An Overview of Keller-Segel Models

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1 Introduction

Multi and unicellular organisms alike have developed mechanisms to regulate their movement in response to their environment. For single-celled organisms, that tends to be in response to stimuli like light, chemicals, temperature, and many more. Their responses to these factors are not random, and over time have been studied by scientists and classified. This project is focused on the movement of organisms, particularly cellular slime molds, specifically due to interactions with chemicals, so called chemotaxis. The study of chemotaxis has led to various mathematical models to represent the movement of organisms, and this project will focus on the Keller-Segel model.

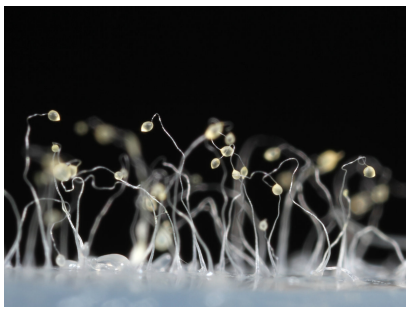

Figure 1: Fruiting bodies of Dictyostelium discoideum (Source).

In the remainder of this section I will introduce chemotaxis and the cellular slime mold Dictyostelium discoideum which is the basis for the Keller-Segel model. In Section 2, I will discuss the derivation of the Keller-Segel model as described in [1] and [2] as well as the proofs of the chemotactic collapse phenomena as laid out in [3]. In Section 3, I will discuss a few chosen variations of the Keller-Segel model, particularly the minimal model, a few models with different diffusion terms, and models coupled to the Navier-Stokes equation. Finally, in Section 4, I will discuss numerical approximations for the Keller-Segel model, and describe a few specific approximations from [4] and [5].

1.1 Chemotaxis & Cellular Slime Mold

Chemotaxis is the movement of cells or organisms in response to some chemical stimulus, often a growth hormone. Movement towards a higher concentration of the chemical is called positive chemotaxis, and the movement towards a lower concentration of the chemical is called negative chemotaxis. For multicellular organisms, chemotaxis of cell populations often plays a crucial role throughout their life cycle.

The cellular slime mold Dictyostelium discoideum is a great example of chemotactic movement. At the start of their life cycle, the cells disperse as if repulsed by one another. They proceed to grow by cell division as long as food sources are available, moving towards the sources of food. After exhausting their food supply, they spread over the entire domain available nearly uniformly. Then, cells will start to exude cyclic Adenosine Monophosphate (cAMP), attracting the other cells. These initial cells will act as collecting points, and the other cells will aggregate toward them. At these points, a slug will form acting as one entity and will move towards light. Eventually a fruiting body is formed and spores are spread, starting the cycle over again. The aggregation phase is of particular interest for this project. The aggregation of the cells is governed by acrasin which is degraded by the enzyme acrasinase.

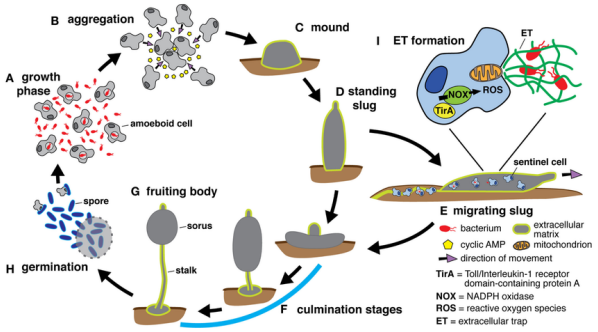

Figure 2: The life cycle of Dictyostelium discoideum (Source).

Chemotactic behavior has been studied for over a century, and is governed by relationships between cells and invariant rules about diffusion. Once scientists were able to understand the movement of things like our slime mold, the natural next step was for mathematicians to make mathematical models.

2 The Keller-Segel Model

$$\begin{aligned} u_t &= \nabla \cdot (D_1 \nabla u - \chi u \nabla v) + f(u,v) \\ v_t &= D_2 \Delta v + g(u,v) - h(u,v) \end{aligned} \tag{1}$$

The classical Keller-Segel model (1) was developed in [1] to describe the cell movement of amoebae in response to the chemical acrasin. u and v are the cell density and acrasin concentration, respectively. D_1 describes the diffusivity of the cells and χ represents the chemotactic sensitivity. D_2 describes the diffusivity of the chemical attractant, f describes cell growth and death, and g and h describe the production and degradation rates of the chemical attractant.

2.1 Model Derivation

The review of Keller-Segel models by Horstmann [2] discusses five different methods to derive this model. Broadly they are 1) based on Fick's law and Fourier's law, 2) based on random walks, 3) based on interacting particle systems, 4) based on transport equations, and 5) based on stochastic processes.

I will reiterate the first derivation, which also seems to be in the original Keller-Segel paper [1]. There are two different perspectives from which we could model the chemotactic movement of our slime: the macroscopic perspective and the microscopic perspective. The macroscopic perspective involves considering the entire population and the density of the population at one place and one time. In 1970, Keller and Segel used a macroscopic perspective

to develop a system of four pdes to describe the aggregation of cellular slime molds like ours. Let $u(t,x)$ be the cellular density and $v(t,x)$ be the chemical attractant concetration.

Assumptions:

- the chemoattractant acrasin is produced at a rate $f(v)$ per amoeba
- the enzyme acrasinase which degrades acrasin, whose concentration is denoted $p(t,x)$, is produced by the amoeba at a rate $g(v,p)$ per amoeba (v is the concentration of acrasin, p the concentration of acrasinase)
- acrasin and acrasinase react to form a complex $\mathcal{E}$ of concentration η which dissociates into the free enzyme plus the degraded product:

$$v + p \underset{r_{-1}}{\overset{r_1}{\rightleftharpoons}} \mathcal{E} \xrightarrow{r_2} p + \text{degraded product}$$

- arcasin, acrasinase, and the complex $\mathcal{E}$ diffuse according to Fick's law
- the amoeba concentration $u(t,x)$ changes as a result of chemotactic motion in the direction of a positive gradient of acrasin and a random motion analogous to diffusion

Now, denote $J^{(u)}(t,x)$ as the flow of the amoeba density, $n(x)$ as the unit exterior normal to surface S, and a control volume D. The balance of the amoeba density $u(t,x)$ tells us

$$\frac{d}{dt}\int_D u(t,x)dx = -\int_{\partial D}\left(J^{(u)}(t,x)\cdot n(x)\right)dS \tag{2}$$

