})(W^2(0)+z+\bar{z}) \\ &\quad +(W^1(0)+(\beta_1 z+\bar{\beta}_1\bar{z})\Omega_0)b_{11}'(W^2(0)+z+\bar{z}) \\ &\quad +b_{12}'(W^1(0)+(\beta_1 z+\bar{\beta}_1\bar{z})\Omega)^2 \\ &= (b_{11}\beta_1+b_{12}'\beta_1{\Omega_0}^2)z^2+(b_{11}\bar{\beta}_1+\bar{\beta}_1 b_{12}'{\Omega_0}^2)\bar{z}^2 \\ &\quad +(b_{11}(\beta_1+\bar{\beta}_1)+2b_{12}'\beta_1\bar{\beta}_1)z\bar{z} \\ &\quad b_{11}'{\beta_1}^2{\Omega_0}^2z^3+b_{11}'{\bar{\beta}_1}^2{\Omega_0}^2\bar{z}^3+b_{11}'({\beta_1}^2+2\beta_1\bar{\beta}_1){\Omega_0}^2z^2\bar{z} \\ &\quad +b_{11}'(2\beta_1\bar{\beta}_1+{\bar{\beta}_1}^2)z\bar{z}^2+O(|z|^4) \end{aligned}\right\}$$

So, after taking the dot product of $f_0$ and $\bar{q}(0)$ and after expanding, we have

$$\begin{aligned} \dot{z} &= i\omega_0 z+\bar{\nu}_1 f_0^1+\bar{\nu}_2 f_0^2 \\ &= i\omega_0 z+\frac{1}{2}g_{20}z^2+\frac{1}{2}g_{02}\bar{z}^2+g_{11}z\bar{z}+\frac{1}{6}g_{30}z^3 \\ &\quad +\frac{1}{6}g_{03}\bar{z}^3+\frac{1}{2}g_{21}z^2\bar{z}+\frac{1}{2}g_{12}z\bar{z}^2+O(|z|^4) \end{aligned}$$

where

$$\left.\begin{aligned} g_{20} &= 2[\bar{\nu}_1(a_{11}\beta_1+a_{20}{\beta_1}^2)+\bar{\nu}_2(b_{11}\beta_1+b_{12}'\beta_1\Omega_0)^2] \\ g_{02} &= 2[\bar{\nu}_1(a_{11}\bar{\beta}_1+a_{20}{\bar{\beta}_1}^2)+\bar{\nu}_2(b_{11}\bar{\beta}_1+b_{12}'\bar{\beta}_1\Omega_0)^2] \\ g_{11} &= \bar{\nu}_1(a_{11}(\beta_1+\bar{\beta}_1)+2a_{20}\beta_1\bar{\beta}_1)+\bar{\nu}_2(b_{11}(\beta_1+\bar{\beta}_1) \\ &\quad +2b_{12}'\beta_1\bar{\beta}_1) \\ g_{12} &= 2\bar{\nu}_2 b_{11}'(2\beta_1\bar{\beta}_1+{\bar{\beta}_1}^2){\Omega_0}^2 \\ g_{21} &= 2\bar{\nu}_2 b_{11}'(2\beta_1\bar{\beta}_1+{\beta_1}^2){\Omega_0}^2 \\ g_{30} &= 6\bar{\nu}_1 b_{11}'{\beta_1}^2{\Omega_0}^2 \\ g_{03} &= 6\bar{\nu}_1 b_{11}'{\bar{\beta}_1}^2{\Omega_0}^2 \end{aligned}\right\}$$

Finally we use the expression of Hassard et al.,1981 :

$$\left.\begin{aligned} C_1(0) &= \tfrac{i}{2\omega_0}(g_{20}g_{11}-2|g_{11}|^2+\tfrac{1}{3}|g_{02}|^2)+\tfrac{1}{2}g_{21} \\ \mu_2 &= -\frac{\mathbf{Re}\ C_1(0)}{\alpha'(0)} \\ \tau_2 &= -\frac{Im\ C_1(0)+\mu_2\omega'(0)}{\omega_0} \end{aligned}\right\}$$

So, the bifurcation is supercritical if $\mu_2>0$ and subcritical if $\mu_2<0$. Further if $\tau_2>0$, the period of the solution increase with $\tau$.

FIG. 4.3 – Up : Numerical solutions of equation 4.9 for $\tau < 18h$ depicting stable situation (decaying oscillation). Down : Numerical solutions of equation 4.9 for $\tau > 18h$ depicting unstable situation (growing oscillation).

FIG. 4.4 – Up : Numerical solutions of equation 4.9 for $\tau = 18h$ depicting periodic situation (stable limit cycle). Down : Numerical solutions of equation 4.9 for $\tau = 6h$ depicting periodic ocsillations (stable limit cycle).

# Introduction au chapitre 5

Dans ce chapitre nous présentons un travail plus général que celui que nous avions présenté dans le chapitre précédent. Il est plus général dans le sens où nous considérons moins de contraintes sur les fonctions introduites dans le modèle. Nous considérons ainsi différentes formes de la fonction de prise du zooplancton, appelée aussi fonction de capture, de la mortalité du zooplancton et de la libération des toxines par des espèces phytoplanctoniques toxiques. Nous étudions différents modèles correspondant à différents choix de ces fonctions et nous comparons les dynamiques associées.

Notre paramètre principal de cette étude est l'efficacité de la toxine $\theta$. Nous montrons que l'équilibre positif est stable ou instable suivant les valeurs des paramètres et suivant la forme des fonctions choisies. Nous montrons aussi que le régime oscillatoire ne peut être observé que si la fonction de prise est du type non linéaire. Nous mettons en évidence une bifurcation de Hopf au voisinage d'une valeur seuil $\theta^*$ et nous prouvons la stabilité des solutions périodiques.
Les méthodes mathématiques adoptées dans ce chapitre sont le critère de Routh-Hurwitz, le critère de divergence de Dulac, l'opérateur de Poincaré puis la méthode de réduction à la forme normale et à la variété centre, méthode qui a été développée dans le chapitre 2.

# Chapitre 5

# Comparisons, by models, of some basic mechanisms acting on the dynamics of the zooplankton-toxic phytoplankton system [1]

**Abstract :** In this paper, classes of mathematical models of phytoplankton-zooplankton systems with toxic phytoplankton are proposed and analysed.
We investigate various situations, by changing the uptake function, the mortality of the zooplankton or the toxin liberation process, and we compare the corresponding dynamics.
We show notably that a stable oscillatory regime (after a Hopf bifurcation) can only be observed if the uptake function is nonlinear.

**Keywords :** Zooplankton ; toxic-phytoplankton ; bloom ; infinite delay ; chain trick ; Hopf-Bifurcation ; Poincaré map ; Routh-Hurwitz Criterion.

[1] This chapter represents our paper published in the journal Mathematical Models and Methods in Applied Sciences (M3AS), (2002). It is realized in collaboration with O. Arino and J. Chattopadhyay.

## 5.1 Introduction

Phytoplankton are unicellular, mostly primitive phytosynthetic, usually microorganisms that float freely near the surfaces in all natural waters. Plankton is the basis of all aquatic food chains and phytoplankton in particular occupies the first trophic level and the fluctuations in its abundance determine the production as a whole marine biological output. Rapid increase and almost equally rapid decrease, separated by periods are the common features of plankton populations. In a broad sense planktonic blooms can be divided into two types, "spring blooms" and "red tides". Spring blooms occur seasonally for the changes in temperature or nutrient availability which are connected with seasonal changes in thermocline depth and strength, and consequent mixing. Red tides are localized outbreaks and occur due to high water temperature (see Truscott and Brindley [27]). The rapid massive growth of algae is, generally, caused by high nutrient levels and favourable conditions. The water must contain high levels of inorganic nutrients (nitrogen and phosphorus) for the algae to feed on and also water temperature and salinity levels must be within a certain range to be conducive to planktonic growth.

Herbivore grazing takes an important role in the bloom dynamics (see Wyatt and Horwood [31], Levin and Segel [17], Uye [28]). A possible interpretation for the contribution of zooplankton to oscillations is as follows : a rather rapid growth of phytoplankton elicits zooplankton growth which depletes the phytoplankton. Depletion of phytoplankton leads to reduced growth (or, even decay) of the zooplankton which in turn allows phytoplankton to flourish again. Compared to the time scale of oceanic events, such as upwelling systems, jets or river plumes, interactions of zooplankton and phytoplankton are a faster process, possibly reflected on higher components of the phytoplankton signals. Toxic substances produced by phytoplankton species reduce the growth of zooplankton by decreasing grazing pressure and this is one of the important common phenomena in plankton ecology (Fay [10]). It is now well established that quite a good number of phytoplankton species produce toxins, such as *Pseudo-nitzschia sp*, *Gambierdiscus toxicus*, *Prorocentrum sp*, *Ostrepsis sp*, *Coolia monotis*, *Thecadinium sp*, *Amphidinium carterae*, *Dinophysis sp*, *Gymnodinium breve*, *Alexandrium sp*, *Gymodinium catenatum*, *Pyrodinium bahamense* , *P.fiesteria piscicida*, *C.polylepis*, *P.patelliferum*, *P.parvum* (see Steidinger et al.[26], Nielsen et al. [21], Aure et al. [1], Hallegraeff [14], etc).

Within the broad perspective drawn above, the paper explores and compares the coupled dynamics of the phytoplankton and the zooplankton in a number of mathematical models. The system phytoplankton-zooplankton has attracted considerable attention from various fields of research. It is an important issue in mathematical ecology.The literature abounds in models focusing on various aspects of the problem (see [9], [19],[25]). Recently, the attention has been focused on the role of the space in explaining heterogeneity and the distribution of the species and the influence of the spatial structure on their abundance (see [22]). However, the very question of the interactions between phytoplankton and zooplankton, independent on space is far from being fully elucidated.

Our purpose here is to perform a comparative study of a variety of spatially homogeneous models and derive some general conclusions about the qualitative features of models.

In order to give an overview of our results, a brief description of the models under consideration is appropriate.

We denote $P(t)$ (resp. $Z(t)$) the density of phytoplankton (resp. zooplankton). We assume that the dynamics of the phytoplantkon alone is governed by a logistic function

$$L(P) = rP(1 - \frac{P}{K}). \tag{5.1}$$

The most general expression we will consider is

$$\begin{cases} \frac{dP}{dt} = L(P) - \alpha f(P)Z \\ \frac{dZ}{dt} = \beta f(P)Z - \mu h(Z) - \theta[\int_{-\infty}^{t} F(t-s)f(P(s))ds]Z(t) \end{cases} \tag{5.2}$$

As already mentioned, the scale of the events considered here is smaller than the one of oceanic events, which is the one of nutrients dynamics. Thus, nutrients are just embodied here as parameters of the phytoplankton dynamics.
The function $f(P)$ represents the consumption rate of phytoplankton by zooplankton. Basic conditions on $f$ are :

$$f(0) = 0, f(P) \geq 0.$$

There is a broad range of possibilities for the choice of $f$ : from $f(P) = P$, which we will refer to as a linear uptake function, to $f(P) = \frac{P}{\gamma+P}$ or $\frac{P^a}{\gamma+P^a}$, for some $a > 0$, which are Holling types nonlinearities, showing convergence to a plateau as $P \to +\infty$ with speed of convergence dependent upon $a$ (see [1],[25]). The case when $f$ is linear, means that the predation rate follows the simple law of mass action. In the case when $f$ is of Holling type, it is assumed that there is a saturation effect in the consumption of phytoplankton by zooplankton : in this case, there is a maximum eating rate, $\alpha$. $h(Z)$ represents a combination of natural mortality and the control that the population exerts on itself in order to avoid crowding. Several choices are feasible : The most frequently cited in the literature is when $h(Z) = cZ$ (see [1]). Another typical model is $h(Z) = dZ^2$ (see [9]). The zooplankton mortality term can also be viewed as a 'closure term' (see [25]) because it closes the model at the top trophic level, and it primarily represents consumption of the zooplankton by higher predators. Natural mortality is usually implicitly assumed to be included in the closure term. The quadratic and linear forms for the closure term represent different ecological assumptions. For $dZ^2$, the specific rate is $dZ$, which assumes that higher predators have a biomass proportional to their prey (see [1]), while the linear function $cZ$ assumes a constant specific higher-predation rate $c$ (see [25]).
General assumptions, satisfied by the above examples, will be made on functions $f$ and $h$.
The integral term is to be interpreted as a result of some toxic substances "secreted" by phytoplankton with a possibly lethal cumulative effect. It covers a variety of situations. If $F$ is a delta function or a combination of delta functions at different points, it is just a combination of values of $f(P)$ at $t$ and possibly different points in the past. In the simplest situation, it just provides a term of the form $\theta f(P(t))Z(t)$, which leads to the equation

$$\frac{dZ}{dt} = (\beta - \theta)f(P(t))Z(t) - \mu h(Z(t)). \tag{5.3}$$

In this case, the effect of this term is just to lower the growth rate of the zooplankton.
In the sequel, we will mainly address two different situations : the case without delay, arising in equation (5.2), and a case with delay distributed all over the past, modeled by a gamma distribution

$$F(t) = \frac{\sigma^{m+1}}{m!}t^m e^{-\sigma t}, \sigma > 0. \tag{5.4}$$

This class of delays has been and is still the subject of active research, in various contexts (see [18],[23], [30]). Interestingly enough, such delay equations can be reduced to an "augmented" system

of ordinary differential equations. We will come back to this in the next section.
We have obtained results in three directions : 1. Steady states (or, equilibriums), 2. Stability, and 3. The onset of oscillations :

1. *Steady states.* Under some normalization assumption on $F$, namely that

$$\int_0^{+\infty} F(s)ds = 1 \tag{5.5}$$

the steady state equations are independent of $F$ and read as

$$\begin{cases} L(P^*) - \alpha f(P^*)Z^* = 0 \\ (\beta - \theta)f(P^*)Z^* - \mu h(Z^*) = 0. \end{cases}$$

Under some general assumptions on $f$ and $h$, we show in Theorem1 that there exists at most one interior equilibrium $(P^*, Z^*)$ such that $P^* \geq \frac{K}{2}$, and that positive solutions are either all on the left or all on the right of $\frac{K}{2}$.
Two different situations come out from the study according to whether $P^* < \frac{K}{2}$ or $P^* \geq \frac{K}{2}$.

2. *Stability* of the interior equilibrium has been investigated using the characteristic equation. We have two general results :
- Linear uptake form leads to local asymptotic stability of the non trivial steady state independent of $F$ and $h$ (Theorem 2).
- $P^* \geq \frac{K}{2}$ implies that the positive equilibrium is locally asymptotically stable independent of $f, h$ and $F$ (Theorem 3).

3. *The onset of oscillations* when $P^* < \frac{K}{2}$ is more involved and has not been dealt with in a general framework. Namely, we have restricted our study to the case when $f(P) = \frac{P}{\gamma + P}$.
The most interesting feature in this case is the onset of stable oscillations via a Hopf bifurcation taking place near some value $\theta_0$. Thus, we are in the situation, evoked earlier on in this introduction, of a succession of highs and lows of phytoplankton density, as a result of zooplankton grazing; the onset of oscillations is just delayed by phytoplankton toxicity. So, oscillations are entailed by the activity of zooplankton and conceivably will be lowered by a reduced activity of the zooplankton.
Most of our work is to do with local dynamics. The only global results obtained in this work are those concerned with equilibriums. In particular, we do not discuss the extent of the Hopf bifurcation. We do not investigate existence of other periodic solutions either.
The techniques used in the study of the characteristic equations combine elementary algebra and the Routh-Hurwitz criterion. Applying this criterion concretely is, in some places, a difficult task though, because the parameters entering the formulas to be checked are in fact intricate functions of the parameters of the equation. The study of stability of the delay equation uses a continuation technique.

The paper is organized as follows. In section 2, we describe the model equations and we state the hypotheses on $f$ and $h$. We also discuss in some length the delay term. In section 3, we study the existence of steady states. Stability analysis is performed in section 4, first in the case when the uptake function is linear and the mortality of zooplankton due to higher predation is unspecified,

secondly in the case when the positive steady state $(P^*, Z^*)$ is such that $P^* \geq \frac{K}{2}$. Finally, we deal with some special cases when the uptake function is of Holling type II. A concluding discussion is carried out in section 5.

## 5.2 A General Model and Main Assumptions

The phytoplankton growth is modelled as a combination of a logistic law with growth rate '$r$' and environmental carrying capacity '$K$' and zooplankton predation, where the uptake rate is modelled according to two classical scenarios : linear, unrestricted and nonlinear, bounded above by maximum food uptake capacity. This leads to the following equation

$$\frac{dP}{dt} = rP(1 - \frac{P}{K}) - \alpha f(P)Z. \tag{5.6}$$

Here $P$ is the density of phytoplankton population and $Z$ is the density of zooplankton population. We now turn our attention to the zooplankton population. '$\beta$' denotes the ratio of biomass consumed by zooplankton for its growth and '$\mu$' is mortality rate of zooplankton due to natural death as well as for higher predation. The mortality function $h(Z)$ may be linear or quadratic in nature. As the process of toxic liberation is not an instantaneous one and the phytoplankton population takes some time for maturity, the delay arises in the system. Let us also assume that the "memory" of the toxicant process is finite and is equal to some positive value $\tau$; that this time is so small compared to the lifespan of the zooplankton that the density of zooplankton is roughly the same during a period of length $\tau$. Assuming that the food uptake rate per unit of zooplankton biomass during the period $[t - \tau, t]$ is described in terms of a function $\rho(s), s \in [t - \tau, t]$, and the probability that the fraction consumed by the zooplankton during the infinitesimal time internal $(s, s + ds)$ be toxic is given by $F(t - s)ds$, the quantity of toxins accumulated per unit of zooplankton biomass during the period $[t - \tau, t]$ is equal to

$$\int_{t-\tau}^{t} \rho(s)F(t - s)ds.$$

The lethal incidence of a quantity $Q$ of toxin on a mass $M$ of zooplankton is given by $\theta QM$ , where $0 < \theta < 1$ is an efficiency parameter and $M = Z(t)dt$. Therefore the lethal incidence rate is given by

$$\theta Z(t) \int_{t-\tau}^{t} \rho(t)F(t - s)ds.$$

In this work we assume

$$\rho(t) = f(P(t)).$$

Hence the evolution of the zooplankton population in this situation may be represented as

$$\frac{dZ}{dt} = \beta f(P)Z - \mu h(Z) - \theta Z(t) \int_{t-\tau}^{t} F(t - s)f(P(s))ds. \tag{5.7}$$

If now $F$ is a function with an exponential decay as $t \to +\infty$, then one can extend the delay to $t \to +\infty$ and equation (5.7) becomes

$$\frac{dZ}{dt} = \beta f(P)Z - \mu h(Z) - \theta Z(t) \int_{-\infty}^{t} F(t - s)f(P(s))ds.$$

Combining both equations (5.6) and (5.7), we arrive at the phytoplankton-zooplankton system (5.2)

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha f(P)Z \\ \frac{dZ}{dt} = \beta f(P)Z - \mu h(Z) - \theta\left[\int_{-\infty}^{t} F(t-s)f(P(s))ds\right]Z \end{cases}$$

We now describe scenarios to be investigated and we give some possible ecological interpretation of each of them.

We will study the system in the case when the production of toxic substances is either instantaneous and follows the Dirac delta function or not instantaneous and follows a gamma distribution. When the production of toxic substances is instantaneous then system (5.2) reduces to

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha f(P)Z \\ \frac{dZ}{dt} = (\beta-\theta)f(P)Z - \mu h(Z) \end{cases} \tag{5.8}$$

In the case when the production of toxic substances is not instantaneous then system (5.2) reads as

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha f(P)Z \\ \frac{dZ}{dt} = \beta f(P)Z - \mu h(Z) - \theta\left[\int_{-\infty}^{t} \frac{\sigma^{m+1}}{m!}(t-s)^m e^{-\sigma(t-s)} f(P(s))ds\right]Z \end{cases} \tag{5.9}$$

Let us now discuss the delay term in more details.

In fact, the production of toxic substance is mediated by some time lag for maturity of phytoplankton population and hence a delay arises in the dynamics. It is also natural to assume that the toxin must reach some threshold value to effect on the grazing pressure of zooplankton and from this point of view the gamma distribution is an appropriate choice. It is to be noted here that the case without delay is a limiting situation of the case with delay as $\sigma \to +\infty$.

Delay differential equations with infinite delay have been considered in several contexts. Amongst others, we mention : applications to mechanics, Coleman and Mizel [5] and applications to biology and ecology, Cushing [6].

The theory of infinite delay equations is well understood now and is discussed notably in the books of Burton [4], Hale and Verduyn Lunel [13], and Hino, Murakami and Naito [16]. One of the issues (Hale and Kato [12]) is the choice of a suitable function space for the initial value. In the problem at hand, it is necessary that the function $s \to F(t-s)f(P(s))$ be integrable on $(-\infty, 0]$, for $t \geqslant 0$.

In fact, in the sequel, we will restrict ourselves to the case when $F$ is a gamma distribution, namely : $F_m(s) = \sigma^{m+1}\frac{s^m}{m!}e^{-\sigma s}, s \geqslant 0, \sigma > 0$. In this case, it is enough to verify that the integral is defined for one value of $t$, say $t = 0$.

Since we work with non-negative functions $P$ and $Z$, integration holds with no further assumption when $f(P) = \frac{P}{\gamma+P}$, since it is bounded. For the case when $f(P) = P$, convergence holds if for example, $P(s) \leq Me^{\sigma'|s|}$, for $s \leq 0$, and some $\sigma' < \sigma$.

In the situation of a gamma distribution, there is a remarkable feature : the equation can be transformed into an "augmented" system of ordinary differential equations. In fact, defining

$$U_j(t) = \int_0^{+\infty} \sigma^{j+1}\frac{s^j}{j!}e^{-\sigma s} f(P(t-s))ds,\ 0 \leq j \leq m.$$

we can see that system (5.2) becomes

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha f(P)Z \\ \frac{dZ}{dt} = \beta f(P)Z - \mu h(Z) - \theta U_m \\ \frac{dU_m}{dt} = \sigma U_{m-1} - \sigma U_m \\ \cdot \\ \cdot \\ \frac{dU_1}{dt} = \sigma U_0 - \sigma U_1 \\ \frac{dU_0}{dt} = \sigma f(P) - \sigma U_0 \end{cases} \tag{5.10}$$

Such equations have been the subject of active research for the last thirty years. We can quote notably Ruan [23], Wolkowicz [30] and Beretta et al. [3]. The transformation is known in the literature as the linear chain trick, MacDonald [18].
We will use this property and the "augmented" system in the sequel. Before we start, it is however necessary to see whether the results obtained for the ordinary system can be transferred to the original equation. Given a solution of system (5.10), defined on the whole real axis, supposedly with all components non-negative, is it associated with a solution of (5.2)?
In fact, if we take the last equation of system (5.10) :

$$U_0' + \sigma U_0 = \sigma f(P),$$

we can write :

$$U_0(t) = e^{-\sigma(t-t_0)}U_0(t_0) + \int_{t_0}^{t} e^{-\sigma(t-s)} f(P(s))ds.$$

Letting $t_0 \to -\infty$, a condition to recover the original expression of $U_0$ is that :

$$\int_{-\infty}^{t} e^{-\sigma(t-s)} f(P(s))ds < \infty$$

and

$$\lim_{t_0 \to -\infty} e^{\sigma t_0} U_0(t_0) = 0$$

which can be fulfilled if $U_0$ and $P$ grow slower than some $e^{\sigma'|t|}$, for $t \leq 0$, and some $\sigma' < \sigma$.
So, solutions of system (5.10) which are either bounded on $IR^-$ or have a small enough exponential growth at $-\infty$ yield solutions of equation (5.2). So, the onset of instability in system (5.10) corresponds to the same phenomenon in equation (5.2). Interestingly, the connection between the infinite delay equation and its O.D.E reduction emanates directly from the characteristic equation associated with any linear infinite delay equation with a gamma distribution kernel.
In fact, consider the linear equation associated with equation (3) near an arbitrary steady state. It takes the form

$$\begin{cases} \frac{dP}{dt} = aP(t) + bZ(t) \\ \frac{dZ}{dt} = cP(t) + dZ(t) + e\int_{-\infty}^{t} F_m(t-s)P(s)ds. \end{cases}$$

where $a, b, c, d$ and $e$ are just constants.
Looking for exponential solutions,

$$P(t) = \pi e^{\lambda t}; Z(t) = \zeta e^{\lambda t}$$

we have

$$\begin{cases} \lambda\pi e^{\lambda t} = a\pi e^{\lambda t} + b\zeta e^{\lambda t}, \\ \lambda\zeta e^{\lambda t} = (c\pi + d\zeta)e^{\lambda t} + e\int_{-\infty}^{t} \frac{\sigma^{m+1}}{m!}(t-s)^m e^{-\sigma(t-s)}\pi e^{\lambda s} ds, \end{cases}$$

$$\begin{aligned} (\lambda - a)\pi &= b\zeta, \\ (\lambda - d)\zeta &= [c + e\int_{-\infty}^{t} \sigma^{m+1}\frac{(t-s)^m}{m!}e^{-(\sigma+\lambda)(t-s)}ds]\pi, \\ (\lambda - a)(\lambda - d) - bc &= be\int_{0}^{+\infty} \sigma^{m+1}\frac{s^m}{m!}e^{-(\sigma+\lambda)s}ds. \end{aligned}$$

The integral term in the last equation is finite if and only if $\mathbf{Re}(\lambda) + \sigma > 0$. Then, the characteristic equation takes on the following polynomial form

$$(\lambda + \sigma)^{m+1}[(\lambda - a)(\lambda - d) - bc] - be\sigma^{m+1} = 0, \tag{5.11}$$

which is a good indication that the underlying dynamics is indeed one of a system of O.D.E.
The stability analysis for the delay equation will be made in section 4, in terms of the characteristic equation computed near each of the equilibria, both in the cases when $F$ is a gamma distribution and when it is a Dirac distribution.
We now consider other parts of the model. Besides the delay part, the system is determined by the functions $f(P)$ and $h(Z)$.
Throughout the paper, we assume the following on these functions :

**(H1)** $f$ is a $C^1$ function, non-negative, $f' > 0$ and $f(0) = 0$.

**(H2)** $h \in C^3, h(0) = 0, h$ is unbounded, $h' > 0, h'' \geqslant 0$ and $h''' \leq 0$. $\widetilde{h}(Z) = \frac{h(Z)}{Z}$ is non decreasing.

