EMERGENT PHENOMENA IN CLASSICAL AND QUANTUM SYSTEMS:
CELLULAR DYNAMICS IN \textit{E. coli}
AND
SPIN-POLARIZATION IN FERMI SUPERFLUIDS

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fulfillment of the requirements for the
degree of Doctor of Philosophy

by

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Science should be fun.
Preface

Emergence is a word used to describe how complex behavior can arise from a large collection of component entities obeying a simple set of rules. It is used to describe phenomena which cannot be predicted from understanding the simple interactions between individual components of the system, which arise because of large numbers of these components interact. A common example of emergent phenomena in classical systems is the shape of snowflakes. Simply knowing how ice forms when water freezes does not allow the prediction of the shapes of snowflakes. These beautifully complex patterns emerge from the collective behavior of many water molecules obeying the simple laws of physics. The phenomenon of superfluidity and superconductivity are examples from the quantum regime. Conway’s Game of Life is a popular example from computer science. In the course of my research these types of emergent phenomena have particularly intrigued me.

The first half of this dissertation will focus on understanding oscillations that emerge in a system of three proteins. These protein oscillations serve a specific function in the cell: determining the location of the division site in the bacterial cell. After giving a brief overview of this protein system, I will present my work studying the effect of dynamic cell geometry on these oscillations. This will begin with a description of models developed to describe elongation and division of bacteria, and discuss the effect of these models on the oscillations of this protein system. Then
I will discuss future experiments which can test some of the predictions which arise from this work.

In the second half of this dissertation, I will focus on understanding the emergent phenomena of superfluidity in the context of Fermi systems with arbitrary population imbalance. I will present a brief introduction to fermi-liquid theory and BCS superconductivity, which will provide a cursory background necessary for a discussion of strongly-correlated Fermi systems. Following this, I will describe the results of some analytic calculations in multi-species Fermi superfluids with arbitrary species population imbalance. Implications of these calculations will be discussed with an eye toward future experiments in a variety of systems.
CHAPTER 1

The Min-system in *Escherichia coli*

The Min-system in *E. coli* is a system of three proteins (*MinC*, *MinD*, and *MinE*) coded by the *minCDE* operon that plays a critical role locating the division site at the center of the cell. The discovery of self-organized spatio-temporal oscillations of proteins in *Escherichia coli* has led to a great deal of understanding about how bacteria divide. These emergent oscillations work in concert with other protein systems to direct the placement of the division site in the bacteria. In this chapter, I will review the essential biological processes behind these protein oscillations. A model which describes these oscillations will be reviewed to motivate my work toward understanding the effect of dynamic cell geometry on this protein system. Finally I will describe the numerical techniques used to perform calculations using this model, and describe how to interpret the results of these calculations.

Before beginning the discussion of the Min-system, I must make a brief comment about the notation employed throughout this dissertation to describe genes and proteins. Though notation varies in the literature, I will try to maintain the following notation throughout this work: Cell lines which are functional and found in the wild (i.e. in nature, not evolved in a laboratory) are labeled wild-type (wt) cells. Phenotypic mutants which lack or have some new characteristic differing from the (wt) cells
are identified by the gene responsible for the mutation. For example, a cell which has the mini-celling defect is identified $min^-$. The minus indicating the defect is associated with the damage of the $min$ gene. Genes associated with this defect are identified as $minA$, $minB$, etc. sequentially by alphabet as they are discovered. (e.g. $minA$ is the first gene identified associated with mini-celling, $minB$ the second, etc.) Upon identification of the gene products, the proteins are labeled in capital italic font (e.g. $MinA$ would be the protein coded by the $minA$ gene). Operons are genes which code for more than one protein. These are identified as any other gene, but all the proteins are included in the description (e.g. The $minCDE$ operon codes for three proteins, $MinC$, $MinD$, and $MinE$). In this work, $Min$-system or $Min$-proteins will be used to refer to the system of proteins responsible for division site selection: $MinC$, $MinD$, and $MinE$.

*E. coli* is a common gram-negative rod-shaped bacteria which is a well understood model system in genetics and molecular biology. A simplified illustration depicting the bacterium is shown in Figure 1.1 (taken from [1]). The structure of this bacterium is very simple: the contents of the cytoplasm are contained by the plasma membrane. The plasma membrane is encased by the rigid cell wall, which is made of murein (peptidoglycan), which gives the bacteria its shape. Murein is a macromolecule composed of subunits made of long glycan threads which are crosslinked by short peptide chains [2]. The cell wall is surrounded by another membrane which contains a variety of receptors that the bacteria uses to sense its environment, and the circular genome
Figure 1.1: Illustration of the basic geometry and cell components of *E. coli*.

lies approximately in the center cytoplasm.

The *Min*-system is one of many systems of proteins which work in concert to accomplish the process of cell division [3–5]. The focus of this work will be on the *Min*-system; though occasionally I will make connection with other protein systems. In Section 1.1, I will describe the biology and biochemistry of the *Min*-system as well as what is known experimentally about the spatio-temporal oscillations of these proteins. Section 1.2 will present in detail the current leading model which describes these oscillations. The results from calculations of (wt) cells using this model are discussed in Section 1.3. Finally, Section 1.4 will discuss open questions in the system which will be addressed by this work.
1.1 Biology and Biochemistry

In 1967, Adler’s discovery of a strain of *E. coli* which produces mini-cells opened the door to understanding cell division using genetic techniques. Mini-cells are non-viable cells which are devoid of nuclear material; they occur when the division site is not correctly localized to the center of the cell [6]. Though the mini-cells themselves are not viable, their parents are; the cells with nuclear material can still grow and divide. This was an important discovery, because it provided the first look at a heritable genetic defect in the cell division machinery.

Location of the division site is determined by the placement of the divisome. This is a “scaffold”-like structure composed of many different proteins organized around a ring of *FtsZ* oligimers, known as the *FtsZ*-ring, which supports the construction of the septum [5]. The septum is the new cell wall which is built to separate the parent cell into two daughters. Cells with the mini-celling defect may fail to correctly localize the *FtsZ*-ring at the center of the cell. Formation of an *FtsZ*-ring in the polar regions of the cell then leads to the creation of mini-cells [3].

Placement of the divisome is regulated by two different systems in *E. coli*. Nucleoid occlusion inhibits formation of the *FtsZ*-ring in regions of the cell where chromosomal material is present [4]. This prohibits the formation of an *FtsZ*-ring in the center of the cell until the fully replicated chromosome has separated. Three proteins: *MinC*, *MinD* and *MinE*, acting together, are responsible for inhibiting *FtsZ*-ring formation at the cell poles in *E. coli* [7]. When *MinC* is localized to the plasma membrane, it
prevents the formation of $FtsZ$-rings by inhibiting $FtsZ$ polymerization [8]. $MinC$ is localized to the plasma membrane by binding to $MinD$. $MinE$ binds to the $MinCD$-complex, releasing the $MinC$ to the cytoplasm and forming a $MinDE$-complex. It then catalyzes a reaction which releases the $MinDE$-complex from the plasma membrane and separates the two proteins, allowing them to diffuse in the cytoplasm [9]. These simple reactions result in the oscillation of these proteins from one end of the cell to the other. This oscillation ensures that the time averaged concentration of $MinC$ is minimized at the center of the cell and maximized at the cell poles, preventing polar $FtsZ$-ring formation [3].

Experimentally, visualization of these oscillations is accomplished by using green fluorescent protein (GFP) labels attached to particular proteins. These labeled proteins may then be observed in vivo using fluorescence microscopy (FM) and differential interference contrast microscopy (DIC) as shown in Figure 1.2. Figure 1.2(a), taken from [10], shows the oscillation of $MinD$-GFP in several cells which are growing and dividing normally. In this figure, frames which are labeled with letters are FM images, and frames labeled with primed letters are DIC images: Frame E shows a typical cell which has recently divided; Frame H shows a typical cell which is undergoing the process of elongation. Frame G shows a typical cell which is in the process of dividing (the septum which is clearly visible in the DIC image $G'$). Time is indicated in seconds in the figure. Figure 1.2(b), taken from [11], shows a series of much higher resolution FM images. Time progression is indicated by a number
on each frame in the figures. In this figure it is much more clear that the proteins are localizing on the plasma membrane at the poles (t=0) and diffusing through the cytoplasm (t=50).

Figure 1.2: MinD-GFP oscillations in different cells observed \textit{in vivo}. (a) FM and DIC images (b) FM images. Time is indicated in seconds.

Genetically, this system of proteins is remarkably well conserved across many species [4]. In bacteria where homologous systems are present, the protein system can function somewhat differently. For example, in \textit{Bacillus subtilis}, which has both MinC and MinD, MinE is missing; though MinC and MinD still play the same roles as they do in \textit{E.coli} (inhibiting FtsZ polymerization and localizing MinC to the plasma membrane, respectively) there are no oscillations [5]. Consequently modeling the function of the Min-system is important for understanding cell division. In the
next section, I will describe a model which captures the essential behavior of the
Min-protein oscillations in *E. coli*.

1.2 A Model for Oscillation

In this section, I will describe the model of Huang and Wingreen (HW-model), which has been a successful continuum reaction-diffusion model that captures many characteristics of the Min-system which have been observed experimentally [12]. Several other models have been put forth to describe the observed oscillations, with varying degrees of success. These models fall into two broad categories: continuum reaction diffusion models [13, 14], and stochastic models [15, 16]. Continuum reaction-diffusion models treat the proteins within the cell as concentrations that are continuous functions of both space and time. Stochastic models treat the proteins as individual localized entities, naturally incorporating spatial fluctuations due to low protein copy number. In my work investigating the effects of dynamic cell geometry, I have employed the HW-model because it only incorporates well established reactions and has been both qualitatively and quantitatively successful in a variety of geometries [12,17,18]. In the following subsections, I will describe this model in four parts: the cell geometry, the biochemistry, a mathematical description, and the numerical techniques used to solve the equations which describe this model.
1.2.1 A Model Cell Geometry

The shape of *E. coli* is effectively determined by the rigid cell wall, a large macromolecule known generically as peptidoglycan (murein). Several strains of *E. coli* with various cell geometries have been observed, such as spherical, and branched cells. These are associated with different defects in one of the protein systems responsible for incorporation of peptidoglycan into the existing cell wall. The cell geometry of (wt) cells is basically cylindrical, as can be seen in the DIC images in Figure 1.2. Mathematically, the cell geometry can then be approximated by cylinder of length $L$ with hemispherical endcaps of radius $R$.

1.2.2 Biochemistry of the HW-model

This model for the oscillations of the *Min*-system contains *MinD* and *MinE*: *MinC* is relegated to the role of a passive spectator during the oscillation. This description of the oscillation is consistent with experiments in various mutants which have shown that *MinC* is not necessary for the system to oscillate, but is necessary to prevent *FtsZ* polymerization [19]. The HW-model also assumes that the action of *MinC* in *FtsZ*-ring suppression is not relevant to the spatio-temporal dynamics observed in the *MinD-GFP* oscillation studies, which show that *Min*-protein oscillations are unaffected in short *FtsZ* mutants [20].

The model consists of a four step cycle of reactions, depicted in Figure 1.3 (reproduced from [12]). This cycle requires the input of energy in the form of adenosine
Figure 1.3: Schematic description of the reactions included in the HW-Model describing the cyclic interactions of two proteins with the cell membrane.

The tri-phosphate (ATP), which is hydrolyzed by MinD after catalysis by MinE. Chemically this set of reactions is described as follows:

**STEP 1:** **Cytoplasmic MinD-ATP may attach to the cell membrane.**

This process is characterized by a binding rate, which describes how quickly this occurs. In principle there is a finite binding rate for all of the proteins to bind to the plasma membrane. The binding rate for MinD MinE and MinD-ADP are taken to be zero, consistent with studies of protein binding to lipid vesicles *in vitro* [21]. Huang and Wingreen have confirmed that allowing binding of MinD-ADP to the membrane does not qualitatively affect the oscillations as long as the binding rate is small and the unbinding rate is large (via private communication). Evidence of polymerization of MinD in lipid vesicles and recently *in vivo* suggests that the binding of MinD-ATP is enhanced by the presence of MinD-ATP already on the membrane [22]. This is
included in the model by making the binding rate dependent upon the membrane-bound MinD-ATP concentration.

STEP 2: **Cytoplasmic MinE may bind to MinD-ATP to form a MinDE-ATP complex.** This complex is stable and does not dissociate from the membrane when ATP hydrolysis is suppressed [21]. Consequently, once the MinDE-complex has formed on the membrane, work must be done to release the proteins from the cell membrane. The energy for this work comes from the hydrolysis of ATP.

STEP 3: **MinE catalyzes the hydrolysis of ATP by MinD.** This results in the release of a phosphate ion, and the separation of MinE and MinD-ADP to diffuse freely in the cytoplasm [23]. In the absence of MinE, MinD and MinC accumulate uniformly on the membrane and do not oscillate [20]. This is where energy is used to do work in the system.

STEP 4: **MinD-ADP is recycled to MinD-ATP through nucleotide exchange.** This is where energy is put into the system to drive the oscillation. The model includes only single step nucleotide exchange, though it has been confirmed that two-step exchange does not qualitatively alter the results [12].

### 1.2.3 Mathematics of the HW-model

Each species of protein is described as by a concentration which is a function of both space and time. Five species concentrations are present in this model: $\rho_{D,ATP}$ and $\rho_{D,ADP}$ describe the cytoplasmic MinD-ATP and MinD-ADP concentrations respectively; $\rho_d$ describes the membrane bound MinD-ATP concentration; $\rho_E$ and $\rho_{de}$
describe the cytoplasmic MinE and membrane bound MinDE-ATP complex concentrations respectively. A mathematical model must describe both the transport and reaction processes.

The motion of the Min-proteins has been studied in vivo and characterized as diffusive transport. Consequently, transport is modeled as random diffusion characterized by a diffusion constant specific to each protein species. These diffusion constants are a measured input to the model, not a prediction. Cytoplasmic diffusion coefficients have been measured to be approximately two orders of magnitude greater than membrane diffusion coefficients for this system [24]. Because of the large disparity between diffusion in the cytoplasm and on the membrane, the membrane diffusion coefficients have been set to zero. ¹

The equation which describes diffusion has long been known:

$$\frac{\partial \rho_i(r, t)}{\partial t} = \mathcal{D}_i \nabla^2 \rho_i(r, t)$$

The rate of change of the concentration of a molecule is proportional to the gradient of the density. This equation describes a mass conserving process so that the integral over all space of the density is time independent. Since this model has five concentrations which need to be described, there will be five of these equations. Each species will diffuse independently with its own diffusion constant \( \mathcal{D}_i \) which characterizes the

¹Allowing membrane diffusion with appropriate diffusion coefficients has no qualitative effect on the results, but incurs significant additional computational cost (K.C. Huang, Private Communication).
rate of diffusion.

Adding reaction terms to the diffusion equation produces a reaction-diffusion equation:

$$\frac{\partial \rho_i(r,t)}{\partial t} = \mathcal{D}_i \nabla^2 \rho_i(r,t) + R1 + R2 + ...$$

The HW-model includes reactions in a manner that ensure mass conservation. This results in a set of five coupled reaction-diffusion equations (RDEs), with reactions illustrated in Figure 1.3 and summarized in Table 1.1. For the sake of clarity, reaction rates are enclosed in brackets $[k]$.

<table>
<thead>
<tr>
<th>Reaction</th>
<th>Description</th>
<th>Reaction-Diffusion Term</th>
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<tr>
<td>1</td>
<td>$MinD$-ATP binding to membrane</td>
<td>$[k_D]\rho_{D\cdot ATP} + [k_{dD} (\rho_d + \rho_{de})]\rho_{D\cdot ATP}$</td>
</tr>
<tr>
<td>2</td>
<td>$MinE$ binding $MinD$-ATP</td>
<td>$[k_E \rho_d] \rho_E$</td>
</tr>
<tr>
<td>3</td>
<td>$MinE$-induced ATP hydrolysis</td>
<td>$[k_{de}] \rho_{de}$</td>
</tr>
<tr>
<td>4</td>
<td>Nucleotide exchange</td>
<td>$[k_{ADP\rightarrow ATP}] \rho_{D\cdot ADP}$</td>
</tr>
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Table 1.1: Mathematical description of the reactions in the HW-Model. Reaction number corresponds to Figure 1.3.

**STEP 1:** Cytoplasmic $MinD$-ATP may attach to the cell membrane.

The concentration of cytoplasmic $MinD$-ATP ($\rho_{D\cdot ATP}$) is reduced via the reaction term:

$$R1 \equiv [k_D]\rho_{D\cdot ATP} + [k_{dD} (\rho_d + \rho_{de})]\rho_{D\cdot ATP} \quad (1.2)$$

The concentration of membrane-bound $MinD$-ATP ($\rho_d$) is increased by the same term. The first term describes $MinD$-ATP binding to the membrane independent
of the presence of other MinD-ATP. This is characterized by a rate parameter $k_D$. The second term describes additional MinD-ATP that binds to the membrane due to the presence of previously bound MinD-ATP. This rate is dependent on the concentration of two species: the membrane bound MinD-ATP ($\rho_d$) and the membrane bound MinDE-ATP complex ($\rho_de$). $k_{dD}$ is the rate parameter associated with this contribution.

**STEP 2:** Cytoplasmic MinE may bind to MinD-ATP to form a MinDE-ATP complex. The MinE that leaves the cytoplasm is described by the reaction term:

$$R2 \equiv [k_E\rho_d]\rho_E \quad (1.3)$$

In addition the amount of MinD-ATP on the membrane is reduced by the same amount as it is converted into the MinDE-ATP-complex. Here again, the rate is dependent on the concentration of MinD-ATP on the membrane ($\rho_d$), which introduces another non-linearity to the RDEs. The rate parameter for this reaction is $k_E$.

**STEP 3:** MinE catalyzes the hydrolysis of ATP by MinD. The concentration of both the cytoplasmic MinE and MinD-ADP increases via the reaction term:

$$R3 \equiv [k_{de}]\rho_{de} \quad (1.4)$$

This is a linear process, characterized by a rate constant $k_{de}$.