According to Fick's law, $J^{(u)}(t,x)$ contains a part that is proportional to the cellular density gradient. By Fourier's law for heat flow, it also contains a part that is proportional to the acrasin concentration gradient. Therefore, we have $J^{(u)}(t,x) = k_2\Delta v - k_1\Delta u$. Our chemoattractant acrasin diffuses, so we can write

$$\frac{d}{dt}\int_D v(t,x)dx = Q^{(v)}(t,D) - \int_{\delta D}\left(J^{(v)}(t,x)\cdot n(x)\right)dS \tag{3}$$

where $Q^{(v)}(t,D)$ is the acrasin created per unit area per unit time. Notice that because we are ignoring reproduction, $Q^{(u)}(t,D) = 0$ in the previous equation. Here, $J^{(v)}(t,x) = -k_c\Delta v$. If we assume the analogous equations for J for acrasinase and the complex, take basic processes into account, and take reaction rates r_{-1},r_1, r_2 from the third assumption, we can derive the following system:

$$\begin{aligned}
u_t &= \nabla(k_1(u,v)\nabla u - k_2(u,v)\nabla v), \qquad x\in\Omega, t>0\\
v_t &= k_c\Delta v - r_1 vp + r_{-1}\eta + uf(v), \qquad x\in\Omega, t>0\\
p_t &= k_p\Delta p - r_1 vp + (r_{-1}+r_2)\eta + ug(v,p), \qquad x\in\Omega, t>0\\
\eta_t &= k_\eta\Delta\eta + r_1 vp - (r_{-1}+r_2)\eta, \qquad x\in\Omega, t>0\\
\frac{\partial u}{\partial n} &= \frac{\partial v}{\partial n} = \frac{\partial p}{\partial n} = \frac{\partial \eta}{\partial n} = 0, \qquad x\in\partial\Omega, t>0\\
u(0,x) &= u_0(x), \quad v(0,x) = v_0(x), \qquad x\in\Omega\\
p(0,x) &= p_0(x), \quad \eta(0,x) = \eta_0(x), \qquad x\in\Omega
\end{aligned} \tag{4}$$

Notice that this is not the same system as (1), though, because more simplification can be done. We start by assuming that the complex is in a steady state with regard to the chemical reaction, then $r_1 vp - (r_{-1} + r_2)\eta = 0$. We also assume that the total concentration of acrasinase is a constant, so $p + \eta = p_0$. These assumptions allow us to simplify (4) to

$$\begin{aligned} u_t &= \nabla\left(k_1(u,v)\nabla u - k_2(u,v)\nabla v\right), \qquad x \in \Omega, t > 0 \\ v_t &= k_c \Delta v - k_3(v)v + uf(v). \qquad x \in \Omega, t > 0 \\ \frac{\partial u}{\partial n} &= \frac{\partial v}{\partial n} = 0, \qquad x \in \partial\Omega, t > 0 \\ u(0,x) &= u_0(x), \quad v(0,x) = v_0(x), \qquad x \in \Omega \end{aligned} \tag{5}$$

If we were to incorporate a nonzero function of cell growth, clearly that would just be added to the first equation in (5), resulting exactly in the first equation in (1). The second equation we must look at more carefully to see that it is the same. Clearly, $D_2\Delta v$ and $k_c\Delta v$ are analogous. Since $f(v)$ in (5) is the rate of production of acrasin, $uf(v)$ corresponds to the term g in (1). Then, we have $k_3(v)v$ in (5) corresponding to h in (1) and thus the two systems are the same, neglecting the boundary and initial conditions.

2.2 Existence and Blow-up of Solutions

The chemotactic collapse is the idea that either the solution of the Keller-Segel system globally exists or the concentration of cells, $u(t,x)$, blows up in finite time. This conjecture was introduced by Childress and Percus in 1981 and Nanjundiah in 1973, and Blanchet et al. validate this conjecture in [3].

Blanchet et al. focus on the simplified Keller-Segel system (6):

$$\begin{aligned} \partial_t u &= \Delta u - \chi\nabla\cdot(u\nabla v), \\ 0 &= \Delta v + u \end{aligned} \tag{6}$$

The main tool they use is the free energy

$$F[u] := \int_{\mathbb{R}^2} u\log u dx - \frac{\chi}{2}\int_{\mathbb{R}} uv dx$$

which provides *a priori* estimates.
Essentially, in [3] they prove

1. solutions of (6) blow-up in finite time when the mass $M > 8\pi/\chi$
2. global time solutions of (6) exist when the mass $M < 8\pi/\chi$

I will briefly go through their arguments as presented in [3]. First, we will make some assumptions and initial observations. We assume our initial data satisfies:

$$u_0 \in L^1_+(\mathbb{R}^2, (1+|x|^2)dx), \quad u_0\log u_0 \in L^1(\mathbb{R}^2, dx) \tag{7}$$

We can see that the total mass is conserved for smooth and sufficiently decaying solutions, ie that

$$M := \int_{\mathbb{R}^2} u_0(x)dx = \int_{\mathbb{R}^2} u(x,t)dx \tag{8}$$

2.2.1 Case 1: $M > 8\pi/\chi$

First we begin by defining what we mean by a weak solution u in the space $L^\infty_{\text{loc}}(\mathbb{R}^+; L^1(\mathbb{R}^2))$. We say that u is a solution to (6) if, for all test functions $\psi \in \mathcal{D}(\mathbb{R}^2)$,

$$\frac{d}{dt}\int_{\mathbb{R}^2}\psi(x)u(x,t)dx$$
$$= \int_{\mathbb{R}^2}\Delta\psi(x)u(x,t)dx - \frac{\chi}{4\pi}\int_{\mathbb{R}^2\times\mathbb{R}^2}[\nabla\psi(x)-\nabla\psi(y)]\cdot\frac{x-y}{|x-y|^2}u(x,t)u(y,t)dxdy$$

Lemma 2.1 *Consider a non-negative L^1 solution u to (6) as defined above on an interval [0,T]. Assume that u also satisfies (8), $\int_{\mathbb{R}^2}|x|^2u_0(x)dx < \infty$. Then it will also satisfy*

$$\frac{d}{dt}\int_{\mathbb{R}^2}|x|^2u(x,t)dx = 4M(1-\frac{\chi M}{8\pi})$$

This can be proven first by taking a smooth function $\varphi_\varepsilon(|x|)$ with compact support which grows nicely to $|x|^2$ as $\varepsilon \to 0$. Then, from our definition of weak solutions we have

$$\frac{d}{dt}\int_{\mathbb{R}^2}\varphi_\varepsilon u dx$$
$$= \int_{\mathbb{R}^2}\Delta\varphi_\varepsilon u dx - \frac{\chi}{4\pi}\int_{\mathbb{R}^2}[\nabla\varphi_\varepsilon(x)-\nabla\varphi_\varepsilon(y)]\cdot\frac{x-y}{|x-y|^2}u(x,t)u(y,t)dxdy$$