In the literature (see [1], [8],[25]), there are several examples of functions $f$ and $h$ which satisfy hypothesis (**H1**) and (**H2**). We quote notably the case when $f$ is linear $f(P) = P$ or of Holling type II functional form $f(P) = \frac{P}{\gamma + P}$. The mortality of zooplankton, which is represented by the function $h$, can be found to be linear or quadratic.

## 5.3 Steady-States

System (5.2) has an invariant subset : $Z = 0$. In this case, the equation reduces to the logistic equation for the phytoplankton, with two equilibria $P = 0$ (unstable) and $P = K$ (stable). Thus the point $(0, 0)$ is unstable with respect to the full system, while the point $(K, 0)$ will be stable or unstable according to whether the expression

$$[(\beta - \theta)]f(K) - \mu h'(0) < 0 \quad \text{or} \quad > 0 \quad \text{(A1)}$$

The equations for the equilibria are :

$$\begin{cases} L(P) - \alpha f(P)Z = 0 \\ (\beta - \theta)f(P)Z - \mu h(Z) = 0 \end{cases} \tag{5.12}$$

Looking for a solution with $Z \neq 0$, therefore $P \neq 0$, the first equation yields

$$Z = \frac{L(P)}{\alpha f(P)} \qquad \text{(A2)}$$

while the second one may be written as

$$(\beta - \theta) f(P) = \frac{\mu h(Z)}{Z} \qquad \text{(A3)}$$

Note that with the general assumptions we made, if the equilibrium $(K, 0)$ is stable, then equation (A3) does not have any feasible solution.
Assuming now that $(K, 0)$ is unstable, that is, (A1) is positive, then the system (A2)-(A3) may have solutions, with both $P$ and $Z > 0$. From equations (A2) and (A3) we obtain :

$$Z = h^{-1}[\frac{(\beta - \theta)}{\alpha\mu} L(P)]$$

Substituting the above expression for $Z$ in (A3), we arrive at

$$f(P) = \frac{\mu}{\beta - \theta} \widetilde{h}(h^{-1}[\frac{(\beta - \theta)}{\alpha\mu} L(P)]) \qquad (A4)$$

From hypothesis (**H2**), we can see that equation $(A4)$ is well defined and is indeed the characteristic equation for the equilibria.
Denote

$$\delta(P) = \frac{\mu}{\beta - \theta} \widetilde{h}(h^{-1}(\frac{(\beta - \theta)}{\alpha\mu} L(P))) \qquad (5.13)$$

then, $(P, Z)$ is a stationary point, with $Z \neq 0$ if and only if :

$$\begin{cases} f(P) = \delta(P) \\ Z = \frac{L(P)}{\alpha f(P)} \end{cases} \qquad (5.14)$$

Existence of non trivial solutions of (5.14) is now discussed in the light of the assumptions made on $f$ and $\delta$.

**Lemma 1.** *Under assumption (H2), we have :* $\delta'' \leq 0$.

**Proof.** By direct inspection of the second order derivative of $\delta$, and noting that, under (**H2**), $\widetilde{h}$ is concave down.

**Theorem 1.** *Under assumptions* (**H1**) *and* (**H2**), *solutions of system* (5.12) *are either all such that* $P^* < \frac{K}{2}$ *or all such that* $P^* \geq \frac{K}{2}$. *Moreover, if a solution* $(P^*, Z^*)$ *is such that* $P^* \geq \frac{K}{2}$, *then it is the unique positive equilibrium.*

**Proof.** We will distinguish two cases according to whether $f'' \leq 0$ or $f'' \geq 0$.
**Case 1.** $f'' \geq 0$ : the function $f$ is concave up and $\delta$ is concave down (see ($\mathbf{H_2}$)).
If $f(K) < \delta(K)$, then the graph of the function $f$ and the graph of the function $\delta$ can not intersect,

thus there is no positive equilibrium. This condition is equivalent to $[(\beta-\theta)]f(K)-\mu h'(0)<0$, which implies that the axial steady state $(K,0)$ is stable.
The biological meaning of this case is that if the maximal rate, $(\beta-\theta)f(K)$, of regeneration of zooplankton by consumption of phytoplankton is less than the minimum rate $\mu h'(0)$, of mortality of zooplankton, then the zooplankton population goes to extinction, which is obvious.
If $f(K)>\delta(K)$, then there is one and only one interior equilibrium.
**Case 2.** $f''\leq 0$ : both the functions $f$ and $\delta$ are concave down
The proof of the second part of the theorem is due to the fact that, on the interval $[\frac{K}{2},K]$, $f$ is increasing while $\delta$ is decreasing.
To prove the first part of the theorem, we consider the equations for the equilibria (5.12).
From the first equation of (5.12), we have

$$Z^*=\frac{L(P^*)}{\alpha f(P^*)} \tag{5.15}$$

Substituting (5.15) in the second equation of (5.12), we have

$$\frac{P^{*2}}{K}-P^*+\frac{\alpha\mu}{r(\beta-\theta)}h(Z^*)=0 \tag{5.16}$$

We may consider $P^*$ as a function of $Z^*$.
From equation (5.16) we obtain

$$\begin{aligned} P_1^*(Z^*) &= \frac{K-\sqrt{K^2-4\frac{K\alpha\mu}{r(\beta-\theta)}h(Z^*)}}{2} \\ P_2^*(Z^*) &= \frac{K+\sqrt{K^2-4\frac{K\alpha\mu}{r(\beta-\theta)}h(Z^*)}}{2} \end{aligned} \tag{5.17}$$

Note here that $P_1^*(Z^*)<\frac{K}{2}<P_2^*(Z^*)$.

*Existence of an equilibrium point* $(P^*,Z^*)$ *with* $0<P^*<\frac{K}{2}$ :
In this case, we have $P^*=P_1^*(Z^*)$ (given by (5.17)).
The functions $P_1^*(Z^*)$ and $f\circ P_1^*(Z^*)$ are increasing with respect to $Z^*$.
From (5.12), $Z^*$ must satisfy :

$$f(P_1^*(Z^*))=\frac{\mu}{\beta-\theta}\frac{h(Z^*)}{Z^*}. \tag{5.18}$$

We have

$$f(P_1^*(0))=0,\ \lim_{Z\to 0}\frac{\mu}{\beta-\theta}\tilde{h}(Z)=\frac{\mu}{\beta-\theta}h'(0)$$

and

$$f(P_1^*(h^{-1}(\frac{Kr(\beta-\theta)}{4\alpha\mu})))=f(\frac{K}{2})$$

A necessary condition for $P^*$, with $P^*<\frac{K}{2}$, to exist is that :

$$h^{-1}(\frac{Kr(\beta-\theta)}{4\alpha\mu})>\frac{Kr}{4\alpha f(\frac{K}{2})}. \tag{5.19}$$

*Existence of an equilibrium point* $(P^*, Z^*)$ *with* $K > P^* > \frac{K}{2}$ :
In this case, $P^* = P_2^*(Z^*)$ (given by (5.17)).
From (5.12), the interior equilibrium must satisfy

$$f(P_2^*(Z^*)) = \frac{\mu}{\beta - \theta} \frac{h(Z^*)}{Z^*} \tag{5.20}$$

The function $f \circ P_2^*(Z^*)$ is decreasing with respect to $Z^*$; on the other hand, $\widetilde{h}(Z) = \frac{h(Z)}{Z}$ is increasing because we suppose that $h'' \geq 0$. Then, equation (5.20) has at most one solution.
Regarding the existence of a solution of (5.20), we have

$$\lim_{Z \to 0} f(P_2^*(Z)) = f(K)$$

and

$$\lim_{Z \to 0} \frac{\mu}{\beta - \theta} \frac{h(Z)}{Z} = \frac{\mu}{\beta - \theta} h'(0)$$

In view of the monotonicity of $f \circ P_2^*$ and $\widetilde{h}$, a necessary and sufficient condition to have existence of $(P^*, Z^*)$, with $P^* > \frac{K}{2}$ is

$$\frac{\mu}{\beta - \theta} h'(0) < f(K) \text{ and } h^{-1}\left(\frac{Kr(\beta - \theta)}{4\alpha\mu}\right) < \frac{Kr}{4\alpha f(\frac{K}{2})} \tag{5.21}$$

where $h^{-1}(\frac{Kr(\beta-\theta)}{4\alpha\mu})$ is the value of $Z^*$ such that $P_1^*(Z^*) = P_2^*(Z^*) = \frac{K}{2}$.
We note that (5.19) and the second condition in (5.21) are mutually exclusive. Therefore, one at most can be satisfied at a time. In the case when $h^{-1}(\frac{Kr(\beta-\theta)}{4\alpha\mu}) = \frac{Kr}{4\alpha f(\frac{K}{2})}$, the unique positive equilibrium is such that $P^* = \frac{K}{2}$. We conclude that interior equilibriums are either all to the right or all to the left of $\frac{K}{2}$, or all are such that $P^* = \frac{K}{2}$.

## 5.4 Stability Analysis

We study the stability of the system near the steady states. We consider three situations : 1. $f$ is linear and $h$ is arbitrary, 2. $P^* \geq \frac{K}{2}$ (whatever $f$ and $h$ may be) and 3. $P^* < \frac{K}{2}$ and $f(P) = \frac{P}{\gamma + P}$.
We will consider the cases when the kernel $F$ is a delta function or a gamma distribution with $m = 1$. In fact, when $m = 0$, we deal with a weak kernel in the sense that, although all values of $P$ in the past are taken into account, only recent values have a strong effect on the system. But, when $m$ increases, values of $P$ further in the past become more important.
These kernels are frequently used in biological modeling (see Cushing [6]).
In cases 1 and 2 we prove asymptotic stability of the non trivial steady state, both in the ordinary and in the delay cases. In case 3 we are not able to derive a result not dependent on $h$. We will focus on two subcases : $h$ linear, and $h$ quadratic, both in the ordinary and the delay cases.

### 5.4.1 $f$ linear : $f(P) = P$

*Unrestricted predation of zooplankton on the phytoplankton* $f(P) = P$.
System (5.8) reads as

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha PZ \\ \frac{dZ}{dt} = (\beta-\theta)PZ - \mu h(Z) \end{cases} \tag{5.22}$$

System (5.9) with $F(t) = \sigma^2 te^{-\sigma t}$ is as follows :

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha PZ \\ \frac{dZ}{dt} = \beta PZ - \mu h(Z) - \theta[\int_{-\infty}^{t} \sigma^2(t-s)e^{-\sigma(t-s)}P(s)ds]Z(t) \end{cases} \tag{5.23}$$

**Theorem 2.** *Existence of a positive equilibrium $E^*(P^*, Z^*)$of system (5.22) - (5.23), ensures that $E^*$ is locally asymptotically stable. In other words, if the uptake functions are linear, then no periodic solution can arise near any steady state of the system whatever the mortality of zooplankton or the distribution of the accumulation of the toxic substances may be.*

The theorem can be rephrased in ecological words as follows :
If the harvesting of phytoplankton by zooplankton is proportional to the abundance of phytoplankton, the oscillatory succession of phytoplankton blooms and zooplankton peaks will not be generated.

**Proof.** We prove the local stability of the steady state in both the ordinary and the delay cases :
*(a)-Ordinary system : $F = \delta_0$.*
System (5.22) has the following non-negative equilibria, namely the trivial equilibrium $E_0(0,0)$ which represents the absence of matter, the axial equilibrium $E_1(K,0)$ which corresponds to the extincion of zooplankton, and an interior equilibrium $E^*(P^*, Z^*)$ which is the coexisting equilibrium.
Linear stability analysis shows that the trivial equilibrium is always unstable. It is not only unstable but also "repulsive" in the sense that any small initial value will lead to solutions escaping from a neighborhood of the origin. Existence of $E^*$ implies that axial equilibrium $E_1$ is also an unstable saddle point and non existence of an interior equilibrium ensures that $E_1$ is a stable point.
In this case the characteristic equation around the positive steady state takes on the following form

$$\lambda^2 + \lambda[\frac{rP^*}{K} + \mu h'(Z^*) - (\beta-\theta)P^*] + \frac{rP^*}{K}(\mu h'(Z^*) - (\beta-\theta)P^*) \tag{5.24}$$
$$+\alpha(\beta-\theta)P^*Z^* = 0$$

From the equation of the equilibria and the fact that $h$ is increasing, we conclude that the positive equilibrium $E^*$ is locally asymptotically stable (LAS).
*(b)-System with delay : $F(t) = \sigma^2 te^{-\sigma t}$.*
System (5.23) has the same equilibria as system (5.22) and the nature of the trivial and the axial equilibria is also the same. In this case, the characteristic equation associated to $E^*(P^*, Z^*)$ takes on the following form

$$\lambda^4 + f_1(\sigma)\lambda^3 + f_2(\sigma)\lambda^2 + f_3(\sigma)\lambda + f_4(\sigma) = 0 \tag{5.25}$$

with

$$\begin{aligned} f_1(\sigma) &= A + B + 2\sigma \\ f_2(\sigma) &= \sigma^2 + 2\sigma(A+B) + AB + \alpha\beta P^*Z^* \\ f_3(\sigma) &= \sigma^2(A+B) + 2\sigma(AB + \alpha\beta P^*Z^*) \\ f_4(\sigma) &= \sigma^2(AB + (\beta-\theta)\alpha P^*Z^*) \end{aligned}$$

where

$$\begin{aligned} A &= \mu h'(Z^*) - (\beta - \theta)P^* > 0 \\ B &= \frac{rP^*}{K} \end{aligned}$$

The sign of the real parts of the roots can be determined by means of the Routh-Hurwitz theorem. When it exists the coexistence equilibrium is LAS.

### 5.4.2 $P^* \geq \frac{K}{2}$, $f$ and $h$ are unspecified

We study the local stability of the positive equilibrium $E^*(P^*, Z^*)$.
Consider system (5.2)

$$\begin{cases} \frac{dP}{dt} = rP(1 - \frac{P}{K}) - \alpha f(P)Z \\ \frac{dZ}{dt} = \beta f(P)Z - \mu h(Z) - \theta \left[\int_{-\infty}^{t} F(t-s)f(P(s))ds\right] Z \end{cases} \tag{5.26}$$

**Theorem 3.** *If the steady state $E^*(P^*, Z^*)$ of system (5.26) is such that $P^* \geq \frac{K}{2}$, then it is locally asymptotically stable (LAS).*

**Proof.** The linearized system of the ordinary differential equation (5.8) near $E^*(P^*, Z^*)$ is

$$\begin{cases} \frac{d\pi}{dt} = L'(P^*)\pi - \alpha f'(P^*)Z^*\pi - \alpha f(P^*)\zeta \\ \frac{d\zeta}{dt} = f'(P^*)(\beta - \theta)Z^*\pi + [(\beta - \theta)f(P^*) - \mu h'(Z^*)]\zeta \end{cases} \tag{5.27}$$

the characteristic equation is given by :

$$\lambda^2 - \lambda[L'(P^*) - \alpha f'(P^*)Z^* + (\beta - \theta)f(P^*) - \mu h'(Z^*)] + L'(P^*)[(\beta - \theta)f(P^*) - \mu h'(Z^*)]$$
$$+\alpha\mu f'(P^*)h'(Z^*)Z^* = 0 \tag{5.28}$$

From system (5.12) we have

$$f(P^*) = \frac{\mu}{\beta - \theta}\frac{h(Z^*)}{Z^*} \leq \frac{\mu}{\beta - \theta}h'(Z^*).$$

because $h'$ is increasing with respect to $Z$.
Also, as $P^* \geq \frac{K}{2}$ thus $L'(P^*) = r(1 - \frac{2P^*}{K}) \leq 0$.
Then we have

$$\begin{cases} L'(P^*) - \alpha f'(P^*)Z^* + (\beta - \theta)f(P^*) - \mu h'(Z^*) < 0 \\ L'(P^*)[(\beta - \theta)f(P^*) - \mu h'(P^*)] + \alpha\mu f'(P^*)h'(Z^*)Z^* > 0 \end{cases}$$

We conclude that the roots of the characteristic equation (5.28) have a negative real part, which implies that if $(P^*, Z^*)$ exists, it is LAS.
To prove the result in the case of a gamma distribution, we perform the "linear chain trick technique" (see MacDonald [18]), then we obtain the following characteristic equation

$$\lambda^4 + f_1(\sigma)\lambda^3 + f_2(\sigma)\lambda^2 + f_3(\sigma)\lambda + f_4(\sigma) = 0$$

where

$$\begin{aligned} f_1(\sigma) &= A + B + 2\sigma \\ f_2(\sigma) &= \sigma^2 + 2\sigma(A+B) + AB + \alpha\beta f(P^*)f'(P^*)Z^* \\ f_3(\sigma) &= \sigma^2(A+B) + 2\sigma(AB + \alpha\beta f(P^*)f'(P^*)Z^*) \\ f_4(\sigma) &= \sigma^2(AB + (\beta-\theta)\alpha f(P^*)f'(P^*)Z^*) \end{aligned}$$

with

$$\begin{aligned} A &= \alpha Z^* f'(P^*) - L'(P^*) > 0 \\ B &= \mu h'(Z^*) - (\beta-\theta) f(P^*) > 0 \end{aligned}$$

In this case, we find that all conditions of the Routh-Hurwitz theorem are satisfied, therefore the steady state is LAS.
One interpretation here is that a certain abundance of the phytoplankton population ($P^* \geq \frac{K}{2}$) leads to the lowering of its growth rate, and then the zooplankton population can only survive and not reproduce new zooplankton.
In the other situation ($P^* < \frac{K}{2}$), the phytoplankton population is growing faster, and permits the zooplankton population to have more and more food, which in turn will decrease the level of phytoplanton leading to a subsequent decrease of the zooplankton population, and will make a new increase of phytoplankton possible. So, in this case, we may have a succession of highs and lows for the zooplankton alternating with a similar succession for the phytoplankton, that is, periodic oscillations take place.

In the remainder of this section, as we find it difficult to obtain general results when $P^* < \frac{K}{2}$, we consider a special case well known in the literature (see [7],[24]), when the uptake function $f$ is of Holling type II functional form. The idea of such a predation function which saturates for higher prey densities is a common one and is supported by experimental observations (Uye,1986).

### 5.4.3 $P^* < \frac{K}{2}$ and $f(P) = \frac{P}{\gamma+P}$.

We consider two main cases according to whether zooplankton mortality is linear or we assume it is dependent upon the density of higher predators. In each case, we examine separately the instantaneous and the delayed (or, cumulative) actions.

#### Linear mortality of zooplankton : $h(Z) = Z$

*(a) Ordinary system : $F = \delta_0$.*
System (5.8) reads as

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha\frac{PZ}{\gamma+P} \\ \frac{dZ}{dt} = (\beta-\theta)\frac{PZ}{\gamma+P} - \mu Z \end{cases} \tag{5.29}$$

System (5.29) has a trivial equilibrium $E_0(0,0)$, an axial equilibrium $E_1(K,0)$ and a positive equilibrium $E^*(P^*,Z^*)$ where

$$P^* = \frac{\gamma\mu}{\beta-\mu-\theta}\ ,\ Z^* = \frac{r}{\alpha}(\gamma+P^*)(1-\frac{P^*}{K}).$$

A necessary and sufficient condition for the existence of a positive equilibrium in this case is

$$\theta < \beta - \mu - \frac{\gamma\mu}{K}.$$

**Theorem 4.** *If the positive equilibrium $E^*(P^*, Z^*)$ of system (5.29) exists then system (5.29) around $E^*$ enters a Hopf bifurcation when $\theta$ passes through a critical value $\theta_0$.*

**Proof.** The characteristic equation around $E^*(P^*, Z^*)$ is given by

$$\lambda^2 + \lambda\left(\frac{r}{K} - \frac{\alpha Z^*}{(\gamma + P^*)^2}\right)P^* + \frac{\alpha\gamma(\beta - \theta)P^*Z^*}{(\gamma + P^*)^3} = 0 \tag{5.30}$$

From the above it is clear that if

$$\theta = \theta_0 = \beta - \frac{K + \gamma}{K - \gamma}\mu \tag{5.31}$$

then we have two imaginary eigenvalues and also

$$\frac{d}{d\theta}\mathbf{Re}(\lambda)|_{\theta=\theta_0} = -\frac{1}{2}\frac{r(K - \gamma)^2}{\mu\gamma K(\gamma + K)} < 0, \tag{5.32}$$

i.e. a transversality condition is satisfied. Hence a Hopf-type bifurcation occurs.
The positive equilibrium $(P^*, Z^*)$ is unstable for $\theta < \theta_0$ and it is stable for $\theta > \theta_0$. The system passes from instability to stability when $\theta$ increases and exhibits a periodic solution near the bifurcation value $\theta_0$.
We have $\beta = \theta + \frac{K+\gamma}{K-\gamma}\mu$, which shows that the effect of $\theta$, that is the force of the toxic effect, is to push forward the value $\beta$ for which bifurcation occurs.
The ecological interpretation of the above results is that the introduction of toxic phytoplankton can reduce the growth rate of zooplankton and eventually the blooms can be delayed.

### 5.4.4 Direction of bifurcation

The main result for this section is summarized in the next theorem.

**Theorem 5.** *The bifurcating branch mentioned in theorem 4 is supercritical, that is to say, stable periodic solutions appear to the left of $\theta_0$.*

**Proof.** We only give the general set up here, a detailed proof is deferred to the Appendix.
We consider system (5.29) in polar coordinates, that is, we put

$$\begin{aligned} P(t) &= \delta(t)\cos(\varphi(t)), \\ Z(t) &= \delta(t)\sin(\varphi(t)), \\ \delta(0) &= r_0, \varphi(0) = 0, \end{aligned}$$

then we expand the parameter $\theta$ in powers of $r_0$, $\theta(r_0)$ can be expanded up to order two :

$$\theta(r_0) = \theta_0 + \theta''(0)r_0^2 + O(r_0^3)$$

Classically (see [11]), the local Hopf bifurcation branch is determined as a curve in the space $(\delta, \theta)$. Here $\theta(0) = \theta_0$.
We obtain

$$\theta''(0) = \frac{1}{\rho'(\theta_0)} \cdot \frac{\alpha^2}{r\mu(K+\gamma)^4}[4\sqrt{Kr\gamma\mu(K-\gamma)^2} + 3r(K-\gamma)^2 + 2K\mu(K-\gamma)]$$

where $\rho'(\theta_0) < 0$.

The direction of the bifurcation branch is given by the sign of the value of $\theta''(0)$, we show in Appendix that : $\theta''(0) < 0$.
*(b) System with delay :* $F(t) = \sigma^2 t e^{-\sigma t}$.
This case is similar to case 4.3.1(a) except for the production of toxic substance which follows the gamma distribution.
System (5.2) reads as

$$\begin{cases} \frac{dP}{dt} = rP(1-\frac{P}{K}) - \alpha\frac{PZ}{\gamma+P} \\ \frac{dZ}{dt} = \beta\frac{PZ}{\gamma+P} - \mu Z - \theta\int_{-\infty}^{t}\sigma^2(t-s)e^{-\sigma(t-s)}P(s)dsZ(t) \end{cases} \tag{5.33}$$

It has the same equilibria as in case 1(a). The characteristic equation takes on the following form

$$\lambda^4 + f_1(\sigma)\lambda^3 + f_2(\sigma)\lambda^2 + f_3(\sigma)\lambda + f_4(\sigma) = 0 \tag{5.34}$$

with

$$\begin{aligned} f_1(\sigma) &= 2\sigma + A \\ f_2(\sigma) &= \sigma^2 + 2\sigma A + B \\ f_3(\sigma) &= \sigma^2 A + 2\sigma B \\ f_4(\sigma) &= \sigma^2 B\frac{(\beta-\theta)}{\beta} \end{aligned}$$

where

$$A = \frac{rP^*}{K} - \frac{\alpha P^* Z^*}{(\gamma+P^*)^2}$$

and

$$B = \frac{\alpha\beta\gamma P^* Z^*}{(\gamma+P^*)^3}$$

In the previous case we observed that when $\theta$ crosses a critical value $\theta_0$ (given by (5.31)) a Hopf bifurcation occurs, but in this case, we observe that for that same value $\theta_0$, all the conditions of the Routh-Hurwitz theorem are satisfied and since the coefficients are non increasing in $\theta$, one can conclude that $E^*$ is LAS at $\theta_0$ and beyond this value, then if a bifurcation occurs, it will take place at a value lower than $\theta_0$.
Thus, the gamma distribution of toxic substances stabilizes the system in a larger range of values of $\theta$ than the Dirac delta distribution.
Starting from $\theta = 0$, systems (5.29) and (5.33) have an unstable interior point : this point becomes stable past a value $\theta^*$ in the case of system (5.33), while it remains unstable up to $\theta = \theta_0 > \theta^*$ in

the case of system (5.29).
Accordingly, if periodic oscillations exist, they must exist for $\theta < \theta^*$ in the case of system (5.33), while they exist for $\theta < \theta_0$ in the case of system (5.29). Thus, the delay acts in such a way that it preserves stability longer than the ordinary system.

