Determining a Model of Stomach Acid Regulation
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Abstract
Acid in the stomach is regulated through a negative feedback mechanism, controlled by the interactions of parietal cell, G cells, D cells, ECL cells, mucosal cells, and the central and enteric nervous systems. This system responds to food intake by increase acid production, thus facilitating digestion. This system negatively impacted by Zollinger Ellison syndrome, when gastronomia cause an overproduction of gastrin by the G cells. Previous studies have developed nonlinear models of these interactions in healthy patients based on Michaelis Menton dynamics. In this paper, a linearized model of the system in the corpus region was generated by assuming a operating range near the Michealis Menton saturation. The system was observed in response to different food volume inputs to ensure similar outputs to the previous nonlinear models. Then using an additional constant stimulus of the parietal cells to mimic the overproduction of gastrin, a Zollinger-Ellison disease model was generated. The same food stimuli were used and the resulting disease response was observed. This showed that as the disease progressed, food became less effective in changing the pH of the stomach, and the body's ability to modify pH in response to food stimuli was diminished.

I. Introduction
The gastric phase of the digestive process is characterized by the secretion of gastric juice. Gastric juice contains hydrochloric acid, which plays a vital role in breaking down proteins. A variety of cells along the stomach lining activate the secretion of gastric acid.

Food entering the system stimulates both hormonal and neural activity to aid digestion. The process of hydrochloric acid secretion begins with the nervous system signaling G-cells of a food input and begins a cascade of hormone production. G-cells secrete the hormone gastrin and causes stimulation of the enterochromaffin-like (ECL) cells to secrete histamine. Histamine binds to parietal cells, which then release hydrochloric acid into the stomach. From stimulation by the enteric nervous system, the hormone acetylcholine is produced to stimulate parietal cells. D-cells lining the stomach contain pH receptors that measure the acidity of the stomach and can control the acidity by the release of the hormone somatostatin, which inhibits the secreted aforementioned hormones of G-cells, ECL cells, and parietal cells. Through these mechanisms, the body controls the stomach acid levels throughout periods of digestion and fasting.

Zollinger-Ellison syndrome is a disease characterized by tumors that overstimulate G-cells to produce excess gastrin. This excess gastrin further stimulates parietal cells to overproduce hydrochloric acid. This excess acid can lead to acid reflux, diarrhea, and damage to the digestive tract.

In this paper, we propose a simplified model of gastric acid regulation in both healthy and Zollinger Ellison diseased individuals using simplified parameters and assumptions.

II. Related Work
We are basing the model of our system on a study by Ian MP Joseph, Yana Zavros, Juanita Merchant, Denise Kirschner. In this study, a virtual model of gastrin secretion and regulation was created using food as an input. This model examines the effects of G-cells, D-cells, ECL cells, parietal cells, and their secretions. The model uses cell population as a measure of activity. Additionally, the study takes neurotransmitters from the ENS and CNS into consideration. The gastrin secretion was modeled in both the antral corpus regions.

From this study differential equations modeling the behavior of cells was determined. In this report, the linearized response of the system around the gastrin and histamine saturation ranges are examined.
### III. Methods
The original model proposed by Joseph et al was modified by assuming a linear response within physiological ranges, which allowed for the linearization of the governing equations. To ensure stability in the system, the saturation range of the Michealis Menten dynamics were used as the center of the linearization. This choice of linearization region resulted in the parietal cell dependence on histamine and gastrin being reduced to a constant value, and the feedback connection to the antrum being severed. Under this linear regime, the regulation of stomach acid could be examined only through reactions and dynamics in the cestrum.

It was also assumed that any premature gastric acid production in response to sensory input, such as smell and taste that would result in the basal ganglia stimulation to increase, could be ignored, and therefore the basal ganglia stimulation was a constant.

Using these assumptions and choice of operating range, the following linearization equations were developed and evaluated at the saturation range. (See Appendix for original equations.)