Since we can always choose our φ_ε such that $\Delta\varphi_\varepsilon$ is bounded and $\nabla\varphi_\varepsilon(x)$ is Lipshitz continuous, we find that

$$\frac{d}{dt}\int_{\mathbb{R}^2}\varphi_\varepsilon u dx \le C\int_{\mathbb{R}^2}u_0 dx$$

for C a positive constant. Then, as $\varepsilon \to 0$, we have

$$\int_{\mathbb{R}^2}\varphi_\varepsilon u dx \le c_1 + c_2 t$$

for c_1 and c_2 positive constants, and thus

$$\int_{\mathbb{R}^2}|x|^2u(x,t)dx < \infty \quad \forall t \in (0,T)$$

Now, the authors cite Lebesgue's dominated convergence theorem (see A.2), and take this to the limit, which completes the proof of Lemma 2.1.

Then, we recover what we wished to prove, that for $M > 8\pi/\chi$ there is a finite time T^* at which the solutions become singular measures. This T^* is our finite blow-up time.

2.2.2 Case 2: $M < 8\pi/\chi$

We begin with the free energy as stated earlier

$$F[u] := \int_{\mathbb{R}^2}u\log u dx - \frac{\chi}{2}\int_{\mathbb{R}}uv dx$$

We can see that the first term is the entropy and the second is a potential energy term. This is also historically called the "entropy method," but the authors chose "free energy" because they felt it was more physically appropriate. For any solution u of (6), $F[u(\cdot,t)]$ is monotonically increasing.

Lemma 2.2 *Consider a non-negative $C^0(\mathbb{R}^+, L^1(\mathbb{R}^2))$ solution u of (6) such that $u(1+|x|^2)$ and $u\log u$ are bounded in $L^\infty_{loc}(\mathbb{R}^+, L^1(\mathbb{R}^2))$, $\nabla\sqrt{u} \in L^1_{loc}(\mathbb{R}^+, L^2(\mathbb{R}^2))$, and $\nabla v \in L^\infty_{loc}(\mathbb{R}^+ \times \mathbb{R}^2)$. Then*

$$\frac{d}{dt}F[u(\cdot,t)] = -\int_{\mathbb{R}^2} u|\nabla(\log u) - \chi\nabla v|^2 dx \tag{9}$$

Let us proceed with a simple proof of this lemma. The potential energy term is $\int_{\mathbb{R}^2} uv dx = \iint_{\mathbb{R}^2\times\mathbb{R}^2} u(x,t)u(y,t)\log|x-y|dxdy$, which is quadratic in u. Using (6), we get

$$\frac{d}{dt}F[u(\cdot,t)] = \int_{\mathbb{R}^2}\left[(1+\log u - \chi v)\,\nabla\cdot\left(\frac{\nabla u}{u} - \chi\nabla v\right)\right]dx$$

Then, an integration by parts completes the proof.
Next, we shall use the fact that our concentration v solves the Poisson equation $-\Delta v = u$ and has the form

$$v(x,t) = -\frac{1}{2\pi}\int_{\mathbb{R}^2}\log|x-y|u(y,t)dy \tag{10}$$

to deduce that

$$\frac{d}{dt}F[n(\cdot,t)] = \frac{d}{dt}\left[\int_{\mathbb{R}^2} u\log u dx + \frac{\chi}{4\pi}\iint_{\mathbb{R}^2\times\mathbb{R}^2} u(x,t)u(y,t)\log|x-y|dxdy\right] \le 0$$

As a part of their argument, [3] utilize the Hardy-Littlewood-Sobolev inequality:

Lemma 2.3 *Let f be a non-negative function in $L^1(\mathbb{R}^2)$ such that $f\log f$ and $f\log(1+|x|^2)$ belong to $L^1(\mathbb{R}^2)$. If $\int_{\mathbb{R}^2} f dx = M$, then*

$$\int_{\mathbb{R}^2} f\log f dx + \frac{2}{M}\iint_{\mathbb{R}^2\times\mathbb{R}^2} f(x)f(y)\log|x-y|dxdy \ge -C(M) \tag{11}$$

with $C(M) := M(1+\log\pi - \log M)$.

Lemma 2.4 *Consider a non-negative $C^0(\mathbb{R}^+, L^1(\mathbb{R}^2))$ solution u of (6) such that $u(1+|x|^2)$ and $u\log u$ are bounded in $L^\infty_{loc}(\mathbb{R}^+, L^1(\mathbb{R}^2))$, $\int_{\mathbb{R}^2}\frac{1+|x|}{|x-y|}u(y,t)dy \in L^\infty((0,T)\times\mathbb{R}^2)$, $\nabla\sqrt{u} \in L^1_{loc}(\mathbb{R}^+, L^2(\mathbb{R}^2))$, and $\nabla v \in L^\infty_{loc}(\mathbb{R}^+\times\mathbb{R}^2)$. If $\chi M \le 8\pi$, then the following estimates hold:*

1. *Entropy:*

$$M\log M - M\log[\pi(1+t)] - K \le \int_{\mathbb{R}^2} u\log u dx \le \frac{8\pi F_0 + \chi M C(M)}{8\pi - \chi M}$$

with $K := max\{\int_{\mathbb{R}^2}|x|^2u_0(x)dx, \frac{M}{2\pi}(8\pi-\chi M)\}$ and $F_0 := F[u_0]$.