**Density-dependent mortality of zooplankton on higher predator**

*(a) Ordinary system : $F = \delta_0$.*

This case is similar to case 4.3.1(a) except for the mortality of zooplankton which is quadratic, system (5.8) reads now as

$$\begin{cases} \frac{dP}{dt} = rP(1 - \frac{P}{K}) - \alpha\frac{PZ}{\gamma+P} \\ \frac{dZ}{dt} = (\beta - \theta)\frac{PZ}{\gamma+P} - \mu Z^2 \end{cases} \tag{5.35}$$

System (5.35) has a trivial equilibrium $E_0$, an axial equilibrium $E_1(K, 0)$ and a positive equilibrium $E^*$, where $P^*$ and $Z^*$ satisfy the system given by

$$\begin{cases} P^{*3} + (2\gamma - K)P^{*2} + (\gamma - 2\gamma K + \frac{\alpha K(\beta-\theta)}{r\mu})P^* - \gamma^2 K = 0 \\ Z^* = \frac{r}{\alpha}(\gamma + P^*)(1 - \frac{P^*}{K}) \end{cases} \tag{5.36}$$

A necessary and sufficient condition for the existence of a positive equilibrium is

$$P^* < K. \tag{5.37}$$

A (sufficient) condition for $P^*$ to satisfy (5.37) is

$$\gamma - \gamma^2 + \frac{\alpha K(\beta - \theta)}{r\mu} > 0.$$

The characteristic equation around $E^*(P^*, Z^*)$ is as follows :

$$\lambda^2 + \lambda(A + \mu Z^*) + A\mu Z^* + \frac{\alpha\gamma(\beta - \theta)P^*Z^*}{(\gamma + P^*)^3} = 0 \tag{5.38}$$

with

$$A = \frac{rP^*}{K} - \frac{\alpha P^* Z^*}{(\gamma + P^*)^2}$$

**Theorem 6.** *If $r(K - \gamma) < \mu K$, then system (5.35) has no nontrivial periodic solution near the positive equilibrium $E^*(P^*, Z^*)$.*
*Otherwise, a Hopf bifurcation takes place at any value $\theta_0$ such that $A + \mu Z^* = 0$ and the following condition holds :*

$$\frac{\alpha}{2} < \frac{1 - \frac{1}{K}}{1 - \frac{1}{\gamma}}.$$

The proof of the first part of theorem 3 is based on an application of the Dulac divergence criterion (Hale and Lunel [10]).(see Appendix).
One can remark that the condition $r(K - \gamma) < \mu K$ is satisfied if in particular the growth rate $r$ of the phytoplankton is less than the mortality rate $\mu$ of zooplankton, hence in this case, we can not

have sustained oscillations.

When the parameter $\theta$ crosses the critical value $\theta_0$, then $A + \mu Z^*$ becomes either $> 0$ (for a small value of the specific predation rate $\alpha$) or $< 0$ (for large value of the specific predation rate $\alpha$ and small value of the intrinsic growth rate $r$), thus $E^*$ is either LAS or $E^*$ is unstable.
At $\theta = \theta_0$, we have two imaginary eigenvalues and the transversality condition

$$\frac{d}{d\theta}\mathbf{Re}\lambda(\theta)\Big|_{\theta=\theta_0} \neq 0$$

is satisfied if the condition of theorem 6 holds. Therefore a Hopf bifurcation occurs at $\theta_0$.

*(b) System with delay : $F(t) = \sigma^2 t e^{-\sigma t}$.*

This case is the same as case 4.3.2(a) except for the production of toxic substance which follows the gamma distribution.
In this case system (5.2) reads as

$$\begin{cases} \frac{dP}{dt} = rP(1 - \frac{P}{K}) - \alpha\frac{PZ}{\gamma+P} \\ \frac{dZ}{dt} = \beta\frac{PZ}{\gamma+P} - \mu Z^2 - \theta[\int_{-\infty}^{t} \sigma^2(t-s)e^{-\sigma(t-s)}\frac{P(s)}{\gamma+P(s)}ds]Z(t) \end{cases} \tag{5.39}$$

We propose first, to study the system with $\sigma$ large enough ($\sigma \rightarrow +\infty$).
The analysis of this case breaks down into several situations (see Appendix for details). For example, for $K$ large enough and either $\theta$ large or $\beta$ small (which in both cases implies a reduced survival rate for zooplankton), we have instability of the non trivial steady state.
Also, if the growth rate $r$ of phytoplankton is small enough, then the system exhibits oscillations around the interior equilibrium with the magnitude of the oscillations of the order of $r$.
One notices that, for a given value of $K$ (large enough), if we vary $\theta$ from large to low values, there is at least one value $\theta_0$ at which the dominant eigenvalue crosses the imaginary axis at $\lambda = 0$ and becomes positive for $\theta < \theta_0$. This suggests the onset, at $\theta = \theta_0$, of a bifurcation branch of steady-states, which could possibly be a stable branch.
Biologically, this would correspond to the loss of stability of an equilibrium at $\theta = \theta_0$ and the stabilization of the system near a different equilibrium.
Search of the bifurcations which may occur when varying one or the other or all of the parameters is beyond the scope of this work and will be addressed in future work.
Now, consider the system in the case 4.3.2 (b) with finite $\sigma$. Under the following condition

$$\begin{aligned} &(-4f_1^2f_2^3 + 16f_2^4 + 18f_1^3f_2f_3 - 80f_1f_2^2f_3 - 6f_1^2f_3^2 + 144f_2f_3^2)f_4 \\ &+(-27f_1^4 + 144f_1^2f_2 - 128f_1^2f_2 - 192f_1f_3)f_4^2 \\ &-f_3^2(-f_1^2f_2^2 + 4f_2^3 + 4f_1^3f_3 - 18f_1f_2f_3 + 27f_3^2) \neq 0 \end{aligned} \tag{5.40}$$

(where $f_i$, $1 \leq i \leq 4$ are given in the Appendix)

we can deduce that the behaviour of the system with $\sigma$ tending to infinity does not change. In fact, define

$$\overline{\sigma} = \max(-\frac{(A+B)}{2}, \sigma_m), \tag{5.41}$$

where

$$A = \frac{rP^*}{K} - \frac{\alpha P^* Z^*}{(\gamma + P^*)^2},$$

$$B = \mu Z^* + 2\theta \frac{P^* Z^*}{(\gamma + P^*)} - \frac{\theta P^*}{(\gamma + P^*)},$$

and $\sigma_m$ is the largest non negative root of (5.40).
For $\sigma > \overline{\sigma}$, the characteristic equation of system (5.39) can not have a root with multiplicity greater than 1; that is the branches $\lambda = \lambda(\sigma)$ can not intersect and are well defined until infinity. Also, if the real roots $\lambda_1$ and $\lambda_2$ (see appendix) cross the imaginary axis, they must cross it on $\lambda = 0$. We note here that either $\lambda = 0$ is a root for all values of $\sigma$ or $\lambda = 0$ is never a root. We add that the other roots $\lambda_+$ and $\lambda_-$ (see Appendix), are independent of $\sigma$.

**Theorem 7.** *For all $\sigma > \overline{\sigma}$, where $\overline{\sigma}$ is defined by (5.41), the behaviour of system (5.39) is the same and is determined in terms of the other parameters of the equation (see ( 5.55) in Appendix for a description of the various cases).*

## 5.5 Conclusion

This work attempted to search for suitable mechanisms by which one can put up the model best fitted to explain the qualitative behavior of the zooplankton-phytoplankton system. To explain this, we used models made up of three main ingredients : phytoplankton-zooplankton (prey-predator) coupling, the density dependent mortality of zooplankton, and the control of zooplankton by toxins. The theoretical results presented in section 4.1 show that if the uptake functions are linear in nature, whatever the mortality of zooplankton may be, and whether the distribution of toxic substances follows a Dirac delta or a gamma distribution, then the positive equilibrium is locally asymptotically stable and consequently there is no periodic solution.
In section 4.2 we proved that in the case when the positive equilibrium $E^*(P^*, Z^*)$ is such that $P^* \geq \frac{K}{2}$, then it is locally asymptotically stable. Therefore oscillations near the non trivial steady state can not be generated. Our interpretation is that the abundance of phytoplankton population leads to a lower growth rate; in this case, zooplankton population can only survive and can not reproduce a new zooplankton.
In section 4.3, we showed that if the uptake function is of Holling type II, then periodic solutions may occur. In the case when we obtain oscillations, the phytoplankton population is growing faster, and allows zooplankton population to have more and more food and thus reproduce, which in turn will decrease the level of phytoplankton leading to a subsequent decrease of zooplankton population. In turn, this allows a new increase of phytoplankton and then a new increase of zooplankton population will follow. In fact, in the case when the mortality of zooplankton is linear, then we proved not only the existence of oscillations but also that the gamma distribution of toxic substances stabilizes the system in a larger range of values of $\theta$ than that of the Dirac distribution. The ordinary equation highlights the role of the toxicant intensity of toxin while the delay equation emphazises

its cumulative effect. If the mortality of zooplankton is quadratic, we obtained different possibilities with respect to parameters and, under some suitable conditions we showed that the behaviour of the system is the same for all $\sigma > \overline{\sigma}$ (given by (5.41)). Here $1/\sigma$ represents the average time lag for the kernel.
The above observations indicate that, of the three components mentioned above, the coupling term of phytoplankton and zooplankton is the leading factor for explaining the oscillatory succession of the phytoplankton-zooplankton system. In fact, if the uptake function $f$ is linear, then neither the presence of toxic substances nor the other ingredients of the system have an effect on the system, but if $f$ is of Holling type II then the presence of toxic phtoplankton reduces the growth rate of zooplankton and may possibly change the behaviour of the system.
The work presented here focused on the interactions of averaged, homogeneous populations of zooplankton and phytoplankton, disregarding the role of spatial heterogeneity as well as the dynamics of nutrients which was not considered a limiting factor.Therefore the dynamics of nutrients was not considered. These issues will be taken into account in future work.

## Appendix

**Theorem A.1.** (Routh-Hurwitz see [20]) *Consider the following equation*

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$$

*A necessary and sufficient condition for* $\mathbf{Re}(\lambda) < 0$*for all roots is that the following four inequalities hold : (i)* $a_1 > 0$, *(ii)* $a_4 > 0$, *(iii)* $a_1a_2 - a_3 > 0$, *(iv)* $a_1(a_2a_3 - a_1a_4) - a_3^2 > 0$.

**Proof of Theorem 5.** In order to determine the direction of bifurcation in the case 4.3.1 (a), we express system (5.29) in polar coordinates $P(t) = \delta(t)\cos(\varphi(t))$ and $Z(t) = \delta(t)\sin(\varphi(t))$.

$$\begin{cases} \frac{d\delta}{dt} = \rho(\theta)\delta(t) + h_1(\theta, \delta(t), \varphi) \\ \frac{d\varphi}{dt} = \sigma(\theta) + o(1) \end{cases} \tag{5.42}$$

where

$$h_1(\theta, \delta, \varphi) = \delta^2 p_3(\theta, \cos(\varphi), \sin(\varphi)) + \delta^3 p_4(\theta, \cos(\varphi), \sin(\varphi)) + O(\delta^4)$$

and $p_j$ is homogeneous polynomial of degree $j$ in $\cos(\varphi)$ and $\sin(\varphi)$, $j = 3, 4$.
$\lambda = \lambda(\theta)$ denotes the eigenvalue of the linearization of (5.29) with a positive imaginary part, we may write

$$\lambda(\theta) = \rho(\theta) + i\sigma(\theta)$$

At $\theta = \theta_0$ we have

$$\rho(\theta_0) = 0\ ,\ \sigma(\theta_0) = \omega > 0$$

Eliminating the time between the two equations of system (5.42), we arrive at

$$\frac{d\delta}{d\varphi}(\varphi) = \frac{\rho(\theta)}{\sigma(\theta)}\delta(\varphi) + \frac{1}{\sigma(\theta)}h_1(\theta, \delta(\varphi), \varphi) \tag{5.43}$$

For the ease of notation, we define

$$\begin{aligned} m(\theta) &= \frac{\rho(\theta)}{\sigma(\theta)} \\ h(\theta,\delta,\varphi) &= \frac{1}{\sigma(\theta)} h_1(\theta,\delta(\varphi),\varphi) \end{aligned}$$

Equation (5.43) becomes

$$\frac{d\delta}{d\varphi}(\varphi) = m(\theta)\delta(\varphi) + h(\theta,\delta(\varphi),\varphi) \tag{5.44}$$

From equation (5.44) we have

$$\delta(\theta,\varphi) = e^{m(\theta)\varphi} r_0 + \int_0^{\varphi} e^{m(\theta)(\varphi-s)} h(\theta,\delta(s),s)ds$$

We construct the Poincaré map

$$\mathcal{P}(\theta,r_0) = e^{2\pi m(\theta)} r_0 + \int_0^{2\pi} e^{m(\theta)(2\pi-s)} h(\theta,\delta(s),s)ds \tag{5.45}$$

We know that $\mathcal{P}(\theta,0) = 0\ \forall\theta$. We are searching $r_0 \neq 0$ for which we have a periodic solution. So, we consider the following function

$$\begin{aligned} Q(\theta,r_0) &= \frac{\mathcal{P}(\theta,r_0)}{r_0} - 1 \\ &= e^{2\pi m(\theta)} - 1 + \int_0^{2\pi} e^{m(\theta)(2\pi-s)} \frac{h(\theta,\delta(s),s)}{r_0} ds \end{aligned}$$

From (5.31) and (5.32) we have

$$\begin{aligned} m(\theta_0) &= 0 \text{ and } m'(\theta_0) \neq 0 \text{ then} \\ Q(\theta_0,r_0) &= \int_0^{2\pi} \frac{h(\theta_0,\delta(s),s)}{r_0} ds \text{ and} \\ Q(\theta_0,0) &= 0 \end{aligned}$$

Or

$$\begin{aligned} \frac{\partial Q}{\partial r_0}(\theta_0,0) &= \lim_{r_0\to 0} \frac{1}{r_0} Q(\theta_0,r_0) \\ &= \lim_{r_0\to 0} \int_0^{2\pi} \frac{h(\theta_0,\delta(s),s)}{r_0^2} ds = 0 \end{aligned}$$

$$\text{because } \int_0^{2\pi} p_3(\theta_0,\cos(s),\sin(s))ds = 0.$$

We have

$$Q(\theta,0) = e^{2\pi m(\theta)} - 1$$

then

$$\frac{\partial Q}{\partial \theta}(\theta_0, 0) = 2\pi m'(\theta_0) \neq 0$$

Then by the implicit function theorem we conclude that there exists a function $\theta = \psi(r_0)$ so that

$$\begin{cases} Q(\psi(r_0), r_0) = 0 \text{ and} \\ \psi(0) = \theta_0 \end{cases} \tag{5.46}$$

From (5.46) we have

$$\psi'(r_0)\frac{\partial Q}{\partial \theta}(\psi(r_0), r_0) + \frac{\partial Q}{\partial r_0}(\psi(r_0), r_0) = 0 \text{ then } \psi'(0) = 0$$

Also from (5.46) we obtain

$$\psi''(0).2\pi m'(\theta_0) + \frac{\partial^2 Q}{\partial r_0^2}(\theta_0, 0) = 0$$

Using the fact that

$$\delta(\theta_0, \varphi) = r_0 + \int_0^{\varphi} h(\theta_0, \delta(s), s)ds$$

and using also the form of $h(\theta, r, \varphi)$ we can find that

$$\frac{\partial^2 Q}{\partial r_0^2}(\theta_0, 0) = 2\int_0^{2\pi} p_4(\theta_0, \cos(s), \sin(s))ds$$

and then

$$\psi''(0) = -\frac{1}{\pi m'(\theta_0)}\int_0^{2\pi} p_4(\theta_0, \cos(s), \sin(s))ds$$

In this case 4.3.1(a) we have

$$\begin{aligned} \int_0^{2\pi} p_4(\theta_0, \cos(s), \sin(s))ds &= \frac{\pi}{4\sigma(\theta_0)}[3a_4(\theta_0) + a_6(\theta_0) + b_5(\theta_0) + 3b_7(\theta_0)] \text{ and} \\ m'(\theta_0) &= \frac{\rho'(\theta_0)}{\sigma(\theta_0)} \end{aligned}$$

Then

$$\psi''(0) = -\frac{1}{4\rho'(\theta_0)}[3a_4(\theta_0) + a_6(\theta_0) + b_5(\theta_0) + 3b_7(\theta_0)]$$

where the quantities $\rho(\theta)$, $\sigma(\theta)$, $a_j(\theta)$ and $b_j(\theta)$ are given by

$$\begin{aligned}
\rho(\theta) &= \frac{rP^*(K-2P^*-\gamma)}{2K(\gamma+P^*)} \\
\sigma(\theta) &= \frac{\{rP^*(K-\gamma)(\gamma+P^*)^2(-rP^*(K-\gamma)(-K+2P^*+\gamma)^2+4K(K-P^*)\gamma(K+\gamma)\mu)\}^{\frac{1}{2}}}{2K(K-\gamma)(\gamma+P^*)^2} \\
a_4(\theta) &= -\frac{(K-P^*)q_{11}^2(\theta)r\gamma(\alpha+(\beta-\theta)q_{12}(\theta))}{K\alpha(\gamma+P^*)^3} \\
a_6(\theta) &= -\frac{q_{12}(\theta)(3(K-P^*)q_{12}(\theta)r-2K\alpha)\gamma(\alpha+(\beta-\theta)q_{12}(\theta))}{K\alpha(\gamma+P^*)^3} \\
b_5(\theta) &= \frac{q_{11}^2(\theta)(3(K-P^*)rq_{12}(\theta)-K\alpha)\gamma(\beta-\theta)}{K\alpha(\gamma+P^*)^3} \\
b_7(\theta) &= \frac{q_{12}^2(\theta)((K-P^*)rq_{12}(\theta)-K\alpha)\gamma(\beta-\theta)}{K\alpha(\gamma+P^*)^3}
\end{aligned}$$

and where $q_{11}(\theta)$ and $q_{12}(\theta)$ are given by the following expressions

$$q_{11}(\theta) = -\alpha(\gamma+P^*)\{-r^2P^{*2}(-KP^*+2P^{*2}-K\gamma+3\gamma P^*+\gamma^2)^2-4KP^*r\gamma(\gamma+P^*)^2(K-P^*)(\beta-\theta)\}^{\frac{1}{2}}/2(K-P^*)r\gamma(K+\gamma)(KP^{*2}+2KP^*\gamma-\gamma P^{*2}+K\gamma^2-2P^*\gamma^2-\gamma^3)\mu$$

$$q_{12}(\theta) = \alpha(K-\gamma)(\gamma+P^*)\{-(r^2P^{*2}(-K^2P^*+2KP^{*2}-K^2\gamma+4KP^*\gamma-2\gamma P^{*2}+2K\gamma^2-3P^*\gamma^2-\gamma^3)^2-4KP^*r\gamma(K^2-KP^*+K\gamma-\gamma P^*)(KP^{*2}+2KP^*\gamma-\gamma P^{*2}+K\gamma^2-2\gamma^2P^*-\gamma^3)\mu)\}^{\frac{1}{2}}/2(K-P^*)r\gamma(K+\gamma)(KP^{*2}+2KP^*\gamma-\gamma P^{*2}+K\gamma^2-2P^*\gamma^2-\gamma^3)\mu$$

If we simplify the expression $3a_4(\theta_0)+a_6(\theta_0)+b_5(\theta_0)+3b_7(\theta_0)$ we obtain the following

$$\psi''(0) = \frac{1}{\rho'(\theta_0)}\cdot\frac{\alpha^2}{r\mu(K+\gamma)^4}[4\sqrt{Kr\gamma\mu(K-\gamma)^2}+(K-\gamma)(3Kr-3r\gamma+2K\mu)]$$

which allows us to annonce the direction of bifurcation summarized in the theorem 5.

**Proof of Theorem 6.** The following Lemma is the well known Dulac criterion (see [10]).

**Lemma A.1.** *Let $B(x,y)$ be $C^1$ on a simply connected region $D \subset IR^2$.*
*If $\frac{\partial(Bf)}{\partial x}+\frac{\partial(Bg)}{\partial y}$ is not identically zero and does not change sign in $D$, then the system*

$$\begin{cases} \frac{dx}{dt} = f(x,y) \\ \frac{dy}{dt} = g(x,y) \end{cases}$$

*(where $f$ and $g$ are $C^1$) has no closed orbits lying entirely in $D$.*

**Proof of 4.4.1(a).** Let $h(P,Z) = \frac{\gamma+P}{PZ}$ which is $C^1$ in the positive quadrant.
we define :

$$\begin{aligned} g_1(P,Z) &= rP(1-\frac{P}{K}) - \alpha\frac{PZ}{\gamma+P} \\ g_2(P,Z) &= (\beta-\theta)\frac{PZ}{\gamma+P} - \mu Z^2 \\ \triangle(P,Z) &= \frac{\partial}{\partial P}(g_1 h) + \frac{\partial}{\partial Z}(g_2 h) \end{aligned}$$

We find that $\triangle(P,Z) = -\frac{2rP}{K} - \frac{\mu\gamma}{P} + r - \mu - \frac{r\gamma}{K} < 0$ for all $P>0, Z>0$ if the condition $r(K-\gamma) < \mu K$ of theorem 6 holds.

**Details for 4.4.1.(b).** In this case the characteristic equation (5.11) becomes

$$\lambda^4 + f_1(\sigma)\lambda^3 + f_2(\sigma)\lambda^2 + f_3(\sigma)\lambda + f_4(\sigma) = 0 \tag{5.47}$$

with

$$\begin{aligned} f_1(\sigma) &= 2\sigma + A + B \\ f_2(\sigma) &= \sigma^2 + \sigma(A+B) + AB \\ f_3(\sigma) &= \sigma^2 + 2\sigma AB \\ f_4(\sigma) &= \sigma^2 C \end{aligned}$$

and

$$\begin{aligned} A &= \frac{rP^*}{K} - \frac{\alpha P^* Z^*}{(\gamma+P^*)^2} \\ B &= \mu Z^* + 2\theta\frac{P^*Z^*}{(\gamma+P^*)} - \frac{\theta P^*}{(\gamma+P^*)} \\ C &= AB + \frac{\alpha\beta\gamma P^* Z^*}{(\gamma+P^*)^3} \end{aligned} \tag{5.48}$$

where the pair $(P^*, Z^*)$ satisfies system (5.36).
We can remark that $A, B$ and $C$ are dependent on all or on some parameters of the system.
We first study the system with $\sigma$ large enough ($\sigma \to +\infty$), and then we will extend the conclusions of the study to all $\sigma > \overline{\sigma}$ ($\overline{\sigma}$ is given by (5.41)), using some continuation argument.
We will perform a change of variables to simplify the study of the case when $\sigma$ is large enough.
We put : $\varepsilon = \frac{1}{\sigma}$, then equation (5.47) becomes

$$(\varepsilon\lambda)^4 + \varepsilon(\varepsilon\lambda)^3(A+B) + 2(\varepsilon\lambda)^3 + \varepsilon(\varepsilon\lambda)^2(A+B) + \varepsilon^2(\varepsilon\lambda)^2 AB \tag{5.49}$$

$$+(\varepsilon\lambda)^2 + \varepsilon(\varepsilon\lambda) + 2\varepsilon^2(\varepsilon\lambda)AB + \varepsilon^2 C = 0.$$

If we put $\mu = \varepsilon\lambda$ and substitute it in the equation (5.49), then we obtain

$$\mu^4 + 2\mu^3 + \mu^2 + \varepsilon[\mu^3(A+B) + \mu^2(A+B) + \mu] + \varepsilon^2[\mu^2 AB + 2\mu AB + C] = 0 \tag{5.50}$$

For $\varepsilon = 0$ the equation reduces to :

$$\mu^4 + 2\mu^3 + \mu^2 = 0$$

which gives $\mu = 0$ or $\mu = -1$. We also notice that the polynomial corresponding to $\varepsilon = 0$ is dominant in the sense that it has the highest degree in $\mu$. So, if we now let $\varepsilon$ to be positive and small, then the roots of the polynomial for $\varepsilon > 0$ will stay close to roots of the unperturbed polynomial.
If we define $\nu = \mu + 1$, equation (5.50) reads

$$(\nu - 1)^2\nu^2 + \varepsilon F(\varepsilon, \nu) = 0 \tag{5.51}$$

with

$$F(\varepsilon, \nu) = (A + B)(\nu - 1)^3 + (A + B)(\nu - 1)^2 + \varepsilon AB(\nu - 1)^2 + (1 + 2\varepsilon AB)(\nu - 1) + \varepsilon C$$

$F(0, 0) = -1$
For $(\varepsilon, \nu)$ close to $(0, 0)$ equation (5.51) gives $\nu^2 \simeq \varepsilon$.
We define

$$\begin{cases} \varepsilon = \delta^2 \\ \nu = \delta q \end{cases}$$

Then equation (5.51) becomes

$$q^2(\delta q - 1)^2 + F(\delta^2, \delta q) = 0 \tag{5.52}$$

We denote

$$G(\delta, q) = q^2(1 - \delta q)^2 + F(\delta^2, \delta q)$$

We have

$$\begin{aligned} G(0, q_0) &= 0, \ q_0 = \pm 1 \\ \frac{\partial G}{\partial q}(0, q_0) &= 2q_0 \neq 0 \end{aligned}$$

The equation $G(\delta, q) = 0$ has in the vicinity of $(0, q_0)$ two solutions for each $\delta$, that is, there are two functions $Q_+$ and $Q_-$, defining two distinct branches crossing at $(0, q_0)$ such that :

$$G(\delta, q) = 0 \leftrightarrow q = Q_\pm(\delta), \ (\delta, q) \text{ close to } (0, q_0).$$

We collect the expressions of the parameters :

$$\nu = \delta q, \mu = \nu - 1, \varepsilon = \delta^2 \text{ and } \lambda = \frac{\mu}{\varepsilon} \tag{5.53}$$

Substituting $Q_\pm(\delta)$ for $q$ in these formulas, we obtain the following two branches of eigenvalues

$$\lambda(\delta) = \frac{1}{\delta^2}(\delta Q_\pm(\delta) - 1)$$

Using the expression of $\delta$ in terms of $\varepsilon$ together with the first order expansion of $Q_\pm$ near 0, we obtain

$$\begin{aligned} \lambda_1(\varepsilon) &\simeq -\frac{1}{\varepsilon} - \frac{1}{\sqrt{\varepsilon}} \\ \lambda_2(\varepsilon) &\simeq -\frac{1}{\varepsilon} + \frac{1}{\sqrt{\varepsilon}} \end{aligned}$$