#### Variables:

<table>
<thead>
<tr>
<th>Location</th>
<th>Name</th>
<th>Variable Name</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corpus</td>
<td>Acid Concentration</td>
<td>Ac</td>
<td>mol/L</td>
</tr>
<tr>
<td></td>
<td>Gastrin Concentration</td>
<td>Gc</td>
<td>mol/L</td>
</tr>
<tr>
<td></td>
<td>Somatostatin</td>
<td>Sc</td>
<td>mol/L</td>
</tr>
<tr>
<td></td>
<td>Histamine Concentration</td>
<td>Hc</td>
<td>mol/L</td>
</tr>
<tr>
<td></td>
<td>Bicarbonate Concentration</td>
<td>Bc</td>
<td>mol/L</td>
</tr>
</tbody>
</table>

| Antral         | Acid Concentration    | Aa            | mol/L |
|                | Gastrin Concentration | Ga            | mol/L |
|                | Somatostatin          | Sa            | mol/L |

| Nervous System | Central Nervous System | Nc            | mol/L |
|                | Acetylcholine          | Ne            | mol/L |

| Corpus Gastric Acid: |
\[
\frac{dA_c}{dt} \approx \frac{\partial A_c}{\partial S_c} \cdot S_c(t) + \frac{\partial A_c}{\partial N_c} \cdot N_c(t) + \frac{\partial A_c}{\partial A_c} \cdot A_c(t) + \frac{\partial A_c}{\partial Fd} \cdot Fd(t) + \frac{\partial A_c}{\partial B_c} \cdot B_c(t) \\
\frac{\partial A_c}{\partial S_c} = D_{cell} \left( \frac{k_{NA}N_c(t)}{N_c(t)-a_{NA}} + K_{GA} + K_{HA} \right) \left( \frac{-k_{SA}}{(k_{SA}+S_c)^2} \right) \\
\frac{\partial A_c}{\partial N_c} = \frac{D_{cell}}{k_{NA}a_{NA}} \left( \frac{1}{k_{SA}} \frac{S_c(t)}{N_c(t)-a_{NA}} \right)^2 \\
\frac{\partial A_c}{\partial Fd} = -k_{gA}Fd(t) - k_{gA}F_{cell} \left( \frac{F_{cell}}{F_{cell}+k_{gA}} \right)^2 \\
\frac{\partial A_c}{\partial B_c} = -h_bA_c(t) \\
\]

| Corpus Bicarbonate Concentration: |
\[
\frac{dB_c}{dt} \approx \frac{\partial B_c}{\partial S_c} \cdot S_c(t) + \frac{\partial B_c}{\partial N_c} \cdot N_c(t) + \frac{\partial B_c}{\partial A_c} \cdot A_c(t) + \frac{\partial B_c}{\partial B_c} \cdot B_c(t) \\
\frac{\partial B_c}{\partial S_c} = \frac{k_{PB}a_{NB}}{\frac{1}{k_{PB}} + \frac{1}{k_{PB}a_{NB}} + \frac{1}{k_{PB}a_{NB}S_c(t)}} \\
\frac{\partial B_c}{\partial N_c} = \frac{1}{N_c(t)-a_{NB}} \left( \frac{k_{PB}a_{NB}}{\frac{1}{k_{PB}} + \frac{1}{k_{PB}a_{NB}} + \frac{1}{k_{PB}a_{NB}S_c(t)}} \right)^2 \\
\frac{\partial B_c}{\partial A_c} = \frac{k_{PB}a_{NB}}{\frac{1}{k_{PB}} + \frac{1}{k_{PB}a_{NB}} + \frac{1}{k_{PB}a_{NB}S_c(t)}} \\
\frac{\partial B_c}{\partial B_c} = -h_bA_c(t) - \beta_b \\
\]

| Corpus Somatostatin: |
\[
\frac{dS_s}{dt} \approx \frac{\partial S_s}{\partial S_c} \cdot S_c(t) + \frac{\partial S_s}{\partial N_c} \cdot N_c(t) + \frac{\partial S_s}{\partial N_e} \cdot N_e(t) \\
\frac{\partial S_s}{\partial S_c} = D_{cell} \left( \frac{k_{NS}N_c(t)}{N_e(t)+a_{NS}} + K_{GS} \right) \left( \frac{-k_{NS}}{(k_{NS}+S_c)^2} \right) \\
\frac{\partial S_s}{\partial N_c} = \frac{D_{cell}}{k_{NS}a_{NS}} \left( \frac{1}{k_{NS}} \frac{S_c(t)}{N_e(t)+a_{NS}} \right)^2 \\
\frac{\partial S_s}{\partial N_e} = \frac{D_{cell}}{k_{NS}a_{NS}} \left( \frac{1}{k_{NS}} \frac{N_e(t)}{N_e(t)+a_{NS}} \right)^2 \\
\frac{\partial S_s}{\partial Fd} = \frac{D_{cell}}{k_{NS}a_{NS}} \left( \frac{1}{k_{NS}} \frac{N_e(t)}{N_e(t)+a_{NS}} \right)^2 \left( \frac{k_{NS}a_{NS}}{(a_{NS}+N_e(t))^2} \right) \\
\]