**STEP 4:** MinD-ADP is recycled to MinD-ATP through nucleotide exchange. MinD-ADP is converted to MinD-ATP; the cytoplasmic concentration of
MinD-ADP decreases via the reaction term:

\[ R4 \equiv [k_{\text{ADP} \rightarrow \text{ATP}}] \rho_{\text{D:ADP}} \] (1.5)

This is a linear approximation to a Michaelis-Menton process, which is justified by the small concentration of the MinD protein compared to the concentration of adenine triphosphate within the cell. Using this approximation introduces a single a rate constant \( k_{\text{ADP} \rightarrow \text{ATP}} \).

One can now form mathematical representation of the reactions shown in Figure 1.3. Adding these reaction terms in a mass preserving manner to the five diffusion equations, produces the set of coupled non-linear RDEs shown in Equation (1.6) [12]. The delta functions serve to force the reactions to occur on the cell membrane, which is located at the cell radius \( R \).

\[
\frac{\partial \rho_{\text{D:ATP}}}{\partial t} = \mathcal{D}_D \nabla^2 \rho_{\text{D:ATP}} + [k_{\text{ADP} \rightarrow \text{ATP}}] \rho_{\text{D:ADP}} - \delta(r - R) [k_D + k_{\text{dD}} (\rho_d + \rho_{de})] \rho_{\text{D:ATP}}
\] (1.6a)

\[
\frac{\partial \rho_{\text{D:ADP}}}{\partial t} = \mathcal{D}_D \nabla^2 \rho_{\text{D:ADP}} - [k_{\text{ADP} \rightarrow \text{ATP}}] \rho_{\text{D:ADP}} + \delta(r - R) [k_{\text{de}}] \rho_{de}
\] (1.6b)

\[
\frac{\partial \rho_E}{\partial t} = \mathcal{D}_E \nabla^2 \rho_E + \delta(r - R) \{ [k_{\text{de}}] \rho_{de} - [k_E \rho_d] \rho_E \}
\] (1.6c)

\[
\frac{\partial \rho_d}{\partial t} = - [k_E \rho_d] \rho_E(R) + \rho_{\text{D:ATP}}(R) [k_D + k_{\text{dD}} (\rho_d + \rho_{de})]
\] (1.6d)

\[
\frac{\partial \rho_{de}}{\partial t} = [k_E \rho_d] \rho_E(R) - [k_{\text{de}}] \rho_{de}
\] (1.6e)
This set of equations has seven parameters, summarized in Table 1.2, corresponding to various reaction parameters and diffusion coefficients. When available, experimentally measured parameters have been used and referenced. Parameters which have not been measured experimentally have been chosen to produce the correct (wt) behavior for the cell. There is considerable freedom in the choice for nucleotide exchange rate due to lack of system specific data; however nucleotide exchange rates are known to span several orders of magnitude in other systems [25]. The value chosen here is well within this established range, and small enough to allow for sufficient diffusion of MinD-ADP between binding events [12].

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Reaction</th>
<th>Value $^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$k_{ADP\rightarrow ATP}$</td>
<td>Nucleotide Exchange</td>
<td>3.5 $sec^{-1}$</td>
</tr>
<tr>
<td>$k_D$</td>
<td>Binding MinD-ATP to Membrane</td>
<td>0.1 $\mu m/sec$</td>
</tr>
<tr>
<td>$k_{dD}$</td>
<td>MinD-ATP Binding Enhancement</td>
<td>0.006 $\mu m^3/sec$</td>
</tr>
<tr>
<td>$k_E$</td>
<td>MinE Binding to membrane-bound MinD-ATP</td>
<td>0.25 $\mu m^3/sec$</td>
</tr>
<tr>
<td>$k_{de}$</td>
<td>MinE-induced ATP Hydrolysis</td>
<td>0.9 $sec^{-1}$</td>
</tr>
<tr>
<td>$D_D$</td>
<td>MinD Cytoplasmic Diffusion Coefficient</td>
<td>14.0 $\mu m^2/sec$ [24]</td>
</tr>
<tr>
<td>$D_E$</td>
<td>MinE Cytoplasmic Diffusion Coefficient</td>
<td>11.0 $\mu m^2/sec$ [24]</td>
</tr>
</tbody>
</table>

$^a$Values given are used in all calculations discussed in the text unless otherwise stated.

Table 1.2: HW-Model Parameters.

The set of RDEs in Equation (1.6) are nonlinear, which makes analytic solutions intractable, though they can be solved numerically. The non-linearity is important
in this system as they build in the feedback mechanisms which give rise to the oscillations which are the non-equilibrium steady state (NESS). The following subsection describes the numerical techniques employed to solve this system of coupled non-linear RDEs.

1.2.4 Numerical Techniques

This set of coupled non-linear RDEs must be solved using numerical techniques. To make this possible, the cell geometry is discretized in cylindrical coordinates and simplified as shown in 1.4. For simplicity of notation, the cell is taken as horizontal with the left edge placed at the origin (x=0). The size of the discretization is characterized by $dx$ along the length of the cell and $dr$ in the radial direction. In all cases described in this work, a discretization spacing of $dx = dr = 0.05\mu m$ has been used to balance accuracy with computational efficiency. This discretization neglects the hemispherical structure of the endcaps; however, it has been confirmed that modeling the full hemispherical endcaps does not qualitatively change any results from
the model (KC Huang, private communication). The choice of spacing was validated by varying the values $dx$ and $dr$ over two orders of magnitude in small cells. This spacing was then chosen to balance accuracy with computational efficiency.

Using this cell discretization, the system of RDEs described in Section 1.2.3 is solved using a four part algorithm at each time step:

1: **Changes in cell concentrations due to reactions are computed**

Starting from the initial concentrations $\rho(r,t)$ (which are stored in a matrix), the change in the concentration of each grid point due to each of the reactions described above is calculated, and the new concentrations are stored in an intermediate matrix.

2: **Crank-Nicholson Diffusion is performed in the x-direction**

Changes in the initial concentrations of each grid point due to diffusion in the x-direction are calculated using a Crank-Nicholson algorithm; the results are added to the new concentration matrix.

3: **Changes in cell concentrations due to reactions are computed**

Changes in concentrations due to reactions are recomputed using the intermediate concentration matrix. The new concentration is stored in a final matrix.

4: **Crank-Nicholson Diffusion is performed in the r-direction**

Changes due to diffusion in the r-direction are computed from the intermediate matrix and added to the final matrix. The calculation then proceeds to the next timestep.

The Crank-Nicholson diffusion algorithm is used to ensure second order accuracy
in time and stability for any choice of time discretization \( dt \) [26]. The changes due to reactions are computed twice to average over the two-step diffusion process. The results presented here employ a uniform initial distribution of proteins with random noise, with \( MinD \) found in the ATP form attached to the membrane and \( MinE \) floating freely in the cytoplasm. Other initial conditions have also been tested and the long-time concentration oscillations are not sensitive to this choice. Model parameters used in these calculations are specified in Table 1.2 above.

1.3 \( Min \)-protein Oscillations in Wild-type Cells

To analyze the effects of the addition of peptidoglycan to the cell wall on the \( Min \)-protein oscillations, comparisons must be made with the static geometry results. Here the results from calculations using static geometry are presented for comparison. An “adult” (wt) single cell is considered as a 4.0\( \mu m \) long cylinder with a 0.5\( \mu m \) radius. This corresponds to a 5.0\( \mu m \) cell with hemispherical endcaps, approximately the size of a typical \( E. coli \) cell before division.

The first surprising thing about this model is that given a random initial distribution, the proteins spontaneously start to oscillate. Figure 1.5 shows the total number of proteins in the cell as a function of position and time. The color bar indicates the total number of proteins at each position and time. The emergence of the oscillation can be seen clearly, with the establishment of \( MinD \) polar localization and a \( MinE \)-ring consistent with experimental observation [11]. In fixed-length cells a pole-to-pole oscillation is stable to arbitrarily long times. This is an example of self-organization
Figure 1.5: Wild-type oscillations of *MinD* and *MinE*. Colorbar indicates total number of proteins. Relaxation to the NESS oscillation occurs in less than 200 seconds. Note the formation of *MinD* polar regions and *MinE* rings.
from the initial random protein distribution to a non-equilibrium steady-state (NESS) oscillation.

By examining the normalized protein number, comparisons can be made between cells of different length, which will have different numbers of proteins. This is the number of proteins in each location (cytoplasm or membrane) of each species (\textit{MinD} or \textit{MinE}) divided by the total number of proteins of the appropriate species. This normalizes the total number of proteins of each species in the cell at any point in time to 1, allowing comparisons between cells of various lengths and protein concentrations. This is shown in Figure 1.6 where it can be seen that most of the proteins of both species are found on the plasma membrane. By construction, the model conserves the total number of proteins of each species, distributing them between the membrane and the cytoplasm. For this choice of parameters, almost all the \textit{MinE} is found attached to the membrane as the \textit{MinDE}-ATP complex. Additionally over 80\% of the total \textit{MinD} is bound to the membrane, either alone or as part of the \textit{MinDE}-ATP complex. After the system has reached the NESS, the normalized protein number in each location (cytoplasm or membrane) is characterized by a small oscillation about a fixed value.

The normalized protein number in the left side of the cell is shown in Figure 1.7(a). The structure within the oscillation is due to the dynamics of the \textit{MinE}-ring which stalls in the polar regions before dispersing and reforming at the cell midline after removing the \textit{MinD} from the plasma membrane of the cell poles. When proteins are
Figure 1.6: Normalized protein number by location in a 4μm (wt) cell, showing small oscillations about a fixed value in NESS.

not allowed to bind to the endcap, this feature is much less pronounced, indicating that the endcaps act as a sink for MinD. In Figure 1.7(b), the MinD oscillation can be clearly seen to be symmetric between both sides of the cell; this is true for the MinE oscillation as well. The system reaches the NESS in approximately 200 seconds, when the oscillation has reached its maximum amplitude. In fixed length cells, once the system has reached a single pole-to-pole oscillation, the NESS oscillation is very stable. The initial relaxation to the NESS was carefully reviewed and alternative initial conditions do not qualitatively change the oscillations observed in the NESS.
Figure 1.7: Normalized distribution of proteins in left half of the cell (a) and $MinD$ in both halves of the cell (b). This shows a symmetric oscillation in a fixed-length 4$\mu m$ (wt) cell.

1.4 Open Questions

There are several open questions which are actively evolving in the literature. What is the effect of dynamic cell geometry on the oscillations which are described by the HW-model? What are the effects of cell division on the oscillations? Do the proteins partition correctly? What are the effects of cell growth on the oscillations? Can the transition between a single pole-to-pole oscillation and multi-node oscillations in filamentous cells be described by this model? In the following chapter, I will construct separate models for elongation and division and examine the effect predicted by these models both independently as well as coupled, on the oscillations described by the HW-model.
CHAPTER 2

Dynamic Geometry

The primary focus of theoretical work in modeling the Min-system has been to establish and characterize the oscillations of the proteins within biologically relevant parameters. Until now theoretical models have considered exclusively fixed cell geometry, typically cylindrical. Recently, other geometries such as round and branched cells have been considered, motivated by *in vivo* studies of Min-protein oscillations in various mutant strains [17,18]. In reality, *E. coli* is a living organism which grows, divides, and reacts to changing environmental conditions. Consequently, the geometry of a living *E. coli* cell must changes due to various processes including elongation, division, osmotic swelling or contraction, lysis, etc. Cell growth by elongation and cell division are two processes which are a natural part of the life cycle of the bacteria. Most other processes are physiological responses to changing environmental conditions, which can be avoided by carefully regulating the environment in which the bacterium is grown.

The phenomenon of Min-protein oscillations is critical for determining proper placement of the division site. Disruption of these oscillations in *E. coli* will render the cell incapable of correctly placing the division site at the cell midline, resulting
in the formation of mini-cells. The absence of experimental observation of mini-cell formation in (wt) cells suggests that the functioning of the Min-system is not disrupted by the processes of cell growth and division, though to date there have been no experiments which have focused on these effects in relation to Min-protein oscillations. Therefore, a successful model of these oscillations should be robust to changes in geometry which are part of the natural life cycle of the bacterium.

In this chapter, I will present two different models: one describing elongation, and one describing cell division; then I will explore them in the context of the HW-model for Min-protein oscillations. I will show that this model offers an incomplete description of the oscillation in the presence of dynamic cell geometry. Section 2.1 will describe a model of elongation and discuss the effect of this model on oscillations described by the HW-model. I will show that this model correctly describes both (wt) and filamentous (FtsZ−) mutants, predicting the emergence of multinode oscillations in filamentous cells. Section 2.2 presents an “aperture” model of cell division, and applies this model to the oscillations of the Min-system. I will discuss the problem of correctly partitioning Min-proteins into daughter cells, and show that this model alone fails to solve the partitioning problem. Finally, since real cells both elongate and divide, Section 2.3 will describe coupling the models from Sections 2.1 and 2.2 and their combined effect on oscillations of the HW-model.

Elongation and division in E. coli are both the result of the incorporation of murein, also known as peptidoglycan, to the existing cell wall. The insertion of
murein during elongation requires a different system of proteins than the assembly of the septum during cell division [27]. For this reason, separate models are constructed to describe elongation and division. Figure 2.1 (reproduced from [2]) illustrates the two processes. During elongation, murein is added to the cylindrical part of the cell wall in a diffuse manner. Septum formation occurs in a localized manner at the division site.

Figure 2.1: Schematic depiction of the addition of murein to the cell wall using two separate protein systems. Elongation is a diffuse process which takes place across the entire length of the cell. Division is a targeted process which is localized to the center of the cell in (wt) bacteria.
2.1 Elongation

Murein synthesis and assembly has been well studied on a molecular level, and has been modeled theoretically since the 1970s [28]. In this section, I will present a coarse-grained model of elongation based on the diffuse insertion of murein into the existing cell wall. Recently the Feingold group made detailed measurements of the growth curves for individual bacteria (reproduced in Figure 2.2) [29]. This provides strong support for the assumption of a linear growth rate, i.e. $\frac{dL}{dt}$ is constant.

Figure 2.2: Growth curve for a single bacteria measured in the Feingold group. Two distinct linear growth regimes exist: the first is purely elongation, and the second is both elongation and division.

However, this is not the only consideration for a model of elongation. In preparation for division, the cell creates an identical copy of itself i.e. the amount of DNA and proteins within the cell doubles. A proper model of elongation must capture this
effect as well. Experimental observations of fluorescence intensity in GFP-oscillation
suggest that there are not abrupt changes in cellular Min-protein concentrations,
though concentration does vary spatially throughout the cell. Theoretically, it has
been shown that varying the relative concentrations of MinD and MinE within the
cell has a dramatic effect on the oscillations of the HW-model [12]. Therefore, in
this work, I will assume that the number of proteins of each species in the model are
produced at a constant rate which maintains the average number of proteins per unit
length as a constant, i.e. \( \frac{N}{L} \) is constant.

Computationally, cell growth is modeled by inserting small slices of volume of
width \( dx \) at random places on both sides of the cell midline, to ensure symmetric
growth. Protein concentrations within this volume slice are determined by averaging
the concentrations in the neighboring slices. Then the protein concentrations in the
entire cell are rescaled to maintain a constant average concentration per unit length
in the cell. This accounts for the average production of additional protein of each
species within the cell as it grows. The cellular growth rate is a parameter in the
model, which is chosen consistent with physical growth rates. All the results reported
here use a growth rate of \( \frac{dL}{dt} = \frac{1}{600} \mu m/sec \), corresponding to typical exponential growth
conditions where the cells are doubling every twenty minutes. Calculations are then
performed using the algorithm described in Section 1.2.4 with initial random concen-
tration distributions of MinD-ATP on the membrane and MinE in the cytoplasm.
The cell is allowed to come to a non-equilibrium steady state (NESS) characterized
by the oscillation period, and then allowed to grow.

2.1.1 Normal Growth

The first test of this model of elongation is whether or not it describes (wt) bacteria undergoing normal growth. For this calculation, the initial state is chosen to be a cell of $2\mu m$ length, corresponding to a freshly divided cell. Model parameters are from Table 1.2. After allowing the cell to reach a NESS (200 seconds for the 2 micron cell), it is made to grow to $4\mu m$ using the experimental growth rate. Figure 2.3 shows the total protein number as a function of position and time.

![Figure 2.3: Protein numbers in a (wt) cell undergoing normal growth from 2-4µm.](image)

As in the fixed-length example above, the number of proteins in the cytoplasm are small compared to the number bound to the membrane as shown in Figure 2.4. In this figure the normalized protein number on the membrane and in the cytoplasm
are plotted as functions of time for each protein species. The total normalized protein number remains 1 even though additional proteins are being added to the cell as it grows. As the cell grows, the amplitude of the oscillations of the number of proteins on the membrane and in the cytoplasm about their average value shows a very slight increase. The oscillation period shows a slight increase with the length of the cell, consistent with calculations in fixed-length cells of similar length [12].

Figure 2.4: Normalized protein number by species and location during (wt) cell growth from 2-4 µm.

To examine the effect of cell growth on the protein oscillations, consider Figure 2.5(a) showing the normalized protein number of each species in the left half of the cell. The NESS is characterized by a stable pole-to-pole oscillation. Figure 2.5(b) shows a very slight increase in the amplitude of the MinD oscillation which begins at $t = 260$ seconds. The MinE oscillation exhibits a slight decrease in the amplitude
of the pole-to-pole oscillation at the same point. Since the amplitude and period of the oscillations in fixed-length cells are functions of the length of the cell, this is the expected behavior. As the cell grows, it evolves from the NESS of the 2µm length to that of the 4µm length in a continuous manner.

![Graphs showing protein dynamics](image)

(a) Protein species in the left side of (wt) cell  (b) MinD in each side of the (wt) cell

Figure 2.5: Distribution of proteins in the left half of the cell (a) and MinD protein in both sides of the cell (b). Data is for a (wt) cell undergoing growth from 2-4µm.

Varying the model parameters appearing in Table 1.2 by ±10% does not qualitatively affect the behavior of the model. Quantitatively, varying these parameters changes the oscillation period consistent with results published in the literature [12]. From this two things can be concluded: first, the oscillations are not sensitive to the growth of the cell on these length scales, and second, the results from calculations using this elongation model are not qualitatively sensitive to the assumptions of a linear growth rate and the average production of new protein. Cells undergoing normal growth have concentration profiles which are qualitatively similar to fixed length cells of similar length.
2.1.2 Filamentous Mutants

Filamentous (FtsZ\textsuperscript{−}) mutants are cells which do not form FtsZ-rings, and consequently do not divide. These cells grow continuously and can reach extraordinary lengths up to ten times the length of a (wt) bacterium. Min-protein oscillations have been observed in filamentous mutants, and they have been seen to exhibit both multi-node standing wave and traveling wave oscillation patterns [10]. These mutants provide an excellent system for testing a model of elongation. In this subsection, I will investigate the elongation model in the context of filamentous mutants such as those shown in Figure 2.6 (taken from [10]). This figure depicts bi-nodal oscillations in short filaments and multi-node oscillations in longer cells. A model for elongation must be able to produce these multi-node patterns.