We note that $\lambda_1(\varepsilon) < 0$ and $\lambda_2(\varepsilon) < 0$ for $\varepsilon > 0$, small enough, while $\lambda_1(\varepsilon) + \lambda_2(\varepsilon) \simeq -\frac{2}{\varepsilon}$.
To find the other two roots $\lambda_+$ and $\lambda_-$ we proceed as follows. We know that

$$\begin{cases} \lambda_1 + \lambda_2 + \lambda_+ + \lambda_- = -(A+B) - \frac{2}{\varepsilon} \\ \lambda_1\lambda_2\lambda_+\lambda_- = \frac{C}{\varepsilon^2} \end{cases}$$

then we conclude that

$$\begin{cases} \lambda_+ + \lambda_- \simeq -(A+B) \\ \lambda_+\lambda_- \simeq C \end{cases}$$

then $\lambda_+$ and $\lambda_-$ are approximated by the solutions of the equation

$$M^2 + (A+B)M + C = 0 \tag{5.54}$$

where expressions of $A, B$ and $C$ are given in (5.48) and do not depend on $\varepsilon$.
We summarize the results of this case in the following :

$$\begin{cases} \textbf{case 1}: \quad (A+B) > 0 \\ \text{If} \begin{cases} C > 0 \text{ then } \mathbf{Re}(\lambda_\pm) < 0 \\ C < 0 \text{ then } \mathbf{Re}(\lambda_+) > 0 \text{ and } \mathbf{Re}(\lambda_-) < 0 \\ C = 0 \text{ then } \lambda_+ = -2(A+B) < 0 \text{ and } \lambda_- = 0 \end{cases} \\ \textbf{case 2}: \quad (A+B) < 0 \\ \text{If} \begin{cases} C > 0 \text{ then } \mathbf{Re}(\lambda_\pm) > 0 \\ C < 0 \text{ then } \mathbf{Re}(\lambda_+) > 0 \text{ and } \mathbf{Re}(\lambda_-) < 0 \\ C = 0 \text{ then } \lambda_+ = -2(A+B) > 0 \text{ and } \lambda_- = 0 \end{cases} \\ \textbf{case 3}: \quad (A+B) = 0 \\ \text{If} \begin{cases} C > 0 \text{ then } \lambda_+ = i\sqrt{C} \text{ and } \lambda_- = -i\sqrt{C} \\ C < 0 \text{ then } \lambda_+ = \sqrt{-C} \text{ and } \lambda_- = -\sqrt{-C} \\ C = 0 \text{ then } \lambda_+ = \lambda_- = 0 \end{cases} \end{cases} \tag{5.55}$$

From these last subcases, we are searching for a possibility to have a Hopf bifurcation. The interesting situation is when $A+B = 0$ and $C > 0$ because we have two imaginary eigenvalues $\pm i\sqrt{C}$. So, we have to search for the transversality condition, that is we have to see if there is some change of stability when the parameter $\theta$ crosses the critical value $\theta^*$; that is the value of $\theta$ for which $A + B = 0$.
We put $\lambda = \lambda(\theta) = \alpha(\theta) + i\beta(\theta)$, with $\alpha(\theta^*) = 0$ and $\beta(\theta^*) = \sqrt{C}$. We substitute it in equation (5.54) then we have

$$\begin{cases} \alpha^2(\theta) - \beta^2(\theta) + \alpha(\theta)(A+B) + C = 0 \\ 2\alpha(\theta)\beta(\theta) + \beta(\theta)(A+B) = 0 \end{cases} \tag{5.56}$$

After derivation of equation (5.56) with respect to $\theta$, we obtain

$$\alpha'(\theta^*) = -\frac{(A+B)'(\theta^*)}{2}$$

hence, if $(A+B)'(\theta^*) \neq 0$ then the transversality condition is satisfied. We conclude that in this case, when $\theta$ crosses the critical value $\theta^*$, a periodic solution appears, that is, a Hopf bifurcation occurs.
In other situations, when the system has 0 as an eigenvalue, the study depends on some reduced system. For example, if $A + B = 0$ and $C = 0$, then the characteristic equation becomes

$$\lambda^4 + 2\sigma\lambda^3 + (\sigma^2 - A^2)\lambda^2 + (\sigma^2 - 2\sigma A^2)\lambda = 0$$

then we have

$$\lambda = 0 \text{ or } \lambda^3 + 2\sigma\lambda^2 + (\sigma^2 - A^2)\lambda + (\sigma^2 - 2\sigma A^2) = 0$$

So, if $\sigma < 2A^2$ then there is at least one eigenvalue with a positive real part which leads to instability.
If $\sigma > \text{Max}(\frac{1}{2}, 2A^2)$, all conditions of the Routh-Hurwitz theorem are satisfied for the polynomial equation and then all the roots have a negative real part; $\mathbf{Re}(\lambda) < 0$, but the presence of the root $\lambda = 0$ does not allow us to conclude. In this case one possibility to study the system is to reduce it to a local center manifold. This approach will be exploited in a future work.

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# Introduction au chapitre 6

Dans ce chapitre, nous explorons la dynamique d'un système phytoplankton-zooplankton évoluant dans un environnement toxique. Ce travail représente une généralisation des travaux présentés dans les chapitres 4 et 5, dans le sens où nous étudions un système structuré.
Dans une première étape, nous considérons la date de naissance comme variable de structure pour formuler les équations du système. Ensuite, et pour des raisons d'interprétations des résultats et leur relation avec les hypothèses, nous transformons le système en un modèle équivalent structuré en âge. L'étude de l'existence, l'unicité et la positivité de la solution a été entreprise en utilisant la méthode des lignes caractéristiques et par la généralisation du principe de contraction. Nous montrons que le semi groupe solution est quasi compact. Cette propriété nous a permis d'étudier le comportement qualitatif du système à partir du spectre ponctuel. Nous analysons ainsi ce comportement qualitatif en fonction du paramètre d'efficacité de la toxine $\nu$. Deux situations ont été traitées ; le cas d'un retard discret, puis le cas d'un retard distribué. Dans les deux cas, en utilisant soit l'équation caractéristique associée dans le cas du retard discret, soit le système des valeurs propres dans le cas du retard distribué, nous montrons qu'au voisinage de la solution stationnaire positive, le système est stable pour $\nu < 0$, instable pour $\nu > 0$ et nous mettons en évidence l'apparition d'une bifurcation lorsque le paramètre $\nu$ traverse la valeur critique $\nu = 0$.

Pour étudier le comportement qualitatif du système au voisinage du point d'équilibre axial, nous avons été amené à réduire notre système à une variété centre en suivant l'approche de Hassard et Kazarinoff dans le cas des systèmes à retard discret. Les résultats de cette étude sont résumés dans la proposition 7.
Les résultats qualitatifs auquels nous aboutissons sont comparés à ceux obtenus dans le chapitre 5.

# Chapitre 6

# A delayed structured model of a Zooplankton-Toxic Phytoplankton system [1]

**Abstract :** In this paper, the dynamics of a phytoplankton-zooplankton system living in a toxic environment is explored. We propose and analyze a delayed age structured population model. We studied the qualitative behaviour, with respect to the toxin efficiency parameter $\nu$. Two situations are investigated; the case of a discrete delay and the case of a distributed delay. In both cases, either using a quasi polynomial characteristic equation, in the case of a discrete delay, or an eigenvalue equation, in the case of a distributed delay, we showed that the system is stable for negative values of the parameter $\nu$, unstable for positive values of the parameter $\nu$ and that a bifurcation occurs when $\nu$ crosses the critical value $\nu = 0$. The qualitative behaviour of the structured model is compared with the results of the global model studied in [4] when the same conditions are assumed.

**Keywords** : Phytoplankton-zooplantkon interactions, age structured model, delayed differential equations.

[1] This chapter is based on a work presented in the second international conference on Mathematical Ecology in Alcalà (Spain 2003). This work is submited to a publication in the journal *Ecological Complexity*. It is realized in collaboration with J. Treuil and O. Arino.

## 6.1 Introduction

In this paper we are interested in the dynamics of a phytoplankton-zooplankton system taking into account the effect of toxic substances released by phytoplankton species. This work is an extension of a previous work by El abdllaoui et al. (2002) and the work by Chattopadhyay et al. (2002).

These previous papers consider the zooplankton population as a kind of individual. This unique super-individual is accumulating and eliminating, along time, toxic substances following some process. This process is modeled here by a delay kernel. In reality, as zooplankton population is composed of individuals which have different birth dates and which evolve in different environmental conditions, these individuals accumulate and eliminate toxic substances at different rates according to their peculiar life histories. So, we can legitimately ask whether the properties described in [2] and [4] are preserved when we model the system taking into account these differences.

In this work, the basic unit is a cohort of individuals, that is, individuals born at the same time. We assume that individuals of a cohort, have the same life histories. In other words, we neglect individual variability within a generation and focus on the differences between generations.
The expression of the relation between the consumption of toxins and the evolution of the density of zooplankton is formulated as a convolution with a delay kernel as in the previous models studied in [2] and [4]. This fact is considered here at the level of each cohort and not at the level of the whole population. In some kind of individual based approach, this would lead us to put forward the birth date as the key variable identifying each cohort, but here we use a classical EDP approach and express the equations of the model in terms of age rather than birth date.
First, we construct the model in a general form and then discuss the details of the introduced functions, the basis hypotheses and the simplifying assumptions we adopted for this study. The main difference between the global model and the structured model discussed here is of course the structure, but also the fact that in the structured model, we do not consider a positive effect of phytoplankton on zooplankton individuals. In other words, we assume that phytoplankton is abundant and thus the birth flux is independent of the consumption. This assumption can be justified if we consider that only a small part of the phytoplankton population is toxic, and consequently that the availibility of wholesome food is sufficient for ensuring the reproduction of zooplankton. A more realistic model must account for the positive effect of the consumption, for example, we have to consider a fecundity rate which is dependent not only on the age of individuals but also on the phytoplankton biomass consumed by these individuals (see for example Clother and Brindley, 1999). This consideration will be adopted in future work. In short, the situation that we are investigating here is : assuming that the system is saturated, that is, the phytoplankton is abundant, some toxic phytoplankton species start to release toxic substances with some efficiency parameter $\nu$ and the aim is to examine the impact of toxic liberation process on the dynamics of the system.

In a first step, we prove existence of the solution of the system following the characteristic lines. We show that the semi group solution is quasi compact which helps when we deal with the asymptotic behaviour of solutions. We then highlight existence of a positive stationary solution which is the analogous, in the structured case, of the positive interior equilibrium in the global case studied in [2] and [4].

We focus then on the local properties of this stationary solution. The study of stability and bifurcation constitute the main result of this work. We use the classical method of linearisation around the stationary solution. First, we study the system in a general case and then in a particular case. In the general case we assume that mortality rate as well as fecundity rate are age dependent functions and we let the delay kernel be unspecified. In the particular case we assume that the delay kernel is a Dirac delta function and that mortality and fecundity rates are constant. Results with small values of the efficiency of the toxin are outlined.

Our analysis shows in both cases, in the particular case as well as in the general case, that under our hypothesis, toxic substances have a destabilizing effect on the phytoplankton-zooplankton system. Indeed, we showed, under some interpretable assumptions on parameters, that if $\nu < 0$ (that is to say that phytoplankton would have some positive effect on the growth of zooplankton) then the system would be stable around the positive stationary solution while for $\nu > 0$ the system is unstable, and when $\nu$ crosses the critical value $\nu = 0$ a bifurcation occurs. These qualitative results of the structured model are equivalent to those we can obtain from the non structured model based on the same assumption concerning the fecundity function of the zooplankton. The differences between the two models are quantitative. They concern the values of parameters characterizing the stationary solutions and the strenght of the unstability. These differences increase when the delay, the efficiency of the toxin or the life time of the zooplankton increases.

The paper is organized as follows : In section 2, we present and explain the mathematical model. In section 3, we study the existence and uniqueness of the solution and determine necessary and sufficient conditions for positive stationary solutions to exist. In section 4, we deal with the problem of stability of the stationary solutions and bifurcation in the general case. Section 5 is devoted to the precise description of the behaviour of the system in the particular case. Section 6 is devoted to a discussion of the impact of the structure, and comparison of the results of the previous sections and those obtained from the non structured model. We end with a discussion on all these results and their interpretations.

## 6.2 Mathematical Model

### 6.2.1 Presentation of the model

Here, we will consider the following model :

$$\begin{cases} \frac{dP}{dt}(t) = rP(t)\left(1 - \frac{P(t)}{K}\right) - f(P(t)) \int_0^\infty \alpha\Big(a, H_\alpha(t-a,t)\Big)\pi(a,t)da \\ \frac{\partial \pi}{\partial t} + \frac{\partial \pi}{\partial a} = -\mu\Big(a, H_\mu(t-a,t)\Big)\pi(a,t) \\ -\gamma\nu\pi(a,t) \int_{t-a}^{t} \alpha\Big(a-(t-s), H_\alpha(t-a,s)\Big)F(t-s,a)f(P(s))ds \\ \pi(0,t) = \int_0^\infty \varphi\Big(a, H_\varphi(t-a,t)\Big)\pi(a,t)da \\ P(\theta) = P_0(\theta), \theta \leq 0 \\ \pi(a,0) = \pi_0(a) \end{cases} \tag{6.1}$$

$P(t)$ represents the density of phytoplankton population at time $t$, while $\pi(a,t)$ represents the density of the cohort of zooplanktonic individuals born at time $\theta = t - a$ (cohort $t - a$) and present at time $t$, having therefore age $a$.
The parameter $r$ is the intrinsic growth rate of the phytoplankton population and $K$ is its carrying capacity.
The coefficient $\alpha\big(a, H_\alpha(t-a,t)\big)$ is the predation rate of an individual of cohort $t - a$ at time $t$ on phytoplankton species. It depends on the age of individuals and on the history $H_\alpha(t-a,t)$ of environmental conditions experienced by the cohort they belong. We represent $H_\alpha(\theta,t)$ (and later $H_\mu(\theta,t)$ and $H_\varphi(\theta,t)$ ) as some convolutions along the trajectories of the whole system from the birth date $\theta$ of a cohort until time $t$.
$\mu\big(a, H_\mu(t-a,t)\big)$ is the mortality rate of an individual of cohort $t - a$ at time $t$. It depends on the age of individuals and on the history $H_\mu(t-a,t)$ of environmental conditions experienced between their birth date $t - a$ and $t$.

$\alpha\big(a-(t-s), H_\alpha(t-a,s)\big)f(P(s))$ represents the effective consumption of each individual of cohort $t - a$ at a given time $s$ in the time interval $[t-a,t]$. The effective consumption of toxins by an individual of cohort $t-a$ at time $s$ is $\gamma\alpha\big(a-(t-s), H_\alpha(t-s,s)f(P(s))\big)$. The quantity of accumulated toxic substances which has, at time $t$ a potential lethal impact on each individual of cohort $t - a$, is as follows :

$$q(a,t) = \int_{t-a}^{t} \gamma\alpha\big(a-(t-s), H_\alpha(t-a,s)\big)f(P(s))F(t-s,a)ds.$$

The delay kernel $F(t-s,a)$ gives the fraction of toxic quantity consumed at time $s$ which has a potential lethal impact at time $t$. This kernel depends on the age and on the time spent $(t-s)$ from time of consumption $s$ until instant $t$. It can be interpreted in two ways : either as an accumulation-elimination process or as a delayed effect (for example, incubation period, the time necessary to toxic substances to be transported to an organ). The actual effect of the potential lethal impact is weighted by a coefficient $\nu$, which represents the efficiency of the toxin.

The function $\varphi\big(a, H_\varphi(t-a,t)\big)$ represents the fecundity of an individual of cohort $t - a$ at time $t$. It depends, as for $\alpha$ and $\mu$, on the age of individuals and on the environmental conditions $H_\varphi(t-a,t)$ experienced between time $t - a$ and time $t$. In system (6.1), the equation

$$\pi(0,t) = \int_0^\infty \varphi\big(a, H_\varphi(t-a,t)\big)\pi(a,t)da$$

describes the birth process of the population or the so-called birth law.

### 6.2.2 Discussion and hypotheses

In system (6.1), we have considered that individuals of the same cohort have the same history of toxicity and therefore, inside a cohort, the process of accumulation and elimination of toxins $F(t-s,a)$ is a common one and the effect on the mortality of zooplankton is the same. As we also assume that phytoplankton is sufficiently abundant so that the uptake function $f(P(t))$ is the same for all individuals at any time $t$. We assume also that nutrients are not a limiting factor : they are introduced as parameters of the model. Another hypothesis that we keep in mind is that the population we are considering here is able to reproduce, in other words that the expectation of reproduction

is greater than 1 when no toxic substance run counter to the survival.

In system (6.1), the dependency of $\mu, \alpha$ and $\varphi$ on the history of environmental conditions can be justified, in a general way, by the fact that these vital parameters can be influenced by conditions prevailing at some key dates in the life history, like birth dates or maturity dates. As we are interested in the impact of the structure on the dynamics of system (6.1), and because we want to compare the results with those of the global model of [4], we choose to remain close to the conditions of the global model. Therefore, we will not consider the entire complexity of the functions considered in system (6.1). We will restrict ourselves to functions which are not dependent on environmental conditions.

So, we assume in this study that the predation rate is constant while the fecundity and the mortality functions are assumed to be positive bounded, dependent on the age of the individuals, that $\varphi$ is locally Lipschitz and there exists $\underline{\mu} > 0$ and $M > 0$ such that : $0 < \underline{\mu} \leq \mu(a) \leq M$ ; $\forall a > 0$; that is, each individual is exposed to mortality with a positive probability. We also suppose that the process of toxicity ( delay kernel $F$ ) is independent on the age of individuals and is dependent only on the time spent from the consumption of toxins until instant $t$. The uptake function $f(P)$ is assumed to be positive and increasing and continuous.

### 6.2.3 The model to be investigated.

Taking the above remarks into account, the system that we consider is :

$$\left\{\begin{array}{c} \frac{dP}{dt}(t) = rP(t)\left(1 - \frac{P(t)}{K}\right) - \alpha f(P(t)) \int_0^{+\infty} \pi(a,t)da \\ \frac{\partial \pi}{\partial t} + \frac{\partial \pi}{\partial a} = -\mu(a)\pi(a,t) - \alpha\gamma\nu\pi(a,t) \int_{t-a}^{t} F(t-s)f(P(s))ds \\ \pi(0,t) = \int_0^{\infty} \varphi(a)\pi(a,t)da \\ P_0 > 0, \pi(a,0) = \pi_0(a) \end{array}\right. \tag{6.2}$$

In order to compare the formulation of this model with that of the global one (see [4]), we must assume that all the parameters are constants. Therefore, integrating (6.2) with respect to $a$, we obtain the equations satisfied by the total population denoted $Z(t) = \int_0^{t\infty} \pi(a,t)da$ :

$$\left\{\begin{array}{c} \frac{dP}{dt}(t) = rP(t)(1 - \frac{P(t)}{K}) - \alpha f(P(t))Z(t) \\ \frac{dZ(t)}{dt} = \varphi Z(t) - \mu Z(t) - \alpha\gamma\nu \int_0^{+\infty} Z^*(a,t)F(a)f(P(t-a))da \\ P_0 > 0, Z(0) = \int_0^{\infty} \pi_0(a)da \end{array}\right. \tag{6.3}$$

where $Z^*(a,t) = \int_a^{\infty} \pi(s,t)ds$ represents the part of the population born before time $t-a$, that is with age greater than $a$ and still alive at time $t$. System (6.3) is similar to the global system (see [4]), except for the term under the integral and the growth term, that is, the expression of the birth flux. We recall that in [4] the birth flux is proportional to the consumption while here it is a function of the fecundity of the generations living before time $t$.

Assuming a constant fecundity function, the global system would be written

$$\left\{\begin{array}{c} \frac{dP}{dt}(t) = rP(t)(1 - \frac{P(t)}{K}) - \alpha f(P(t))Z(t) \\ \frac{dZ(t)}{dt} = \varphi Z(t) - \mu Z(t) - \alpha\gamma\nu Z(t) \int_0^{+\infty} F(a)f(P(t-a))da \\ P_0 > 0, Z(0) = Z_0 > 0 \end{array}\right. \tag{6.4}$$

One explanation of the term under the integral in system (6.3) is that the mortality caused by toxicity consumed at time $t-a$ only affects the proportion of the population present at time $t$ but born before $t-a$, that is $Z^*(a,t)$ and not the whole population $Z(t)$ as in the system (6.4).

One can remark that the two systems : the system (6.4) and the system (6.3) would be governed by equations of similar structure and thus having the same dynamics, if the age pyramid (which is expressed by the function $Z^*$) is kept constant.

## 6.3 Solutions

In this section, first we prove existence and uniqueness of the solution of system (6.2) using the method of characteristic lines and then we prove that the semi group solution is quasi compact. We then study the existence and uniqueness of stationary solutions.

### 6.3.1 Existence and uniqueness

Solving the problem following the characteristic lines means that we consider the equation along the trajectories made by the vector field, in our case $(1,1)$, in the *time* $\times$ *age* space.
The new variable $s$ is a local time that we confound usually with $t$. In our case we have : $a = s + c$ where $c$ is a constant.

If we denote

$$u(s) = \pi(s+c, s)$$

then the main equation of system (6.2) can be written as

$$\frac{du}{ds} = -\left(\mu(s+c) + \overline{\nu}\int_0^{s+c} F(\xi) f(P(s-\xi))d\xi\right) u(s), \forall s \geq t_c. \tag{6.5}$$

where $t_c = \max(0, -c)$.
Thus we obtain the following solution of equation (6.5)

$$\begin{aligned} u(t) &= u(t_c)\exp\left(-\int_{t_c}^{t}\mu_c(s)ds\right), t \geq t_c \\ \text{where} \quad &: \quad \mu_c(t) = \mu(t+c) + \overline{\nu}\int_0^{t+c} F(\xi) f(P(t-\xi))d\xi \end{aligned}$$

Therefore the solution $u(t)$ can be written as

$$u(t) = \begin{cases} u(0)\exp\left(-\int_0^t \mu_c(s)ds\right) & \text{si } s+c \geq t \\ u(-c)\exp\left(-\int_{-c}^t \mu_c(s)ds\right) & \text{si } s+c < t \end{cases}$$

which implies that $\pi(a,t)$ has the following expression

$$\pi(a,t) = \begin{cases} \pi_0(a-t)\exp\left(-\int_0^t \mu_{a-t}(s)ds\right) & \text{si } a \geq t \\ \pi(0,t-a)\exp\left(-\int_{t-a}^t \mu_{a-t}(s)ds\right) & \text{si } a < t \end{cases} \tag{6.6}$$

The solution $\pi(a,t)$ in (6.6) is expressed in terms of $\pi(0,t)$ which is given by the boundary condition

$$\pi(0,t) = \int_0^{+\infty} \varphi(a)\pi(a,t)da \tag{6.7}$$

If we substitute the expression of $\pi(a,t)$ given by (6.6) in equation (6.7), we obtain

$$\begin{aligned} \pi(0,t) &= \int_0^t \varphi(a)\pi(0,t-a)\exp\left(-\int_{t-a}^t \mu_{a-t}(s)ds\right)da \\ &\quad + \int_t^{+\infty} \varphi(a)\pi_0(a-t)\exp\left(-\int_0^t \mu_{a-t}(s)ds\right)da \end{aligned} \tag{6.8}$$

Now, if we put

$$\phi(t) = \pi(0,t)$$

which is the population of age 0 at time $t$, then equation (6.8) will be written as a Volterra equation as follows :

$$\begin{aligned} \phi(t) &= \int_0^t \varphi(a)\phi(t-a)\exp\left(-\int_{t-a}^t \mu_{a-t}(s)ds\right)da \\ &\quad + \int_t^{+\infty} \varphi(a)\pi_0(a-t)\exp\left(-\int_0^t \mu_{a-t}(s)ds\right)da \end{aligned} \tag{6.9}$$

To prove the existence of a solution of equation (6.9), we construct a linear operator $\mathcal{K} : L^1(0,+\infty) \to C$, then we prove that $(\mathcal{I} - \mathcal{K})$ is invertible, which will confirm that equation (6.9) has one and only one solution.
Let $\mathcal{K}$ be the operator given by

$$\mathcal{K}(\phi)(t) = \int_0^t \varphi(a)\phi(t-a)\exp\left(-\int_{t-a}^t \mu_{a-t}(s)ds\right)da \tag{6.10}$$

$$= \int_0^t \varphi(t-a)\phi(a)\exp\left(-\int_a^t \mu_{-a}(s)ds\right)da \tag{6.11}$$

then the problem becomes : find a function $\phi$ such that

$$(\mathcal{I} - \mathcal{K})(\phi) = \int_.^{+\infty} \varphi(a)\pi_0(a-.)\exp\left(-\int_0^. \mu_{a-.}(s)ds\right)da \tag{6.12}$$

Let $\pi_0 \in L^1(0,+\infty)$ and let $T > 0$.
The operator $\mathcal{K}$ is continuous :

$$\exists C_1 > 0, |\mathcal{K}(\phi)|_\infty \leq \frac{C_1}{\underline{\mu}} |\phi|_\infty$$

where $\underline{\mu}$ is the lower bound of $\mu(.)$ as mentioned previously.
A straightforward calculation leads to

$$\exists C_2 > 0 \ / \ \left|\mathcal{K}^{n+1}(\phi)\right|_\infty \leq C_2 \frac{T^n}{n!} |\phi|_\infty$$

thus, for each $T > 0$ we can find an integer $n = n(T)$ such that $\mathcal{K}^n$ is a strict contraction. Therefore $(\mathcal{I} - \mathcal{K})$ is invertible and equation (6.9) has a unique solution given by :

$$\phi = (\mathcal{I} - \mathcal{K})^{-1}\left(\int_.^{+\infty} \varphi(a)\pi_0(a-.)\exp\left(-\int_0^. \mu_{a-.}(s)ds\right)da\right)$$