2. *Fisher information: For all $t>0$, with $C_1 := F_0 + \frac{\chi M}{8\pi}C(M)$ and $C_2 := \frac{\chi M - 8\pi}{8\pi}$,*

$$\begin{aligned} 0 &\le \int_0^t ds\int_{\mathbb{R}^2} u(x,s)|\nabla(\log u(x,s)) - \chi\nabla v(x,s)|^2 dx \\ &\le C_1 + C_2[M\log\left(\frac{\pi(1+t)}{M}\right) + K] \end{aligned}$$

Now we shall prove this lemma. From (9), we have

$$(1-\theta)\int_{\mathbb{R}^2} u\log u dx + \theta\left[\int_{\mathbb{R}^2} u\log u dx + \frac{\chi}{4\pi\theta}\iint_{\mathbb{R}^2\times\mathbb{R}^2} u(x,t)u(y,t)\log|x-y|dxdy\right] \le F_0$$

We choose $\frac{\chi}{4\pi\theta} = \frac{2}{M} \iff \theta = \frac{\chi M}{8\pi}$ and apply (11):

$$(1-\theta)\int_{\mathbb{R}^2} u(x,t)\log u(x,t)dx - \theta C(M) \le F_0$$

If $\chi M < 8\pi$, then $\theta < 1$ and

$$\int_{\mathbb{R}^2} u(x,t)\log u(x,t)dx \le \frac{F_0 + \theta C(M)}{1-\theta}$$

Thus, we have an upper bound for the entropy. We can also bound $\int_{\mathbb{R}^2} u\log u dx$ from below. By Lemma 2.1:

$$\frac{1}{1+t}\int_{\mathbb{R}^2} |x|^2 u(x,t)dx \le K \quad \forall t > 0.$$

Through some rewriting and simplifying, specifically with $\mu(x,t) := \frac{1}{\pi(1+t)}\exp(-\frac{|x|^2}{1+t})$, we have

$$\begin{aligned}\int_{\mathbb{R}^2} u(x,t)\log u(x,t)dx &\ge \frac{1}{1+t}\int_{\mathbb{R}^2}|x|^2u(x,t)dx - K + \int_{\mathbb{R}^2} u(x,t)\log u(x,t)dx \\ &= \int_{\mathbb{R}^2}\frac{u(x,t)}{\mu(x,t)}\log\left(\frac{u(x,t)}{\mu(x,t)}\right)\mu(x,t)dx - M\log[\pi(1+t)] - K\end{aligned}$$

By Jensen's inequality,

$$\int_{\mathbb{R}^2}\frac{u(x,t)}{\mu(x,t)}\log\left(\frac{u(x,t)}{\mu(x,t)}\right)\mu(x,t)dx > X\log X$$

where $X = \int_{\mathbb{R}^2}\frac{u(x,t)}{\mu(x,t)}\mu(x,t)dx = M$. Thus we have a lower estimate for the entropy term. From (9) and (11) we have

$$(1-\theta)[M\log\left(\frac{M}{\pi(1+t)}\right) - K] + \theta C(M) + \int_0^t ds\int_{\mathbb{R}^2} u(x,s)|\nabla(\log u(x,s)) - \chi\nabla v(x,s)|^2 dx \le F_0$$

Then we have that $\sqrt{u}|\nabla(\log u) - \chi\nabla v|$ is bounded in $L^2_{\text{loc}}(\mathbb{R}^+, L^2(\mathbb{R}^2))$ and we have the bound.

Next, we want to piece together our bound on $\int_{\mathbb{R}^2} u\log u dx$ and the $|x|^2$ moment bound of Lemma 2.1 in the next lemma.

Lemma 2.5 *For any $w \in L^1_+(\mathbb{R}^2)$, if $\int_{\mathbb{R}^2}|x|^2 w dx$ and $\int_{\mathbb{R}^2} w\log w dx$ are bounded from above, then $w\log w$ is uniformly bounded in $L^\infty(\mathbb{R}^+_{loc}, L^1(\mathbb{R}^2))$ and*

$$\int_{\mathbb{R}^2} w|\log w|dx \le \int_{\mathbb{R}^2} w\left(\log w + |x|^2\right)dx + 2\log(2\pi)\int_{\mathbb{R}^2} w dx + \frac{2}{e}$$

I will omit the proof to the lemma because I did not fully understand it, but see the Appendix A.2 for the proof from [3] and my thoughts.
Now, we can state the existence result for the subcritical case $M < 8\pi/\chi$.

Theorem 2.6 *Assume that $u_0 \in L^1_+(\mathbb{R}^2, (1+|x|^2)dx)$ and $u_0 \log u_0 \in L^1(\mathbb{R}^2, dx)$. If $M < 8\pi/\chi$, then the Keller-Segel system (6) has a global weak non-negative solution u with initial data u_0 such that, for any $T > 0$,*

$$(1+|x|^2+|\log u|)u \in L^\infty(0,T;L^1(\mathbb{R}^2)),$$
$$\iint_{[0,T]\times\mathbb{R}^2} u|\nabla \log u - \chi \nabla v|^2 dxdt < \infty,$$

3 Variations of the Keller-Segel Model

The standard Keller-Segel model is often used as scaffolding to describe a biological scenario from which more nuanced relationships can be described. The system itself encapsulates many of the relationships necessary, but more specific variations exist to represent specific systems. The standard system also presents challenges when it comes to solving and simulating cell behavior, so often researchers will instead focus on a specific variation called the minimal model.

The minimal model is only one of a plethora of variations. Variations of the Keller-Segel system could be coupling the system with other equations like Navier-Stokes or simply changing one element like the source type, secretion or diffusion behavior, type of motility, etc. The review paper [6] contains a list of many different variations for further reading.

Broadly, the non-coupled variations of the model can be written in the form

$$\begin{aligned} u_t &= \nabla(D(u)\nabla u - A(u)B(v)C(\nabla v)) + f(u) \\ v_t &= \Delta v + ug(u) - v \end{aligned} \tag{12}$$

with zero-flux boundary conditions:

$$\mathbf{n} \cdot (D(u)\nabla u - A(u)B(v)C(\nabla v)) = \mathbf{n} \cdot \nabla v = 0 \tag{13}$$

The choices for A, B, C, D, f, and g depend on the specific model.

3.1 The Minimal Model

The classical Keller-Segel model is quite complicated, and in order to perform numerical simulations or produce numerical solutions, many papers instead focus on the so-called minimal Keller-Segel model. This model comes about through making the following more restrictive assumptions about the behavior of the cells and the chemical:

1. individual cells experience a combination of chemotaxis and random motion towards the chemical attractant
2. cells do not die or divide, ie $f = 0$

3. the attractant is produced at a constant rate, ie g is a constant dependent only on u
4. the degradation rate of the attractant, h, is linearly dependent on its concentration v
5. the attractant diffuses passively over the domain

These assumptions led us to the minimal model (14), with D_1, D_2, and χ positive constants.

$$\begin{aligned} u_t &= \nabla \cdot (D_1 \nabla u - \chi u \nabla v) \\ v_t &= \nabla^2 D_2 v + g(u) - h(v) \end{aligned} \tag{14}$$

It has been proved in various works (ie, [2] [4] [7]) that this model has globally existing solutions in 1D, finite time blow-up, and spacial pattern formation. In higher dimensions there is a threshold phenomenon with blow-up solutions.