| Central Neural Activation: |
\[
\frac{dN_c}{dt} \approx \frac{\partial N_c}{\partial Fd} \cdot Fd(t) - k_{Ne}N_c(t) + Bas_1 \\
\frac{\partial N_c}{\partial Fd} = \frac{k_{max_1}}{(Fd(t)+k_{Fd})^2} \\
\]

| Enteric Neural Activation: |
\[
\frac{dN_e}{dt} \approx \frac{\partial N_e}{\partial Fd} \cdot Fd(t) - k_{Ne}N_e(t) + Bas_2 \\
\frac{\partial N_e}{\partial Fd} = \frac{k_{max_2}}{(Fd(t)+k_{Fd})^2} \\
\]

These equations were then used to a Simulink model of the stomach. This system was built of subsystems that incorporated the dynamics of the parietal cells, mucosal cells, D cells, and the central and enteric nervous systems. The schematic of this model can be seen in Figure 2.

The Laplace transform of these equations was also taken to develop the transfer functions of the system. As this system was controlled by three independent inputs (food...
volume, Fd(t), central basal ganglia activation, Bas1(t), and enteric basal ganglia activation, Bas2(t), this system is a MISO (multi-input, single output) system and thus requires three independent transfer functions. The total transfer function can be written:

\[ A_c(s) = X(s)Bas1(s) + Y(s)Bas2(s) + Z(s)Fd(s) \]

Where:

\[ X(s) = \frac{A_c(s)}{Bas1(s)} = \frac{459.4s^2 - 7596s + 17270}{s^4 - 18.3s^3 + 63.5s^2 - 19.75s - 102.5} \]

\[ Y(s) = \frac{A_c(s)}{Bas2(s)} = \frac{4.11s + 4.38}{s^4 - 18.3s^3 + 63.5s^2 - 19.75s - 102.5} \]

\[ Z(s) = \frac{A_c(s)}{Fd(s)} = \frac{0.01952s - 0.03237s + 0.0768}{s^4 - 18.3s^3 + 63.5s^2 - 19.75s - 102.5} \]

Using these transfer functions, the Bode response of the system to each stimulus could be independently observed.

For the Simulink experiments, the Bas1 and Bas2 levels were set such that the equilibrium pH of the stomach was 2.4. To observe the response induced by food, three “meals” were introduced to the system with different durations and food volumes. The associated change in stomach pH to accommodate this increase stomach volume and to facilitate digestion was observed.

To examine the effects of Zollinger Ellison Syndrome on the body’s ability to regulate stomach acid concentrations, a steady stimulus was added to the parietal subsystem to model the effect of the gastrinoma on the secretion of stomach acid. The level of stimulus was changed so that the resulting equilibrium change and regulator effects of the body could be observed at different stages of the disease.

IV. Results

The Simulink model that was developed for this investigation is shown in Figure 2. The output of this model for both healthy and diseased states are also shown responding to three meals of three different sizes at different times throughout the day is shown in Figure 3. The system responses are shown in the Bode Plots of Figure 4 which were found using the transfer functions X(s), Y(s), and Z(s). These transfer functions and Bode Plots were made using MATLAB 2020b.

![Simulink Model of Linearized Gastric Acid Regulation](image.png)

Figure 2: Simulink Model of Linearized Gastric Acid Regulation. This figure shows the full Simulink model that was developed to simulate the body’s linear gastric acid regulation in the saturation region. This model was also used to determine the effects of Zollinger Ellison Syndrome on stomach acid equilibrium concentration and the body’s ability to regulate acid within this disease state. Each subsystem in the model is used to capture the dynamics of one cell type or one section of the nervous system.

V. Discussion

From the graphs in Figure 3, we were able to see stomach pH decrease proportionally to food volume intake. Stomach pH between 2.4 and 2.1 falls within expected ranges of stomach pH during digestion. Additionally, these sudden decreases in pH correspond to increased digestive activity as the body breaks down the food. This relationship indicates that our model developed is valid given the parameters used.

To create a Zollinger-Ellison disease model, a constant concentration of acid input was added to the acid output of parietal cells. This constant concentration of acid
would simulate the overproduction of hydrochloric acid due to gastrin overproduction from the gastronomes that characterize the disease.