### 6.3.2 Quasi compactness of the semi group solution

Now we will prove that the semi group solution is quasi compact, that is to say that it is the sum of a compact operator and a small operator in some sense to be explained below. This property is very useful when we deal with the problem of asymptotic behaviour. It permits us to obtain the behaviour of the solutions from the study of the point spectrum only instead of the study of the whole spectrum of the generator of the semi group solution. Indeed, the objective is to show that the trajectories remain in a compact set when time evolves.
As mentioned before, the first equation of $P(t)$ is scalar and as $P(t)$ is bounded, the set which contains solutions $P(t)$ is compact. That is why we will concentrate ourselves on the equation of $\pi$ instead of $(P(t), \pi(t,.))$.
From (6.6), we can write the solution as follows :

$$\begin{aligned}\pi(a,t) &= \chi_{(t,+\infty)}(a)\pi_0(a-t)\exp(-\int_0^t \mu_{a-t}(s)ds) \\ &\quad +\chi_{(0,t)}(a)\pi(0,t-a)\exp(-\int_{t-a}^t \mu_{a-t}(s)ds)\end{aligned}$$

Let us denote

$$\begin{aligned}\left(U(t)\pi_0\right)(a) &= \chi_{(t,+\infty)}(a)\pi_0(a-t)\exp(-\int_0^t \mu_{a-t}(s)ds) \\ \left(W(t)\pi_0\right)(a) &= \chi_{(0,t)}(a)\pi(0,t-a)\exp(-\int_{t-a}^t \mu_{a-t}(s)ds)\end{aligned}$$

then

$$\pi(a,t) = \left(U(t)\pi_0\right)(a) + \left(W(t)\pi_0\right)(a)$$

It is easy to show that $\|U(t)\pi_0\| \underset{t\to+\infty}{\to} 0$ in $L^1$. Indeed, we have

$$\|U(t)\pi_0\|_{L^1} \leq \exp(-\underline{\mu}t)\,\|\pi_0\|\,, \text{ for every } \pi_0 \in L^1$$

which implies that

$$\|U(t)\|_{\mathcal{L}(L^1)} \leq \exp(-\underline{\mu}t)$$

For the compactness of the operator $W(t)$, it is sufficient that the following operator be compact.

$$L(\phi)(t) = \left(\int_0^{+\infty} \varphi(a+t)\phi(a)\exp\left(-\int_0^t \mu_a(s)ds\right)da\right)$$

Indeed, we want to prove that the map $\pi_0 \to \phi = (\mathcal{I}-\mathcal{K})^{-1}L(\pi_0)$ is compact. As the operator $(\mathcal{I}-\mathcal{K})^{-1}$ is bounded, it is sufficient to prove that operator $L$ is compact.
Before proving that the operator $L$ is compact, we note that

$$L : L^1(0,+\infty) \to C([0,+\infty))$$

Let $R > 0$, and let $B(0,R) \subset L^1(0,+\infty)$ be a bounded set. Define

$$\mathcal{F} = \{L(\phi); \phi \in B(0,R)\}$$

Our objective is to show that the family $\mathcal{F}$ is relatively compact. For that, let $\phi \in B(0,R)$. We can easily show that

$$|L(\phi)|_\infty \leq R \|\varphi\|_\infty$$

and thus the family $\mathcal{F}$ is bounded.
Now let us show that $\mathcal{F}$ is equicontinuous : Let $t, t' \in [0,+\infty)$. Under the conditions on $\mu$, $f$ and $\varphi$, we can prove that there exists a constant $k > 0$ such that :

$$\left|\varphi(\sigma+t)\exp(-\int_0^t \mu_\sigma(s)ds) - \varphi(\sigma+t')\exp(-\int_0^{t'} \mu_\sigma(s)ds)\right| \leq k\left|t-t'\right|$$

Therefore, we have

$$\left|L(\phi)(t) - L(\phi)(t')\right| \leq kR\left|t-t'\right|$$

$\forall \phi \in B(0,R)$. Thus, the family $\mathcal{F}$ is equicontinuous. By the Ascoli-Arzela theorem (see [1]) we conclude that $\mathcal{F}$ is relatively compact. That is to say that the operator $L$ is compact.

### 6.3.3 Stationary solutions

In the stationary mode, system (6.2) reads as

$$\begin{cases} r\overline{P}(1-\frac{\overline{P}}{K}) = \alpha f(\overline{P})\int_0^{+\infty}\overline{\pi}(a)da \\ \frac{d\overline{\pi}}{da} = -\left(\mu(a) + \nu\alpha\gamma f(\overline{P})\int_0^a F(s)ds\right)\overline{\pi}(a) \\ \overline{\pi}(0) = \int_0^{+\infty}\varphi(a)\overline{\pi}(a)da \end{cases} \tag{6.13}$$

It is easy to see that $(\overline{P}=0, \overline{\pi}=0)$ and $(\overline{P}=K, \overline{\pi}=0)$ are trivial stationary solutions. It is also evident that no stationary solution exists with a positive population of zooplankton and with suitable properties of $f(P)$, fullfilling $\overline{P}=K$. Based on the assumption that when there is no toxic phytoplankton the zooplankton population increases, we can see that a 2-tuple of the form $(\overline{P}=0, \overline{\pi}\neq 0)$ cannot be a stationary solution.
Let us now focus on the existence of non trivial stationary solutions $(\overline{P} \in ]0,K[, \overline{\pi}\neq 0)$.
Let us denote by $M(\overline{P},a)$ the total rate of mortality at age $a$ if the phytoplankton is at a constant level $\overline{P}$

$$M(\overline{P},a) = \left(\mu(a) + \alpha\gamma\nu f(\overline{P})\int_0^a F(\tau)d\tau\right)$$

It includes both natural mortality and mortality by toxin at the equilibrium. Therefore the stationary equation satisfied by $\overline{\pi}$ can be written as

$$\begin{cases} \frac{d\overline{\pi}}{da}(a) = -M(\overline{P},a)\overline{\pi}(a) \\ \overline{\pi}(0) = \int_0^{+\infty}\varphi(a)\overline{\pi}(a)da \end{cases}$$

which gives

$$\overline{\pi}(a) = \overline{\pi}(0)e^{-\int_0^a M(\overline{P},s)ds} \tag{6.14}$$

Now, if we substitute expression (6.14) in the expression of $\overline{\pi}(0)$, then we can easily see that existence of a non trivial stationary solution leads to the following neccessary and sufficient equilibrium relation

$$\int_0^{+\infty} \varphi(a) e^{-\int_0^a M(\overline{P},s)ds} da = 1$$

Let us denote by $\Phi(\overline{P})$ is the total rate of reproduction of each cohort of zooplankton population if the phytoplankton population remains at a level $\overline{P}$

$$\Phi(\overline{P}) = \int_0^{+\infty} \varphi(a) e^{-\int_0^a M(\overline{P},s)ds} da$$

The equilibrium relation expresses that, for a stationary solution $(\overline{P}, \overline{\pi})$ to exist, the total output of each cohort is exactly equal to its initial size. If we return to the first equation of (6.13) we have

$$\begin{aligned} r\overline{P}(1 - \frac{\overline{P}}{K}) &= \alpha f(\overline{P}) \int_0^{+\infty} \overline{\pi}(a) da \\ &= \alpha f(\overline{P}) \overline{\pi}(0) \int_0^{+\infty} e^{-\int_0^a M(\overline{P},s)ds} da \end{aligned}$$

we can then express $\overline{\pi}(0)$ in terms of $\overline{P} \neq 0$ as follows :

$$\overline{\pi}(0) = \frac{r\overline{P}(1 - \frac{\overline{P}}{K})}{\alpha f(\overline{P}) \int_0^{+\infty} e^{-\int_0^a M(\overline{P},s)ds} da}$$

**Proposition 1.** *Given a fecundity function $\varphi(.)$, a mortality function $\mu(.)$, a toxic efficiency parameter $\nu$ and a delay kernel $F(.)$, then necessary and sufficient conditions to a positive stationary solution ($\overline{P} \in ]0, K[, \overline{\pi} \neq 0$) to exist are as follows :*

$$\Phi(\overline{P}) = \int_0^{+\infty} \varphi(a) e^{-\int_0^a M(\overline{P},s)ds} da = 1 \tag{6.15}$$

$$\overline{\pi}(a) = \overline{\pi}(0) e^{-\int_0^a M(\overline{P},s)ds} \tag{6.16}$$

$$\overline{\pi}(0) = \frac{r\overline{P}(1 - \frac{\overline{P}}{K})}{\alpha f(\overline{P}) \int_0^{+\infty} e^{-\int_0^a M(\overline{P},s)ds} da} \tag{6.17}$$

Existence of a stationary solution is related to the existence of some $\overline{P}$ satisfying the equilibrium relation. As soon as such a $\overline{P}$ exists, the stationary solution is entirely determined in terms of $\overline{P}$. For positive values of the efficiency parameter $\nu$, $\Phi(\overline{P})$ is continuous and a strictly decreasing function. Then a necessary and sufficient condition for the existence of a $\overline{P} \in ]0, K[$ satisfying the equilibrium relation is

$$\Phi(0) > 1 > \Phi(K)$$

We have already assumed that if there is no toxic phytoplankton, the population of zooplankton increases, that is, that $\Phi(0) > 1$. The second constraint $\Phi(K) < 1$ means that when the phytoplankton is at its maximum $K$ the population of zooplankton decreases and goes to extinction. If this constraint is fullfilled, then there exists a unique value $\overline{P} \in ]0, K[$ which satisfies the equilibrium relation and, therefore, there exists a unique positive stationary solution.

**Proposition 2.** *Assuming :*

1) *in the absence of toxic phytoplankton, the population of zooplankton increases, and*
2) *in presence of toxic phytoplankton, given a fecundity function $\varphi(.)$ and a natural mortality function $\mu(.)$, the toxic efficiency parameter is positive and large enough (greater than some threshold value depending on $\varphi(.)$ and $\mu(.)$), that is, it satisfies*

$$\int_0^{+\infty} \varphi(a) e^{-\int_0^a M(K,s)ds} da < 1$$

*then there exists a unique positive stationary solution ($\overline{P} \in ]0, K[, \overline{\pi} \neq 0$).*

We have focused on the conditions for a positive stationary solution to exist in terms of constraints on the $3-$tuple $(\varphi(.), \mu(.), \nu)$. Now we can remark that given a level of phytoplankton $\overline{P} \in ]0, K[$, a positive mortality function $\mu(.)$, and a positive toxic efficiency parameter $\nu$, there exists a set of positive fecundity functions $\varphi(.)$ denoted here $\mathcal{F}(\overline{P}, \mu(.), \nu)$ that satisfie the equilibrium relation. Moreover, as $\varphi(.)$ is not explicitly involved in expression of $\overline{\pi}(a)$, the resulting stationary solution is the same for all functions $\varphi(.)$ chosen in $\mathcal{F}(\overline{P}, \mu(.), \nu)$. Therefore, for each 3-tuple $(\overline{P} \in ]0, K[, \mu(.) > 0, \nu > 0)$ there exists one and only one positive stationary solution associated with this 3-tuple. This stationary solution is given by

$$\overline{\pi}(a) = \overline{Z}(\overline{P}) \frac{e^{\int_0^a -M(\overline{P},s)ds}}{\int_0^{\infty} e^{-\int_0^a M(\overline{P},s)ds} da}$$

where $\overline{Z}(\overline{P})$ represents the total zooplankton population at the equilibrium and is given by

$$\overline{Z}(\overline{P}) = \frac{r\overline{P}(1 - \frac{\overline{P}}{K})}{\alpha f(\overline{P})}$$

The space $(\overline{P}, \mu(.), \nu)$ is less natural than the space of $(\varphi(.), \mu(.), \nu)$. But from the above remarks it is technically suitable for characterizing the behaviour of the studied system, and we will use it in the sequel for these reasons.

## 6.4 Stability analysis in the general case

In order to study the asymptotic behaviour of the solutions of system (6.2), we are going to linearise it around the stationary solution $(\overline{P}, \overline{\pi})$. We will then study the behaviour of the largest eigenvalue of the characteristic equation for small values of the toxin efficiency parameter.

### 6.4.1 Linearized system around stationary solutions

We perform the following change of coordinates

$$\begin{cases} \pi(a,t) = \widetilde{\pi}(a,t) + \overline{\pi}(a) \\ P(t) = \widetilde{P}(t) + \overline{P} \end{cases}$$

Recall that

$$M(\overline{P}, a) = \left( \mu(a) + \alpha\gamma\nu f(\overline{P}) \int_0^a F(\tau) d\tau \right)$$

and denote

$$\begin{aligned} R(\overline{P}) &= \left(r(1-\frac{2\overline{P}}{K}) - \alpha f'(\overline{P})\overline{Z}(\overline{P})\right) \\ &= \alpha f(\overline{P})\overline{Z}'(\overline{P}) \end{aligned}$$

where $f'(\overline{P})$ is the derivative of $f(\overline{P})$, and $\overline{Z}'(\overline{P})$ the derivative of $\overline{Z}(\overline{P}))$ with respect to $\overline{P}$
The linearised system around the equilibrium $(\overline{P}, \overline{\pi})$ is

$$\left\{ \begin{array}{c} \frac{d\widetilde{P}}{dt}(t) = R(\overline{P})\widetilde{P}(t) - \alpha f(\overline{P}) \int_0^{+\infty} \widetilde{\pi}(a,t)da \\ \frac{\partial\widetilde{\pi}}{\partial t}(a,t) + \frac{\partial\widetilde{\pi}}{\partial a}(a,t) = -M(\overline{P},a)\widetilde{\pi}(a,t) - \alpha\gamma\nu f'(\overline{P})\overline{\pi}(a) \int_0^a \widetilde{P}(t-s)F(s)ds \\ \widetilde{\pi}(0,t) = \int_0^{+\infty} \varphi(a)\widetilde{\pi}(a,t)da \end{array} \right. \tag{6.18}$$

As mentioned before, we consider $(\overline{P}, \mu(.), \nu)$ where $\overline{P} \in ]0, K]$ is the level of phytoplankton, $\mu(.) > 0$ is the mortality function in the absence of toxin and $\nu > 0$ is the efficiency of the toxin. For every point $(\overline{P} \in ]0, K], \mu(.), \nu > 0)$, we have seen that it is possible to find a set of fecundity functions $\overline{\varphi}(.)$ satisfying $\Phi(\overline{P}) = 1$. Therefore every point $(\overline{P} \in ]0, K], \mu(.), \nu > 0)$ is associated with a unique stationary solution. Consider now also points with $\nu \leq 0$. It is easy to show that for every point $(\overline{P} \in ]0, K], \mu(.) > 0, \nu = 0)$ there exists a stationary solution associated with a set of functions $\overline{\varphi}(.)$. It is also easy to show that for every $(\overline{P} \in ]0, K], \mu(.) > 0$ and $\nu < 0$, but still sufficiently small, there also exists a unique stationary solution with positive functions $\varphi(.)$. We will now examine the stability of the stationary solutions near $\nu = 0$.

### 6.4.2 Eigenvalues at $\nu = 0$

Given $\overline{P}$ and $\mu(.)$, the stationary solution for $\nu = 0$ is given by

$$\overline{\pi}(a) = \overline{Z}(\overline{P}) \frac{e^{-\int_0^a \mu(s)ds}}{\int_0^{\infty} e^{-\int_0^a \mu(s)ds} da}$$

for the fecundity functions $\varphi(.)$ satisfying the equilibrium relation

$$\int_0^{+\infty} \varphi(a) e^{-\int_0^a \mu(s)ds} da = 1$$

The linearized system around such stationary solutions at $\nu = 0$ is

$$\left\{ \begin{array}{c} \frac{d\widetilde{P}}{dt}(t) = R(\overline{P})\widetilde{P}(t) - \alpha f(\overline{P}) \int_0^{+\infty} \widetilde{\pi}(a,t)da \\ \frac{\partial\widetilde{\pi}}{\partial t}(a,t) + \frac{\partial\widetilde{\pi}}{\partial a}(a,t) = -\mu(a)\widetilde{\pi}(a,t) \\ \widetilde{\pi}(0,t) = \int_0^{+\infty} \varphi(a)\widetilde{\pi}(a,t)da \end{array} \right. \tag{6.19}$$

**Proposition 3.** *Considering the eigenvalues $\lambda$ and eigenvectors $(\widetilde{P}_0, \widetilde{\pi}_0(.))$ of the linearized system at $\nu = 0$, we can easy establish that :*

- *$\lambda = R(\overline{P})$ is an eigenvalue, with eigenvectors component $\widetilde{\pi}_0(.)$ satisfying $\widetilde{\pi}_0(.) = 0$*
- *$\lambda = 0$ is an eigenvalue, with eigenvectors component $\widetilde{\pi}_0(.)$ proportionnal to the corresponding component of stationary solution $\overline{\pi}(.)$*

- *the real parts of other eigenvalues, if they exist, are negative, and less than some negative upper bound.*

**Proof.** Seeking solutions of the form

$$\begin{aligned} \widetilde{P}(t) &= \exp(\lambda t)\,\widetilde{P}_0 \\ \widetilde{\pi}(a,t) &= \exp(\lambda t)\widetilde{\pi}_0(a) \end{aligned}$$

we obtain the following equations for the eigenvalues $\lambda$

$$\begin{aligned} (\lambda - R(\overline{P}))\widetilde{P}_0 &= -\alpha f(\overline{P}) \int_0^\infty \widetilde{\pi}_0(a)da \\ \frac{d\widetilde{\pi}_0(a)}{da} &= -(\lambda + \mu(a))\widetilde{\pi}_0(a) \\ \widetilde{\pi}_0(0) &= \int_0^\infty \varphi(a)\widetilde{\pi}_0(a)da \end{aligned}$$

It is easy to see that we have a first eigenvalue $\lambda = R(\overline{P})$ with $\widetilde{\pi}_0(.) = 0$. For the other eigenvalues, we have the following characteristic equation :

$$\int_0^\infty \varphi(a)e^{-\int_0^a \mu(s)ds}e^{-\lambda a}da = 1 \tag{6.20}$$

with $\widetilde{\pi}_0(.)$ part of eigenvectors satisfying

$$\widetilde{\pi}_0(a) = \widetilde{\pi}_0(0)e^{-\int_0^a (\lambda+\mu(s))ds}$$

Thus 0 is also an eigenvalue, with eigenvectors part $\widetilde{\pi}_0(.)$ satisfying

$$\begin{aligned} \widetilde{\pi}_0(a) &= \widetilde{\pi}_0(0)e^{-\int_0^a \mu(s)ds} \\ &= \frac{\widetilde{\pi}_0(0)}{\overline{\pi}(0)}\overline{\pi}(a) \end{aligned}$$

### 6.4.3 Behaviour of the system around $\nu = 0$

Consider now values of the efficiency of the toxin near 0. We will denote $(\overline{P}, \overline{\pi}(\nu, a))$ the $\nu$ dependent stationary solutions for varying $\nu$, the level of phytoplankton $\overline{P}$ and the natural mortality function $\mu(.)$ being fixed. Let $\varphi(\nu, a)$ be the $\nu$ dependent fecundity functions satisfying the equilibrium relations ensuring the existence of these stationary solutions. In the same way, we will use $\widetilde{P}(\nu, t)$, $\widetilde{\pi}(\nu, a, t)$ for designing the $\nu$ dependence of the solution in the linearized system 6.18 which, with these notations, becomes :

$$\left\{ \begin{array}{l} \frac{\partial}{\partial t}\widetilde{P}(\nu,t) = R\widetilde{P}(\nu,t) - \alpha f(\overline{P}) \int_0^{+\infty} \widetilde{\pi}(\nu,a,t)da \\ \\ \frac{\partial}{\partial t}\widetilde{\pi}(\nu,a,t) + \frac{\partial}{\partial a}\widetilde{\pi}(\nu,a,t) = -M(\nu,\overline{P},a)\widetilde{\pi}(\nu,a,t) \\ \qquad -\nu\alpha\gamma f'(\overline{P})\overline{\pi}(\nu,a) \int_0^a \widetilde{P}(\nu,t-s)F(s)ds \\ \\ \widetilde{\pi}(\nu,0,t) = \int_0^{+\infty} \overline{\varphi}(\nu,a)\widetilde{\pi}(\nu,a,t)da \end{array} \right. \tag{6.21}$$

We consider solutions of the form

$$\begin{aligned} \widetilde{P}(\nu,t) &= \exp(\lambda(\nu)t)\,\widetilde{P}_0(\nu) \\ \widetilde{\pi}(\nu,a,t) &= \exp(\lambda(\nu)t)\widetilde{\pi}_0(\nu,a) \end{aligned} \tag{6.22}$$

The eigenvalues $\lambda(\nu)$ and the eigenvectors $(\widetilde{P}_0(\nu),\widetilde{\pi}_0(\nu,a))$ are also $\nu$ dependent. We consider the eigenvalues branch $\lambda(\nu)$ in continuity with the 0 eigenvalue of system (6.19), that is the eigenvalues branch $\lambda(\nu)$ satisfying $\lambda(0)=0$, and corresponding eigenvectors. In examining the sign of the derivative $\lambda'(0)$ we come to

**Proposition 4.** *For a level of phytoplankton $\overline{P}\in]0,K[$ sufficiently large, that is, statisfying $\overline{Z}'(\overline{P})<0$ and for each natural mortality function $\mu(.)>0$, there exists a $\nu^*>0$ such that system (6.2), around the positive stationary solution associated with the point $\left(\overline{P},\mu(.),\nu\right)$, is stable for $\nu\in]-\nu^*,0[$, is unstable for $\nu\in]0,\nu^*[$ and a bifurcation occurs when $\nu$ crosses the value $\nu=0$.*

**Proof.** Substituting(6.22) into system (6.21), we obtain

$$\left\{\begin{array}{l} \lambda(\nu)\widetilde{P}_0(\nu)=R\widetilde{P}_0(\nu)-\alpha f(\overline{P})\int_0^{+\infty}\widetilde{\pi}_0(\nu,a)da \\ \lambda(\nu)\widetilde{\pi}_0(\nu,a)+\frac{\partial}{\partial a}\widetilde{\pi}_0(\nu,a)=-M(\nu,\overline{P},a)\widetilde{\pi}_0(\nu,a) \\ -\nu\alpha\gamma f'(\overline{P})\overline{\pi}(\nu,a)\left(\int_0^a\exp(-\lambda(\nu)s)F(s)ds\right)\widetilde{P}_0(\nu) \\ \widetilde{\pi}_0(\nu,0)=\int_0^{+\infty}\overline{\varphi}(\nu,a)\widetilde{\pi}_0(\nu,a)da \end{array}\right. \tag{6.23}$$

Eliminating $\widetilde{P}_0(\nu)$ from the combination of the first and second equations of system (6.23) leads to equation

$$\begin{aligned} &\left\{\alpha f(\overline{P})\int_0^{\infty}\widetilde{\pi}_0(\nu,a)da\right\}\left\{\nu\alpha\gamma f'(\overline{P})\overline{\pi}(\nu,a)\left(\int_0^a\exp(-\lambda(\nu)s)F(s)ds\right)\right\} \\ =\;&\{\lambda(\nu)-R\}\left\{\frac{\partial}{\partial a}\widetilde{\pi}_0(\nu,a)+\lambda(\nu)\widetilde{\pi}_0(\nu,a)+M(\nu,\overline{P},a)\widetilde{\pi}_0(\nu,a)\right\} \end{aligned} \tag{6.24}$$

We compute the derivative of (6.24) with respect to $\nu$ and evaluate it at $\nu=0$. We obtain :

$$\left\{\begin{array}{l} -R\left\{\frac{\partial^2}{\partial\nu\partial a}\widetilde{\pi}_0(0,a)+\lambda'(0)\widetilde{\pi}_0(0,a)+\mu(a)\frac{\partial}{\partial\nu}\widetilde{\pi}_0(0,a)+\left(\alpha\gamma f(\overline{P})\int_0^a F(s)ds\right)\widetilde{\pi}_0(0,a)\right\} \\ \qquad +\lambda'(0)\left\{\frac{\partial}{\partial a}\widetilde{\pi}_0(0,a)+\mu(a)\widetilde{\pi}_0(0,a)\right\} \\ \qquad =\left\{\alpha f(\overline{P})\int_0^{\infty}\widetilde{\pi}_0(0,a)da\right\}\left\{\alpha\gamma f'(\overline{P})\overline{\pi}(0,a)\left(\int_0^a F(s)ds\right)\right\} \end{array}\right. \tag{6.25}$$

while (differentiating the expression of $\widetilde{\pi}_0(\nu,0)$)

$$\frac{\partial}{\partial\nu}\widetilde{\pi}_0(0,0)=\int_0^{+\infty}\varphi(0,a)\left(\frac{\partial}{\partial\nu}\widetilde{\pi}_0(0,a)\right)da+\int_0^{\infty}\frac{\partial}{\partial\nu}\varphi(0,a)\widetilde{\pi}_0(0,a)da \tag{6.26}$$

But for the chosen branch of eigenvalues starting with $\lambda(0)=0$, we know the expression of the eigenvectors $\widetilde{\pi}_0(\nu,a)$ at $\nu=0$. These eigenvectors $\widetilde{\pi}_0(0,a)$ satisfy the same equations as $\overline{\pi}(0,a)$, as we have seen before. We have

$$\frac{\partial}{\partial a}\widetilde{\pi}_0(0,a)+\mu(a)\widetilde{\pi}_0(0,a)=0$$

and we can take $\widetilde{\pi}_0(0,a)=\overline{\pi}(0,a)$. In these conditions, the equation (6.25) can be written as a differential equation governing a function $\psi(.)$ defined by $\psi(a)=\frac{\partial}{\partial\nu}\widetilde{\pi}_0(0,a)$. This function represents the $\nu$ derivative of $\widetilde{\pi}(\nu,a)$ at $\nu=0$ for a given age $a$. We have

$$\psi'(a)=-\mu(a)\psi(a)+g(a)$$

where $g(a)$ is given as

$$g(a)=-\overline{\pi}(0,a)\left(\lambda'(0)+\alpha\gamma\int_0^a F(s)ds\left(\frac{f(\overline{P})\overline{Z}'(\overline{P})+f'(\overline{P})\overline{Z}(\overline{P})}{\overline{Z}'(\overline{P})}\right)\right)$$