3.2 Different types of Diffusion

Some variations of the Keller-Segel model involve only changing the chemical diffusion term. This intuitively makes sense as we expect different chemicals to diffuse differently. While there are dozens of different ways to model how a chemical diffuses, I will briefly discuss the cases of no diffusion, nonlinear diffusion, and Fokker-Planck diffusion.

3.2.1 Non-Diffusive Chemical

While at first it may seem odd to discuss a non-diffusive chemical, Keller-Segel systems with non-diffusive chemicals are valuable for representing haptotaxis and angiogenesis. Haptotaxis is cell movement based on gradients of adhesion, and angiogenesis is the process of new blood vessels forming out of existing blood vessels. Studying angiogensis is important for understanding tumor growth, but is a process that occurs throughout our life cycle. These Keller-Segel systems are also related to the dynamics of self-reinforced related walks.

One such Keller-Segel system with a non-diffusive chemical is given in [8] as

$$\begin{aligned} \partial_t u &= \Delta u - \Delta \cdot \left(u \frac{\Delta v}{v} \right) \\ \partial_t v &= u v^{\lambda} \end{aligned} \tag{15}$$

In [8], they prove the existence of global solutions to (15) with $\lambda < 1$ and small initial data.

Another interesting example which can be modeled by such a system is the movement of myxobacteria, whose life cycle is remarkably similar to the slime mold Keller and Segel studied. When food sources are scarce, the myxobacteria aggregate into fruiting bodies and develop myxospores, eventually repeating the process again. Myxobacteria move through gliding on "slime trails" and when a myxobacteria encounters another's slime trail, it will often choose to go down that trail, speeding up on the way. The authors in [9], discuss the blow-up and collapse conditions for a specific model similar to (15). One of the main conclusions in [9] is that aggegration can occur even without a diffusive chemical, in their case due to the tendency of the random walking myxobacteria to follow the slime trails left by others.

3.2.2 Nonlinear Diffusion

Typically researchers assume constant diffusion, but more accurately diffusion would depend nonlinearly on something like the chemical concentration or cell density. The dependence on cell density has been frequently studied in ecological applications to describe "population-induced" movement for insect populations.

Generally, Keller-Segel systems with nonlinear diffusion could be of the form:

$$\begin{aligned} u_t &= \nabla \cdot (D(u)\nabla u - \chi u \nabla v) \\ v_t &= \Delta v + u - v \\ D(u) &= d_1 u^n, \quad n \geq 0 \end{aligned} \tag{16}$$

Telling us that the rate of diffusion would increase as the cell density increases.

In [10], Kowalczyk specifically works with $D(u) = uh'(u)$ where $h'(u) \geq \frac{c}{u}$ and h is a stress function. This diffusion term rapidly grows to infinity for large enough cell density. This phenomena is related to the fact that the closer the cells are, the more stress (h) will be exerted between them. If instead they were all far enough away from each other, the stress is essentially zero, so there is no finite time blow-up. Due to the nonlinearity, we cannot use all results from Keller-Segel models in general to discuss this model, but Kowalczyk works towards proving global existence of solutions for this specific model.

3.2.3 Fokker-Planck Diffusion

In class we discussed the Fokker-Planck equation $\partial_t p = -\nabla \cdot (up - D\nabla p)$, where $up - D\nabla p$ is a probability flux. Fokker-Planck diffusion in systems like the Keller-Segel system allows us to take into account some stochastic noise, modeling when the cells undergo some random Brownian-like motion. For instance, Yoon and Kim in [11] incorporate Fokker-Planck diffusion into the following Keller-Segel system

$$\begin{aligned} \partial_t u &= \Delta(\gamma(v)u) \\ \partial_t v &= \epsilon \Delta v + bu - av \end{aligned} \tag{17}$$

The chemical is produced by the cell with a rate $b > 0$, degraded at a rate $a > 0$, and diffused at a rate $\epsilon > 0$. Instead of the usual chemotactic term χ seen in (1), we have the motility function $\gamma(v)$ which decreases as the density of the chemical increases, ie $\gamma'(v) < 0$. The form of the motility function used in [11] is $\gamma(v) = \frac{c_0}{v^k}$ for $c_0 > 0$ and $k > 0$. The idea behind adding Fokker-Planck diffusion is that the organisms decrease motility as the density of the chemical increases. This naturally creates an advection term in the first equation, which we can see is equivalent to

$$\nabla \cdot \left(\gamma(v) \left(\nabla u - \frac{k}{v} u \Delta v \right) \right)$$

In their paper, Yoon and Kim establish the global existence of solutions to this problem and discuss pattern formation in order to establish that there is a pattern of cell aggregation. They then perform numerical simulations and focus on confirming their theory of cell aggregation.

3.3 Coupling with Navier-Stokes

One of the most common variations that I found in my research was the Keller-Segel system coupled with the Navier-Stokes equation. This not surprising, for if we wish to extend beyond observing a cellular slime mold to observing a bacteria like Escherichia coli (E. coli), we must also consider the movement of the fluid the bacteria lives in.

The general chemotaxis-fluid system can be written as in [12]:

$$\begin{aligned} \partial_t n + u \cdot \nabla n &= \Delta n - \nabla \cdot (n \nabla c) + f(n), && (x,t) \in \Omega \times (0,T) \\ \partial_t c + u \cdot \nabla c &= \Delta c - c + n, && (x,t) \in \Omega \times (0,T) \\ \partial_t u + \kappa (u \cdot \nabla) u + \nabla P &= \Delta u - n \nabla \phi + g(x,t), && (x,t) \in \Omega \times (0,T) \\ \nabla \cdot u &= 0, && (x,t) \in \Omega \times (0,T) \end{aligned} \tag{18}$$

Note there is a change in variable names here: n is the concentration of cells, c the concentration of our chemical, and u is the fluid velocity field. P is the pressure of the fluid, f is the degradation of the chemical, g an external force, and ϕ a potential function (e.g. gravitational or centrifugal force). Ω is a bounded smooth domain with no flux boundary conditions, and we should have $T < \infty$. We can see that if we take away the fluid interaction, we can reduce this back down to the Keller-Segel model as defined in (1).

In [13], Tao and Winkler show that if $f(n) = rn - \mu n^2$ and $\kappa = 1$ in the two dimenational case, then there exists a global classical solution. In three dimensions, the conditions are $\mu \geq 23$, $r \geq 0$, and $\kappa = 0$ for a global classical solution.