Fixed-length cells which have lengths greater than approximately 8\(\mu m\) form multi-node standing wave patterns. It has been argued that these patterns are consistent with experimental observation [12]. Figure 2.7, shows MinD and MinE protein numbers in a 20\(\mu m\) fixed-length cell; the data are truncated at 4000 seconds for clarity. Though pattern formation occurs in the first 200 seconds, similar to the time necessary to establish pole-to-pole oscillations in wild-type cells, the multi-nodal pattern is unstable, and decays to a single pole-to-pole oscillation at very long times. Examining this in more detail, Figure 2.8 shows both the short (a), intermediate (b), and long (c) time behavior of the oscillation in the 20\(\mu m\) cell. For short times, the appearance of a multi-node oscillation then evolves to a traveling wave at intermediate times. At
Figure 2.6: FM visualization of $GFP$-$MinD$ oscillations in filamentous $FtsZ^{-}$ mutants taken. Scale bar is 2.5$\mu m$ in A and 5.0$\mu m$ in B. B’ is DIC image. Times are given in seconds.
long times, the pole-to-pole oscillation has formed and the cell has transitioned to the NESS.

Figure 2.7: Relaxation to the NESS pole-to-pole oscillation for a 20µm fixed-length cell. Colorbar indicates number of proteins.

These long-time calculations show that fixed-length cells which exhibit multi-node standing wave or traveling wave patterns are not in the non-equilibrium steady state. For time scales on the order of several thousand seconds for “short” filaments to several tens of thousands of seconds in “long” filaments, these multi-nodal patterns all relax into single pole-to-pole oscillations corresponding to the NESS of the system. The relaxation time is dependent on the length, so future experiments which can arrest the growth of filamentous cells may be able to resolve whether or not this relaxation time is physically meaningful.

Figure 2.9 shows the normalized protein number by side of the cell for a 20µm
Figure 2.8: Behavior of fixed-length 20µm cell at various time scales, showing transition to the NESS pole-to-pole oscillation. Colorbar indicates number of proteins.
fixed length cell. At short times, there is no pole-to-pole oscillation. One can observe a transition from the chaotic short-time behavior to a pole to pole oscillation at approximately 2300 seconds. Referring to Figure 2.7, this occurs when the last multi-node coalesces at the left pole of the bacterium. Though there is still some noise in the actual protein distribution (which decays away at longer times), the pole-to-pole oscillation is quite stable. The structure in the MinE oscillations in fixed-length cells persists for much longer times than in the MinD oscillations (data not shown). 

![Graph showing normalized MinD and MinE protein numbers](image.png)

Figure 2.9: Normalized MinD and MinE protein numbers in the left side of a 20µm fixed-length cell. The NESS pole-to-pole oscillation that emerges from the chaos of the short time behavior is quite stable in the long time regime.

This suggests that the multi-nodal structure which is present in filamentous cells is a consequence of a transition out of the NESS. To test this, the HW-model was implemented in a cell which is allowed to grow from 2µm to 10µm. The results of this
calculation are shown in Figure 2.10, where the transition to a multi-node oscillation occurs as the cell grows longer than $7 \mu m$.

Figure 2.11 shows the normalized number of proteins in the left half of a cell growing from 2-10$\mu m$ in various time regimes. The $MinE$ oscillation starts to develop additional structure long before additional structure appears in the $MinD$ oscillation, which can be seen in 2.11(a). 2.11(b) highlights the transition from a single pole-to-pole oscillation to a bi-nodal structure. This transition occurs consistently at $7 \mu m$, when the length of the cell has become long enough that $MinD$ starts to collect at the cell midline. In the long time regime 2.11(c), the cell exhibits a multi-node oscillation which is qualitatively quite different from the short-time multi-node behavior of fixed-length cells. The short-time behavior of fixed-length cells exhibit chaotic multi-node oscillations, with no well defined pole-to-pole oscillation. In the growing cell, the multi-node oscillations emerge in a manner which simply adds additional structure to the the pole-to-pole oscillation.

In this section, I have presented a coarse-grained model of cellular growth manifested as elongation of the cylindrical part of the cell. This model was investigated in combination with the HW-model of $Min$-protein oscillations. The model correctly describes the elongation of (wt) cells undergoing normal growth. Further, the model qualitatively describes the multi-nodal oscillation of the $Min$-proteins in $FtsZ^-$ mutants. Finally model predicts a transition out of the NESS pole-to-pole oscillation in filamentous mutants that occurs around $L=7 \mu m$. 
Figure 2.10: Protein numbers in a cell growing from 2-10µm. (a) Total number of proteins for the entirety of the growth regime. (b) Highlights the transition from pole-to-pole to bi-nodal oscillation. Colorbar indicates number of proteins.
Figure 2.11: Normalized protein numbers in left side of a filamentous cell undergoing linear growth from 2-10\(\mu\)m in various time regimes. (b) Highlights the transition to multi-node oscillations.
2.2 Division

In this section, I will construct a geometric model for cell division based on recent experiments, and examine this model for cell division in the conjunction with the HW-model describing Min-protein oscillations. The process of cell division occurs through the construction of the septum which separates the two daughter cells. This forms two new hemispherical endcaps for the two daughter cells. The septum is localized to the center of the cell through the action of nucleoid occlusion and the oscillations of the Min-proteins [7] [30] [31]. A suite of filamentous temperature-sensitive gene products (FtsZ, FtsA, FtsB, FtsQ, etc.) organize in a well described order to form the divisome around the FtsZ-ring. Finally a signal causes the FtsZ-ring to shrink as peptidoglycan is added, forming the septum which will become the new endcaps as the new daughter cells separate [32].

Recently, the Feingold group has combined high-resolution phase-contrast microscopy measurements with fluorescence images of the cell membrane during growth and division to obtain very accurate measurements of cell wall growth during septation. Using the cell geometry shown in Figure 2.12 (reproduced from [29]), they find that the ratio of the radius of the opening between the two daughters during the process of constriction to the radius of the cell can be fit to the following expression:

\[ W \equiv \frac{r}{R} = \sqrt{1 - \left(\frac{t - \tau_c}{\tau_g - \tau_c}\right)^2 } \]  

(2.1)

where \( \tau_c \) is the time when the formation of the septum starts and \( \tau_g \) is the generation time. After averaging their data from several bacteria from different populations, they
determine best estimates of $\tau_g = 22.8 \pm 1.3$ minutes, and $\tau_c = 10.6 \pm 1.1$ minutes.

Figure 2.12: Model cell geometry during septation.

Min-protein oscillations are only coupled to the cell geometry through the boundary conditions in the HW-model. The purely geometrical construction of Reshes and colleagues fits the observed growth dynamics of the septum. Division will therefore be modeled in a manner which incorporates these ideas using an “aperture” model. Computationally, the endcaps have been approximated as circular disks as described in Section 1.2.4. Consequently, in this model, the geometry of the nascent septum will be approximated in the same manner. The nascent septum will be modeled as an impenetrable circular barrier with a circular opening which is closed radially inward at the cell midline, analogous to the closing of a camera aperture. Constriction is modeled so that the radius of the opening between the cells fits Equation (2.1) with the parameters $\tau_c$ and $\tau_g$ discussed above. Min-proteins in the cell are allowed to bind to the newly formed septum as it is constricting, and comparisons are made with the same system when proteins are not allowed to bind to the septum.
2.2.1 Division of Fixed-Length Wild-type Cells

To understand the effects of cell division on the oscillations, consider first Figure 2.13. This figure shows the total number of proteins of each species in the cell as a function of position and time for a typical (wt) dividing geometry. In (a) the oscillating proteins are allowed to bind to the septum as it is forming, in (b) they are not. Both cells begin from the same initial random protein distribution. The onset of division begins at \( t = 200 \) seconds and the cell has completed division at \( t = 740 \) seconds.

![Figure 2.13: Total protein count by species for a 4 \( \mu m \) fixed-length cell undergoing division.](image)

(a) Binding to septum  
(b) No binding to septum

In Figure 2.13(a) notice that the oscillations in the daughter cell are symmetric. In Figure 2.13(b), the absence of protein binding to the nascent septum leads to a distinct asymmetry in the protein oscillation of the daughter cells. This is inconsistent with experiments, which have not reported such an asymmetry in oscillations of daughter cells. From this result it is reasonable to conclude that Min-proteins binding to the
nascent septum as the cell divides allows for symmetric oscillation in the daughter cells.

It is clear from 2.13 that after division is complete most of the proteins are trapped in the right daughter cell. Further, there are no oscillation at all in the left daughter, which is not observed in (wt) cells. This is known as the partitioning problem: how does the cell arrange to have equal concentrations of both Min-proteins in both daughters? This problem was recently addressed in the literature for the first time [33] [34]. Sengupta and Rutenberg have shown that it is possible for the cell to divide with both daughters exhibiting oscillations. They speculated that coupling the onset of division to a particular time in the oscillation cycle could solve the problem producing oscillations in both daughter cells.

Figure 2.14: Daughter concentrations (from a 4µm parent) as a function of division start time.
I tested this hypothesis by varying the time for the onset of division in the cell. The result of this calculation is shown in Figure 2.14. Here one can see the final total normalized protein number in each daughter cell at the completion of septation as a function of the time at which division is started. Equal partitioning would be a value of 0.5. Though there are times during the oscillation cycle for MinD and MinE where the number of each respective protein in both daughters is equal, these times are not the same for both protein species. By comparing the final daughter concentrations in this figure to the results of Sengupta and Rutenberg, one can see that though it is possible to partition two oscillating daughters, there is a significant disparity in the number of proteins in each daughter. This will have the following experimental consequence: one daughter would glow brighter than the other in GFP-oscillation experiments. The absence of this observation in (wt) cells suggests that some other mechanism is at work to solve the partitioning problem [10] [11] [35].

2.3 Coupled Elongation and Division

Recent data have indicated that septation begins much earlier in the cell cycle while the cell is still undergoing lateral growth [29]. Previous investigations of cellular division have considered the cell to be of fixed length during septation, and noted that there is a significant problem correctly partitioning the oscillating proteins [33] [34]. In Section 2.2 it was shown that coupling the onset of cell division to one point in the oscillation cycle does not solve this problem. In this section, I test the hypothesis that coupling growth to division will result in the correct partitioning of the proteins.
between the daughter cell. Results of calculations which couple the geometric models of elongation and division, described in Section 2.1 and Section 2.2), respectively.

Figure 2.15: Combined effect of elongation and division on a (wt) cell which grows from 2-4\(\mu m\) and then divides.

In Section 2.1 it was shown that (wt) elongation caused a small perturbation to the NESS. Is this perturbation enough to cause the proteins to correctly partition? Figure 2.15 shows the total protein numbers of each species for a cell which grows from 2-4\(\mu m\) and divides. The results are qualitatively similar to those of division in fixed length cells. Though there is an oscillation in both daughter cells shown in the figure, the left daughter has considerably more protein than the right. The oscillations in both daughters are an artifact of the time chosen to begin division.

This can be seen quantitatively in Figure 2.16(a), which shows the normalized protein number in the left half of the cell for each species for both short times and
Figure 2.16: Protein numbers in left half of a (wt) cell undergoing both growth from 2-4µm and division.

times near the completion of cell division. Figure 2.16(a) shows that there is little effect on the oscillation at early times due to elongation, though there is a small disturbance in the NESS. As in the fixed-length case, there is no time at which both species of proteins are equally divided between the two daughters. At long times Figure 2.16(b) shows that the MinE oscillation has developed a small feature to the oscillation. However, that small feature at the extreme of the oscillation is related to protein binding on the endcaps and does not provide a mechanism for proper partitioning. From this figure, it is clear that even with both elongation and division, there is no point in the oscillation cycle where both proteins are equally partitioned. Therefore coupling the onset of division to a point in the oscillation cycle won’t solve the partition problem with both elongation and division.

Further, in this figure, it is clear that the left daughter has over 60% of the MinD and almost 75% of the MinE. This is a significant protein asymmetry which would
be readily visible in GFP-oscillation experiments. From these results, I conclude that combining elongation and septation does not solve the partitioning problem even when division is coupled to a particular phase of the oscillation.
CHAPTER 3

Min System - Summary and Conclusions

I have studied the effect of dynamic cell geometry on the spatio-temporal oscillations of the Min-system of proteins in Escherichia coli. Results have presented from three separate studies of the effects of elongation, division and coupled elongation and division on the HW-model of Min-protein oscillations. The process of elongation was studied using a linear growth model which was developed based on experimental observations. Cell division was studied with an “aperture” model which was developed based upon recent observations of septal growth. This model was investigated both with and without protein binding to the septum. These two models were then coupled to try to correctly describe the behavior of these oscillations during the cell cycle of wild-type bacteria. The results of these studies are summarized below.

The linear growth model presented here correctly captures the behavior of (wt) cell growth. The process of elongation is a small perturbation on (wt) cells, and the oscillation of the The HW-model of Min-protein oscillations is robust against this small perturbation. This is in good agreement with the FM studies of GFP oscillations which have been reported [10,20].

A diffuse linear growth model predicts a transition from pole-to-pole oscillations to multi-node oscillations. In the study of filamentous mutants,
the emergence of multi-node *Min*-protein oscillations was shown to be a consequence of a transition out of a nonequilibrium steady-state (NESS). This transition occurs consistently when the cell grows to lengths greater than 7μm. This work has shown that the multinode oscillation which emerges does not exhibit the chaotic behavior shown in fixed-length cells which have not reached the NESS. Though there is a multi-node structure to the oscillation, it still manages to move a significant portion of the proteins from one side of the cell to the other with a well defined frequency.

The “aperture” model of cell division presented here suggests that septal protein binding is necessary for symmetric oscillations in daughter cells. During cell division, protein binding to the septum prevents asymmetrical oscillation in daughter cells. Experimentally, asymmetrical oscillations have not been reported in (wt) cells. It is reasonable to conclude that *Min*-proteins bind to the septum during cell division.

The “aperture” model fails to solve the partitioning problem. It has been shown that the HW-model does not correctly partition both the *MinD* and *MinE* proteins equally between the two daughters using the “aperture” model developed here. Experimentally, non-oscillating (wt) daughters have not been reported in the literature.

Synchronizing division with the oscillation cycle produces daughters which both oscillate. Others have hypothesized that some mechanism for synchronizing the oscillations with the onset of cell division could result in daughter cells
which both oscillate [34]. This work has shown that this is possible; however, the resulting daughters have significantly different protein concentrations which would be readily visible in GFP-oscillation experiments. This concentration asymmetry in daughter cells has not been reported in (wt) cells, though it has not been thoroughly investigated.

It is clear that there is still work to be done to solve the partitioning problem. One avenue to pursue to address this problem is to consider some of the problems with the HW-model. The model is a mean-field description of the system: proteins are treated as concentrations that may be arbitrarily diffuse. In this model, the concentration associated with a single protein molecule can be spread over several discrete sites in the computational geometry. That is to say, that some parts of the cell there can be a finite concentration which is much less that the concentration associated with a single molecule. This smoothes out effects due to the spatial locations of individual proteins within the cell. It also masks some interaction dynamics, as individual proteins do not have to find each other to interact. These problems have been addressed in stochastic implementations of the HW-model, and have been shown to be important for describing certain mutant phenotypes [15,36].

The oscillation model, by construction, conserves total protein number. Non-mass conserving interactions are not considered, and may be important within the cell. This is especially unrealistic when trying to describe (wt) cells which grow. Equally, it may be important when the cell’s environment changes, for example when
the bacterium runs out of food. As the cell grows, proteins are being manufactured and recycled. To date no model has been put forward that allows for fluctuations in the number of proteins. Stochastic models have considered spatial fluctuations, so particle number fluctuations may be a natural thing to investigate in the context of cell growth and division.

There are many other avenues to extend and expand upon this work: By focusing on changes in the cell geometry which are a result of the life cycle of the bacterium, I have neglected other processes which are responses to changing environmental conditions. There is some evidence for fluctuations in the size of the cell radius in response to environmental stimulus. It would be interesting to investigate the effects of radial perturbations on these protein oscillations. Another direction could be applying these growth models to stochastic systems. This would discover if cell growth has an effect on Min-protein oscillations in mutants which are especially sensitive to noise in the oscillation. There is some recent evidence for helical MinD-ATP polymerization [22] which has been included in some models [16, 37]. The HW-model does not include polymer dynamics, which may be involved in correctly partitioning the proteins. In conclusion, the oscillations Min-system of proteins is a beautiful example of emergent phenomena about which much can still be learned.
CHAPTER 4

An Overview of Superfluidity in Fermi Systems

The superfluid transition is an example of emergent behavior in quantum systems whereby the system enters a state which has zero viscosity. In charged Fermi systems, this phenomena is known as superconductivity because charged particles in a superfluid state flow with no resistance. The first microscopic theory was described by Bardeen, Cooper and Schrieffer \[38\]. Uncharged Fermi systems such as He\(^3\) may undergo a superfluid transition to a state which flows with zero viscosity. The zero field phase diagram for He\(^3\) is illustrated in Figure 4.1 reproduced from \[39\]. This shows a rich structure in the superfluid state, with multiple phases (A-phase, B-phase). In all superfluid systems, flow without resistance arises due to collective effects in these many-body systems. These collective effects are result of fluctuations in the number of fermions in the system which are purely quantum mechanical in nature and have no classical analogue. The work in the subsequent chapters is motivated by the recent surge of interest in superfluidity in exotic Fermi systems, ranging from quark matter to systems of ultra-cold atomic fermions.

Superfluidity in quark matter systems has become a hot topic in recent years \[40\]. Figure 4.2 (adapted from \[41\]) shows a qualitative rendering of the QCD phase diagram as a function of temperature and baryon chemical potential. A variety of
Figure 4.1: Zero field phase diagram for He\textsuperscript{3} depicting the Normal state, the Superfluid A and B phases, and Solid phase.

interesting phases are depicted, from a Quark-Gluon Plasma (QGP) state which has been the focus of some attention at the Relativistic Heavy Ion Collider [41], to color superconducting states which are of particular interest in the study of neutron stars [42].