Solving this differential equation gives the expression of $\frac{\partial}{\partial\nu}\widetilde{\pi}_0(0,a)$, as

$$\frac{\partial}{\partial\nu}\widetilde{\pi}_0(0,a)=\frac{\partial}{\partial\nu}\widetilde{\pi}_0(0,0)\exp\left(-\int_0^a\mu(s)ds\right)+\int_0^a\exp\left(-\int_s^a\mu(\sigma)d\sigma\right)g(s)ds$$

Substituting this expression of $\frac{\partial}{\partial\nu}\widetilde{\pi}_0(0,a)$ into equation (6.26), we arrive at a relation between $\lambda'(0)$ and the $\nu$ derivative of the fecundity function $\frac{\partial}{\partial\nu}\overline{\varphi}(0,a)$ at $\nu=0$. This relation can be written as :

$$\begin{aligned}\lambda'(0)\left(\int_0^{\infty}a\overline{\pi}(0,a)\overline{\varphi}(0,a)da\right)&=\int_0^{\infty}\overline{\pi}(0,a)\frac{\partial}{\partial\nu}\overline{\varphi}(0,a)da\\-\int_0^{\infty}\overline{\pi}(0,a)\overline{\varphi}(0,a)&\left(\alpha\gamma\frac{f(\overline{P})\overline{Z}'(\overline{P})+f'(\overline{P})\overline{Z}(\overline{P})}{\overline{Z}'(\overline{P})}\int_0^a\int_0^s F(\sigma)d\sigma ds\right)da\end{aligned}$$

But using now the equilibrium relation when $\nu\neq 0$ that is

$$\int_0^{+\infty}\overline{\varphi}(\nu,a)\exp\left(-\int_0^a M(\nu,\overline{P},s)ds\right)da=1 \tag{6.27}$$

we can compute the $\nu$ derivative of the fecundity function $\frac{\partial}{\partial\nu}\overline{\varphi}(0,a)$ at $\nu=0$. Taking the derivative of (6.27) and evaluating it at $\nu=0$, we obtain

$$\int_0^{\infty}\overline{\pi}(0,a)\frac{\partial}{\partial\nu}\overline{\varphi}(0,a)da=\alpha\gamma f(\overline{P})\int_0^{\infty}\overline{\pi}(0,a)\overline{\varphi}(0,a)\left(\int_0^a\int_0^s F(\sigma)d\sigma ds\right)da$$

Substituting this expression into the equation giving $\lambda'(0)$ we obtain finally

$$\begin{aligned}&\lambda'(0)\left(\int_0^{\infty}a\overline{\pi}(0,a)\overline{\varphi}(0,a)da\right)=\\-&\frac{f'(\overline{P})\overline{Z}(\overline{P})}{f(\overline{P})\overline{Z}'(\overline{P})}\left(\alpha\gamma f(\overline{P})\int_0^{\infty}\overline{\pi}(0,a)\overline{\varphi}(0,a)\left(\int_0^a\int_0^s F(\sigma)d\sigma ds\right)da\right)\end{aligned}$$

which can be written as

$$\lambda'(0) = -\frac{f'(\overline{P})\overline{Z}(\overline{P})}{f(\overline{P})\overline{Z}'(\overline{P})}\alpha\gamma f(\overline{P})\frac{\int_0^\infty \overline{\pi}(0,a)\overline{\varphi}(0,a)\left(\int_0^a (a-s)F(s)ds\right)da}{\left(\int_0^\infty \overline{\pi}(0,a)\overline{\varphi}(0,a)ada\right)} \tag{6.28}$$

When $\overline{Z}'(\overline{P}) < 0$, $\lambda'(0)$ is positive. When $\nu$ goes from small negative values to small positive ones, so does also $\lambda$ on the eigenvalues branch $\lambda(\nu)$ satisfying $\lambda(0) = 0$. But we know from the study of the system (6.19) ($\nu = 0$) for negative values of $\overline{Z}'(\overline{P})$ (namely for $R(\overline{P} < 0)$, that the 0 eigenvalue is the eigenvalue with the largest real part. We conclude by continuity that $\lambda(\nu)$ will preserve this property in a neighborhood of $\nu = 0$. In this neighborhood, the stationary solutions with $\nu < 0$ will be stable, and the stationary solutions with $\nu > 0$ will be unstable.

## 6.5 A particular case : Delay kernel as a Dirac function

In the particular case, we assume that the fertility and the mortality functions are constant : $\varphi(a) = \overline{\varphi}$, $\mu(a) = \mu$, and that the delay kernel $F(t)$ is a Dirac delta function. In other words, we assume that there exists $a^* > 0$ such that

$$\int_0^a F(s)ds = \begin{cases} 0 \text{ si } a < a^* \\ 1 \text{ si } a > a^* \end{cases}$$

### 6.5.1 The positive stationary solution

Let us denote by $\overline{\mu}$ for a given $3-$tuple $(\overline{P}, \mu, \nu)$, the total mortality, both natural and by toxin, for every age greater than $a^*$

$$\overline{\mu} = \mu + \alpha\gamma\nu f(\overline{P}).$$

Thus, if the phytoplankton is maintained at a constant level, then the total reproduction rate of a cohort is given by

$$\Phi(\overline{P}) = \overline{\varphi}\left[\frac{1-e^{-\mu a^*}}{\mu} + \frac{e^{-\mu a^*}}{\overline{\mu}}\right]$$

Therefore, the equilibrium relation $\Phi(\overline{P}) = 1$ leads, for a given $3-$tuple $(\overline{P}, \mu, \nu)$, to a fecundity $\overline{\varphi}$ given by

$$\overline{\varphi} = \frac{\mu\overline{\mu}}{\mu e^{-\mu a^*} + \overline{\mu}(1-e^{-\mu a^*})}$$

$\overline{\varphi}$ and $\overline{\mu}$ are functions of $\overline{P}, \mu, \nu$ and also of the delay $a^*$.
It is easy to see that

$$\begin{aligned} \overline{\varphi} - \mu &= \frac{\mu(\overline{\mu}-\mu)e^{-\mu a^*}}{\mu e^{-\mu a^*} + \overline{\mu}(1-e^{-\mu a^*})} \\ \overline{\varphi} - \overline{\mu} &= -\frac{\overline{\mu}(\overline{\mu}-\mu)(1-e^{-\mu a^*})}{\mu e^{-\mu a^*} + \overline{\mu}(1-e^{-\mu a^*})} \end{aligned}$$

Then, for $\nu > 0$,

$$\overline{\varphi} - \mu > 0\,;\ \overline{\varphi} - \overline{\mu} < 0\,;\ \lim_{a^* \to \infty} \overline{\varphi} - \mu = 0\,;\ \lim_{a^* \to 0} \overline{\varphi} - \overline{\mu} = 0$$

Note also that $\overline{\varphi} - \mu$ increase with $\overline{P}$ and $\nu$.
The stationary solution is expressed as follows

$$\overline{\pi}(a) = \begin{cases} \overline{Z}(\overline{P})\overline{\varphi}e^{-\mu a} \text{if } a < a^* \\ \overline{Z}(\overline{P})\overline{\varphi}e^{-\mu a^*}e^{-\overline{\mu}(a-a^*)} \text{if } a > a^* \end{cases}$$

### 6.5.2 The linearized system around the stationary solution

The equation of $\widetilde{\pi}$ becomes

$$\begin{aligned} \frac{\partial \widetilde{\pi}}{\partial t}(a,t) + \frac{\partial \widetilde{\pi}}{\partial a}(a,t) &= \begin{cases} -\mu\widetilde{\pi}(a,t) \text{ if } a < a^* \\ -\overline{\mu}\widetilde{\pi}(a,t) - \alpha\gamma\nu f'(\overline{P})\overline{\pi}(a)\widetilde{P}(t-a^*) \text{ if } a > a^* \end{cases} \\ \widetilde{\pi}(0,t) &= \overline{\varphi}\int_0^{+\infty} \widetilde{\pi}(a,t)da \end{aligned}$$

Let us denote

$$\begin{cases} \widetilde{Z}_1(t) = \int_0^{a^*} \widetilde{\pi}(a,t)da \\ \widetilde{Z}_2(t) = \int_{a^*}^{+\infty} \widetilde{\pi}(a,t)da \end{cases} \tag{6.29}$$

$\widetilde{Z}_1(t)$ (respectively $\widetilde{Z}_2(t)$) represents the difference between the current number and the stationary number of individuals of age less than $a^*$ (respectively greater than $a^*$), at time $t$.
If we express system (6.18) in terms of these new variables, we obtain the following equations

$$\begin{cases} \frac{d\widetilde{P}}{dt}(t) = R(\overline{P})\widetilde{P}(t) - \alpha f(\overline{P})\left(\widetilde{Z}_1(t) + \widetilde{Z}_2(t)\right) \\ \frac{d\widetilde{Z}_1}{dt}(t) + \widetilde{\pi}(a^*,t) - \overline{\varphi}(\widetilde{Z}_1(t) + \widetilde{Z}_2(t)) = -\mu\widetilde{Z}_1(t) \\ \frac{d\widetilde{Z}_2}{dt}(t) - \widetilde{\pi}(a^*,t) = -\overline{\mu}\widetilde{Z}_2(t) - \alpha\gamma\nu f'(\overline{P})\left(\int_{a^*}^{+\infty}\overline{\pi}(a)da\right)\widetilde{P}(t-a^*) \end{cases} \tag{6.30}$$

As we are interested in the asymptotic behaviour of system (6.2), we consider large values of $t$, that is we assume that $t > a^*$. From the equation of $\widetilde{\pi}(a,t)$ for $a^* > a$ we have

$$\begin{aligned} e^{\mu a}\widetilde{\pi}(a, t-a^*+a) &= \text{constant} \\ \widetilde{\pi}(0, t-a^*) &= e^{\mu a^*}\widetilde{\pi}(a^*,t) \\ \widetilde{\pi}(a^*,t) &= e^{-\mu a^*}\widetilde{\pi}(0,t-a^*) \\ \widetilde{\pi}(a^*,t) &= e^{-\mu a^*}\overline{\varphi}\left(\int_0^{\infty}\widetilde{\pi}(a,t)da\right) \\ \widetilde{\pi}(a^*,t) &= e^{-\mu a^*}\overline{\varphi}\left(\widetilde{Z}_1(t) + \widetilde{Z}_2(t)\right) \end{aligned}$$

Therefore, system (6.30) can be written as

$$\begin{cases} \frac{d\widetilde{P}}{dt}(t) = R(\overline{P})\widetilde{P}(t) - \alpha f(\overline{P})\left(\widetilde{Z}_1(t) + \widetilde{Z}_2(t)\right) \\ \frac{d\widetilde{Z}_1}{dt}(t) = (\overline{\varphi} - \mu)\widetilde{Z}_1(t) + \overline{\varphi}\widetilde{Z}_2(t) - e^{-\mu a^*}\overline{\varphi}\left\{\widetilde{Z}_1(t-a^*) + \widetilde{Z}_2(t-a^*)\right\} \\ \frac{d\widetilde{Z}_2}{dt}(t) = -\overline{\mu}\widetilde{Z}_2(t) + e^{-\mu a^*}\overline{\varphi}\left\{\widetilde{Z}_1(t-a^*) + \widetilde{Z}_2(t-a^*)\right\} \\ -\alpha\gamma\nu f'(\overline{P})\left(\int_{a^*}^{+\infty}\overline{\pi}(a)da\right)\widetilde{P}(t-a^*) \end{cases} \tag{6.31}$$

In fact, system (6.31) can be written in a different way, which will give interpretable entities on the one hand, and a relatively simple characteristic equation on the other hand. Now let us consider the following change of coordinates :

$$\begin{aligned} \widetilde{Z}(t) &= \widetilde{Z}_1(t) + \widetilde{Z}_2(t) \\ \widetilde{U}(t) &= (\varphi - \mu)\widetilde{Z}(t) - (\overline{\mu} - \mu)\widetilde{Z}_2(t) \end{aligned}$$

$\widetilde{Z}(t)$ represents the difference between the total population of zooplankton at time $t$ and the total population at equilibrium. $\widetilde{U}(t)$ represents the growth of $\widetilde{Z}(t)$ per unit of time, or the difference between the crude reproduction and the total mortality when the phytoplankton population is maintained at a constant level $\overline{P}$. Recall that $\overline{Z}(\overline{P})$ is the total population of zooplankton at equilibrium. With these new variables system (6.31) becomes :

$$\left\{ \begin{array}{c} \frac{d\widetilde{P}}{dt}(t) = \alpha f(\overline{P}) \left\{ \overline{Z}'(\overline{P})\widetilde{P}(t) - \widetilde{Z}(t) \right\} \\ \frac{d\widetilde{Z}}{dt}(t) = \widetilde{U}(t) - \frac{f'(\overline{P})}{f(\overline{P})}\overline{Z}(\overline{P})(\overline{\varphi} - \mu)\widetilde{P}(t - a^*) \\ \frac{d\widetilde{U}}{dt}(t) = (\overline{\varphi} - \mu - \overline{\mu})\widetilde{U}(t) - \frac{f'(\overline{P})}{f(\overline{P})}\overline{Z}(\overline{P})(\overline{\varphi} - \mu)\widetilde{P}(t - a^*)(\overline{\varphi} - \overline{\mu}) \\ +\overline{\mu}(\overline{\varphi} - \mu) \left\{ \widetilde{Z}(t) - \widetilde{Z}(t - a^*) \right\} \end{array} \right. \tag{6.32}$$

Note here that system (6.32) is expressed in terms of global variables. To obtain the formulation of system (6.31) and then (6.32), the calculation involves a tedious but straightforward computation using the expression of $\overline{\varphi}$.

### 6.5.3 The characteristic equation and its properties

Now, we search solutions in exponential form

$$\left\{ \begin{array}{l} \widetilde{P}(t) = e^{\lambda t}\widetilde{P}_0 \\ \widetilde{Z}(t) = e^{\lambda t}\widetilde{Z}_0 \\ \widetilde{U}(t) = e^{\lambda t}\widetilde{U}_0 \end{array} \right. \tag{6.33}$$

If we substitute expressions (6.33) into system (6.32), we arrive at the characteristic equation, which can be written as follows :

$$-p(\lambda)e^{\lambda a^*} + q(\lambda) = 0 \tag{6.34}$$

where

$$\begin{aligned} p(\lambda) &= \left(\lambda - \alpha f(\overline{P})\overline{Z}'(\overline{P})\right)(\lambda - (\overline{\varphi} - \mu))(\lambda + \overline{\mu}) \\ q(\lambda) &= \left(\alpha f'(\overline{P})\overline{Z}(\overline{P}) - \overline{\mu}\right)(\overline{\varphi} - \mu)\lambda \\ &+ \left(\alpha f'(\overline{P})\overline{Z}(\overline{P})\mu + \overline{\mu}\alpha f(\overline{P})\overline{Z}'(\overline{P})\right)(\overline{\varphi} - \mu) \end{aligned}$$

In order to study the stability of system (6.2) in the case where the delay kernel is a Dirac distribution, we focus on equation (6.34). It can be written as

$$\exp(\lambda a^*) = \frac{q(\lambda)}{p(\lambda)}$$

We have

$$\begin{aligned}\lim_{\lambda\to 0}\frac{q(\lambda)}{p(\lambda)} &= 1+\frac{\mu}{\overline{\mu}}\frac{f'(\overline{P})\overline{Z}(\overline{P})}{f(\overline{P})\overline{Z}'(\overline{P})}\\ \lim_{\lambda\to+\infty}\frac{q(\lambda)}{p(\lambda)} &= 0\end{aligned}$$

Then, if $\overline{Z}'(\overline{P}) < 0$ the functions $\exp(\lambda a^*)$ and $\frac{q(\lambda)}{p(\lambda)}$ will intersect in the positive octant because $\lim_{\lambda\to 0}\frac{q(\lambda)}{p(\lambda)} < 1$ and $\lambda = (\overline{\varphi}-\mu) > 0$ is an asymptote of $\frac{q(\lambda)}{p(\lambda)}$.
If $\overline{Z}'(\overline{P}) > 0$, then the function $\frac{q(\lambda)}{p(\lambda)}$ has two asymptotes and of course the functions $\exp(\lambda a^*)$ and $\frac{q(\lambda)}{p(\lambda)}$ will also intersect in the positive octant. In conclusion, we have the following result :

**Proposition 5.** *The characteristic equation (6.34) has at least one positive real eigenvalue for each* $\nu > 0$.

### 6.5.4 Stability analysis

We consider the extended space of points $(\overline{P},\mu,\nu)$ where $\nu$ can be null or negative. We follow the same framework as in the general case (see section 6.4), but now, both the natural mortality $\mu$ and the fecundity function $\varphi$ are independent on the age. We will examine the stability of the stationary solutions near $\nu = 0$

**Proposition 6.** *Assuming that* $\varphi$ *and* $\mu$ *are independent on the age, and that the delay kernel is a Dirac delta function, then for each level of phytoplankton* $\overline{P}\in]0,K[$ *and each natural mortality rate* $\mu > 0$, *there exists a* $\nu^* > 0$ *such that system (6.2), around the positive stationary solution associated with the points* $(\overline{P},\mu,\nu)$, *is stable for* $\nu\in]-\nu^*,0[$, *is unstable for* $\nu\in]0,\nu^*[$ *and a bifurcation occurs when* $\nu$ *crosses the value* $\nu = 0$.

**Proof.** We will study the characteristic equation for $\nu = 0$ and then for $\nu$ in the vicinity of 0. We start from a point $[\overline{P},\mu,0]$ and progress in the space toward $\nu > 0$. We will denote $\overline{\varphi}(\nu)$ the function giving the value of $\overline{\varphi}$ satisfiying the equilibrium relation for a given $\nu$, $\overline{P}$ and $\mu$ being fixed. We have $\overline{\varphi}(0) = \mu$ and $\overline{\varphi}'(0) = \alpha\gamma f(\overline{P})e^{-\mu a^*}$

When $\nu = 0$, the characteristic equation reduces to :

$$\lambda\left(\lambda-\alpha f(\overline{P})\overline{Z}'(\overline{P})\right)(\lambda+\mu) = 0$$

and the eigenvalues are

$$\lambda_1 = 0,\ \lambda_2 = \alpha f(\overline{P})\overline{Z}'(\overline{P}),\ \lambda_3 = -\mu$$

When $\overline{Z}'(\overline{P}) < 0$, the 0 eigenvalue is the largest one. Let $\lambda_{max}(\nu)$ be the largest eigenvalue for a given $\nu$. We have $\lambda_{max}(0) = 0$. Let us compute the derivative of $\lambda_{max}(\nu)$ at $\nu = 0$.From a straightforward calculation on the characteristic equation we obtain

$$\lambda'_{max}(0) = -\left(\frac{f'(\overline{P})\overline{Z}(\overline{P})}{f}(\overline{P})\overline{Z}'^{(\overline{P})}\right)\left\{\alpha\gamma f(\overline{P})e^{-\mu a^*}\right\}$$

Thus, as $\overline{Z}'(\overline{P}) < 0$ the sign of $\lambda'_{max}(0)$ is positive. As $\nu$ goes from small negative values to small positive values, the largest eigenvalue goes from small negative values to small positive values, and the system goes from a stable behaviour around the stationary solution to an unstable one.

### 6.5.5 Stability analysis in the case of trivial solutions

It is easy to see that $E_0(0,0)$ and $E_1(K,0)$ are trivial stationary solutions. The linearisation of system (6.2) around $E_0(0,0)$ shows that $E_0$ is always unstable. Indeed, the linearisation of the system around $E_0$ and in the case of Dirac delta function is as follows :

$$\begin{cases} \frac{d\widetilde{P}}{dt}(t) = r\widetilde{P}(t) \\ \frac{dZ(t)}{dt} = u(t) \\ \frac{du}{dt}(t) = -\mu u(t) + \mu(\overline{\varphi} - \mu)(Z(t) - Z(t-a^*)) \end{cases}$$

then, $\lambda = r$ is an eigenvalue, which implies that $E_0$ is unstable.
The linearisation around $E_1$ is as follows :

$$\begin{cases} \frac{d\widetilde{P}}{dt}(t) = -r\widetilde{P}(t) - \alpha f(K)Z(t) \\ \frac{dZ(t)}{dt} = u(t) \\ \frac{du}{dt}(t) = (\overline{\varphi} - \mu - \overline{\mu})u(t) + \overline{\mu}(\overline{\varphi} - \mu)(Z(t) - Z(t-a^*)) \end{cases}$$

The characteristic equation associated with $E_1$ is

$$\left\{\lambda^2 - \lambda(\overline{\varphi} - \mu - \overline{\mu}) - \overline{\mu}(\overline{\varphi} - \mu)\right\} e^{\lambda a^*} + \overline{\mu}(\overline{\varphi} - \mu) = 0 \tag{6.35}$$

therefore $\lambda = 0$ is an eigenvalue whatever the parameter values be. We can easily prove that all other eigenvalues have negative real part.
To conclude the behaviour near $E_1$, one method is to reduce the system to a center manifold. We will use the method developed in [5]. For this end and for a technical reason, we use system (6.31) in terms of $(\widetilde{P}, \widetilde{Z}_1, \widetilde{Z}_2)$ and we rewrite it as follows :

$$\frac{d}{dt}\begin{pmatrix} \widetilde{P}(t) \\ \widetilde{Z}_1(t) \\ \widetilde{Z}_2(t) \end{pmatrix} = L\begin{pmatrix} \widetilde{P}(t) \\ \widetilde{Z}_1(t) \\ \widetilde{Z}_2(t) \end{pmatrix} + M\begin{pmatrix} \widetilde{P}(t-a^*) \\ \widetilde{Z}_1(t-a^*) \\ \widetilde{Z}_2(t-a^*) \end{pmatrix} + \begin{pmatrix} g_1(\widetilde{P}, \widetilde{Z}_1, \widetilde{Z}_2) \\ 0 \\ g_3(\widetilde{P}(t-a^*), \widetilde{Z}_2(t)) \end{pmatrix}$$

where

$$L = \begin{pmatrix} -r & -\alpha f(\overline{P}) & -\alpha f(\overline{P}) \\ 0 & (\overline{\varphi} - \mu) & \overline{\varphi} \\ 0 & 0 & -(\mu + \alpha\gamma\nu f(\overline{P})) \end{pmatrix}$$

$$M = \begin{pmatrix} 0 & 0 & 0 \\ 0 & -\overline{\varphi}e^{-\mu a^*} & -\overline{\varphi}e^{-\mu a^*} \\ -\alpha\gamma\nu f'(\overline{P})\overline{Z}_2 & \overline{\varphi}e^{-\mu a^*} & \overline{\varphi}e^{-\mu a^*} \end{pmatrix}$$

$$\begin{aligned} g_1(X,Y,Z) &= -\frac{r}{K}X^2 - \alpha[\overline{Z}_1 + \overline{Z}_2][f(\overline{P}+X) - f(\overline{P}) - f'(\overline{P})X] \\ &- \alpha[f(\overline{P}+X) - f(\overline{P})][Y+Z] \\ g_3(X,Y) &= -(\alpha\gamma\nu)Y[f(\overline{P}+X) - f(\overline{P})] \\ &- (\alpha\gamma\nu)\overline{Z}_2[f(\overline{P}+X) - f(\overline{P}) - f'(\overline{P})X] \end{aligned}$$

Here, $(\overline{P}, \overline{Z}_1, \overline{Z}_2)$ is an equilibrium point of system (6.31).
In the case of the axial equilibrium $E_1(K,0)$, a long and tedious calculation, following the method developed in [5] leads to the reduced scalar equation :

$$\frac{ds}{dt}(t) = -(\alpha\gamma\nu)\phi_1\psi_3[\phi_3 f'(K)]s^2(t) - (\alpha\gamma\nu)\phi_1^2\psi_3[\phi_3\frac{f''(K)}{2}]s^3(t) + o(s^3)$$

where

$$\begin{aligned}\phi_1 &= -\frac{\alpha\mu f(K)(\mu+\alpha\gamma\nu f(K))}{r}; \phi_2 = \overline{\varphi}(\mu+\alpha\gamma\nu f(K))(1-e^{-\mu a^*}); \phi_3 = \overline{\varphi}\mu e^{-\mu a^*};\\ \psi_1 &= 0; \psi_2 = (\mu+\alpha\gamma\nu f(K)) - \overline{\varphi}e^{-\mu a^*}; \psi_3 = \overline{\varphi}(1-e^{-\mu a^*})\end{aligned}$$

We summarize the results in the following proposition.