4 Numerical Approximations

Most of the work on Keller-Segel models since the original paper published in 1970 has been focused on analytical results for the system, not on numerical results. This is because the classical Keller-Segel model (1) has solutions that do not lend themselves well to discretization. More recently, though, researchers have begun working with numerical approximations of many of the variations of the model, in particular the minimal model.

While we have seen that solutions to the minimal Keller-Segel model do exist, they are nontrivial to find. Numerical approximations, however, may allow us to still accurately illustrate the behavior of solutions without having to do anything too complicated. The assumptions made to reach the minimal model from the classical model also set the stage much better for numerical approximations.

4.1 My Simple Finite Differences

My first idea when discussing numerical approximations is to simply use finite differences. So, I attempted to make a finite difference scheme for the 1D minimal model (14). We can rewrite

$$u_t = \frac{u_i^{n+1} - u_i^n}{\Delta t}, \quad u_{xx} = \frac{u_{i-1}^n - 2u_i^n + u_{i+1}^n}{\Delta x^2}, \quad u_x = \frac{u_{i+1}^n - u_{i-1}^n}{2\Delta x}$$

(similar for v_{xx} and v_x). Thus our system (14) becomes

$$\begin{aligned}\frac{u_i^{n+1}-u_i^n}{\Delta t} =& D_1\frac{u_{i-1}^n-2u_i^n+u_{i+1}^n}{\Delta x^2}\\ &-\chi\left(\frac{u_{i+1}^n-u_{i-1}^n}{2\Delta x}\cdot\frac{v_{i+1}^n-v_{i-1}^n}{2\Delta x}+u_i^n\frac{v_{i-1}^n-2v_i^n+v_{i+1}^n}{\Delta x^2}\right)\\ \frac{v_i^{n+1}-v_i^n}{\Delta t} =& D_2\frac{v_{i-1}^n-2v_i^n+v_{i+1}^n}{\Delta x^2}+u_i^n-v_i^n\end{aligned} \tag{19}$$

I used a simple forward Euler step for the time discretizations, and centered difference schemes for the space derivative, so I would expect this scheme to be first order accurate in time and space due to local truncation analysis (see Appendix A.3.1).

4.2 Saito and Suzuki Numerics

Beyond my own attempts at constructing a finite difference scheme, I also read through a paper by Saito and Suzuki [4] where they implemented a 1D and 2D finite difference scheme for the following model, which they refer to as the simplified Keller-Segel system

$$\begin{aligned}u_t &= u_{xx}-(uv_x)_x\\ 0 &= v_{xx}-av+u\end{aligned} \tag{20}$$

with boundary conditions

$$\begin{aligned}&u_x-uv_x=0, \quad v_x=0, \quad x=0,1\\ &u(t=0)=u_0(x)\geq 0\end{aligned} \tag{21}$$

Their scheme is much more involved than my simple scheme, but still understandable. I will reiterate their construction.

Choose a positive integer N and mesh size $h=\frac{1}{N}$. Then we can define two kinds of mesh points with associated subintervals:

$$\begin{aligned}&x_i=(i-\frac{1}{2})h \quad \text{for } i=0,\cdots,N+1 \qquad I_i=(\hat{x_{i-1}},\hat{x_i}) \quad \text{for } i=1,\cdots,N\\ &\hat{x_i}=ih, \qquad \text{for } i=-1,\cdots,N+1 \qquad \hat{I_i}=(x_i,x_{i+1}), \quad \text{for } i=0,\cdots,N\end{aligned}$$

We also define

$$X_h=\{\sum_{i=1}^{N}\alpha_i\chi_i|\{\alpha_i\}_{i=1}^N\subset\mathbb{R}\}, \qquad \hat{X_h}=\{\sum_{i=0}^{N}\beta_i\hat{\chi_i}|\{\beta_i\}_{i=0}^N\subset\mathbb{R}\}$$

where χ_i and $\hat{\chi_i}$ are the characteristic functions of I_i and $\hat{I_i}\cap[0,1]$, respectively. From this

we want to find the unknowns from the forms:

$$u_h^n = \sum_{i=1}^{N} u_i^n \chi_i \approx u(x,t_n) \quad \text{for } u_i^n \in \mathbb{R}$$
$$b_h^n = \sum_{i=1}^{N} b_i^n \chi_i \approx v_x(x,t_n) \quad \text{for } b_i^n \in \mathbb{R}$$
$$F_h^n = \sum_{i=0}^{N} F_i^n \hat{\chi}_i \approx F^n \equiv (u - uv_x)(x,t_n) \quad \text{for } F_i^n \in \mathbb{R}$$
$$v_h^n = \sum_{i=0}^{N} v_i^n \hat{\chi}_i \approx v(x,t_n) \quad v_i^n \in \mathbb{R}$$

Using these notations, we can now describe the finite differences scheme proposed for (20). First, assume we have calculated u_h^{n-1}. Thinking of G_h as an operator from X_h to $\hat{X}_h$, we can calculate v_h^n by $v_h^{n-1} = G_h u_h^{n-1}$. We can compute b_h^{n-1} by

$$b_i^{n-1} = \frac{v_i^{n-1} - v_{i-1}^{n-1}}{h}, \text{ and set } b_i^{n-1,+} = \max\{0, b_i^{n-1}\}, \quad b_i^{n-1,-} = \max\{0, -b_i^{n-1}\}$$

Using an upwind technique, we can imagine that u_i^n and u_{i+1}^n are carried to $\hat{x}_h$ by $b_i^{n-1,+}$ and $-b_i^{n-1,-}$, respectively. So, the flux F_i^n can be approximated by

$$F_i^n = \frac{u_{i+1}^n - u)i^n}{h} - b_i^{n-1,+} u_i^n + b_i^{n-1,-} u_{i+1}^n \quad \text{for } i = 1, \cdots, N-1$$

with boundary conditions $F_0^n = 0$ and $F_N^n = 0$. Then, their proposed scheme for (20) with $\theta \in [0,1]$ is

$$\frac{u_i^n - u_i^{n-1}}{\tau_h} = \theta \frac{F_i^n - F_{i-1}^n}{h} + (1-\theta)\frac{F_i^{n-1} - F_{i-1}^{n-1}}{h} \quad \text{for } i = 1, \cdots, N \tag{22}$$