Recent experiments in cold atoms systems provide a rich experimental playground for testing theories attempting to explain superfluidity in Fermi systems. The discovery of Feshbach resonances, combined with the ability to adjust the population of hyperfine states, as well a controlling interparticle interactions, has led to the ability to produce imbalanced populations in s-wave superfluids [43]. These experimental techniques have opened the door to understanding population imbalanced Fermi systems, further motivating this work. Additionally, this work provides a framework to begin a discussion of superfluidity in arbitrarily spin-polarized He\textsuperscript{3}. In this system
Figure 4.2: QCD Phase diagram

however, it is known that additional effects such as spin-orbit coupling are important for describing both normal state and superfluid properties. These effects can be included in the analysis presented here, but are beyond the scope of this work.

The remainder of this chapter will discuss background material from Fermi-Liquid theory, basic BCS pairing phenomena, Bogoliubov equations and some elementary results from the liquid He^3 system. I will also give a brief overview of the elegant d-vector and D-matrix formalisms for describing the states of Fermi superfluids. In the subsequent chapter, I will describe how to consider superfluidity in Fermi systems with arbitrary population imbalance. This population imbalance can be quasi-spin in the case of cold atoms systems such as Li^6 or K^{40}, or it could be color imbalance in the case of a quark system. I will show that such systems exhibit a variety of interesting properties associated with population imbalance in both the singlet and
triple pairing channels. Finally the effects of anisotropic spin-dependent interactions in these systems will be described. This is done with an eye toward understanding superfluidity in cold atoms systems, but the formalism is general and applicable to more exotic systems such as those discussed earlier. Throughout this work, I will consider a system with unit volume, and set $\hbar = 1$.

4.1 Fermi-Liquid Theory

Landau’s Fermi-Liquid theory is the cornerstone for understanding systems of interacting fermions. The core idea behind this theory is that we can understand most of the physics by understanding the free Fermi gas, and then quasi-perturbatively add the effects of interactions. Below I present a cursory sketch of the important concepts of Fermi-Liquid Theory, following the work of Gordon Baym and Christopher Pethick [44].

Consider a non-interacting gas with two species of fermions at $T=0$ with equal numbers of each species. Due to the Pauli-Exclusion principle, the particles cannot all occupy a state with zero momentum. As more and more particles are added to the system, one can plot the occupied states as a function of momentum. These occupied states are all enclosed within a surface in the momentum space of each species known as the Fermi surface. There is a separate Fermi surface associated with each species of fermion. In the case of equal populations, the Fermi surface of both species are identical. For free particles, these surfaces are spheres with a radius given by the momentum of the last fermion of that species added to the system $k_F$ (See Figure
4.3). Since the energy of this fermion is proportional to the square of its momentum,

\[ E_F \]

one can equivalently characterize the system by this energy designated \( E_F \). This is the energy necessary to add a single particle to the system, so it is also the chemical potential for the system, \( \mu \). As the temperature increases, the occupation number for each state is given by the Fermi-Dirac distribution with the appropriate chemical potential, \( \mu(T) \).

To include the effects of interactions in the system, the problem is cast not in terms of the bare particles but in terms of “dressed” quasiparticles. These quasiparticles have an energy which contains the effects of the interactions and can be written as:

\[
\varepsilon_{k\alpha} = \varepsilon_{k\alpha}^0 + \sum_{k'\alpha'} f_{k\alpha,k'\alpha'} \delta n_{k'\alpha'}
\]

(4.1)

Here \( \varepsilon_{k\alpha} \) is the energy of the quasiparticle with momentum \( k \) and spin \( \alpha \) (\( \uparrow \) or \( \downarrow \)),

![Figure 4.3: Illustration of a 3D Fermi Sphere.](image-url)
\( \varepsilon_{k\alpha}^0 \) is the energy of the bare particle, given by:

\[
\varepsilon_{k\alpha}^0 = \frac{k^2}{2m},
\]

\( \delta n_{k\alpha} \) is the variation of the occupation number of the state with momentum \( k \) and spin \( \alpha \), and \( f_{k\alpha,k'\alpha'} \) is the second variation of the total energy of the system \( (E) \) with respect to the occupation number of the interacting particle states:

\[
f_{k\alpha,k'\alpha'} = \frac{\delta^2 E}{\delta n_{k\alpha} \delta n_{k'\alpha'}}
\]

In the absence of spin-orbit interactions this interaction energy for an isotropic system can be written in a manner which separates the spin-dependent and spin-independent parts of the interaction:

\[
f_{k\alpha,k'\alpha'} = f_{kk}^s + f_{kk'}^a \sigma \cdot \bar{\sigma}
\]

where \( \bar{\sigma} \) is a vector composed of the Pauli matrices. Here \( s \) and \( a \) indicate spin symmetric and spin anti-symmetric respectively. These can then be expanded in Legendre Polynomials about the Fermi surface, since we are considering small variations from the \( T=0 \) ground state.

\[
f_{kk'}^s = \sum_l f_l^s P_l(\cos \theta)
\]

\[
f_{kk'}^a = \sum_l f_l^a P_l(\cos \theta)
\]

The moments of this expansion are then related to the conventional Landau Fermi-Liquid parameters \( (F_l^s \text{ and } F_l^a) \) by a factor of the density of states at the Fermi
energy:

\[
F_s^i = g(E_F) f_s^i \quad (4.4a)
\]

\[
F_a^i = g(E_F) f_a^i \quad (4.4b)
\]

The conventional Landau Fermi-Liquid parameters are important because many of them can be obtained from physical measurements. The lowest order Landau parameters are the most important: \(F_0^s\) is related to the compressibility of the material, accessible in zero-sound experiments; the effective mass of the quasiparticles is related to \(F_1^s\), obtained from specific heat measurements; the spin susceptibility is related to \(F_0^a\) which can be determined by measuring linear response to an applied field; and \(F_1^a\) can be determined from spin-diffusion experiments [44,45].

4.2 Superfluidity in Systems with Equal Populations

Superfluidity arises in Fermi systems due to the existence of a residual attractive interaction that remains after renormalizing the fermions to create the Fermi-Liquid ground state. The residual interaction is not attractive for all systems, which accounts for the rarity of superfluid or superconducting states. The physical mechanism for this attractive interaction is system dependent, and in many systems the actual nature of the interaction is a matter of some debate. The standard BCS analysis exploits the delicate interplay between the repulsive coulomb interaction and the attractive electron-phonon interaction [38]. At low enough temperature, this residual interaction is attractive, leading to quasiparticle pairing in crystalline systems. Obviously
an electron-phonon interaction is not present in liquid systems such as He\textsuperscript{3} or cold Fermion gases such as Li\textsuperscript{6} or K\textsuperscript{40}. In these systems the actual nature of the pairing interaction is more exotic.

Now allow an arbitrarily attractive residual interaction between the Fermi-Liquid quasiparticles which were described in Section 4.1. Such an interaction allows the formation of a bound state which will have a lower energy than that of the non-interacting quasiparticles. The momenta of the particles in this bound state has not changed, and therefore the chemical potential remains constant. Bound quasiparticle pairs may then condense into the superfluid state, which has a lower energy than the Fermi-liquid, due to the attractive nature of the residual interaction. This phase transition is characterized by an order parameter related to the energy difference between the paired state and the unpaired Fermi-Liquid. Below, I present a cursory sketch of the analysis for a Fermi superfluid with two species of equal population. In the interest of brevity of notation, I will employ the Einstein summation convention, where repeated indices are summed over.

The following Hamiltonian describes a system of translationally invariant, interacting fermions with no spin-orbit coupling in the absence of any external fields:

\[ \hat{H} = \hat{H}_0 + \hat{V}_2 \]  

(4.5)

where \( \hat{H}_0 \) describes free fermions plus 1-body interactions (e.g. a magnetic field), and
$\hat{V}_2$ describes 2-body interactions between the fermions:

$$\hat{H}_0 = \int d^3r \left\{ \hat{\Psi}_\alpha^\dagger (\mathbf{r}) \left[ \hat{\xi}_{\alpha\beta} + U^{(1)}_{\alpha\beta}(\mathbf{r}) \right] \hat{\Psi}_\beta (\mathbf{r}) \right\}$$  (4.6a)

$$\hat{V}_2 = \int d^3r \int d^3r' \left\{ \hat{\Psi}_\delta^\dagger (\mathbf{r}) \hat{\Psi}_\gamma^\dagger (\mathbf{r}') \left[ \frac{1}{2} U^{(2)}_{\delta\gamma,\alpha\beta}(\mathbf{R}) \right] \hat{\Psi}_\alpha (\mathbf{r}') \hat{\Psi}_\beta (\mathbf{r}) \right\}$$  (4.6b)

Here, the bare particle energy operator may be written as:

$$\hat{\xi}_{\alpha\beta} = \left[ \frac{k^2}{2m} - \mu \right] \hat{\delta}_{\alpha\beta} = \xi_0(k) \hat{\delta}_{\alpha\beta}$$  (4.7)

where $m$ and $\mu$ are the effective mass and chemical potential of the fermions and subscripts $\alpha$ or $\beta$ indicate Fermion species (either $\uparrow$ or $\downarrow$). For a translationally invariant systems, the two-body interaction, may only be a function of $\mathbf{R} = \mathbf{r} - \mathbf{r}'$ [46].

A general form for a two-body interaction which preserves these symmetries can be written as:

$$\frac{1}{2} U^{(2)}_{\alpha\beta,\delta\gamma}(\mathbf{R}) = \Gamma_{\rho}(\mathbf{R}) \hat{\delta}_{\alpha\gamma} \hat{\delta}_{\beta\delta} + \Gamma_{s}(\mathbf{R}) \vec{\sigma}_{\delta\beta} \cdot \vec{\sigma}_{\gamma\alpha}$$  (4.8)

where $\Gamma_{\rho}(\mathbf{R})$ accounts for the spin independent interactions and $\Gamma_{s}(\mathbf{R})$ accounts for the spin-dependent interactions. $\vec{\sigma}_{\mu\nu}$ indicates the vector of Pauli matrices connecting spins $\mu$ and $\nu$; $\hat{\delta}_{\alpha\gamma}$ and $\hat{\delta}_{\beta\delta}$ are delta-functions in spin space (formally necessary in the density term) [46]. Making standard mean field approximation for the two-body interaction, self-consistent spin-dependent mean potentials $U^{(2)}_{\alpha\beta}(\mathbf{R})$ and pair potentials $\Delta_{\alpha\beta}(\mathbf{R})$ are defined as follows:

$$U^{(2)}_{\alpha\beta}(\mathbf{R}) = -U^{(2)}_{\alpha\delta,\beta\gamma}(\mathbf{R}) \left\langle \hat{\Psi}_\delta^\dagger (\mathbf{r}') \hat{\Psi}_\gamma (\mathbf{r}) \right\rangle$$  (4.9a)

$$+ \delta^3(\mathbf{R}) \int d^3r'' U^{(2)}_{\alpha\delta,\gamma\beta}(\mathbf{r} - \mathbf{r}'') \left\langle \hat{\Psi}_\delta^\dagger (\mathbf{r}'') \hat{\Psi}_\gamma (\mathbf{r}'') \right\rangle$$

$$\Delta_{\alpha\beta}(\mathbf{R}) = -\frac{1}{2} U^{(2)}_{\alpha\beta,\delta\gamma}(\mathbf{R}) \left\langle \hat{\Psi}_\gamma (\mathbf{r}) \hat{\Psi}_\delta (\mathbf{r}') \right\rangle$$  (4.9b)
where $\langle \hat{\Psi}^\dagger(r')\hat{\Psi}(r) \rangle$ is the normal density matrix and $\langle \hat{\Psi}(r')\hat{\Psi}(r) \rangle$ is the anomalous density matrix for the system [45].

This allows the two-body interaction to be separated into two parts: mean potentials, which dress the bare fermions and form the Fermi liquid quasiparticles; and pair potentials, which contain the residual interaction responsible for BCS-type quasiparticle pairing. Since the one-body potential has not been restricted to be diagonal in the spin indices, the notation can be simplified by combining the one-body potential with the mean potential term from the two-body potential:

$$U_{\alpha\beta}(R) = U^{(2)}_{\alpha\beta}(R) + U^{(1)}_{\alpha\beta}(r)\delta(R) \quad (4.10)$$

The total mean potential, which contains both contributions from 1-body and 2-body interactions, describes the additional contribution to the bare fermion energy due to renormalizing the system into quasiparticles and forming a Fermi-Liquid. The total mean potential $U_{\alpha\beta}(R)$ can then be related to the symmetric and anti-symmetric Landau parameters $f^s$ and $f^a$ [46]. These parameters can then be explicitly calculated from a microscopic model once the functions appearing in the interaction in Equation (4.8) are specified. Using these definitions, the Hamiltonian may be written generally as:

$$\hat{H} = \int d^3r \int d^3r' \left\{ [\xi_0\delta_{\alpha\beta} + U_{\alpha\beta}(R)] \hat{\Psi}_\beta^\dagger(r')\hat{\Psi}_\alpha(r) + \Delta_{\alpha\beta}(R)\hat{\Psi}_\beta^\dagger(r')\hat{\Psi}_\alpha^\dagger(r) - \Delta^*_{\alpha\beta}(R)\langle \hat{\Psi}_\beta(r')\hat{\Psi}_\alpha(r) \rangle \right\} \quad (4.11)$$
By expanding the field operators in a plane wave basis:

\[
\hat{\Psi}^\dagger_\alpha \,(r) = \sum_k \frac{e^{i k \cdot r}}{L^{3/2}} \hat{c}^\dagger_{k \alpha} \\
\hat{\Psi}_\alpha \,(r) = \sum_k \frac{e^{-i k \cdot r}}{L^{3/2}} \hat{c}_{k \alpha}
\] (4.12)

where \( \hat{c}_{k \alpha} \) and \( \hat{c}^\dagger_{k \alpha} \) are annihilation/creation operators for fermions of momentum \( k \) and spin \( \alpha \), and integrating over the volume of the system, the Hamiltonian can be written entirely in momentum space. This calculation is detailed in Appendix A. In this way, the Hamiltonian can be written in a more compact form:

\[
\hat{H} = \sum_k \left\{ [\xi(k) \delta_{\alpha\beta} + U_{\alpha\beta}(k)] \hat{c}^\dagger_{k \beta} \hat{c}_{k \alpha} + \left( \Delta^*_{\alpha\beta}(k) \hat{c}_{k \beta} \hat{c}_{-k \alpha} + h.c. \right) - \Delta^*_{\alpha\beta}(k) \langle \hat{c}_{k \beta} \hat{c}_{-k \alpha} \rangle \right\}
\] (4.13)

where \( \xi(k) \) is the bare particle energy measured from the Fermi surface:

\[
\xi(k) = \frac{k^2}{2m} - \mu
\]

and the mean and pair potentials are given by Equations (A.8) and (A.10) respectively.

4.3 The Bogoliubov Equations

The Bogoliubov Equations are a convenient way to calculate the superfluid excitation spectrum as well as the order parameter variation in \( r \)-space. These equations can be derived by considering the commutators of the fermion field operators with the Hamiltonian and applying a transformation of the Bogoliubov-Valatin type [47] [48]:

\[
\hat{c}_{k \alpha} = \sum_{\beta} U_{\alpha\beta} \hat{c}_{k \beta}
\]
\[ \hat{c}_{k\uparrow} = u_{k\nu\uparrow} \hat{\gamma}_{k\nu} - v_{k\nu\uparrow} \hat{\gamma}_{k\nu} \]
\[ \hat{c}_{k\downarrow}^{\dagger} = u_{k\nu\downarrow}^{\ast} \hat{\gamma}_{k\nu} - v_{k\nu\downarrow}^{\ast} \hat{\gamma}_{k\nu} \]
\[ \hat{c}_{k\downarrow} = u_{k\nu\downarrow} \hat{\gamma}_{k\nu} + v_{k\nu\downarrow} \hat{\gamma}_{k\nu} \]
\[ \hat{c}_{k\uparrow}^{\dagger} = u_{k\nu\uparrow}^{\ast} \hat{\gamma}_{k\nu} + v_{k\nu\uparrow}^{\ast} \hat{\gamma}_{k\nu} \]  (4.14a)
\[ \hat{c}_{k\downarrow} = u_{k\nu\downarrow} \hat{\gamma}_{k\nu} + v_{k\nu\downarrow} \hat{\gamma}_{k\nu} \]
\[ \hat{c}_{k\uparrow}^{\dagger} = u_{k\nu\uparrow}^{\ast} \hat{\gamma}_{k\nu} + v_{k\nu\uparrow}^{\ast} \hat{\gamma}_{k\nu} \]  (4.14b)

Here, \( \hat{\gamma}^{\dagger} \) and \( \hat{\gamma} \) are creation and annihilation operators for so-called “Bogolons,” \(^1\) and the coefficients \( u_{k\nu\alpha} \) and \( v_{k\nu\alpha} \) (where \( \alpha \) is the spin index of the Fermi-Liquid quasiparticles and \( \nu \) corresponds to the spin-eigenvalues associated with the Bogolons) are chosen to diagonalize the Hamiltonian \([49]\).

Since the operators \( \gamma_{k\nu} \) and \( \gamma_{k\nu}^{\dagger} \) are fermion operators, the Bogoliubov coefficients must satisfy appropriate orthonormality conditions:

\[ \bar{u}_{k} \bar{v}_{k} - \bar{v}_{k} \bar{u}_{k} = 0 \]  (4.15a)
\[ \bar{u}_{k} \bar{u}_{k}^{\dagger} + \bar{v}_{k} \bar{v}_{k}^{\dagger} = 1 \]  (4.15b)

where \( \bar{u}_{k} \) and \( \bar{v}_{k} \) are 2 \times 2 matrices (in spin-space) of the Bogoliubov coefficients. These coefficients depend on the bare particle energies, the mean potentials, and the gap. The calculation of these coefficients is detailed in several texts, including \([46]\).