**Proposition 7.** *If $\nu = 0$ then $E_1(K,0)$ is stable but not asymptotically stable. Assume $\nu \neq 0$. Then we have :*

1) *If $f'(K) \neq 0$ then the axial equilibrium $E_1(K,0)$ is unstable.*
2) *If $f'(K) = 0$ then we have two cases :*
   a) *If $\nu f''(K) > 0$ then $E_1(K,0)$ is asymptotically stable*
   b) *If $\nu f''(K) < 0$ then $E_1(K,0)$ is unstable.*

## 6.6 Discussion

### 6.6.1 Comparison with non structured models

As mentioned before, one objective of this work is to compare the present structured model with the non structured models considered in [2] and [4]. But we investigated here a situation where the birth flux is determined by a fecundity function in terms of age, without dependence on the consumption. Therefore, we must compare with a simplified version of the global models outlined in [4]. This version is given by system (6.3) :

$$\left\{\begin{array}{c}\frac{dP}{dt}(t) = rP(t)(1-\frac{P(t)}{K}) - \alpha f(P(t))Z(t)\\ \frac{dZ(t)}{dt} = \overline{\varphi}Z(t) - \mu Z(t) - \alpha\gamma\nu Z(t)\int_0^{+\infty} F(a)f(P(t-a))da\\ P_0 > 0, Z(0) = Z_0 > 0\end{array}\right.$$

The same kind of calculations made before for the structured model in the particular case, leads to the corresponding equations and properties for system (6.3) :

- equilibrium relation $\Phi(\overline{P}) = 1 : \overline{\varphi} = \overline{\mu}$
- stationary solution for a given $\overline{P}$

$$\overline{Z}(\overline{P}) = \frac{r\overline{P}(1-\frac{\overline{P}}{K})}{\alpha f(\overline{P})}$$

- linearized system around stationary solution, with non specified delay kernel

$$\begin{cases} \frac{d\tilde{P}}{dt}(t) = \alpha f(\overline{P}) \left\{ \overline{Z}'(\overline{P})\tilde{P}(t) - \tilde{Z}(t) \right\} \\ \frac{d\tilde{Z}}{dt}(t) = -\frac{f'(\overline{P})}{f(\overline{P})}\overline{Z}(\overline{P})(\overline{\varphi} - \mu) \int_0^\infty F(a)\tilde{P}(t-a)da \end{cases}$$

- linearized system around stationary solution, with a Dirac delay kernel

$$\begin{cases} \frac{d\tilde{P}}{dt}(t) = \alpha f(\overline{P}) \left\{ \overline{Z}'(\overline{P})\tilde{P}(t) - \tilde{Z}(t) \right\} \\ \frac{d\tilde{Z}}{dt}(t) = -\frac{f'(\overline{P})}{f(\overline{P})}\overline{Z}(\overline{P})(\overline{\varphi} - \mu)\tilde{P}(t-a^*) \end{cases}$$

- characteristic equation, with a Dirac delay kernel

$$\lambda\left(\lambda - \alpha f(\overline{P})\overline{Z}'(\overline{P})\right) = \alpha f'(\overline{P})\overline{Z}(\overline{P})(\overline{\varphi} - \mu)e^{-\lambda a^*}$$

- derivative of $\lambda(\nu)$ at $\nu = 0$ on the eigenvalues branch starting from $\lambda(0) = 0$, in both cases (non specified delay kernel and Dirac delay kernel)

$$\lambda'(0) = -\left(\frac{f'(\overline{P})\overline{Z}(\overline{P})}{f(\overline{P})\overline{Z}'(\overline{P})}\right)\{\alpha\gamma f(\overline{P})\}$$

These results show that both systems, the non structured and the structured one, are qualitatively equivalent when we deal with the existence and stability of stationary solutions. Indeed, in both cases there exists under suitable conditions a unique positive stationary solution. In both cases, this stationary solution is unstable. In both cases also, we find the same threshold value of the phytoplankton population. Beyond this threshold the same bifurcation appears : When the efficiency of toxin goes from small negative values (that is , values which were propitious for the zooplankton) to positive ones, the stationary solutions go from stable to unstable states.

The differences between the two systems, from the stationary solutions point of view, are rather quantitative. These quantitative differences are coming from the absence of any reference to the delay or delay kernel made by the non structured model in some important expressions.
This absence appears first in the equilibrium relation ensuring the existence of the stationary solution. For a given level of phytoplankton population, a given natural mortality and a given efficiency of the toxin, the non structured model overrates the level of fecundity necessary for the equilibrium to exist. In the case of a Dirac delay kernel for example, this relative overrating is expressed by

$$\frac{(\overline{\mu} - \mu)}{\mu}(1 - e^{-\mu a^*})$$
$$\frac{\alpha\gamma\nu f(\overline{P})\tau}{\ln(2)}(1 - (\frac{1}{2})^{\frac{a^*}{\tau}})$$

where $\tau$ is the mean life of zooplankton individuals in a cohort. It is the time necessary to population to be in its half. The overrating is negligible for small values of the delay, but it becomes important for a delay comparable with the mean life. It increases also with the mean life, and with the efficiency of the toxin.

The absence of reference to the delay appears also in the measure of the instability around stationary solutions, that is in the expression of the derivative $\lambda'(0)$. The non structured model overrates this instability. The relative overratings are expressed by

$$e^{\mu a^*} - 1$$

in the Dirac delay kernel case, and by

$$\frac{\int_0^\infty \overline{\pi}_0(0,a)ada}{\int_0^\infty \overline{\pi}_0(0,a)\left(\int_0^a (a-s)F(s)ds\right)da} - 1$$

in the non specified delay kernel case (with, for comparison, constant mortality and fecundity functions). In both cases, these quantities are positive, and are increasing with the delay.

### 6.6.2 On the behaviour of the structured model around stationary solutions

We have seen that stationary solutions are unstable. The source of this instability does not lie in the technical way used for taking in account the delay. It lies in the nature of the interactions betweeen zooplankton and toxic phytoplankton. This nature is the same in the current version of both models, the structured model as well as the non structured one, following their common basis assumption concerning the fecundity function of zooplankton. The interactions between zooplankton and toxic phytoplankton do not present indeed any positive feedback : The zooplankton kills the toxic phytoplankton in the predation process, and the toxic phytoplankton kills the zooplankton in the toxin production process. This is not the usual schema which occurs in prey predator models, where the growth of predators population depends on the predation.

Examining now the details of the results on the structured model, we reach the following conclusions :

- In the Dirac case, we proved that there always exists a positive eigenvalue of the linearized system around the stationary solution.
- In the general case, we proved this property locally, for small values of the efficiency of the toxin.

In both cases however, two different situations occur clearly. These two situations correspond to values of phytoplankton level $\overline{P}$ fullfilling $\overline{Z}'(\overline{P}) < 0$ and $\overline{Z}'(\overline{P}) > 0$, respectively. For the interpretation of the conditions of this transition, let $L(\overline{P})$ be the logistic function $L(\overline{P}) = r\overline{P}(1 - \frac{\overline{P}}{K})$.
$\overline{Z}'(\overline{P}) < 0$ is equivalent to $\frac{L'(\overline{P})}{L(\overline{P})} < \frac{f'(\overline{P})}{f(\overline{P})}$. This last condition implies that, when the level of phytoplankton population equilibrium varies, the relative variation of the zooplankton individual uptake is greater than the relative variation of the growing rate of the phytoplankton population. For assumed properties of $f(\overline{P})$, $\frac{f'(\overline{P})}{f(\overline{P})}$ decreases as $\overline{P}$ increases, but $\frac{L'(\overline{P})}{L(\overline{P})}$ decreases more rapidly, being negative when $\overline{P} > \frac{K}{2}$. For stationary solutions with a level of phytoplankton population exceeding a threshold less than the half carrying capacity, $\overline{Z}'(\overline{P}) < 0$ is fullfilled. The exact value of the threshold is strongly dependent on the behaviour of the uptake function for $\overline{P} < \frac{K}{2}$, and in some cases the opposite situation $\overline{Z}'(\overline{P}) > 0$ may never occur.

As regards the meaning of the two situations, we emphasize the following results : The instability for $Z'(\overline{P}) < 0$ has its origin in the response of the dynamic of the zooplankton to the mortality by toxin, that is as $\nu > 0$. If were possible to maintain the population of zooplankton at the same level $\overline{Z} = \frac{L(\overline{P})}{\alpha f(\overline{P})}$, the system would return to the state $\overline{P}, \overline{Z}$ after a small perturbation on the phytoplankton. When in these situation ($Z'(\overline{P}) < 0$) the efficiency of the toxin becomes negative (and then propitious to the zooplankton), the system becomes stable around the stationary solution. The instability for $Z'(\overline{P}) > 0$ has on the contrary its origin in the response of the phytoplankton itself. If it were possible to maintain the population of zooplankton at the same level $\overline{Z} = \frac{L(\overline{P})}{\alpha f(\overline{P})}$ the system would diverge from $\overline{P}, \overline{Z}$ after a small perturbation on the zooplankton. And when in these second situation the efficiency of the toxin becomes negative, the system remains unstable around the stationary solution. It would be very interesting to study and compare in both cases the global behaviour of the system, but it is outside the scope of this paper.

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# Introduction au chapitre 7

Dans ce chapitre, nous continuons à étudier le système phytoplancton-zooplankton et l'impact de la toxine libérée par des espèces phytoplanctoniques toxiques sur le comportement du système.
Nous nous focalisons dans cette partie sur le processus spatial. En fait, ce travail vient compléter les travaux effectués dans la deuxième partie de la thèse dans laquelle nous avons considéré et analysé des modèles homogènes et des modèles structurés par âge.

Nous proposons un modèle spatialisé décrivant le mouvement nycthéméral du plancton. Pendant la journée, le phytoplancton qui se trouve dans la couche de mélange profite de la lumière pour synthétiser de la matière organique et se reproduire par photosynthèse. Pendant ce temps là, le zooplancton migre vers les couches les plus profondes pour éviter les prédateurs. Pendant la nuit, la reproduction cesse et l'absence de lumière permet au zooplancton de se diriger vers la surface de l'eau pour se nourrir. Au lever du jour, le zooplancton se dirige à nouveau vers les couches les plus profondes et la reproduction du phytoplancton recommence.

Nous présentons ici une modélisation du phénomène et nous donnons ensuite une esquisse de la preuve de l'existence des solutions.
Le modèle est un système d'équations aux dérivées partielles à retard infini. Ce retard infini provient du fait que nous considérons un effet cumulatif de la toxine.

La première étape consiste à écrire les équations sous formes abstraites. Puis à appliquer la théorie des semi groupes pour montrer l'existence des solutions dans un espace de banach adéquat.
La difficulté mathématique réside dans le choix de cet espace du fait que l'intervalle du retard est infini et que l'état du système à chaque instant $t$ contient toujours la donnée initiale.
Ce problème a été considéré par Hale et Kato (voir [7]) qui ont introduit une axiomatique concernant ces espaces de phase et cette classe d'équations différentielles à retard infini.
Le travail d'Arino *et al.* (voir [1]) nous a donné une construction de tels espaces. L'espace dans lequel on se propose de faire l'étude en est un cas particulier. Nous considérons ainsi l'espace des fonctions continues à croissance au plus exponentielle.

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# Chapitre 7

# A Spatio-temporal Zooplankton-Toxic Phytoplankton System with unbounded delay [1]

**Abstract :** In this paper, we are intersted in the dynamics of a phytoplankton-zooplankton system in a toxic environment. We proposed a structured model. The variable of structure is the space. We take into account many biological and physical processes. We consider infinite delay to describe the accumulation and elimination of toxic substances. Our aim at this level, is to propose the modelling of the spatio-temporal process and to sketch a method for existence of solutions.

**Key words :** Phytoplankton, Zooplankton, Toxin, Diffusion, Transportation, infinite delay.

[1] This chapter is based on an ongoing work.

## 7.1 Introduction

In this work we are interested in the dynamics of phytoplankton-zooplankton system in the presence of toxic substances. This work is a continuation of other works which have been done in this context (see for example Chattopadhyay et al. (2002), El abdllaoui et al. (2002) and El abdllaoui et al. (2004)). Our aim is to have a complete view of the problem of toxicity arising in interactions of Zooplankton-toxic phytoplankton system. In this work, the model is made up of equations describing the dynamical processes, both physical and biological, taking place in the domain of interest notably diffusion, transportation, predation, natural mortality and mortality by toxins, and made up also of equations having their rise from exchanges at the boundary of the domain : initial values at time $t = 0$, and boundary values at $z = -z_0$ and $z = 0$.
We focus on the migration of the zooplankton between the bottom and the surface of the water. In fact, along the day, the phytoplankton reproduces on the surface while the zooplankton goes into the bottom to avoid predators. In the night, the zooplankton moves into the surface to feed on the produced phytoplankton while the reproduction of a new phytoplankton ceases because of the abscence of the light. We proposed a delayed structured model. The variable of structure is the water column $z$. We introduce an infinite delay in order to describe accumulation and elimination of toxic substances by zooplankton individuals in all their life cycle, that is from their birth date until time $t$. For more details and explanations on the problem of toxicity, the modelling of this phenomenon and the mathematical analysis in the case of homogenous model as well as in the case of age-structured model, we refer the reader, for example, to the papers cited above.

## 7.2 Mathematical Model and Main Assumptions

The model to be investigated reads as follows :

$$\begin{cases} \dfrac{\partial P}{\partial t}(t,z) = \dfrac{\partial}{\partial z}(d(z)\dfrac{\partial}{\partial z}P(t,z)) + r(I(t,z))P(t,z)(1-\dfrac{P(t,z)}{K}) \\ \qquad -\alpha H(I_2 - I(t,z))f(P(t,z))Z(t,z) \\ \dfrac{\partial Z}{\partial t}(t,z) + div(V(z,I(t,z),P(t,z)).Z(t,z)) = \dfrac{\partial}{\partial z}(\rho(z)\dfrac{\partial}{\partial z}Z(t,z)) \\ \qquad +\beta H(I_2 - I(t,z))f(P(t,z))Z(t,z) - \mu(z)h(Z(t,z)) \\ -\theta\int_{-\infty}^{0} F(\tau,\zeta(z,t,t+\tau))f(P(t+\tau,\zeta(z,t,t+\tau)))Z(t+\tau,\zeta(z,t,t+\tau))d\tau \\ \dfrac{\partial I}{\partial t}(t,z) - \dfrac{\partial I}{\partial z}(t,z) = \omega J(t,z) - I(t,z) \\ \dfrac{\partial J}{\partial t}(t,z) - \dfrac{\partial J}{\partial z}(t,z) = -\omega I(t,z) - J(t,z) \\ \dfrac{\partial \zeta}{\partial s}(z,t,s) = V(\zeta(z,t,s),I(s,\zeta(z,t,s)),P(s,\zeta(z,t,s))),\forall s < t \end{cases} \tag{7.1}$$

**Boundary conditions :**

$$\begin{cases} \dfrac{\partial P}{\partial z}(t,0) = 0 \ , \ \lim\limits_{z \to -z_0} d(z)\dfrac{\partial P}{\partial z}(t,z) = 0, \forall t > 0 \\ \dfrac{\partial Z}{\partial z}(t,-z_0) = \dfrac{\partial Z}{\partial z}(t,0) = 0, \forall t > 0 \\ P(\theta,z) = P_0(\theta,z), \forall \theta \in ]-\infty, 0], \forall z \in [-z_0, 0] \\ Z(\theta,z) = Z_0(\theta,z), \forall \theta \in ]-\infty, 0], \forall z \in [-z_0, 0] \\ I(0,z) = \phi_1(z), J(0,z) = \phi_2(z) \\ I(t,0) = h_1(t), J(t,0) = h_2(t) \\ \zeta(z,t,t) = z, \zeta(z,0,\theta) = \varphi(z,\theta), \forall \theta \in ]-\infty, 0], \forall z \in [-z_0, 0], \forall t \geq 0 \end{cases}$$

where $V$ represents the speed of zooplankton individuals. We assume that zooplankton move according to the light intensity and food availability. Therefore, we assume that the speed $V$ at depth $z$ is given by

$$V(z,I,P) = -l(z)H(I - I_1)V_d + k(z)g(P)H(I_2 - I)V_m, \forall -z_0 \leq z \leq 0 \tag{7.2}$$

We assumed that the positive direction is from the bottom into the surface.

The function $g$ is assumed decreasing with respect to $P$. It means that the speed of zooplankton individuals decreases when they meet phytoplankton in different layers. The zooplankton individuals have to feed when they meet the phytoplankton on their trajectories.

$d(z)$ (resp. $\rho(z)$) is the rate of vertical diffusion of phytoplankton (resp. Zooplankton). For simplicity, we assume that $d(.)$ and $\rho(.)$ depend on $z$ only, while in general, they are functions of the three space variables and time.

$r(I)$ is the growth rate of phytoplankton in the absence of zooplankton. It is assumed to be a function of light availablility (photosynthesis).

$f$ is the uptake function of zooplankton. It models the consumption of zooplankton.

$\alpha$ and $\beta$ are respectively the predation rate of zooplankton on phytoplankton and the conversion rate of zooplankton. They are assumed to be constants.

$\mu(z)$ is the mortality rate of zooplankton at depth $z$. It is assumed to be positive and bounded.

$h$ is the mortality function of zooplankton. Often, $h$ is considered in the litterature as a linear or quadratic function (see for example [4]) . It describes the natural mortality of zooplankton as well as the mortality by higher predation on zooplankton individuals.

$\zeta(z,t,s)$ represents the position of zooplankton at time $s < t$ such that its position at time $t$ is $z : \zeta(z,t,t) = z$.

$H$ is the Heaviside function; $H(x) = 1$ if $x > 0$ and $H(x) = 0$ if $x < 0$. We choose the Heaviside function to describe the fact that the predation is occuring in the night only.

$I_1$ (resp. $I_2$) is the minimum (resp. the maximum) intensity of the light from which the zooplankton begins to move into the bottom (resp. to go into the surface). We have $I_2 < I_1$.

$V_d$ (resp. $V_m$) is the maximum speed of the descent (resp. increase) of zooplankton.

The original problem is not regular because of the Heaviside function $H$. For that, we assume that there is a regular sequence $(H_n)$ which converges into $H$ when $n \to \infty$, and we consider the problem with this regular functions that we still denote $H$. We can choose $H_n$ as follows

$$H_n(x) = \begin{cases} 1 \text{ si } x > 0 \\ \dfrac{x + \frac{1}{n}}{\frac{1}{n}} \text{ si } -\dfrac{1}{n} \leq x \leq 0 \\ 0 \text{ si } x < -\dfrac{1}{n} \end{cases}$$

or we can also choose a $C^\infty$ functions with compact support, we think about convolution of $H$ with some mollifiers sequence (see for example Brezis [2]).

The boundary conditions at the surface $z = 0$ as well as at $z = -z_0$ express the fact that there is no flux of phytoplankton and zooplankton.
The initial conditions $P_0$ and $Z_0$ are functions defined on $(-\infty, 0] \times [-z_0, 0]$. In fact, as the delay is infinite, it is necessary for the resolution of the system, to know all the past of the populations. Two further natural properties of such functions are that they are non negative and such that $P_0(\theta,.), Z_0(\theta,.) \in L^1(-z_0, 0)$ for each $\theta \leq 0$. Often, and for technical reason, the space $L^1$ is replaced by the space $L^2$.
The production of new phytoplankton is assumed to follow the logistic law

$$r(I(t,z))P(t,z)(1 - \frac{P(t,z)}{K}) \tag{7.3}$$

where $I(t,z)$ is the light intensity at time $t$ and depth $z$.
The term (7.3) can be viewed as a simplified model of photosynthesis.
The term

$$\beta H(I_2 - I(t,z))f(P(t,z))Z't,z)$$

represents the predation of zooplankton on phytoplankton. We introduce the Heaviside function $H$ in order to separate the day and the night.

The model has to be investigated assuming the following hypothesis.

**(H1)** $r(.)$ and $H(.)$ are $C^1$ functions, positive and bounded.

**(H2)** $f$ is a $C^2$ function non negative, increasing and $f(0) = 0$. These hypotheses are satisfied by the Michaelis-Menten function

$$f(P) = \frac{P}{k+P}$$

where $k$ is the half saturation-constant.

**(H3)** $l(z)$ and $k(z)$ are positive functions, continuous, bounded and lipschitzian and satisfy : $l(-z_0) = 0$ and $k(0) = 0$.

**(H4)** $d(.) \in L^\infty$ and $\frac{1}{d(.)} \in L^\infty$. We assume also that $\rho(z) > 0$. For simplification, we will consider that $\rho(z)$ is a constant ; $\rho(z) = \rho$.

**(H5)** $g$ and $h$ are $C^1$ functions, positive bounded such that $g$ is decreasing with respect to $P$ and $h$ is such that $h(0) = 0$.

In what follows, we attempt to give some steps to follow so that to prove existence of solutions.

First, remark that equations of $I$ and $J$ are uncoupled with the other variables.
Let us denote

$$\begin{aligned} I &= \widetilde{I} + h_1(t) \\ J &= \widetilde{J} + h_2(t) \end{aligned}$$

The system with $(\widetilde{I}, \widetilde{J})$ reads as

$$\left\{ \begin{array}{c} \dfrac{\partial \widetilde{I}}{\partial t}(t,z) - \dfrac{\partial \widetilde{I}}{\partial z}(t,z) = \omega \widetilde{J}(t,z) - \widetilde{I}(t,z) + [\omega h_2(t) - h_1(t) - h_1'(t)] \\ \dfrac{\partial J}{\partial t}(t,z) - \dfrac{\partial J}{\partial z}(t,z) = -\omega \widetilde{I}(t,z) - \widetilde{J}(t,z) - [\omega h_1(t) + h_2(t) + h_2'(t)] \\ \widetilde{I}(t,0) = \widetilde{J}(t,0) = 0 \end{array} \right. \tag{7.4}$$

System (7.4) can be writen in an abstract form as follows

$$\left\{ \begin{array}{c} \dfrac{du}{dt} = A_1 u(t) + f(t) \\ u(0) = 0 \end{array} \right. \tag{7.5}$$

where

$$f(t) = ([\omega h_2(t) - h_1(t) - h_1'(t)], [\omega h_1(t) + h_2(t) + h_2'(t)])$$

and

$$M = \begin{pmatrix} -1 & \omega \\ -\omega & -1 \end{pmatrix}$$

The operator $A_1$ is given by

$$\begin{aligned} A_1 \begin{pmatrix} \varphi \\ \psi \end{pmatrix} &= \frac{d}{dz} \begin{pmatrix} \varphi \\ \psi \end{pmatrix} + M \begin{pmatrix} \varphi \\ \psi \end{pmatrix} \\ \mathcal{D}(A_1) &= \left\{ (\varphi, \psi) \in \left(H^1\right)^2 / \varphi(0) = \psi(0) = 0 \right\} \end{aligned}$$

From the theory of inhomogenous initial value problem (see Pazy [8]), we can apply theorem 8 (see Annex) in the case of system (7.5) to prove existence of a classical solution.
Then, we propose to use the function $I$ as a given function in the other equations.

For the existence of solutions $(P, Z)$, we suggest the following steps :
First, begin by proving existence of solution of the trajectory equation $\zeta$. Then, write the equations of $P$ and $Z$ as abstract equations, show that the linear operator associated with the two abstract equations, considered together, is the infinitesimal generator of a $C_0$-semigroup in some adequate space. Then, write the equations as integral equations using the semi group operators, and finally show, using the method of successive approximation, that the sequence made up from the integral form is convergent.

## 7.3 Conclusion

The present paper attempts to describe the problem of toxicity in phytoplankton-zooplankton interactions. We take into account physical and biological processes. Our aim is to study the role of toxic substances released by phytoplankton species. This problem was considered in previous works (see for example [3], [5], [6]) with different hypotheses. The main difference here is the introduction of space as the variable of structure. The fact that we considered not only diffusion and transportation to describe the movement of individuals, but also unbounded delay to describe the cumulative effect of toxic substances added more difficulties in the study of the present model compared with models studied in the above references. At this level of study, we concentrated ourselves on modeling and tried to give some helpful steps to prove existence of solutions.
Therefore, in the first part of this work, we gave explanations on the model and processes taken into account for modelling. Then, we sketched a method to follow, using semi-group theory, to prove existence of solutions under feasible hypotheses.
This work is an ongoing one. We think that the complete study of the model presented here will help to have a clear view and understanding of the role of toxic substances in phytoplankton-zooplankton system. This study is beyond the scope of this work and will be addressed in future work.

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# Conclusion et perspectives

Les travaux qui ont été présentés dans cette thèse font partie du cadre plus général de l'écologie mathématique. Le problème principal étudié à travers ce travail est l'influence d'une infection, d'une maladie ou encore d'une production de substances toxiques présents dans un système, sur le comportement qualitatif de ce dernier. Dans le but de comprendre cette influence, deux étapes ont été considérées : la modélisation mathématique puis l'analyse mathématique des modèles.

Dans la première partie de la thèse, nous avons considéré des systèmes prédateurs-proies : le problème entrepris est le suivant : nous avons supposé la présence d'une infection virale ou d'une maladie transmissible dans la population des proies et nous avons etudié le comportement asymptotique des solutions du système en fonction du paramètre de transmission $\lambda$. Ce problème est abordé à partir de différentes hypothèses sur la réponse fonctionnelle. Dans le premier modèl, une réponse fonctionnelle classique a été considérée : la fonction de prise du prédateur y est proportinnelle à l'abondance de la proie (modèle classique). Dans le deuxième modèle a été considérée une réponse fonctionnelle basée sur le principe de la ratio-dépendance : la fonction de prise du prédateur est une fonction du rapport de l'abondance de la proie par l'abondance du prédateur.
Du point de vue mathématique, les équations qui ont été étudiées dans cette première partie sont des équations ordinaires. L'analyse de stabilité de ces systèmes a été entreprise par linéarisation au voisinage des solutions stationnaires lorsque cette linéarisation était possible. Dans l'étude du modèle ratio-dépendant, un problème difficile se pose au voisinage de l'origine : le champ de vecteurs n'est pas défini en ce point et par suite la linéarisation n'est plus possible. L'analyse de stabilité au voisinage de ce point critique (origine) a été menée par l'étude du système de valeurs propres non linéaires.

Le résultat principal auquel nous avons abouti dans la première partie montre que la présence d'une infection dans la population des proies peut entrainer la coexistence des espèces et peut donc agir comme un contrôle biologique. Bien évidemment, ce résultat dépend des valeurs du paramètre de transmission. Ainsi dans les deux modèles, un seuil de ce paramètre a été mis en évidence. Quelques simulations numériques ont illustré ces résultats analytiques et ont clairement reproduit le comportement qualitatif mentionné plus haut.