Equivalently, we can write

$$\begin{aligned} \frac{u_i^n - u_i^{n-1}}{\tau_h} =& \theta[\Delta_h u_i^n - D_h\{b_i^{n-1,+} u_i^n\} + D_h^*\{b_i^{n-1,-} u_i^n\}] \\ &+ (1-\theta)[\Delta_h u_i^{n-1} - D_h\{b_i^{n-1,+} u_i^{n-1}\} + D_h^*\{b_i^{n-1,-} u_i^{n-1}\}] \end{aligned} \tag{23}$$

with

$$D_h u_1^n - b_0^{n-1,+} u_0^n + b_1^{n-1,-} u_1^n = 0, \qquad D_h u_N^n - b_N^{n-1,+} u_N^n + b_{N+1}^{n-1,-} u_{N+1}^n = 0$$

where

$$D_h \varphi_i = \frac{\varphi_i - \varphi_{i-1}}{h}, \quad D_h^* \varphi_i = \frac{\varphi_{i+1} - \varphi_i}{h}, \quad \Delta_h = D_h D_h^* = D_h^* D_h$$

From here, the authors prove conservation of mass for this scheme, uniqueness of a solution u_h^n, as well as error estimates and convergence analysis. Then, they extend to a 2D case, which is very similar so will be omitted for an attempt at brevity. They also prove conservation of mass and uniqueness of solutions for the 2D case.

4.3 Strehl Finite Element

Finite element methods are based on the weak or variational formulation of a problem, expressing unknowns as linear combinations of a finite basis. I have limited experience with finite element methods, so I read through a paper by Strehl et al. ([5]) that discusses an implementation of a FEM for the generic three-dimensional Keller-Segel model

$$\begin{aligned} u_t &= \nabla \cdot (D(u)\nabla u - A(u)B(v)C(\nabla v)) + q(u) \\ v_t &= d\Delta v - s(u)v + g(u)u \end{aligned} \tag{24}$$

As before, u is the cell density, and v is the chemoattractant concentration. To specify a specific model, choices of D, A, B, C, q, d, s, and g would be made. They proceed with the standard process for a Galerkin orthogonalization, and eventually arrive at two decoupled subproblems discretized in time:

$$[\mathbf{M}(1) + \Delta t\mathbf{L}(D(u^n)) - \Delta t\mathbf{K}(v^n)]u^{n+1} = \mathbf{M}(1)u^n + \Delta t\mathbf{q}^n \tag{25}$$

$$[\mathbf{M}(1) + \Delta t\mathbf{L}(d) - \Delta t\mathbf{M}(s(u^n))]v^{n+1} = \mathbf{M}(1)v^n + \Delta t\mathbf{M}(g(u^n))u^n \tag{26}$$

Strehl et al. describe $\mathbf{M}$ as the consistent mass matrix, $\mathbf{L}$ as the discrete diffusion operator, and $\mathbf{K}(v)$ as a discrete transport operator due to the chemotactic flux $A(u)B(v)C(\nabla v)$. We can define the entries of $\mathbf{M}$, $\mathbf{L}$, $\mathbf{K}(v)$, and $\mathbf{q}^n$ in terms of the finite element basis functions φ:

$$m_{ij}(\psi) = \int_\Omega \varphi_i\varphi_j\psi dx, \quad \psi \in \{1, s(u), g(u)\} \tag{27}$$

$$l_{ij}(\psi) = \int_\Omega \nabla\varphi_i \cdot \nabla\varphi_j\psi dx, \quad \psi \in \{D(u), d\} \tag{28}$$

$$k_{ij}(v) = \int_\Omega \nabla\varphi_i \cdot A(\varphi_j)B(v)C(\nabla v)dx \tag{29}$$

$$d_i^n = \int_\Omega \varphi_i q_j(u^n)dx \tag{30}$$

Through this formulation, one of their main aims was to create a positivity-preserving scheme. From equation (25), we can see that we must examine the matrix $\mathbf{A} = \mathbf{M} + \Delta t(\mathbf{L} - \mathbf{K})$ to make any claims about positivity. Strehl et al. establish a condition on this matrix that would ensure $\mathbf{A}^{-1} \geq 0$, and go on to prove that condition is satisfied, meaning this scheme is positivity-preserving.

They go on to perform numerical experiments with specific choices of the variables in (24) to prove the robustness of their model. Since the scheme was developed from the generic model, this framework can be used to explore a variety of three dimensional Keller-Segel models.

4.4 Implementations of the Finite Differences

Attempts to implement both my simple finite difference scheme and the Saito finite difference scheme were made, but without promising results were abandoned in the pursuit of other elements of this project.

Generally, the finite difference schemes translate extremely well to code, with only the boundary and initial conditions left to understand. For the minimal model and scheme (19), the boundary conditions can be taken from (5), ie no flux boundary conditions. The initial concentrations and other terms like diffusion and chemotactic sensitivity can be taken from [7]. For example, $u(x,0) = 1$, $v(x,0) = 1 + 0.1e^{-10x^2}$, $D_1 = 0.1$, $D_2 = 1$, and $\chi = 5.0$.

5 Discussion

Keller-Segel models have a rich history and are still an important part of biological and mathematical research today. This project gave an overview of Keller-Segel models from the initial derivation of the model in [1] to more current research of variations of the model. The Keller-Segel model not only allows mathematicians to model the behavior of things like bacteria, it also has fascinating blow-up behavior that is valuable to study on its own. Indeed, there are a number of papers that focus solely on the blow-up phenomenon, which was touched on in this project.

Future directions for this project could be to study more variations of the model, in particular to more fully explore the coupled Navier-Stokes models. Another avenue that would be worthwhile to explore more rigorously is numerical approximations to the model. Due to time constraints, I chose not to focus on the newer insights that can be offered through a numerical lens, but in recent years the Keller-Segel model has had some interesting developments numerically. Certainly future work on this project would include implementations of numerical schemes and an analysis of their accuracy and stability.

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A Appendix

A.1 Additional Details for Model Derivation

Definition A.1 (Fick's Law) *[Wikipedia] The movement of particles from high to low concentration (the diffusion flux J) is proportional to the concentration gradient ($\frac{d\varphi}{dx}$) of the particle.*

$$J = -D\frac{d\varphi}{dx}$$

Definition A.2 (Fourier's Law) *[Wikipedia] The rate of heat transfer through a material is proportional to the negative gradient in the temperature and to the area, at right angles to that gradient, through which the heat flows.*

$$q = -k\nabla T$$

A.2 Additional Details from Section 2.2

A.2.1 Lebesgue's Dominated Convergence Theorem

Theorem A.1 *(From [14]) Suppose that f_n is a sequence of measurable functions such that f_n pointwise converges to f almost everywhere as $n \to \infty$, ie that $\lim_{n\to\infty} f_n(x) = f(x)$ exists. Assume also that there is an integrable function g such that $|f_n| \leq g$ for all n. Then f and f_n are integrable and we have*

$$\int f d\mu = \lim_{n\to\infty} \int f_n d\mu = \int f d\mu$$

Why was this important? In our proof of Lemma 2.1, we have a series of functions φ_ε that converge to $f = |x|^2$ which is clearly bounded by an integrable function. Thus, we are able to make the final leap to the answer we want via the limit.