The Bogoliubov equations can be written as the following matrix equation:

\[
\hat{\Omega} \begin{pmatrix} u_{\uparrow} \\ u_{\downarrow} \\ v_{\uparrow} \\ v_{\downarrow} \end{pmatrix} = \varepsilon \begin{pmatrix} u_{\uparrow} \\ u_{\downarrow} \\ v_{\uparrow} \\ v_{\downarrow} \end{pmatrix} \]  (4.16)

\(^1\)It is important to distinguish Bogolons, which are quasiparticle excitations of the superfluid phase, from the quasiparticles which form the underlying Fermi-Liquid.
where the Bogolon eigenvalues $k\nu$ have been suppressed on the Bogoliubov coefficients $u$ and $v$. After making the plane wave expansion discussed in Section 4.2 and integrating over the system volume, the matrix $\hat{\Omega}$ can be written in momentum space as:

$$
\hat{\Omega}_k = 
\begin{pmatrix}
\xi(k) + U_{\uparrow\uparrow}(k) & U_{\uparrow\downarrow}(k) & \Delta_{\uparrow\uparrow}(k) & \Delta_{\uparrow\downarrow}(k) \\
U_{\uparrow\downarrow}(k) & \xi(k) + U_{\downarrow\uparrow}(k) & \Delta_{\downarrow\uparrow}(k) & \Delta_{\downarrow\downarrow}(k) \\
-\Delta_{\uparrow\uparrow}^*(-k) & -\Delta_{\uparrow\downarrow}^*(-k) & -\xi(-k) - U_{\uparrow\downarrow}^*(-k) & -U_{\uparrow\uparrow}^*(-k) \\
-\Delta_{\downarrow\uparrow}^*(-k) & -\Delta_{\downarrow\downarrow}^*(-k) & -U_{\downarrow\uparrow}^*(-k) & -\xi(-k) - U_{\downarrow\downarrow}^*(-k)
\end{pmatrix}
$$

(4.17)

The Bogolon excitation spectrum is given by the eigenvalues of this matrix. Defining

$$
\tilde{\xi}(k) \equiv \begin{pmatrix} [\xi(k) + U_{\uparrow\uparrow}(k)] & U_{\uparrow\downarrow}(k) \\ U_{\uparrow\downarrow}(k) & [\xi(k) + U_{\downarrow\uparrow}(k)] \end{pmatrix}
$$

(4.18)

$$
\tilde{\Delta}(k) \equiv \begin{pmatrix} \Delta_{\uparrow\uparrow}(k) & \Delta_{\uparrow\downarrow}(k) \\ \Delta_{\downarrow\uparrow}(k) & \Delta_{\downarrow\downarrow}(k) \end{pmatrix}
$$

(4.19)

simplifies the operator $\hat{\Omega}_k$ to the following form:

$$
\hat{\Omega}_k = \begin{pmatrix} \tilde{\xi}(k) & \tilde{\Delta}(k) \\ \tilde{\Delta}^\dagger(k) & -\tilde{\xi}(k) \end{pmatrix}
$$

(4.20)

This relies on two facts: first, that the gap has the property $\Delta^\dagger(k) = -\Delta^*(-k)$ due to the symmetry of the anomalous density matrix; second, the bare quasiparticle energies are real and depend on $k^2$. At this point, one generally assumes that the
matrix $\tilde{\xi}(k)$ is diagonal with the diagonal elements equal:

$$
\tilde{\xi}(k) = \begin{pmatrix}
\xi(k) & 0 \\
0 & \xi(k)
\end{pmatrix}
$$

This corresponds to neglecting spin-flip scattering in the Fermi-Liquid state, which are described by the off-diagonal mean potentials, and assuming that the mean potentials $U_{\uparrow\uparrow}(k)$ and $U_{\downarrow\downarrow}(k)$ are equal. These approximations are reasonable in an unpolarized superfluid [46].

4.4 Density Matrix Formalism and Equation of Motion Method

The density matrix formalism is very useful due to the direct connection between these matrices and the normal and anomalous Greens’ functions. The normal and anomalous density matrices have elements given by:

$$
\rho_{\alpha\beta}(r, r') \equiv \left\langle \Psi_\beta^\dagger(r') \Psi_\alpha(r) \right\rangle = G_{\alpha\beta}(r, r')
$$

$$
\rho_{\alpha\beta}^A(r, r') \equiv \left\langle \Psi_\beta^\dagger(r') \Psi_\alpha(r) \right\rangle = F_{\alpha\beta}(r, r')
$$

where $G_{\alpha\beta}(r, r')$ is the normal Green’s function and $F_{\alpha\beta}(r, r')$ is the anomalous Green’s function. After making the same plane wave expansion from Equation (4.12) and rearranging, these become:

$$
\rho_{\alpha\beta}(R) = \sum_k \frac{e^{ik\cdot R}}{L^3} \left\langle \hat{c}_{k\beta}^\dagger \hat{c}_{k\alpha} \right\rangle
$$

$$
\rho_{\alpha\beta}^A(R) = \sum_k \frac{e^{ik\cdot R}}{L^3} \left\langle \hat{c}_{k\beta}^\dagger \hat{c}_{-k\alpha} \right\rangle
$$
where these have been written to emphasize the translational symmetry. Here, the expectation value is taken over the Fock space of the bare fermions. Fourier transforming these with respect to the position difference yields:

\[
\rho_{\alpha\beta}(k) = \langle \hat{c}_{-k\alpha}^\dagger \hat{c}_{k\alpha} \rangle \equiv n_{\alpha\beta}(k) \tag{4.23a}
\]

\[
\rho^A_{\alpha\beta}(k) = \langle \hat{c}_{k\beta} \hat{c}_{-k\alpha} \rangle \equiv n^A_{\alpha\beta}(k) \tag{4.23b}
\]

The density matrix corresponds to the number of fermions and the anomalous density matrix is referred to as the “pair amplitude” [46].

One can write the density matrices in terms of the Bogoliubov coefficients by applying the transformation in Equation (4.14). The elements of the regular and anomalous density matrices then take the form [46]:

\[
n_{\alpha\beta}(k) = \sum_{\nu} \left[ u_{k\nu\beta}^* v_{k\nu\alpha} f_{k\nu} + v_{-k\nu\beta}^* u_{-k\nu\alpha} (1 - f_{k\nu}) \right] \tag{4.24a}
\]

\[
n^A_{\alpha\beta}(k) = \frac{1}{2} \sum_{\nu} \left[ u_{-k\nu\alpha} v^*_{-k\nu\beta} - u_{k\nu\beta} v^*_{k\nu\alpha} \right] (1 - 2 f_{k\nu}) \tag{4.24b}
\]

The simple relationship between the density matrix and the regular and anomalous Green’s Functions allows the use of the equation of motion method to calculate the density matrix elements. By writing the commutators of the field operators and the Hamiltonian, one obtains a set of coupled equations for the regular and anomalous Green’s functions, which can be solved with a clever application of Fourier transforms. In this way one can show that the normal and anomalous Green’s functions depend on both the quasiparticle energy \(\xi(k)\) as well as the gap [50].
4.5 Singlet-Triplet Separation

Because of the different symmetry involved in singlet and triplet pairing, different interactions are responsible for each type of pairing. Superfluids can pair in the $\vec{S} = 0$ channel and the $\vec{S} = 1$ channel. Examples of these different pairing symmetries include the conventional $S=0, l=0$ (s-wave singlet), and the unconventional $S=0, l=2$ (d-wave singlet) and $S=1, l=1$ (p-wave triplet) superfluids [46]. Since both effects are contained in the Hamiltonian developed in this section, it is useful to separate them.

This separation is accomplished by writing the order parameter as $\tilde{\Delta}(k) = \tilde{\Delta}_s(k) + \tilde{\Delta}_t(k)$ where:

$$\tilde{\Delta}_s(k) = \begin{pmatrix} 0 & \Delta_{\uparrow\downarrow}^s(k) \\ \Delta_{\downarrow\uparrow}^s(k) & 0 \end{pmatrix} \quad (4.25a)$$

$$\tilde{\Delta}_t(k) = \begin{pmatrix} \Delta_{\uparrow\uparrow}^t(k) & \Delta_{\uparrow\downarrow}^t(k) \\ \Delta_{\downarrow\uparrow}^t(k) & \Delta_{\downarrow\downarrow}^t(k) \end{pmatrix} \quad (4.25b)$$

Here the pairing interaction in the $\uparrow\downarrow$ channel is separated into a singlet and triplet part:

$$\Delta_{\uparrow\downarrow}(k) = \Delta_{\uparrow\downarrow}^s(k) + \Delta_{\uparrow\downarrow}^t(k)$$

The singlet pairing interaction must be anti-symmetric under particle exchange, so $\Delta_{\uparrow\downarrow}^s(k) = -\Delta_{\downarrow\uparrow}^s(k)$, whereas the triplet pairing interaction is symmetric under particle exchange and $\Delta_{\uparrow\downarrow}^t(k) = \Delta_{\downarrow\uparrow}^t(k)$. This separation provides two advantages: first, it simplifies the calculation in clean systems which only have one kind of pairing symmetry; second, it provides a general formalism for the treatment of dirty systems.
which may contain multiple pairing interactions with different symmetry.

4.6 The d-vector and D-matrix Formalism

The d-vector formalism is an elegant tool for describing many properties of triplet order parameters in a very compact way. Among these are the symmetries of the order parameter and structure of the energy gap, as well as the spin and orbital angular momentum of the paired fermions. The d-vector is defined by the following relations:

\[ \tilde{\Delta}_s(k) = d_0(k)\chi_0 \quad (4.26a) \]
\[ \tilde{\Delta}_t(k) = d(k) \cdot \vec{\chi} \quad (4.26b) \]

where \( \chi_i \) are 2x2 matrices in spin-space given by:

\[
\begin{align*}
\chi_0 &= \begin{pmatrix} 0 & 1 \\ -1 & 0 \end{pmatrix} \\
\chi_1 &= \begin{pmatrix} -1 & 0 \\ 0 & 1 \end{pmatrix} \\
\chi_2 &= \begin{pmatrix} i & 0 \\ 0 & i \end{pmatrix} \\
\chi_3 &= \begin{pmatrix} 0 & 1 \\ 1 & 0 \end{pmatrix}
\end{align*}
\]

\(|d_0(k)|\) is the magnitude of the singlet order parameter (which may be complex) and \(d(k)\) is a vector in spin space.

Superfluid states which have the property \(d(k) \times d^*(k) = 0\) are referred to as unitary states, i.e. states for which all the fermions which can pair are paired. The d-vector has a simple physical meaning in the case of unitary states, where its magnitude is proportional to the energy gap, and it points in a direction normal to the plane of equal spin polarization. Non-unitary states are states for which \(d(k) \times d^*(k) \neq 0\), and is composed of pairs with multiple energy gaps. These states are energetically unfavorable in the absence of a symmetry breaking field [51].
Though the d-vector gives us a simple way to understand the symmetries of the order parameter, it hides a great deal. Define the elements of the D-matrix, $D_{\alpha i}$, in the following way:

$$\Delta_i(k) = D_{\alpha i} \chi_\alpha \hat{k}_i$$ (4.28)

Here $\hat{k}$ is a unit vector in the orbital space, and $\chi$ is the vector of spinors described above. $D_{\alpha i}$ is a complex 3x3 matrix which describes the magnitude of the gap and the coupling between the spin of the cooper pair and its angular momentum. Consequently, there are 18 possible states for a triplet superfluid: 9 complex numbers uniquely characterize the order parameter for a given state. There is a simple relationship between the D-matrix and the d-vector:

$$d = D_{\alpha i} \hat{k}_i^{-1}$$ (4.29)

consequently they are equivalent descriptions of the order parameter for the system.

To better understand the d-vector, consider the example of Strontium Ruthenate ($\text{Sr}_2\text{RuO}_4$) which is thought to pair as a p-wave triplet. Figure 4.4 (reproduced from [51]) shows an example of the d-vector construction for the A-phase of this material. The large arrow denotes the angular momentum of the Cooper pair. The small arrows in plane denote the spin of each fermion. In this plane equal spin pairing (ESP) exists for any choice of spin-quantization axis (illustrated by the use of multiple arrows). The d-vector has a magnitude equal to that of the gap and points in a direction perpendicular to the plane of equal-spin polarization (in this case the
\[ \mathbf{d}_A(k) = \Delta_0 (k_x + i k_y) \hat{\mathbf{z}} \]  

(4.30)

where \( k_x \) and \( k_y \) are the angular momentum components of the cooper pair.

### 4.7 Free Energy

To determine both ground state and \( T \neq 0 \) properties of superfluids, it is often useful to construct a Ginzburg-Landau (GL) free energy expansion. In balanced population systems, the s-wave singlet ground state can be obtained directly from the BCS equations. Consequently a free-energy analysis is not employed in this case. However, a proper free energy minimization analysis is necessary to correctly determine the stable ground state in polarized s-wave systems. For systems with unconventional pairing (d-wave, p-wave), minimizing the GL free energy and testing
the stability of the states is necessary to obtain the correct ground state [46].

The GL free energy is constructed as an expansion in powers of invariants of the order parameter. Since the order parameter is a complex matrix in spin-space, invariants must be constructed out of products of real scalars. The lowest order of these terms is given by \( Tr(\tilde{\Delta}\tilde{\Delta}^\dagger) \). In the absence of an applied field, there are four 4\(^{th}\) order invariants, which are described in most textbooks [45]. For unitary states in a balanced population system, the GL free energy can be written in the following way:

\[
\Delta \tilde{\mathcal{F}} = \tilde{\mathcal{F}} - \tilde{\mathcal{F}}_N = -\alpha Tr[\Delta\Delta^\dagger] + \beta_1 |Tr[\Delta\Delta^T]|^2 + \beta_2 (Tr[\Delta\Delta^\dagger]) \\
+ \beta_3 Tr[(\Delta\Delta^T)(\Delta\Delta^T)^*] + \beta_4 Tr[(\Delta\Delta^\dagger)^2] + \beta_5 Tr[(\Delta\Delta^\dagger)(\Delta\Delta^\dagger)^*]
\] (4.31)

where \( \tilde{\mathcal{F}}_N \) is the free energy of the normal state, and \( \alpha \) and \( \beta_i \) are parameters which can be calculated from microscopic models of the interaction potential. When \( \Delta \tilde{\mathcal{F}} < 0 \) the superfluid state has a lower free energy than the normal state. In practice, testing this stability must be done numerically for specific models and systems. This work will analyze the population imbalance Fermi superfluid at the level of general formalism, without specifying a particular form for the interaction. In this way, the work remains as general as possible, and can be applied to a number of different systems.
Population imbalance in Fermi systems refers to having more of one species of fermion than another. A simple example of this would be a system composed of electrons, having more spin up electrons than spin down. In this example, the spin of the fermion describes the species, however in other systems this might not be the case. In quark systems, for example, population imbalance might refer to color, e.g. a 3-species model with up, down and strange quarks where the populations of up and down quarks are equal, but different from the population of strange quarks. For clarity of discussion, this work will refer to population imbalanced two-level systems as “quasi-spin systems.” Examples of such quasi-spin systems include coupled hyperfine states with imbalanced populations, such as in Li$^6$ and K$^{40}$, nuclear matter with isospin imbalances, and spin-polarized He$^3$. In quasi-spin systems the difference between the chemical potentials of the two species acts to create an effective “internal” field. In this work, the majority species will be taken to be the up ($\uparrow$) quasi-spin species.

When there is a population imbalance in the system, each species of fermion will form it’s own Fermi surface (with spectra shown in Figure 5.1), characterized by a chemical potential unique to that species, $\mu_{\alpha}$, where $\alpha$ indicates the species in question: up ($\alpha = \uparrow$) or down ($\alpha = \downarrow$). The energy of the fermions will be measured from the
Figure 5.1: Energy spectrum of two species of non-interacting fermions with different chemical potentials.

chemical potential of the relevant species, i.e.

$$\xi_\alpha^0(k) = \frac{k^2}{2m} - \mu_\alpha$$

(5.1)

In principle, one can allow different species to have different masses, e.g. recent experiments using mixtures of Li$^6$ and K$^{40}$. In such systems, the calculation proceeds in the same way; one must simply include a species index on the mass. The population imbalance can therefore be characterized by two parameters: the average chemical potential $\bar{\mu}$ and the chemical potential difference $h$, defined as:

$$\bar{\mu} \equiv \frac{\mu^\uparrow + \mu^\downarrow}{2} \quad \text{and} \quad h \equiv \frac{\mu^\uparrow - \mu^\downarrow}{2}$$

(5.2)

I will now extend the analysis described in the previous chapter to a population imbalanced Fermi system characterized by these parameters.
A Hamiltonian of the same form discussed in Section 4.2 which neglects one-body and spin-orbit interactions describes such a system quite well. In quasi-spin systems, the population imbalance picks a preferential direction in spin-space which is taken as the z-axis. Since the population imbalance creates a distinct anisotropy in spin space, the effects of anisotropic spin-dependent interactions can be considered by taking a 2-body interaction of the form:

$$\frac{1}{2} U^{(2)}_{\alpha\beta,\delta\gamma}(R) = \Gamma^\rho_{\alpha\gamma}(R) \hat{\sigma}^\rho_{\alpha\gamma} \hat{\sigma}^\rho_{\beta\delta} + \Gamma^{xy}_{s}(R) \left[ \hat{\sigma}^x_{\alpha\gamma} \hat{\sigma}^x_{\beta\delta} + \hat{\sigma}^y_{\alpha\gamma} \hat{\sigma}^y_{\beta\delta} \right] + \Gamma^z_{s}(R) \hat{\sigma}^z_{\alpha\gamma} \hat{\sigma}^z_{\beta\delta}$$

(5.3)

where $\Gamma^{xy}_{s}(R)$ and $\Gamma^z_{s}(R)$ are the perpendicular and parallel components of the spin-dependent interaction relative to the preferred axis, respectively, and $\Gamma^{xy}_{s}(R) \neq \Gamma^z_{s}(R)$. When these components of the interaction are equal, the physics of the isotropic case is recovered. It should be noted that (5.3) is generic and can be written in the iso-spin or color imbalanced cases as well. In Section 5.1 the Bogoliubov equations for population imbalanced systems are developed. Sections 5.3 and 5.4 focus on understanding the effect of isotropic spin-dependent potentials for imbalanced singlet and triplet superfluids respectively. Section 5.5 addresses the effects of spin-dependent anisotropy in population imbalanced systems.
5.1 Bogoliubov Equations for Population Imbalanced Systems