Figure 3 also models a sudden onset and proliferation of ZES on a subject. The first day is a healthy model of stomach pH as a result of food stimuli, the second day displays the onset of Zollinger Ellison disease, and the third day displays a critical progression of the disease model. The onset and progression of the disease is input as increasing magnitudes of added acid to the parietal cell output. From the figure, the regulatory functions of the patient began to fail as the disease progressed.

In our disease model in Figure 3, two different magnitudes of the Zollinger-Ellison disease were input. It should be noted that with a stronger magnitude of Zollinger-Ellison disease, food input began to have a decreasing impact on stomach pH in comparison to the disease input. Instead, the disease caused G cells to overproduce gastrin, which in turn overstimulated parietal cells to produce more gastric acid. The effects of the overproduction of acid from the disease overpowered the system’s regulatory system, and the steady state pH decreased.

Future studies would need to be done regarding updating model parameters and simplifications to lie more closely to physiological ranges/systems. While the base model displayed relevant results, the many assumptions we made in creating this model adds instability to the system when operating outside of the parameters we’ve set such as the beginning of Michaelis-Menten interactions.

Our model ignores the effects of stimuli other than volumetric food intake, the regulatory processes facilitated by gastrin, and cells within the antrum portion of the stomach. Because of the simplifications present in our model, this model cannot be considered reliable for use in comprehensive studies. Additional improvements to our model would include incorporation of other stimuli and sub-processes involved in the gastric acid regulation model. Another improvement would be to include cell population dynamics over a larger period of multiple days, as cell populations have relatively little change over 24 hours.

VI. References
Figure 4: Bode Plots comparing acid between each of the two nervous system inputs and the food stimulus input. The Bode Plots show the system behaves as a low pass filter with varying cutoff frequencies and gains to the response of the system, depending on the input. The phase shifts also vary between inputs. (A) corresponds to Bas1 input, (B) corresponds to Bas2 input, and (C) corresponds to food input.

MATLAB code:

```
% 122A Project: A Model of Gastric Acid Regulation in Both Healthy and Zollinger Ellison Syndrome Systems

% Determining the linearization coefficients

MM = @(x,v,k) v*x/(x+k); % General form of the Michealis Menten
dMM = @(x,v,k) v*k/(x+k)^2; % General MM derivative
AC = @(A,k) 1 + (A^2)/(A^2 + k^2); % acetylcholine func
dAC = @(A,k) -2*(k^2)*A/(A^2 + k^2)^2; % acetylcholine derivative
M2 = @(x,k) 1+(x/k); % simplified MM
dM2 = @(x,k) -k/(k+x)^2; % simplified MM derivative

% Steady state cell populations
G_cell = 8.75e6;
Da_cell = 3.70e6;
E_cell = 8.68e6;
Dc_cell = 2.69e8;
P_cell = 1e8;

k_NG1 = 6.28e-17;
k_NG2 = 8.75e-17;
a_NG1 = 1e-10;
a_NG2 = 1e-10;
k_SG = 9e-11;
```
\[ k_G = 11.88; \]
\[ \text{beta}_G = 1.5; \]
\[ kG\_plus\_betaG = k_G + \text{beta}_G; \]
\[ k_{AS} = 8.04e-15; \]
\[ k_{GS} = 2.54e-18; \]
\[ a_{AS} = 0.05; \]
\[ a_{GS} = 5.20e-12; \]
\[ k_{NS} = 1e-9; \]
\[ k_{S} = 13.86; \]
\[ k_{NS1} = 1.14e-15; \]
\[ k_{NS2} = 1.54e-17; \]
\[ a_{NS1} = 6.28e-7; \]
\[ a_{NS2} = 8.98e-11; \]
\[ k_{ss} = 9e-11; \]
\[ k_{NH} = 7.59e-16; \]
\[ k_{GH} = 7.77e-16; \]
\[ a_{NH} = 3.25e-8; \]
\[ a_{GH} = 3.0e-10; \]
\[ k_{SH} = 9e-10; \]
\[ k_h = 11.89; \]
\[ k_{NA} = 2.33e-11; \]
\[ k_{GA} = 4.98e-11; \]
\[ k_{AG} = k_{GA}; \]
\[ k_{HA} = 7.96e-10; \]
\[ a_{NA} = 5e-6; \]
\[ a_{GA} = 1.8e-10; \]
\[ a_{AG} = a_{GA}; \]
\[ a_{HA} = 2e-8; \]
\[ k_{SA} = 9e-10; \]
\[ \text{beta}\_a = 2.72; \]
\[ k_a = 2.72; \]
\[ b=1e-10; \]
\[ h=1e-10; \]
\[ N_{max1} = 4.26e-15; \]
\[ N_{max2} = N_{max1}; \]