A.2.2 Hardy-Littlewood-Sobolev Inequality

Lemma A.2 *(From [3]) Let f be a non-negative function in $L^1(\mathbb{R}^2)$ such that $f \log f$ and $f \log(1 + |x|^2)$ belong to $L^1(\mathbb{R}^2)$. If $\int_{\mathbb{R}^2} f dx = M$, then*

$$\int_{\mathbb{R}^2} f \log f dx + \frac{2}{M} \iint_{\mathbb{R}^2\times\mathbb{R}^2} f(x)f(y) \log|x-y| dxdy \geq -C(M) \tag{31}$$

with $C(M) := M(1 + \log\pi - \log M)$.

It seems that most sources I read, ie [15] and [16] do not write the inequality in this same form, but I think some transformations, for instance via integration by parts, will result in the form above. For example, in [15] they write the inequality as

$$\int_{\mathbb{R}^2}\int_{\mathbb{R}^2} f(x)g(y)|x-y|^{-\lambda} dxdy \leq C(n,\lambda) \|f\|_p \|g\|_p$$

with a C involving the Gamma function.

This inequality is founded in analysis and specifically useful when discussing Sobolev spaces, so it is not something that I was able to fully understand. From my reading, I believe it often appears in applications of PDEs and physics, so it is not surprising that it appeared through the course of this project.

A.2.3 Proof of Lemma 2.5

Lemma A.3 *For any $w \in L^1_+(\mathbb{R}^2)$, if $\int_{\mathbb{R}^2} |x|^2 w dx$ and $\int_{\mathbb{R}^2} w \log w dx$ are bounded from above, then $w \log w$ is uniformly bounded in $L^\infty(\mathbb{R}^+_{loc}, L^1(\mathbb{R}^2))$ and*

$$\int_{\mathbb{R}^2} w|\log w|dx \leq \int_{\mathbb{R}^2} w\left(\log w + |x|^2\right)dx + 2\log(2\pi)\int_{\mathbb{R}^2} wdx + \frac{2}{e}$$

Proof: (From [3] Let $\bar{w} := w\mathbb{1}_{\{w\leq 1\}}$ and $m = \int_{\mathbb{R}^2} \bar{w}dx \leq M$. Then

$$\int_{\mathbb{R}^2} \bar{w}(\log \bar{w} + \frac{1}{2}|x|^2)dx = \int_{\mathbb{R}^2} W\log W d\mu - m\log(2\pi)$$

where $W := \bar{w}/\mu, d\mu(x) = \mu(x)dx$ and $\mu(x) = (2\pi)^{-1}e^{-|x|^2/2}$. By Jensen's inequality,

$$\int_{\mathbb{R}^2} W\log W d\mu \geq \left(\int_{\mathbb{R}^2} W d\mu\right)\log\left(\int_{\mathbb{R}^2} W d\mu\right) = m\log m,$$

$$\int_{\mathbb{R}^2} \bar{w}\log\bar{w}dx \geq m\log(\frac{m}{2\pi}) - \frac{1}{2}\int_{\mathbb{R}^2}|x|^2\bar{w}dx \geq -\frac{1}{e} - M\log(2\pi) - \frac{1}{2}\int_{\mathbb{R}^2}|x|^2\bar{w}dx$$

.

Using

$$\int_{\mathbb{R}^2} w|\log w|dx = \int_{\mathbb{R}^2} w\log w dx - 2\int_{\mathbb{R}^2}\bar{w}\log\bar{w}dx,$$

completes the proof.

A.3 Additional details from Section 4

A.3.1 Local Truncation Analysis of My Scheme

$$\begin{aligned}
\frac{u_i^{n+1}-u_i^n}{\Delta t} =& D_1\frac{u_{i-1}^n - 2u_i^n + u_{i+1}^n}{\Delta x^2} \\
&- \chi\left(\frac{u_{i+1}^n - u_{i-1}^n}{2\Delta x}\cdot\frac{v_{i+1}^n - v_{i-1}^n}{2\Delta x} + u_i^n\frac{v_{i-1}^n - 2v_i^n + v_{i+1}^n}{\Delta x^2}\right) \\
\frac{v_i^{n+1}-v_i^n}{\Delta t} =& D_2\frac{v_{i-1}^n - 2v_i^n + v_{i+1}^n}{\Delta x^2} + u_i^n - v_i^n
\end{aligned}$$

Let's Taylor expand the various terms

$$\begin{aligned}
u_i^n &= u \\
u_i^{n+1} &= u + ku_t + \frac{1}{2}k^2u_{tt} \\
u_{i+1}^n &= u + hu_x + \frac{1}{2}h^2u_{xx} + \frac{1}{6}h^3u_{xxx} + \frac{1}{24}h^4u_{xxxx} \\
u_{i-1}^n &= u - hu_x + \frac{1}{2}h^2u_{xx} - \frac{1}{6}h^3u_{xxx} + \frac{1}{24}h^4u_{xxxx}
\end{aligned}$$

We can see that the terms for v are the same. Then, after some cancellation the first equation becomes

$$\frac{1}{k}[ku_t + \frac{1}{2}k^2u_{tt}] = \frac{1}{h^2}[h^2u_{xx} + \frac{1}{12}h^4u_{xxxx}] - \frac{1}{2h}[2hu_x + \frac{1}{3}h^3u_{xxx}]\frac{1}{2h}[2hv_x + \frac{1}{3}h^3v_{xxx}] - \frac{1}{h^2}u[h^2v_xx + \frac{1}{12}h^4v_{xxxx}]$$

We can subtract away our original equation and we are left with

$$\tau = \frac{1}{2}ku_t - \frac{1}{12}h^2u_{xxxx} + \frac{1}{12}uh^4v_{xxxx} + O(h) \in O(k+h)$$

The second equation becomes

$$\frac{1}{k}[kv_{tt} + \frac{1}{2}k^2v_{tt}] = \frac{1}{h^2}[h^2v_{xx} + \frac{1}{12}h^4v_{xxxx}] + u - v$$

and once again we subtract away the original equation to be left with

$$\tau = \frac{1}{2}kv_{tt} - \frac{1}{12}h^2u_{xxxx} \in O(k+h^2)$$