In the mean-field limit described in Chapter 4, the Bogoliubov operator $\hat{\Omega}$ for a population imbalanced Fermi superfluid becomes:

$$
\hat{\Omega} = \begin{pmatrix}
\xi^0_{\uparrow}(k) + U_{\uparrow\uparrow}(k) & U_{\uparrow\downarrow}(k) & \Delta_{\uparrow\uparrow}(k) & \Delta_{\uparrow\downarrow}(k) \\
U_{\downarrow\uparrow}(k) & \xi^0_{\downarrow}(k) + U_{\downarrow\downarrow}(k) & \Delta_{\downarrow\uparrow}(k) & \Delta_{\downarrow\downarrow}(k) \\
\Delta^*_{\uparrow\uparrow}(k) & \Delta^*_{\uparrow\downarrow}(k) & -\xi^0_{\downarrow}(k) - U^*_{\downarrow\uparrow}(k) & -U^*_{\downarrow\downarrow}(-k) \\
\Delta^*_{\downarrow\uparrow}(k) & \Delta^*_{\downarrow\downarrow}(k) & -U^*_{\uparrow\downarrow}(-k) & -\xi^0_{\uparrow}(k) - U^*_{\uparrow\uparrow}(k)
\end{pmatrix}
$$

(5.4)

The difference between this equation and Equation (4.17) come in the species index associated with the bare particle energies $\xi^0_{\alpha}$. In the case of arbitrary spin-polarization, several of the simplifying assumptions of the balanced population case are no longer valid. The assumption that $U_{\uparrow\uparrow}(k) = U_{\downarrow\downarrow}(k)$ is not valid because scattering between majority spins will be much more common than between minority spins. Further, neglecting the off-diagonal mean potentials is unwise, as spin-flip scattering provides a mechanism for quasiparticles to switch fermi-surfaces which may be important for pairing. One can, however, still assume that the pairing interactions are independent of the interactions dressing the underlying Fermi-Liquid. With these assumptions, the Bogoliubov operator $\hat{\Omega}$ can be simplified to the following form:

$$
\hat{\Omega} = \begin{pmatrix}
\xi_{\uparrow}(k) & 0 & \Delta_{\uparrow\uparrow}(k) & \Delta_{\uparrow\downarrow}(k) \\
0 & \xi_{\downarrow}(k) & \Delta_{\downarrow\uparrow}(k) & \Delta_{\downarrow\downarrow}(k) \\
\Delta^*_{\uparrow\uparrow}(k) & \Delta^*_{\uparrow\downarrow}(k) & -\xi_{\downarrow}(k) & 0 \\
\Delta^*_{\downarrow\uparrow}(k) & \Delta^*_{\downarrow\downarrow}(k) & 0 & -\xi_{\uparrow}(k)
\end{pmatrix}
$$

(5.5)
where
\[
\xi_\uparrow(k) = \frac{k^2}{2m} + U_+(k) - \bar{\mu} + \sqrt{(U_-(k) - h)^2 + U_{1\parallel}(k)U_{1\downarrow}(k)} \tag{5.6a}
\]
\[
\xi_\downarrow(k) = \frac{k^2}{2m} + U_+(k) - \bar{\mu} - \sqrt{(U_-(k) - h)^2 + U_{1\parallel}(k)U_{1\downarrow}(k)} \tag{5.6b}
\]

and
\[
U_+(k) = \frac{U_{1\parallel}(k) + U_{1\downarrow}(k)}{2} \tag{5.7a}
\]
\[
U_-(k) = \frac{U_{1\parallel}(k) - U_{1\downarrow}(k)}{2} \tag{5.7b}
\]

\(\xi_\alpha(k)\) are the renormalized quasiparticle energies for each species which include the effect of the Fermi-Liquid interactions. \(U_\pm(k)\) are the average and difference of the parallel scattering potentials respectively.

It is useful to define the following quantities:
\[
\xi_\pm(k) = \frac{\xi_\uparrow(k) \pm \xi_\downarrow(k)}{2} \tag{5.8}
\]

as the average and difference of the quasiparticle energies for the two species. These take the simple forms:
\[
\xi_+(k) = \frac{k^2}{2m} + U_+(k) - \bar{\mu} \tag{5.9a}
\]
\[
\xi_-(k) = \sqrt{(U_-(k) - h)^2 + U_{1\parallel}(k)U_{1\downarrow}(k)} \tag{5.9b}
\]

The interpretations of these quantities are straightforward: \(\xi_+(k)\) is the energy of a dressed Fermi-Liquid quasiparticle measured from the average chemical potential. This quantity is independent of species. \(\xi_-(k)\) is the shift in the energy of the quasiparticle due to its species. This shift is due to population imbalance through \(h\) and
$U_\pm(k)$ and spin-flip scattering through $U_{\uparrow\downarrow}(k)$ and $U_{\downarrow\uparrow}(k)$. $\xi_\pm(k)$ depend on the polarization explicitly through $\bar{\mu}$ and $h$ as well as implicitly through the mean potentials. As discussed in Section 4.4 the mean potentials depend on the density matrices, which are given by the normal Green’s functions describing the propagation of quasiparticles in the underlying Fermi-Liquid, as well as the anomalous Green’s functions which describes the pair amplitude [45]. These Green’s functions explicitly depend on the quasiparticle energies of each species as well as the gap, and can be obtained using the equation of motion method [52]. With these caveats, the Bogolon excitation spectrum can be obtained from the eigenvalues of the matrix $\hat{\Omega}$. The results for the singlet and triplet pairing symmetries are discussed in Sections 5.3 and 5.4 respectively.

5.2 Free Energy Analysis of Population Imbalanced Systems

In cold atoms systems, p-wave superfluids have not yet been realized. Recent experiments with s-wave superfluids having imbalanced populations and the experimental realization of p-wave Feshbach resonances provide a motivation for making predictions about similar systems with p-wave symmetry. Recent experiments with population imbalanced s-wave superfluids have observed phase separation between normal and superfluid phases as functions of polarization in cold atomic gases. This separation can be seen in Figure 5.2 (taken from [43]) where $\delta$ is the measure of polarization. As the polarization is increased, the superfluid core of the gas decreases until it vanishes at large polarization. The singlet case will be discussed but the focus will be on the effects of population imbalance on the lowest energy states of p-wave
triplet superfluids.

Figure 5.2: Phase separation in s-wave superfluid observed by the MIT group. The dark grey area in the center is the superfluid, and the unpaired normal state in yellow surrounding it in the phase-separated regime. $\delta$ is the measure of the polarization.

Recall that for unpolarized Fermi superfluids, the free energy is given by Equation (4.31). In systems where the population imbalance does not create an internal field, this is the free energy. The polarization dependence is hidden in the parameters $\alpha$ and $\beta_i$ which must be calculated from the microscopic 2-body interactions. If it is assumed that there is no internal field, or if the internal field is negligible, one can get a picture of the ground state structure as a function of the polarization by considering the free energy of each state as a function of the relative strength of each parameter.

Quasi-spin systems with imbalanced populations have an effective internal magnetic field due to the number difference between the two spin species. This effective internal field is given by:

$$B \equiv \mu_B (n_\uparrow - n_\downarrow) \hat{z} \quad (5.10)$$
where $\mu_B$ is the magnetic dipole moment per fermion, obviously in the same direction as the preferential direction in spin space. The internal field couples to the order parameter, contributing the following term to lowest order in the internal field [45]:

$$\Delta \mathcal{F}^B = \frac{1}{4} \tilde{\alpha} B_i B_j \text{Tr} \left( \sigma_i \tilde{\Delta} \left[ \sigma_j, \tilde{\Delta}^\dagger \right] \right)$$

(5.11)

where $\tilde{\alpha}$ is a parameter which can be calculated from the 2-body interaction potential and depends both explicitly and implicitly on the chemical potential difference between the two species. Since the induced internal field is in the $\hat{z}$ direction defined by the quasi-spin imbalance, these become:

$$\Delta \mathcal{F}^B = \frac{1}{4} \tilde{\alpha} \mu_B^2 (n_\uparrow - n_\downarrow)^2 \text{Tr} \left( \sigma_z \tilde{\Delta} \left[ \sigma_z, \tilde{\Delta}^\dagger \right] \right)$$

(5.12)

After this simplification, the additional contribution to the change in the free energy is:

$$\Delta \mathcal{F}^B = \frac{1}{2} \tilde{\alpha} \mu_B^2 (n_\uparrow - n_\downarrow)^2 (|\Delta_{\uparrow\downarrow}|^2 + |\Delta_{\downarrow\uparrow}|^2)$$

(5.13)

The free energy contribution due to the internal field only depends on the interspecies pairing. Consequently it will only affect superfluid states which depend on $m_s = 0$ angular momentum components, and will therefore be important in both the singlet and triplet quasi-spin systems. In nuclear or quark matter, the “internal” field will couple to the relevant order parameter defined in the appropriate isospin or quark basis, analogous to the spin-basis discussed above. At this point it is useful to separate the singlet and triplet interactions as discussed earlier; each is discussed in Sections 5.3 and 5.4 respectively.
5.3 Singlet Case

After making the singlet-triplet separation discussed in Section 4.5, the Bogoliubov equations for a singlet-paired population imbalanced superfluid take the following form:

\[
\begin{pmatrix}
\xi^\uparrow(k) & 0 & 0 & d_0(k) \\
0 & \xi^\downarrow(k) & -d_0(k) & 0 \\
0 & -d_0^*(k) & -\xi^\uparrow(k) & 0 \\
d_0^*(k) & 0 & 0 & -\xi^\downarrow(k)
\end{pmatrix}
\begin{pmatrix}
u^\uparrow \\
u^\downarrow \\
v^\uparrow \\
v^\downarrow
\end{pmatrix}
= \varepsilon_s(k)
\begin{pmatrix}
u^\uparrow \\
u^\downarrow \\
v^\uparrow \\
v^\downarrow
\end{pmatrix}
\]

The singlet excitation spectrum is given by the eigenvalues of the matrix $\hat{\Omega}$:

\[
\varepsilon^\uparrow_s(k) = \sqrt{\xi^2_+(k) + |d_0(k)|^2 + \xi^\uparrow(k)}
\]

\[
\varepsilon^\downarrow_s(k) = \sqrt{\xi^2_+(k) + |d_0(k)|^2 - \xi^\downarrow(k)}
\]

Assuming that the gap is small compared to $\xi^\pm(k)$ allows $\varepsilon_s(k)$ to be expanded as a Taylor Series. In this expansion, the positive eigenvalues become:

\[
\varepsilon^\uparrow_s(k) = \xi^\uparrow(k) + \frac{|d_0|^2}{2\xi^\pm(k)} - \frac{|d_0|^4}{8\xi^3_+(k)}
\]

\[
\varepsilon^\downarrow_s(k) = \xi^\downarrow(k) + \frac{|d_0|^2}{2\xi^\pm(k)} - \frac{|d_0|^4}{8\xi^3_+(k)}
\]

In the singlet case, pairing can only occur between quasiparticles with opposite spin. For these quasiparticles to pair in the conventional manner, they must have momenta which are equal in magnitude and opposite in direction. If this is not the case, the Cooper pair will have a non-zero center of mass momentum. This is a so-called FFLO state; these states will not be discussed here because to date they have not been observed in 3D systems [43].
In the case of a population imbalanced system, the Fermi surfaces for the two species are separated by an energy difference which is given by $2\hbar$. As the polarization increases, a degeneracy in the singlet excitation spectrum is lifted. This occurs because there are two processes which can create singlet pairs. First, an up spin can be scattered from the up Fermi surface, and pair with a down spin on the down Fermi surface. Second, a down spin can be scattered from the down Fermi surface and pair with an up spin on the up Fermi surface. When there is a difference between the energies of the two Fermi surfaces, there is a different energy associated with these two processes. The energy of pairs on the up Fermi surface is larger than the energy of pairs on the down Fermi surface.

Taking $d_0 = \frac{\Delta_0}{\sqrt{2}}$, the free energy of the singlet superfluid in the absence of an internal field obtained from (4.31) is given by:

$$
\Delta F_s^* = -\alpha |\Delta_0|^2 \left( 1 - \frac{\beta_0}{\alpha} |\Delta_0|^2 \right)
$$

(5.17)

where $\beta_0$ is simply $2(2\beta_1 + 2\beta_2 + \beta_3 + \beta_4 + \beta_5)$, and can be calculated from the 2-body interaction. As the population imbalance is varied, there will be a delicate interplay between the magnitude of the gap and the relative strength of the parameters $\alpha$ and $\beta_0$. From this expression, it is clear that large gaps will destroy the superfluid state in cases where the relative size of $\frac{\beta_0}{\alpha} > \frac{1}{|\Delta_0|^2}$.

In quasi-spin systems, the free energy difference depends on the internal field as well:

$$
\Delta F^* = -\alpha |\Delta_0|^2 \left( 1 + \frac{\tilde{\alpha} B^2 - \beta_0}{\alpha} |\Delta_0|^2 \right)
$$

(5.18)
In this situation, there are four parameters which compete to stabilize the singlet superfluid: The magnitude of the gap, the internal field, and the ratios \( \frac{\tilde{\alpha}}{\alpha} \) and \( \frac{\beta_0}{\alpha} \). Since these quantities must all be determined self consistently by solving the coupled gap and number equations, these parameters are not all free. In most systems, these quantities must be calculated numerically for model-specific interaction potentials. This work lays the foundation for such model-dependent calculations in the future.

### 5.4 Triplet Case

In the triplet case, the Bogoliubov equations take the form (suppressing \( k \) dependence):

\[
\begin{pmatrix}
\xi_\uparrow & 0 & -d_x - id_y & d_z \\
0 & \xi_\downarrow & -d_z & d_x + id_y \\
-d_x^* - id_y^* & -d_z^* & -\xi_\uparrow & 0 \\
d_x^* & d_z^* - id_y^* & 0 & -\xi_\downarrow \\
\end{pmatrix}
\begin{pmatrix}
u_\uparrow \\
u_\downarrow \\
v_\uparrow \\
v_\downarrow \\
\end{pmatrix}
= \varepsilon(k)
\begin{pmatrix}
u_\uparrow \\
u_\downarrow \\
v_\uparrow \\
v_\downarrow \\
\end{pmatrix}
\tag{5.19}
\]

where the gap has been written in terms of the d-vector. In the triplet case, the energy spectrum is considerably more complex. The square of the eigenvalues of \( \hat{\Omega} \) has the form (suppressing \( k \) dependence):

\[
\varepsilon_t^2 = \xi_+^2 + \xi_-^2 + |d|^2 \pm \sqrt{4\xi_+^2 \xi_-^2 + 4\xi_+^2 |d_z|^2 + 4i\xi_+ \xi_- (d \times d^*)_z + |d \times d^*|^2} \tag{5.20}
\]

In the balanced population limit \( \xi_- = 0 \) and we recover the unpolarized result:

\[
\varepsilon_t^2 = \xi^2 + |d|^2 \pm |d \times d^*|
\]
In the balanced population case, the degeneracy of the Bogolon excitation spectrum is only split in the case of non-unitary states through the term involving $|d \times d^*|$. 

In triplet-paired superfluids, population imbalance has two effects described in Equation (5.20). The terms $4\xi^2 \xi^2$ and $4\xi^2 |d_z|^2$ contribute to splitting the triplet excitation degeneracy in the case of both unitary and non-unitary states. The first term is a contribution which is only *implicitly* dependent on the order parameter via the mean potentials. The second term has a direct dependence on the component of the d-vector which lies along a preferential direction in spin-space. This term arises even in cases where the population imbalance does not create an additional internal field due to spin-imbalance. The term $4i\xi \xi_+(d \times d^*)_z$ is an additional contribution to split the degeneracy from the nonunitary states, i.e. it only contributes when $|d \times d^*| \neq 0$. For unitary phases the triplet excitation spectrum in the population imbalanced case is then simply:

$$\varepsilon^2_t = \xi^2_+ + \xi^2_- + |d|^2 \pm 2\xi_- \sqrt{\xi^2_+ + |d_z|^2}$$  

(5.21)

Restricting the discussion to unitary states, now consider the free energy analysis for the triplet case. The GL free energy from Equation 4.31 can be written in terms of the d-vector for unitary states as:

$$\Delta \mathcal{F} = -\alpha |d|^2 + 2\beta_{12}(d \cdot d)(d^* \cdot d^*) + \beta_{345}|d|^4$$  

(5.22)

where $\beta_{12} = \beta_1 + \beta_2$ and $\beta_{345} = \beta_3 + \beta_4 + \beta_5$ are parameters calculated from the two-body interaction. These parameters combine because of the relationship between the order parameter and the d-vector when using the d-vector representation.
For population imbalanced quasi-spin systems, the change in the free energy associated with the coupling of the internal field to the order parameter can be written in terms of the d-vector:

$$\Delta \tilde{\mathbf{f}} = \tilde{\alpha} B^2 |\mathbf{d}_z|^2$$

(5.23)

The internal field couples only to the z-component of the d-vector, i.e. the inter-species pairing component of the order parameter. Noting that the internal field depends on the order parameter through the normal Green’s function, one can then minimize the change in the free energy with respect to the order parameter. In practice, this must be done numerically, as the parameters $\alpha$, $\tilde{\alpha}$, $\beta_{12}$ and $\beta_{345}$ all depend on the 2-body interaction potential.

At this point, a great deal of intuition can be gained by considering the change in the free energy of the known lowest energy states of a triplet superfluid in zero applied field, the B-phase and A-phase observed in liquid He$^3$. At normal pressure, that A-phase is unstable, and at high pressure it is stabilized by strong-coupling contributions to the free energy [45]. In a potential independent way, the ground state structure can be investigated by considering the relative free energy difference between these phases. The d-vectors for each of these states are given by:

$$\mathbf{d}_B = \Delta_0 (\sin \theta \cos \phi \hat{x} + \sin \theta \sin \phi \hat{y} + \cos \theta \hat{z})$$

(5.24a)

$$\mathbf{d}_A = \Delta_0 \sin \theta e^{i\phi} \hat{z}$$

(5.24b)

where $\theta$ and $\phi$ are the Euler angles which describe the orbital momentum of the fermion wavefunction. In this description, the preferential direction in spin-space is
taken to be the z-direction in orbital space. Systems for which spin-orbit coupling is
important require more careful consideration about the relationship between direc-
tionality in spin-space and orbital-space; such systems are not considered here.