Dans la deuxième partie de la thèse, des modèles représentant des interactions entre le phytoplancton et le zooplancton ont été présentés . Le problème qui a été étudié est le suivant : Quelques espèces phytoplanctoniques sont toxiques, ces espèces quand elles sont mangées par le zooplancton vont agir comme un poison et augmentent ainsi la mortalité des individus zooplanctoniques. La toxine libérée par ces espèces peut avoir un effet instantanée ou retardé. Les modèles qui ont été considérés dans

cette partie sont d'abord des modèles globaux non structurés puis des modèles structurés en âge ou en espace. Dans les travaux qui ont été achevés, nous avons retrouvé le comportement de la première partie, à savoir l'existence d'un seuil, cette fois-ci de l'efficacité de la toxine, en dessous duquel le système est instable et au dessus duquel le système est stable. Au voisinage de la valeur critique (le seuil), nous avons montré l'apparition d'une bifurcation de Hopf. Encore une fois, et comme dans la première partie, notre conclusion est que la présence des substances toxiques contribue à la stabilisation du système, contrôle les efflorescences (blooms) du plancton et peut donc agir comme un contrôle biologique de ces blooms.

Du point de vue mathématique, la formulation des problèmes aux dérivées partielles sous forme de problème de Cauchy et la démonstration de l'existence et l'unicité des solutions ont fait l'objet de la première étape de l'étude.
Dans le cas du modèle structuré en âge : La méthode des lignes caractéristiques a été utilisée. Nous avons montré que le semi groupe solution est quasi compact. Cette propriété nous a permis de faire l'analyse de stabilité à partir du système aux valeurs propres.
Dans le cas du modèle structuré en espace, la théorie des semi groupes a été proposée. La présence du retard infini dans les équations constitue un obstacle non négligeable pour l'étude. L'écriture du modèle sous forme d'un problème de Cauchy n'est pas chose facile. Nous avons formulé notre problème dans un espace de phase (un espace de Banach) satisfaisant les axiomes de Hale et Kato. Puis nous avons proposé une méthode pour montrer que le problème de Cauchy admet bien une solution intégrale. L'étude de ce problème structuré en espace n'est pas encore menée à son terme. Nous comptons entreprendre le reste de l'étude dans un futur proche afin de comparer ses résultats à ceux obtenus dans les cas des modèles précédents. L'analyse et la comparaison des résultats de tous les modèles étudiés dans cette deuxième partie pourront contribuer à une meilleure compréhension du système phytoplancton- zooplancton et du rôle de la toxine dans ce système.

# Annexe : Outils Mathématiques

## 7.4 Rappels sur les semi-groupes

Ce chapitre est scindé en deux parties :
La première partie rappelle et expose des résultats sur la théorie des semi-groupes d'opérateurs linéaires bornés (voir Henry [3], Pazy [4], Engel et Nagel [2]).
La deuxième partie présente des méthodes qui servent à étudier des équations caractéristiques du type polynômiale ou quasi-polynômiale. Ce sont des équations qui proviennent souvent de l'étude qualitative des équations différentielles ordinaires ou à retard (voir Pontriaguine [5], Bellman et Cooke [1], Stépan [6]).

### 7.4.1 Semi-groupe fortement continu

**Définition 1.** *Soit $X$ un espace de Banach, muni de la norme $\|.\|$ .*
*Etant donné une famille $(T(t))_{t\geq 0}$ d'opérateurs linéaires bornés sur $X$, nous dirons que cette famille est un semi-groupe fortement continu sur $X$ si et seulement si les propriétés suivantes sont satisfaites :*
(i) $T(0) = I$, *($I$ est l'opérateur identité sur $X$),*
(ii) $T(t+s) = T(t)T(s)$ *pour tout* $t \geq 0, s \geq 0$,
(iii) *Pour tout $x \in X$ fixé, l'application $t \to T(t)x$ est continue sur $[0, +\infty[$.*

Un semi-groupe fortement continu d'opérateurs linéaires bornés est aussi appelé un $C_0$ semi-groupe. Au semi-groupe $T(t)$, on fait correspondre la famille d'opérateurs $A_h$, pour $h > 0$, définie par

$$A_h x = \frac{1}{h}\{T(h)x - x\}, \quad pour \quad x \in X.$$

Cette famille nous permet de définir le générateur infinitésimal d'un semi-groupe.

**Définition 2.** *Le générateur infinitésimal du semi-groupe $(T(t))_{t\geq 0}$ est l'opérateur $A$ défini sur le domaine*

$$D(A) = \{x \in X, \lim_{h\to 0} A_h x \ existe \ \}$$

*par*

$$Ax = \lim_{h\to 0} A_h x, \quad pour \ tout \quad x \in D(A)$$

Dans ce qui suit, nous rappelons quelques propriétés fondamentales d'un $C_0$ semi-groupe $(T(t))_{t\geq 0}$ de génétrateur infinitésimal $A$.

**Théorème 1.** *Soit $(T(t))_{t\geq 0}$ un $C_0$ semi-groupe et soit $A$ son générateur infinitésimal. Alors les propriétés suivantes sont satisfaites :*

(i) *$T(t)$ est exponentiellement borné : il existe deux constantes réelles $M$ et $\omega$ telles que pour tout $t \geq 0$*

$$\|T(t)\| \leq Me^{\omega t}.$$

(ii) *Pour tout $x \in X$,*

$$\lim_{h\to 0} \frac{1}{h} \int_t^{t+h} T(s)xds = T(t)x.$$

(iii) *Pour tout $x \in X$,*

$$\int_0^t T(s)xds \in D(A) \quad et \quad A\Big(\int_0^t T(s)xds\Big) = T(t)x - x.$$

(iv) *Pour tout $x \in D(A)$,*

$$T(t)x - T(s)x = \int_s^t T(\tau)Axd\tau = \int_s^t AT(\tau)xd\tau.$$

Si $\omega = 0$ dans (i) du théorème (1), le semi-groupe est dit uniformément borné. Si de plus $M = 1$, le semi-groupe est dit de contractions.

**Corollaire 1.** *Si $A$ est le générateur infinitésimal d'un $C_0$ semi-groupe $(T(t))_{t\geq 0}$ sur $X$, alors $D(A)$ est dense dans $X$ et $A$ est un opérateur fermé.*

De la définition d'un générateur infinitésimal d'un semi-groupe, on voit bien qu'un semi-groupe définit au plus un générateur. Réciproquement, à chaque générateur correspond un et un seul semi-groupe. Ceci est donné par le théorème suivant :

**Théorème 2.** *Soient $(T(t))_{t\geq 0}$ et $(S(t))_{t\geq 0}$ deux semi-groupes du même générateur $A$. Alors $T(t) = S(t)$ pour tout $t \geq 0$.*

Dans la suite, nous allons donner des conditions nécessaires et suffisantes pour qu'un opérateur linéaire $A$ soit le générateur infinitésimal d'un $C_0$ semi-groupe de contractions. Ceci en passant par le comportement de la résolvante de $A$ que nous définissons dans ce qui suit.

**Définition 3.** *Soit $A$ un opérateur linéaire fermé dans $X$.*
*(a) L'ensemble résolvant de $A$, noté $\rho(A)$ est défini par*

$$\rho(A) = \left\{\lambda \in \mathbf{C} \ / \ (\lambda I - A)^{-1} \text{ existe et est borné dans } X\right\}$$

*(b) Le spectre de $A$ est l'ensemble $\sigma(A) = \mathbf{C}\backslash\rho(A)$.*
*(c) La famille $R(\lambda : A) = (\lambda I - A)^{-1}$, $\lambda \in \rho(A)$ d'opérateurs linéaires bornés est appelée la résolvante de $A$.*

**Théorème 3. [Hille-Yosida]** *Soit $A$ un opérateur linéaire (non borné). $A$ est le générateur infinitésimal d'un $C_0$ semi-groupe de contractions si et seulement si :*
*(i) $A$ est fermé et $\overline{D(A)} = X$.*
*(ii) L'ensemble résolvant de $A$, $\rho(A)$ contient $IR^+$, et pour tout $\lambda > 0$, on a*

$$\|R(\lambda : A)\| \leq \frac{1}{\lambda}$$

**Semi-groupe compact**

**Définition 4.** *Un $C_0$ semi-groupe $(T(t))_{t\geq 0}$ sur $X$ est dit compact pour $t > t_0$ si $T(t) : X \to X$ est un opérateur compact pour tout $t > t_0$. $(T(t))_{t>0}$ est dit compact sur $X$ si $T(t)$ est compact pour tout $t > 0$.*

**Théorème 4.** *Soit $(T(t))_{t\geq 0}$ un $C_0$ semi-groupe. Si $T(t)$ est compact por tout $t > t_0$, alors $T(t)$ est continu au sens de la norme des opérateurs pour tout $t > t_0$.*

**Théorème 5.** *Soit $(T(t))_{t\geq 0}$ un $C_0$ semi-groupe et soit $A$ son générateur infinitésimal. $(T(t))_{t\geq 0}$ est compact si et seulement si $T(t)$ est continu au sens de la norme des opérateurs pour tout $t > 0$ et la résolvante $R(\lambda : A)$ est compacte pour $\lambda \in \rho(A)$.*

**Corollaire 2.** *Soit $(T(t))_{t\geq 0}$ un $C_0$ semi-groupe et soit $A$ son générateur infinitésimal. Si $R(\lambda : A)$ est compact pour un certain $\lambda \in \rho(A)$ et si $T(t)$ est continu au sens de la norme des opérateurs pour $t > t_0$, alors $T(t)$ est compact pour $t > t_0$.*

**Semi-groupe analytique**

**Définition 5.** *Soit $\triangle = \{z \in \mathbf{C} : \varphi_1 < \arg(z) < \varphi_2, \varphi_1 < 0 < \varphi_2\}$. Pour tout $z \in \triangle$, soit $T(z)$ un opérateur linéaire borné. La famille $(T(z))_{z\in\triangle}$ est dite analytique dans $\triangle$ si :*
*(i) $z \to T(z)$ est analytique dans $\triangle$.*
*(ii) $T(0) = I$ et $\lim_{z\to 0} T(z)x = x$ pour tout $x \in X$.*
*(iii) $T(z_1 + z_2) = T(z_1)T(z_2)$, pour tout $z_1$ et $z_2 \in \triangle$.*

Dans le théorème qui va suivre, nous allons donner des assertions équivalentes qui permettent d'étendre un $C_0$ semi-groupe à un semi-groupe analytique.

**Théorème 6.** *Soit $(T(t))_{t\geq 0}$ un $C_0$ semi-groupe uniformément borné. Soit $A$ le générateur infinitésimal de $(T(t))_{t\geq 0}$ et supposons que $0 \in \rho(A)$. Les assertions suivantes sont équivalentes :*
*(i) $T(t)$ peut être étendu à un semi-groupe analytique dans un secteur $\triangle_\delta = \{z \in \mathbf{C} : |\arg z| < \delta\}$ et $\|T(z)\|$ est uniformément borné dans tout sous secteur fermé $\overline{\triangle}_{\delta'}, \delta' < \delta$, de $\triangle_\delta$.*
*(ii) Il existe une constante $C$ telle que pour tout $\sigma > 0$ et $\tau \neq 0$, on a*

$$\|R(\sigma + i\tau)\| \leq \frac{C}{|\tau|}.$$

*(iii) Il existe $\delta \in ]0, \frac{\pi}{2}[$ et $M > 0$ tels que*

$$\rho(A) \supset \Sigma = \{\lambda \in \mathbf{C} : |\arg \lambda| < \frac{\pi}{2} + \delta\} \cup \{0\}$$

*et*

$$\|R(\lambda : A)\| \leq \frac{M}{|\lambda|} \text{pour } \lambda \in \Sigma, \lambda \neq 0.$$

*(iv) $T(t)$ est différentiable pour $t > 0$ et il existe une constante $C$ telle que*

$$\|AT(t)\| \leq \frac{C}{t} \text{ pour } t > 0.$$

### 7.4.2 Problème de Cauchy

Soit $A$ un opérateur linéaire non borné, fermé et de domaine $D(A)$ dense dans un espace de Banach $X$.
On considère le problème suivant : Trouver $u$ tel que

$$(P) \quad \begin{cases} \dfrac{du}{dt} = Au(t) + f(t) \\ u(0) = u_0 \end{cases}$$

où $u_0$ et $f$ sont donnés.
Le problème $(P)$ est connu sous le nom du problème de Cauchy.

**Cas homogène** $(f = 0)$

**Théorème 7.** *Soit $(A, D(A))$ le générateur infinitésimal d'un $C_0$ semi-groupe $(T(t))_{t\geq 0}$ sur l'espace de Banach $X$.*

*Pour tout $u_0 \in D(A)$, le problème de Cauchy*

$$\begin{cases} \dfrac{du}{dt} = Au(t) \quad t > 0 \\ u(0) = u_0 \end{cases}$$

*admet une unique solution $u : IR^+ \to D(A)$. $u(.)$ est de classe $C^1$ de $IR^+ \to X$. De plus, $u(t) = T(t)u_0$.*

**Cas non homogène** ($f \neq 0$)

Nous commencons par donner des définitions de solutions du problème de Cauchy. Nous donnerons aussi des résultats d'existence de solutions du problème non homogène et nous finirons par donner des résultats de régularité de ces solutions.

**Définition 6.** *Soit $u_0 \in D(A)$ et soit $f \in L^1(0,T,X)$. La fonction $u \in C(0,T,X)$ définie par*

$$u(t) = T(t)u_0 + \int_0^t T(t-s)f(s)ds, \quad 0 \leq t \leq T \tag{7.6}$$

*est appelée la solution intégrale du problème $(P)$ sur $[0,T]$.*

**Définition 7.** *Une fonction $u : [0,T] \to X$ est une solution classique du problème $(P)$ sur l'intervalle $[0,T]$ si $u$ est continue de $[0,T] \to X$, $u(t) \in D(A)$ pour tout $t \in ]0,T[$ et $u$ est de classe $C^1$ sur $]0,T[$ et $u$ satisfait les équations de $(P)$ pour tout $t \in ]0,T[$.*

**Théorème 8.** *Soit $A$ le générateur infinitésimal d'un $C_0$ semi-groupe $(T(t))_{t\geq 0}$.*
*(i) Si $f \in L^1(0,T,X)$, alors pour tout $u_0 \in X$, le problème $(P)$ admet au plus une solution. Si cette solution existe alors elle est donnée par (7.6).*
*(ii) Si $f \in L^1(0,T,X)$ et $f$ est continue sur $]0,T[$. Si $f(s) \in D(A)$ pour tout $0 < s < T$ et $Af(s) \in L^1(0,T,X)$, alors pour tout $x \in D(A)$, le problème $(P)$ admet une solution sur $[0,T[$.*
*(iii) Si $f$ est de classe $C^1$ de $[0,T]$ vers $X$, alors le problème $(P)$ admet une solution classique $u(.)$ sur $[0,T]$ pour tout $u_0 \in D(A)$.*

Nous avons vu que si l'on exige plus de régularité sur le terme non linéaire du problème $(P)$, on obtiendra plus de régularité sur la solution de ce problème lorsque l'opérateur $A$ est le générateur infinitésimal d'un $C_0$ semi-groupe. Dans le cas où $A$ est le générateur infinitésimal d'un semi-groupe analytique, nous avons des résultats de régularité plus forts. Nous résumons ces résultats dans ce qui suit après avoir donné la définition d'une fonction Hölderienne.
Soit $I$ un intervalle.

**Définition 8.** *On dit qu'une fonction $f : I \to X$ est Hölderienne d'exposant $\theta$, $0 < \theta < 1$ sur $I$ s'il existe une constante $L$ telle que*

$$\|f(t) - f(s)\| \leq L\,|t - s|^{\theta} \quad \textit{pour tout } s, t \in I.$$

*$f$ est dite localement Hölderienne sur $I$ si pour tout $t \in I$, il existe un voisinage de $t$ dans lequel $f$ est Hölderienne.*

L'ensemble des fonctions Hölderiennes sur $I$, d'exposant $\theta$, est noté $C^{\theta}(I : X)$.

**Corollaire 3.** *Soit $A$ le générateur infinitésimal d'un semi-groupe analytique $T(t)$. Si $f \in L^1(0, T, X)$ est localement Hölderienne sur $]0, T]$, alors pour tout $u_0 \in X$, le problème $(P)$ admet une unique solution.*

**Théorème 9.** *Soit $A$ le générateur infinitésimal d'un semi-groupe analytique $T(t)$ et soit $f \in C^{\theta}([0, T] : X)$. Si $f$ est la solution du problème de Cauchy $(P)$ alors :*
*(i) Pour tout $\delta > 0$, $Au \in C^{\theta}([\delta, T] : X)$ et $du/dt \in C^{\theta}([\delta, T] : X)$.*
*(ii) Si $u_0 \in D(A)$ alors $Au$ et $du/dt$ sont continus sur $[0, T]$.*
*(iii) Si $u_0 = 0$ et $f(0) = 0$ alors $Au$ et $du/dt \in C^{\theta}([0, T] : X)$.*

Un autre type d'équations que nous rappellons ici sont les équations semi-linéaires, c'est à dire les équations suivantes :

$$(P') \quad \begin{cases} \dfrac{du(t)}{dt} + Au(t) = f(t, u(t)), & t > 0 \\ u(0) = u_0 \end{cases}$$

Nous nous contentons pour ce type d'équations de donner un résultat d'existence local dans le cas où $(-A)$ génère un semi-groupe compact.

**Théorème 10.** *Soit $X$ un espace de Banach et $U \subset X$ un ouvert. Soit $(-A)$ le générateur infinitésimal d'un semi-groupe compact $T(t), t \geq 0$. Si $0 < a \leq \infty$ et $f : [0, a[ \times U \to X$ est continue alors pour tout $u_0 \in U$ il existe $t_1 = t_1(u_0), 0 < t_1 < a$ tel que le problème $(P')$ admet une solution $u \in C([0, t_1], U)$.*

## 7.5 Quelques méthodes pour étudier les équations caractéristiques :

Dans cette partie nous exposons quelques méthodes pour étudier des équations caractéristiques provenant des équations différentielles ordinaires ou à retard discret. Nous commencçons par rappeler la méthode de Sturm permettant de localiser les racines d'une équation polynômiale. Nous présentons en suite la méthode de Routh-Hurwitz qui donne des conditions nécéssaires et suffisantes de stabilité d'une équation polynômiale. Enfin, nous présentons la méthode de Pontriaguine qui est une généralisation du résultat de Routh-Hurwitz. La méthode de Pontriaguine concerne les équations quasi-polynômiales.

### 7.5.1 Méthode de Routh-Hurwitz

Le critère de Routh-Hurwitz donne des conditions nécessaires et suffisantes pour que tous les zéros du polynôme

$$P(\lambda) = a_0\lambda^n + a_1\lambda^{n-1} + .. + a_n, \quad a_0 \neq 0 \text{ et } a_i \in IR,\ i = 0, n$$

soient à partie réelle négative.
Nous nous contentons de donner le résultat dans le cas des équations caractérisques provenant de la linéarisation de systèmes ordinaires. Ainsi, nous avons le critère de Routh-Hurwitz suivant :
Toutes les racines de l'équation

$$\lambda^n + a_1\lambda^{n-1} + .. + a_n = 0$$

ont des parties réelles négatives si et seulement si les inégalités suivantes sont satisfaites

$$a_1 > 0, \quad \begin{vmatrix} a_1 & a_3 \\ 1 & a_2 \end{vmatrix} > 0, ..., \quad \begin{vmatrix} a_1 & a_3 & a_5 & . & . & 0 \\ 1 & a_2 & a_4 & . & . & 0 \\ 0 & & a_3 & & & . \\ . & & & . & & . \\ . & & & & . & . \\ 0 & & & & & a_n \end{vmatrix} > 0. \tag{7.7}$$

**Exemple** Si $n = 3$, l'équation s'écrit

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$$

Dans ce cas les conditions (7.7) s'écrivent

$$a_1 > 0,\ a_1a_2 - a_3 > 0,\ a_3 > 0.$$

Si $n = 4$, l'équation s'écrit

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$$

Dans ce cas les conditions (7.7) s'écrivent

$$a_1 > 0,\ a_1a_2 - a_3 > 0,\ a_1(a_2a_3 - a_1a_4) - a_3^2 > 0,\ a_4 > 0.$$

### 7.5.2 Méthode de Pontriaguine

La méthode de Pontriaguine concerne des équations du type quasi-polynômiales ; c'est à dire des équations de la forme

$$h(z, e^z) = 0$$

où $h(x, y)$ est un polynôme en $x$ et $y$. Nous commencons par donner des définitions qui nous seront utiles dans la suite.

**Définition 9.** *Soit $f(z, u, v)$ un polynôme en $z, u$ et $v$, que nous écrivons sous la forme*

$$f(z, u, v) = \sum_{m,n} z^m \phi_m^{(n)}(u, v), \quad m, n \in IN$$

*où $\phi_m^{(n)}(u,v)$ est un polynôme de degré $n$, homogène en $u$ et $v$. On appelle terme principal dans le polynôme $f(z,u,v)$ le terme $z^r\phi_r^{(s)}(u,v)$ tel que : pour tout les termes $\phi_m^{(n)}(u,v)$ de $f(z,u,v)$ on ait soit $r > m$ et $s > n$ ou bien $r = m$ et $s > n$ ou bien $r > m$ et $s = n$.*

**Remarque 1.** *Evidemment, il existe des fonctions quasi-polynômiales qui n'admettent pas de terme principal.*

Avant de rappeler les résultats dûs à Pontriaguine, nous introduisons les notations suivantes : Soit $z^r\phi_r^{(s)}(u,v)$ le terme principal de $f(z,u,v)$.
On note par :

$$\phi^{*(s)}(u,v) = \sum_{n\leq s} \phi_r^{(n)}(u,v)$$

et on note aussi

$$\phi^{*(s)}(z) = \phi^{*(s)}(\cos z, \sin z)$$

**Théorème 11.** *Soit $f(z,u,v)$ un polynôme et soit $z^r\phi_r^{(s)}(u,v)$ son terme principal. Si $\varepsilon$ est tel que $\phi^{*(s)}(\varepsilon + iy)$ est différent de zéro pour tout $y \in IR$, alors dans la bande $-2k\pi \leq x \leq 2k\pi$, $z = x + iy$, la fonction $F(z) = f(z, \cos z, \sin z)$ possède exactement $(4sk + r)$ zéros pour toute valeur assez grande de $k$. Ainsi, pour que toutes les racines de la fonction $F(z)$ soient réelles, il faut et il suffit qu'elle admette $(4sk+r)$ racines réelles dans la bande $-2k\pi \leq x \leq 2k\pi$ pour $k$ assez grand.*

Afin d'annoncer le théorème principal de Pontriaguine, nous avons besoin de la définition suivante :

**Définition 10.** *Soient $p(y)$ et $q(y)$ deux fonctions à variable réelle. On dit que les zéros de ces fonctions sont alternés si tous les zéros de $p(y)$ et $q(y)$ sont simples, si entre deux zéros de l'une il existe au moins un zéro de l'autre et si les fonctions $p(y)$ et $q(y)$ ne s'annullent pas simultanément.*

**Théorème 12.** *Soit $H(z) = h(z, e^z)$, où $h(z,t)$ est un polynôme en $z$ et $t$ et admettant un terme principal. On sépare la fonction $H(iy)$ en partie réelle et imaginaire :*

$$H(iy) = F(y) + iG(y)$$

*Si tous les zéros de la fonction $H(z)$ sont à partie réelle négative, alors les zéros des fonctions $F(y)$ et $G(y)$ sont réels, alternés et on a*

$$G'(y)F(y) - G(y)F'(y) > 0 \tag{7.8}$$

*pour tout $y$. De plus, pour que tous les zéros de $H(z)$ soient à partie réelle négative, il suffit que l'une des conditions suivantes soit satisfaite :*
*(a) Tous les zéros de $F(y)$ et $G(y)$ sont réels, alternés et l'inégalité (7.8) est satisfaite pour au moins*

*une valeur de $y$.*
*(b) Tous les zéros de $F(y)$ sont réels et pour chaque zéro $y = y_0$, la condition (7.8) est satisfaite ; c'est à dire : $F'(y_0)G(y_0) < 0$.*
*(c) Tous les zéros de $G(y)$ sont réels et pour chaque zéro $y = y_0$, l'inégalité (7.8) est satisfaite ; c'est à dire $G'(y_0)F(y_0) > 0$.*

**Références**


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**Modélisation et Etude Mathématique de quelques problèmes de dynamique des populations**

**Résumé :** Le travail présenté dans cette thèse se situe dans la ligne des travaux d'écologie mathématique initiée dans les année 1920 par les travaux de Lotka et Volterra, travaux dans lesquels a été introduite la représentation des interactions entre espèces par des systèmes d'équations différentielles.

La thèse est scindée en deux parties : La première partie est consacrée aux systèmes Prédateurs-Proies en présence d'une infection transmissible entre les proies. Le but de cette partie est d'étudier l'impact de l'infection sur le comportement du système. Notre paramètre principal de l'étude de cette partie est le taux de transmission $\lambda$.
Deux modèles ont été élaborés et étudiés mathématiquement : un modèle classique dont la réponse fonctionnelle ne dépend que de l'abondance de la proie. Puis un modèle ratio-dépendant où la réponse fonctionnelle est considérée dépendante du rapport proie par prédateur.

Dans la deuxième partie, nous avons étudié le système Phytoplancton-Zooplancton en présence de substances toxiques libérées par quelques espèces phytoplanctoniques toxiques.
Le but est d'étudier l'effet de la toxine sur le comportement qualitatif du système. Nos paramètres principaux dans cette étude sont l'efficacité de la toxine et le retard.
Plusieurs modèles ont été élaborés et étudiés : des modèles homogènes mais aussi des modèles structurés, des modèles à retard fini mais aussi des modèles à retard infini, des retards discrets ou distribués.

Dans les deux parties, nous avons pu mettre en évidence la stabilité, l'instabilité et la bifurcation en fonction des paramètres. Notre résultat principal est que la présence d'infection ou de substances toxiques dans les systèmes que nous avions étudiés peut être bénéfique au système et peut agir comme un contrôle biologique.

**MOTS CLEFS :** Prédateur-Proie, Zooplancton-Phtoplancton Toxique, Stabilité, Bifurcation, Equations à retard.