Using the effective field parameter $B$ defined in Equation 5.10 and substituting
these into Equation (5.22), we obtain:

$$\Delta \tilde{\mathcal{F}}_{B-phase} = -\alpha |\Delta_0|^2 \left( 1 + \frac{\tilde{\alpha}}{\alpha} B^2 \cos^2(\theta) - \frac{\beta_0}{\alpha} |\Delta_0|^2 \right)$$  \hspace{1cm} (5.25a)

$$\Delta \tilde{\mathcal{F}}_{A-phase} = -\alpha |\Delta_0|^2 \sin^2(\theta) \left( 1 + \frac{\tilde{\alpha}}{\alpha} B^2 - \frac{\beta_0}{\alpha} |\Delta_0|^2 \sin^2(\theta) \right)$$  \hspace{1cm} (5.25b)

where $\beta_0 = 2(\beta_1 + \beta_2) + \beta_3 + \beta_4 + \beta_5$.

In the balanced population case, the B-phase is the ground state of the system
in the absence of an applied field. The A-phase is the next lowest energy state, and
is only stabilized in the presence of high pressure. Figure 5.3(a) shows this structure
as a function of the angle $\theta$ and the parameter $\beta_0$. This angle specifies to the phase
space angle where the order parameter has a node, i.e. the angle for which the gap
vanishes. The value of the gap has been chosen such that $|\Delta_0|^2 = 4$ and the parameter
$\alpha = 1$ in the figure. The black plane corresponds to $\Delta F = 0$; the red surface is the
free energy difference of the A-phase from the normal state; and the blue surface is
the free energy difference of the B-phase from the normal state. For large values of $\beta_0$
the A-phase has the lowest free energy; for small values the B-phase has the lowest
free energy.

The cross over value of $\beta_0$ is determined by setting the free energy difference
Figure 5.3: Free energy difference between superfluid states with B-phase (blue) and A-phase (red) order parameters and the normal state, plotted as a function of the angular momentum angle $\theta$ and the ratio of the parameters $\beta_0$ and $\alpha$. The black plane at $\Delta F = 0$ denotes the point where the free energy of the superfluid state is the same as the normal state. (a) No effective internal field (b) Small effective internal field.
between the B-phase and the A-phase equal to zero. This occurs when:

$$\beta_0 = \frac{-\alpha \cos^2(\theta) - \tilde{\alpha} B^2 \cos(2\theta)}{|\Delta_0|^2 (\sin^4(\theta) - 1)}$$  \hspace{1cm} (5.26)

Taking the $B = 0$ case (no internal field) this expression can be plotted as a function of $\theta$, which is shown in Figure 5.4(a). For values of $\beta_0$ which are smaller than the curve, the B-phase has a lower energy; larger values of $\beta_0$ lead to the A-phase having lower energy. This shows that even if the population imbalance does not cause an effective internal field, the ground state structure can change if the polarization changes the ratio of $\beta_0$ to $\alpha$ sufficiently. As $\frac{\beta_0}{\alpha} \rightarrow \infty$ the angle $\theta$ where $\Delta \Phi_A = 0$ goes to 0 or $\pi$. This corresponds to the angle of the node in the A-phase gap.

![Figure 5.4](image)

(a) $B=0$  \hspace{1cm} (b) $B=0.01$

Figure 5.4: Critical value of $\beta_0$ for which the imbalanced population Superfluid A and B-phases have the same free energy for zero internal field (a) and non-zero internal field (b).

For the case where the population imbalance induces an effective internal field,
the ground state structure changes significantly. In population imbalanced quasi-spin systems which have an induced internal field \((B \neq 0)\), the expression (5.26) has a singularity at \(\theta = \frac{\pi}{2}\). This is shown in Figure 5.4(b). The consequence of this singularity is that the A-phase has a lower free energy for all values of \(\beta_0\), as shown in Figure 5.3(b).

To examine in detail the effect of the internal field, consider Figure 5.5. These figures show slices of Figures 5.3(a) and 5.3(b), respectively, taken at various values of \(\beta_0\). The induced internal field (which is a measure of the polarization) acts to bend the free energy curve for the B-phase and suppress the appearance of the double well which appears in the free energy curve for the A-phase to larger values of \(\beta_0\). The appearance of this double well in the A-phase free energy coincides with the value of \(\beta_0\) for which the A-phase and the B-phase have the same free energy.

5.5 Anisotropic Spin-Dependent Interactions

Now consider the effects of an anisotropic spin-dependent interaction in population imbalanced systems. The two-body interaction described by Equation (5.3), has spin-dependent terms:

\[
\Gamma^{xy}_s(R) \left[ \sigma_{\alpha\gamma}^x \sigma_{\beta\delta}^x + \sigma_{\alpha\gamma}^y \sigma_{\beta\delta}^y \right] + \Gamma^z_s(R) \left[ \sigma_{\alpha\gamma}^z \sigma_{\beta\delta}^z \right]
\]

Using the following relationships between Pauli matrices:

\[
\left[ \sigma_{\alpha\gamma}^x \sigma_{\beta\delta}^x + \sigma_{\alpha\gamma}^y \sigma_{\beta\delta}^y \right] = \frac{1}{2} \left[ \sigma_{\alpha\gamma}^+ \sigma_{\beta\delta}^- + \sigma_{\alpha\gamma}^- \sigma_{\beta\delta}^+ \right] = \frac{1}{2} \left[ S_{\alpha\gamma}^+ S_{\beta\delta}^- + S_{\alpha\gamma}^- S_{\beta\delta}^+ \right]
\]
Figure 5.5: Free energy difference of the B-phase and A-phase order parameters from the normal state for particular values of $\beta_0$ both with and without an induced internal field $B$. 
Equation (5.3) can be rewritten as:

\[
\frac{1}{2} U^{(2)}_{\alpha\beta,\delta\gamma}(\mathbf{R}) = \Gamma_\rho(\mathbf{R}) [\delta_{\alpha\gamma}\delta_{\beta\delta}] + 2\Gamma_{xy}(\mathbf{R}) \left[ S^+_{\alpha\gamma} S^-_{\beta\delta} + S^-_{\alpha\gamma} S^+_{\beta\delta} \right] + \Gamma_z(\mathbf{R}) [\sigma^z_{\alpha\gamma} \sigma^z_{\beta\delta}] \quad (5.27)
\]

The various possibilities for this quantity are enumerated in Table 5.1. All terms not listed in the table are identically zero. When substituting these expressions into

<table>
<thead>
<tr>
<th>Potential</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\frac{1}{2} U^{(2)}_{11,11}(\mathbf{R}))</td>
<td>(\Gamma_\rho(\mathbf{R}) + \Gamma_z(\mathbf{R}))</td>
</tr>
<tr>
<td>(\frac{1}{2} U^{(2)}_{11,11}(\mathbf{R}))</td>
<td>(\Gamma_\rho(\mathbf{R}) + \Gamma_z(\mathbf{R}))</td>
</tr>
<tr>
<td>(\frac{1}{2} U^{(2)}_{11,11}(\mathbf{R}))</td>
<td>(\Gamma_\rho(\mathbf{R}) - \Gamma_z(\mathbf{R}))</td>
</tr>
<tr>
<td>(\frac{1}{2} U^{(2)}_{11,11}(\mathbf{R}))</td>
<td>(\Gamma_\rho(\mathbf{R}) - \Gamma_z(\mathbf{R}))</td>
</tr>
<tr>
<td>(\frac{1}{2} U^{(2)}_{11,11}(\mathbf{R}))</td>
<td>(2\Gamma_{xy}(\mathbf{R}))</td>
</tr>
<tr>
<td>(\frac{1}{2} U^{(2)}_{11,11}(\mathbf{R}))</td>
<td>(2\Gamma_{xy}(\mathbf{R}))</td>
</tr>
</tbody>
</table>

Table 5.1: Evaluation of the two body terms for the mean and pair potentials.

Equations (4.9a) and (4.9b) care must be taken to correctly sum over \(\delta\) and \(\gamma\). The pair potential has the free summation indices \(\delta\) and \(\gamma\) both in the initial spin states, whereas the mean potential has one summation index in the initial state and one in the final state.

As in the balanced population case, the mean potentials can be related to the Landau parameters. In this case, define the following integral operators (where the integrals are understood to act on the quantity \(\left\langle \hat{\Psi}_{\beta}^+ (\mathbf{r}^\prime) \hat{\Psi}_{\alpha} (\mathbf{r}^\prime) \right\rangle\) appearing in (4.9a))
These definitions, together with the results from Table 5.1 allow us to write the mean potentials as:

\[
\begin{align*}
U^{(2)}_{↑↑}(\mathbf{R}) &= \frac{1}{2} \left\{ [f^{sp}(\mathbf{R}) + f^{a}(\mathbf{R})] \langle \tilde{\Psi}^{\dagger}_{↑}(\mathbf{r}') \tilde{\Psi}_{↑}(\mathbf{r}) \rangle \\
&+ [f^{sa}(\mathbf{R}) - f^{a}(\mathbf{R})] \langle \tilde{\Psi}^{\dagger}_{↑}(\mathbf{r}') \tilde{\Psi}_{↓}(\mathbf{r}) \rangle \right\} \\
U^{(2)}_{↑↓}(\mathbf{R}) &= \frac{1}{2} \left\{ f^{a}(\mathbf{R}) \langle \tilde{\Psi}^{\dagger}_{↑}(\mathbf{r}') \tilde{\Psi}_{↓}(\mathbf{r}) \rangle \right\} \\
U^{(2)}_{↓↑}(\mathbf{R}) &= U^{(2)}_{↑↓}(\mathbf{R}) \text{ where } ↑→↓ \\
U^{(2)}_{↓↓}(\mathbf{R}) &= U^{(2)}_{↑↑}(\mathbf{R}) \text{ where } ↑→↓
\end{align*}
\]

Here we have used the fact that \( f^{(i)}(\mathbf{R}) = f^{(i)}(-\mathbf{R}) \) and written them in terms of half of the sum of the two. In the balanced population limit, \( f^{sp} = f^{sa} = f^{a} \) which is the form of the isotropic case.
To examine the effect of this anisotropy on pairing interactions, define the following quantities for convenience:

\[ V^{tp}(\mathbf{R}) = [\Gamma^\rho(\mathbf{R}) + \Gamma^z_s(\mathbf{R})] \] (5.30a)

\[ V^{ta}(\mathbf{R}) = [\Gamma^\rho(\mathbf{R}) - \Gamma^z_s(\mathbf{R}) + 2\Gamma^{xy}_s(\mathbf{R})] \] (5.30b)

\[ V^s(\mathbf{R}) = [\Gamma^\rho(\mathbf{R}) - \Gamma^z_s(\mathbf{R}) - 2\Gamma^{xy}_s(\mathbf{R})] \] (5.30c)

We do this so we can make comparisons to an analysis using the singlet-triplet separation. Here (5.30c) is the singlet potential, while (5.30a) and (5.30b) are the triplet potentials. Note that for the case where \( \Gamma^z_s(\mathbf{R}) = \Gamma^{xy}_s(\mathbf{R}) \) the two triplet potentials are equal, leaving a single triplet, as in the isotropic case. Using these definitions and the fermion anti-commutation relations, the pair potentials (4.9b) are then:

\[ \Delta^{\uparrow\uparrow}(\mathbf{R}) = V^{tp}(\mathbf{R}) \left[ \langle \Psi_{\uparrow}(\mathbf{r}') \Psi_{\uparrow}(\mathbf{r}) \rangle - \langle \Psi_{\uparrow}(\mathbf{r}) \Psi_{\uparrow}(\mathbf{r}') \rangle \right] \] (5.31a)

\[ \Delta^{\uparrow\downarrow}(\mathbf{R}) = V^{ta}(\mathbf{R}) \left[ \langle \Psi_{\uparrow}(\mathbf{r}') \Psi_{\uparrow}(\mathbf{r}) \rangle + \langle \Psi_{\uparrow}(\mathbf{r}) \Psi_{\downarrow}(\mathbf{r}') \rangle \right] \] (5.31b)

\[ + V^s(\mathbf{R}) \left[ \langle \Psi_{\downarrow}(\mathbf{r}') \Psi_{\uparrow}(\mathbf{r}) \rangle - \langle \Psi_{\uparrow}(\mathbf{r}') \Psi_{\downarrow}(\mathbf{r}) \rangle \right] \]

\[ \Delta^{\downarrow\downarrow}(\mathbf{R}) = \Delta^{\uparrow\uparrow}(\mathbf{R}) \text{ where } \uparrow \rightarrow \downarrow \] (5.31c)

\[ \Delta^{\downarrow\uparrow}(\mathbf{R}) = \Delta^{\uparrow\downarrow}(\mathbf{R}) \text{ where } \uparrow \rightarrow \downarrow \] (5.31d)

For completeness, the Fourier transformations of the anisotropic mean and pair potentials are presented in Appendix B.

Anisotropic spin-dependent interactions may have interesting effects on both the normal phase as well as the superfluid properties of a material. The normal state properties are influenced only by the mean potentials, which can be related to the
Landau parameters. The anti-symmetric Landau Fermi-Liquid parameter remains unchanged in form, although there is a small correction due to the anisotropy. This effect could be seen in spin-current experiments as a correction to the $F_1^a$ parameter, as well as in spin-susceptibility experiments which measure $F_0^a$. The effect on the symmetric Fermi-Liquid parameters may be more dramatic. This parameter splits into two pieces $f^{sp}$ and $f^{sa}$, which are associated with intra and inter-species scattering in the Fermi-Liquid, respectively. This splitting occurs because the population imbalance creates an asymmetry in the Fermi surfaces of the two species, which can lead to different effective masses for each species. This effect could be observed in specific heat experiments.

There are significant effects on the superfluid properties as well. The singlet order parameter receives a small correction due to the spin-dependent anisotropy. For this reason it is expected that s-wave superfluids will not be dramatically affected by this anisotropy. Triplet superfluids, may experience a variety of new interesting physics. Anisotropic spin-dependent interactions cause the triplet component of the pair potential is split into two pieces: one which affects the intra-species pairing, and one which affects inter-species pairing in the triplet state. This is shown in Equations 5.30a and 5.30b. In such a system the consequences for pairing are two-fold: pairs of the same species ($\uparrow\uparrow$ or $\downarrow\downarrow$) have a different gap than pairs of different species ($\uparrow\downarrow$). Because of these different gaps, the complexity of the coupling between the number equations and the gap equations will increase. For this reason, ultra-cold atomic
systems may be an ideal place to explore anisotropic spin-dependent interactions. By properly tuning a Feschbach resonance, the coupling strength to each part of the order parameter can be well controlled. In this way one can selectively turn on and turn off various parts of the interaction by tuning the resonance. This might allow for a clean way to experimentally characterize the anisotropy in the spin-dependent interaction.
I have presented a general formalism for describing some of the ground-state properties of Fermi superfluids with arbitrary population imbalance. Starting from a general model-independent form of the 2-body interaction, a mean field theory was employed to separate the Fermi-Liquid and pairing interactions for Fermi systems with arbitrary population imbalance. This work has been done in a model independent manner in the interest of generality, making the results applicable to a wide variety of systems ranging from color superconductivity in neutron stars to spin-polarized He$^3$. The key results are highlighted and discussed below.

The Bogolon excitation spectra for both singlet and triplet paired superfluids were calculated. The Bogoliubov equations were used to calculate the superfluid excitation spectrum of both the singlet and triplet interaction symmetries for the population imbalanced case. Population imbalance causes a degeneracy to be lifted in both the singlet and triplet excitation spectra. Singlet paired superfluids with population imbalances have the excitation spectra split by an amount $2\xi_-(k)$. The triplet excitation spectrum is split by three terms: two affect both unitary states and non-unitary states, and one affects only non-unitary states. The splitting affecting both cases is linearly dependent on $\xi_-(k)$. 

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The Ginzburg-Landau free energy was calculated for the A-phase and B-phase triplet order parameters. A GL-free energy calculation was performed for general two-body potentials with arbitrary population imbalance on the known lowest energy unitary p-wave triplet states, the A-phase and B-phase. Using the d-vector formalism, the energy difference for each of these phases was calculated and compared to each other as well as the normal state. When the population imbalance creates an effective internal field, this lowers the free energy of the A-phase below that of the B-phase. It was seen that the lowest energy state depends on the relative strength of two parameters $\alpha$ and $\beta_0$, as well as the angle $\theta$ describing the angular momenta of the fermions.

Anisotropic spin-dependent interactions in population imbalanced superfluids were described. Anisotropy in the spin-dependent two-body interaction has been shown to have effects on both singlet and triplet paired superfluids. In the singlet case, the anisotropy causes a small correction to the gap parameter; the triplet order parameter splits into two pieces: one associated with the intra-species gap, and one with the inter-species gap. Triplet p-wave superfluids with anisotropic spin-dependent interactions will therefor have two gaps associated with different pair species. Additionally, there is a splitting in the symmetric Landau Fermi liquid parameter. This should be observable in experiments which can measure effective mass, as well as specific heat measurements.

These model independent results provide an excellent framework to extend the
work which has been presented here. In the future, a complete study of all 18 possible p-wave states could be carried out in a model independent manner. To date this has not been done, and many of the non-unitary states have not been realized physically. The ability to obtain arbitrary population imbalances in cold atoms systems may make these non-unitary states easier to realize in p-wave superfluids. It would also be very interesting to apply this formalism to the problem of color superconductivity, to understand the effects of large population asymmetries in a three species model. Finally it should be possible to apply this framework to highly spin-polarized He$_3$ superfluids. This formalism is an excellent place to begin the numerical work to make predictions in these systems.
BIBLIOGRAPHY


APPENDIX A

Momentum Space Hamiltonian

Here I present the calculation of the momentum space Hamiltonian. The Hamiltonian can be separated into three distinct terms, writing it as:

\[ \hat{H} = \hat{H}_{BP} + \hat{H}_{MP} + \hat{H}_{PP} \]  

(A.1)

where the subscripts refer to the bare particle, mean potential and pair potential terms, respectively. Each term is considered separately.

A.1 Bare Particle Term

The Bare Particle term given by:

\[ \hat{H}_{BP} = \sum_{\alpha,\beta} \int d^3r \ \hat{\Psi}_\alpha^\dagger(r) \hat{\xi} \hat{\Psi}_\alpha(r) \]  

(A.2)

Here we have explicitly noted that the bare fermion energy \( \xi_\alpha \) has no spatial dependence and is a constant for the purposes of integration. Substituting (4.12) into (A.2):

\[ \hat{H}_{BP} = \sum_{\alpha,\beta} \int d^3r \ e^{-i(k-k') \cdot r} L^3 c_{k'\alpha}^\dagger \hat{\xi}(k) c_{k\alpha} \]

where now the momentum dependence of the bare-particle energy is stated explicitly. Performing the integration over \( r \) and the sum over \( k' \), the bare-particle term
becomes:

$$\hat{H}_{BP} = \sum_{k,\alpha} \xi^0(k) \hat{c}^\dagger_{k\alpha} \hat{c}_{k\alpha}$$  \hspace{1cm} (A.3)$$

where

$$\xi^0(k) = \left[ \frac{k^2}{2m} - \mu \right]$$

is the bare free particle energy of a fermion with momentum $k$ and spin $\alpha$.

A.2 Mean Potential Term

The mean potential term is given by:

$$\hat{H}_{MP} = \sum_{\alpha,\beta} \int d^3r \int d^3r' \left[ U^{(2)}_{\alpha\beta}(R) + U^{(1)}_{\alpha\beta}(r) \delta(R) \right] \hat{\psi}^\dagger_{\beta}(r') \hat{\psi}_{\alpha}(r)$$  \hspace{1cm} (A.4)$$

Substituting for the field operators and simplifying:

$$\hat{H}_{MP} = \sum_{\alpha,\beta} \sum_{k,k'} \int d^3R \int d^3r' \ e^{-ik\cdot r} \ e^{i k' \cdot r'} \ \frac{L}{3^{3/2}} \left[ U^{(2)}_{\alpha\beta}(R) + U^{(1)}_{\alpha\beta}(r) \delta(R) \right] \hat{c}^\dagger_{k'\beta} \hat{c}_{k\alpha}$$

Now consider each term separately. First, the one-body term:

$$\hat{H}_{MP1} = \sum_{\alpha,\beta} \sum_{k,k'} \int d^3R \int d^3r' \ e^{-ik\cdot r} \ e^{i k' \cdot r'} \ \frac{L}{3^{3/2}} \left[ U^{(1)}_{\alpha\beta}(r) \delta(R) \right] \hat{c}^\dagger_{k'\beta} \hat{c}_{k\alpha}$$

Integrating the delta function, and then performing the remaining Fourier transform, this becomes:

$$\hat{H}_{MP1} = \sum_{\alpha,\beta} \sum_{k,k'} U^{(1)}_{\alpha\beta}(k-k') \hat{c}^\dagger_{k'\beta} \hat{c}_{k\alpha}$$

Finally, the two-body term:

$$\hat{H}_{MP2} = \sum_{\alpha,\beta} \sum_{k,k'} \int d^3R \int d^3r' \ e^{-ik\cdot r} \ e^{i k' \cdot r'} \ \frac{L}{3^{3/2}} \left[ U^{(2)}_{\alpha\beta}(R) \right] \hat{c}^\dagger_{k'\beta} \hat{c}_{k\alpha}$$
Integrating with respect to $R$ and $r'$ gives:

$$
\hat{H}_{MP2} = \sum_{\alpha, \beta} \sum_{k, k'} U^{(2)}_{\alpha \beta}(k) \delta^3(k - k') \hat{c}^\dagger_{k', \beta} \hat{c}_{\alpha}
$$

Putting these together, the mean potential term becomes:

$$
\hat{H}_{MP} = \sum_{\alpha, \beta} \sum_{k, k'} \left[ U^{(1)}_{\alpha \beta}(k - k') + U^{(2)}_{\alpha \beta}(k) \delta^3(k - k') \right] \hat{c}^\dagger_{k', \beta} \hat{c}_{\alpha}
$$

(A.5)

### A.3 Pair Potential Terms

Consider the pair potential terms, given by:

$$
\hat{H}_{PP} = \int d^3r \int d^3r' \left[ \Delta^*_{\alpha \beta}(R) \hat{\psi}^\dagger_\beta(r') \hat{\psi}_\alpha(r) + \Delta_{\alpha \beta}(R) \hat{\psi}^\dagger_\alpha(r) \hat{\psi}^\dagger_\beta(r') \right] - \Delta^*_{\alpha \beta}(R) \left\langle \hat{\psi}^\dagger_\beta(r') \hat{\psi}^\dagger_\alpha(r) \right\rangle
$$

(A.6)

Substituting for the field operators and simplifying as above, this becomes:

$$
\hat{H}_{PP} = \sum_k \Delta^*_{\alpha \beta}(k) \hat{c}_{-k, \beta} \hat{c}^\dagger_{k, \alpha} + \Delta_{\alpha \beta}(k) \hat{c}^\dagger_{k, \alpha} \hat{c}_{-k, \beta} - \Delta^*_{\alpha \beta}(k) \left\langle \hat{c}_{-k, \beta} \hat{c}^\dagger_{k, \alpha} \right\rangle
$$

(A.7)

The mean potentials are given by [46]:

$$
U^{(2)}_{\uparrow \uparrow}(k) = \frac{1}{2V} \sum_{k'} \left\{ [f^s(k, k') + f^a(k, k')] \left\langle \hat{c}^\dagger_{k', \uparrow} \hat{c}_{k, \uparrow} \right\rangle + [f^s(k, k') - f^a(k, k')] \left\langle \hat{c}^\dagger_{k', \downarrow} \hat{c}_{k, \downarrow} \right\rangle \right\}
$$

$$
U^{(2)}_{\uparrow \downarrow}(k) = \frac{1}{2V} \sum_{k'} f^a(k, k') \left\langle \hat{c}^\dagger_{k', \uparrow} \hat{c}_{k, \downarrow} \right\rangle
$$

$$
U^{(2)}_{\downarrow \downarrow}(k) = U^{(2)}_{\uparrow \uparrow}(k) \text{ with spin indices interchanged.}
$$

$$
U^{(2)}_{\downarrow \uparrow}(k) = U^{(2)}_{\uparrow \downarrow}(k) \text{ with spin indices interchanged.}
$$

(A.8)

where

$$
f^s(k, k') = 2\Gamma_\rho(0) - \Gamma_\rho(k - k') - 3\Gamma_s(k - k')
$$

$$
f^a(k, k') = 2\Gamma_s(0) - \Gamma_\rho(k - k') + \Gamma_s(k - k')
$$

(A.9)
The pair potentials are given by [46]:

\[
\Delta_{\uparrow\uparrow}(\mathbf{k}) = \frac{1}{V} \sum_{\mathbf{k}'} V^t(\mathbf{k}, \mathbf{k}') \left[ \langle c_{\mathbf{k}'\uparrow} c_{\mathbf{k}\uparrow} \rangle + \langle c_{\mathbf{k}\uparrow} c_{\mathbf{k}'\uparrow} \rangle \right]
\]

\[
\Delta_{\uparrow\downarrow}(\mathbf{k}) = \frac{1}{V} \sum_{\mathbf{k}'} \left\{ V^t(\mathbf{k}, \mathbf{k}') \left[ \langle c_{\mathbf{k}'\uparrow} c_{\mathbf{k}\downarrow} \rangle + \langle c_{\mathbf{k}\uparrow} c_{\mathbf{k}'\downarrow} \rangle \right] + V^s(\mathbf{k}, \mathbf{k}') \left[ \langle c_{\mathbf{k}'\uparrow} c_{\mathbf{k}\downarrow} \rangle - \langle c_{\mathbf{k}\downarrow} c_{\mathbf{k}'\uparrow} \rangle \right] \right\}
\]

\[
\Delta_{\downarrow\downarrow}(\mathbf{k}) = \text{same as } \Delta_{\uparrow\uparrow}(\mathbf{k}) \text{ with spin indices interchanged.}
\]

\[
\Delta_{\downarrow\uparrow}(\mathbf{k}) = \text{same as } \Delta_{\uparrow\downarrow}(\mathbf{k}) \text{ with spin indices interchanged.}
\]

where

\[
V^t(\mathbf{k}, \mathbf{k}') = \Gamma_\rho(\mathbf{k} - \mathbf{k}') + \Gamma_s(\mathbf{k} - \mathbf{k}') - \Gamma_\rho(\mathbf{k} + \mathbf{k}') - \Gamma_s(\mathbf{k} + \mathbf{k}')
\]

\[
V^s(\mathbf{k}, \mathbf{k}') = \Gamma_\rho(\mathbf{k} - \mathbf{k}') - 3\Gamma_s(\mathbf{k} - \mathbf{k}') + \Gamma_\rho(\mathbf{k} + \mathbf{k}') - 3\Gamma_s(\mathbf{k} + \mathbf{k}')
\]
APPENDIX B

Fourier Transforms of Anisotropic Potentials

In this appendix, the full calculation of the Fourier Transformations of the anisotropic mean and pair potentials is presented. Section B.1 details the calculation of the mean potentials and Section B.2 details the calculation of the pair potentials.

B.1 Mean Potentials

Begin by considering the mean potential term $U^{11}(\mathbf{R})$. In this calculation the one-body contribution is neglected, but including it is a straightforward extension of the method described below. With this caveat, one can then write the following:

$$U^{11}(\mathbf{k}) = \int d^3 \mathbf{R} \ e^{-i \mathbf{k} \cdot \mathbf{R}} U^{(2)}_{11}(\mathbf{R})$$  \hspace{1cm} (B.1)

Substituting (5.29b), we have:

$$U^{11}(\mathbf{k}) = \int d^3 \mathbf{R} \ e^{-i \mathbf{k} \cdot \mathbf{R}} \frac{1}{2} \left\{ f^{(a)}(\mathbf{r} - \mathbf{r}') \left\langle \hat{\psi}_1^+(\mathbf{r}') \hat{\psi}_1(\mathbf{r}) \right\rangle \right\}$$

Substituting the integral operator gives:

$$U^{11}(\mathbf{k}) = \frac{1}{2} \int d^3 \mathbf{R} \ e^{-i \mathbf{k} \cdot \mathbf{R}} \left[ -\Gamma_\rho(\mathbf{r} - \mathbf{r}') + \Gamma_\phi^z(\mathbf{r} - \mathbf{r}') \right] \left\langle \hat{\psi}_1^+(\mathbf{r}') \hat{\psi}_1(\mathbf{r}) \right\rangle$$

$$+ \frac{1}{2} \int d^3 \mathbf{R} \ e^{-i \mathbf{k} \cdot \mathbf{R}} \delta^3(\mathbf{R}) \int d^3 \mathbf{r}'' \left[ 2\Gamma_{xy}(\mathbf{r} - \mathbf{r}'') \right] \left\langle \hat{\psi}_1^+(\mathbf{r}'') \hat{\psi}_1(\mathbf{r}'') \right\rangle$$

The first term is simply:

$$U^{11}_1(\mathbf{k}) \equiv \frac{1}{2} \int d^3 \mathbf{R} \ e^{-i \mathbf{k} \cdot \mathbf{R}} \left\{ -\Gamma_\rho(\mathbf{r} - \mathbf{r}') \left\langle \hat{\psi}_1^+(\mathbf{r}') \hat{\psi}_1(\mathbf{r}) \right\rangle \right\}$$
Expanding the field operators using (4.12) this becomes:

\[ U_{11}^1(k) = \frac{1}{2} \int d^3R \ e^{-ik \cdot R} \left\{ -\Gamma_\rho(R) \sum_{k'} \frac{e^{ik' \cdot R}}{L^3} \left\langle \hat{c}_{k'\downarrow}^\dagger \hat{c}_{k'\uparrow} \right\rangle \right\} \]

Switching the order of integration and summation gives:

\[ U_{11}^1(k) = -\frac{1}{2L^3} \sum_{k'} \left\langle \hat{c}_{k'\downarrow}^\dagger \hat{c}_{k'\uparrow} \right\rangle \int d^3R \ e^{-i(k-k') \cdot R} \Gamma_\rho(R) \]

Performing the final integration yields:

\[ U_{11}^1(k) = -\frac{1}{2L^3} \sum_{k'} \Gamma_\rho(k - k') \left\langle \hat{c}_{k'\downarrow}^\dagger \hat{c}_{k'\uparrow} \right\rangle \]

The second term follows in exactly the same manner:

\[ U_{11}^2(k) = \frac{1}{2L^3} \sum_{k'} \Gamma_{s}(k - k') \left\langle \hat{c}_{k'\downarrow}^\dagger \hat{c}_{k'\uparrow} \right\rangle \]

Finally, examining the third term:

\[ U_{11}^3(k) = \frac{1}{2} \int d^3R \ e^{-ik \cdot R} \delta^3(R) \int d^3r'' \left[ 2\Gamma_{xy}^s(r - r'') \right] \left\langle \hat{\Psi}_{\uparrow}(r'') \hat{\Psi}_{\downarrow}(r'') \right\rangle \]

Performing the same expansion, integrating over \( R \), and rearranging as before gives:

\[ U_{11}^3(k) = \frac{1}{2L^3} \sum_{k'} \left\langle \hat{c}_{k'\downarrow}^\dagger \hat{c}_{k'\uparrow} \right\rangle 2\Gamma_{xy}^s(0) \]

Collecting these terms, the mean potential becomes:

\[ U_{11}(k) = \frac{1}{2L^3} \sum_{k'} \left\langle \hat{c}_{k'\downarrow}^\dagger \hat{c}_{k'\uparrow} \right\rangle \left[ -\Gamma_\rho(k - k') + \Gamma_{s}(k - k') + 2\Gamma_{xy}^s(0) \right] \]

This can be written in terms of the Bogoliubov coefficients using the relation for the density matrix:
Notice that the delta-function in the third term has been dropped. It is understood that the zero-momentum contribution from the potential only contributes when \( k = 0 \).

Identify of the Fourier transforms of the integral operators defined earlier:

\[
f^{a}(k, k') = [-\Gamma_\rho (k - k') + \Gamma_z (k - k') + 2\Gamma_x^y (0)]
\]

\[
f^{sp}(k, k') = [-\Gamma_\rho (k - k') - 3\Gamma_z (k - k') + 2\Gamma_\rho (0) - 2\Gamma_x^y (0) + 2\Gamma_z^y (0)]
\]

\[
f^{sa}(k, k') = [-\Gamma_\rho (k - k') + \Gamma_z (k - k') - 4\Gamma_x^y (k - k') + 2\Gamma_\rho (0) + 2\Gamma_x^y (0) - 2\Gamma_z^y (0)]
\]

Since the calculation of the other mean potentials proceeds in a similar manner, the results are collected below:

\[
U^{\uparrow\uparrow}(k) = \frac{1}{2L^3} \sum_{k'} [f^{sp}(k, k') + f^{a}(k, k')] \langle \hat{c}_{k' \uparrow}^{\dagger} \hat{c}_{k' \uparrow} \rangle + \frac{1}{2L^3} \sum_{k'} [f^{sa}(k, k') - f^{a}(k, k')] \langle \hat{c}_{k' \downarrow}^{\dagger} \hat{c}_{k' \downarrow} \rangle
\]

\[
U^{\uparrow\downarrow}(k) = \frac{1}{2L^3} \sum_{k'} f^{a}(k, k') \langle \hat{c}_{k' \downarrow}^{\dagger} \hat{c}_{k' \uparrow} \rangle
\]

\[
U^{\downarrow\uparrow}(R) = U^{\uparrow\downarrow}(R) \text{ where } \uparrow \rightarrow \downarrow
\]

\[
U^{\downarrow\downarrow}(R) = U^{\uparrow\uparrow}(R) \text{ where } \uparrow \rightarrow \downarrow
\]

B.2 Pair Potentials

As with the mean potentials, the Fourier transform of \( \Delta^{\uparrow\uparrow}(k) \) is presented, and the results for the remainder of the pair potentials are collected at the end. Proceeding as with the mean potentials:

\[
\Delta^{\uparrow\downarrow}(k) = \int d^3R \ e^{-ik \cdot R} \Delta^{\uparrow\downarrow}(R)
\]
Substituting (5.31a) we obtain:

\[
\Delta_{\uparrow\uparrow}(k) = \int d^3R \ e^{-ik \cdot R} \ V^t(R) \left[ \langle \Psi_\uparrow(r') \Psi_\uparrow(r) \rangle - \langle \Psi_\uparrow(r)\Psi_\uparrow(r') \rangle \right]
\]

Expanding the field operators as plane waves in \(k'\) and \(-k'\):

\[
= \frac{1}{L^3} \sum_{k'} \int d^3R \ e^{-ik \cdot R} V^t(R) \left( e^{ik' \cdot R} - e^{-ik' \cdot R} \right) \langle c_{-k'} c_k \rangle
\]

Finally, performing the integration yields:

\[
= \frac{1}{L^3} \sum_{k'} \left[ V^t(k - k') - V^t(k + k') \right] \langle c_{-k'} c_k \rangle
\]

This can be expressed in terms of the Bogoliubov coefficients as:

\[
= \frac{1}{L^3} \sum_{k',\nu} \left[ V^t(k - k') - V^t(k + k') \right] u_{k',\nu} v_{k,\nu} (1 - 2f_{k',\nu})
\]

The remaining pair potentials are calculated in a similar manner and are collected here:

\[
\Delta_{\uparrow\downarrow}(k) = \frac{1}{L^3} \sum_{k',\nu} \left[ V^t(k - k') - V^t(k + k') \right] u_{k',\nu} v_{k,\nu} (1 - 2f_{k',\nu}) \quad (B.7)
\]

\[
\Delta_{\downarrow\uparrow}(k) = \frac{1}{L^3} \sum_{k',\nu} \left[ V^s(k - k') + V^s(k + k') \right] \left[ u_{k',\nu}^* u_{k,\nu} + v_{k',\nu}^* v_{k,\nu} \right] (1 - 2f_{k',\nu})
\]

\[
+ \frac{1}{L^3} \sum_{k',\nu} \left[ V^s(k - k') + V^s(k + k') \right] \left[ v_{k',\nu}^* u_{k,\nu} - u_{k',\nu}^* v_{k,\nu} \right] (1 - 2f_{k',\nu}) \quad (B.8)
\]

\[
\Delta_{\downarrow\downarrow}(k) = \Delta_{\uparrow\uparrow}(k) \text{ where } \uparrow \rightarrow \downarrow \quad (B.9)
\]

\[
\Delta_{\uparrow\downarrow}(k) = \Delta_{\downarrow\uparrow}(k) \text{ where } \uparrow \rightarrow \downarrow \quad (B.10)
\]