\[ \text{Ga} = 1e-12; \]
\[ Gc = (\text{beta}_G/k_G)\*\text{Ga}; \]
\[ Ac = 4e-3; \]
\[ Aa = (\text{beta}\_a/k_a)\*\text{Ac}; \]
\[ Nc = 1.3e-8; \]
\[ Ne = 6e-11; \]
\[ Hc = 1.06e-9; \]
\[ Sc = 1.07e-11; \]
\[ Sa = 1.7e-12; \]
\[ Bc = 0; \]
\[ Ba = 0; \]

%% Gains
\[ Fd = 0; \]
\[ k_{Fd}=1e-10; \]
\[ a_{FA}=1; \]
\[ k_{Bc}=1; \]
\[ a_{NB}=1; \]
\[ k_{Ba}=1; \]
\[ \text{kappa}\_b= 1; \]
\[
\begin{align*}
\text{k1\_fd} &= 1e-10; \\
\text{k\_AN1} &= 1e-6; \\
\text{k2\_fd} &= 1e-10; \\
\text{k\_AN2} &= 1e-6; \\
\text{k\_FG} &= 1e-10; \\
\text{a\_FG} &= 1e-6; \\
\text{k\_Ns} &= 1; \text{ %CNS} \\
\text{k\_Ne} &= 1; \text{ %ENS} \\
\text{Bas1} &= 2.35e-5; \text{ %CNS constant} \\
\text{Bas2} &= 2.3e-5; \text{ %ENS constant} \\
\% \text{Antral Gastrin} \\
\text{dGdSa} &= (G\_cell)*(\text{MM}(\text{Ne},k\_NG1,a\_NG1) + \text{MM}(\text{Nc},k\_NG2,a\_NG2) + \text{MM}(\text{Fd},k\_FG,a\_FG))*\text{dM2}(\text{Sa},k\_SG) \\
\text{dGdNe} &= (G\_cell/(\text{M2}(\text{Sa},k\_SG)))*\text{dMM}(\text{Ne},k\_NG1,a\_NG1) \\
\text{dGdNC} &= (G\_cell/(\text{M2}(\text{Sa},k\_SG)))*\text{dMM}(\text{Nc},k\_NG2,a\_NG2) \\
\text{dGdFd} &= (G\_cell/(\text{M2}(\text{Sa},k\_SG)))*\text{dMM}(\text{Fd},k\_FG,a\_FG) \\
\% \text{Antral Somatostatin} \\
\text{dSdSa} &= (\text{Da\_cell}/\text{M2}(\text{Nc},k\_NS1))*(\text{MM}(\text{Ne},k\_NS1,a\_NS1) + k\_AS)*\text{dM2}(\text{Sa},k\_ss) - k\_S \\
\text{dSdNC} &= (\text{Da\_cell}/\text{M2}(\text{Sa},k\_ss))*(\text{MM}(\text{Ne},k\_NS1,a\_NS1) + k\_AS)*\text{dM2}(\text{Nc},k\_NS1) \\
\text{dSdNe} &= (\text{Da\_cell}/(\text{M2}(\text{Nc},k\_NS1)*\text{M2}(\text{Sa},k\_ss)))*\text{dMM}(\text{Ne},k\_NS1,a\_NS1) \\
\% \text{Corpus Somatostatin} \\
\text{dScdSc} &= (\text{Dc\_cell}/\text{M2}(\text{Nc},k\_NS2))*(\text{MM}(\text{Ne},k\_NS2,a\_NS2) + k\_GS)*\text{dM2}(\text{Sc},k\_ss) - k\_S \\
\text{dScdNC} &= (\text{Dc\_cell}/\text{M2}(\text{Sc},k\_ss))*(\text{MM}(\text{Ne},k\_NS2,a\_NS2) + k\_GS)*\text{dM2}(\text{Nc},k\_NS2) \\
\text{dScdNe} &= (\text{Dc\_cell}/(\text{M2}(\text{Nc},k\_NS2)*\text{M2}(\text{Sc},k\_ss)))*(\text{dMM}(\text{Ne},k\_NS2,a\_NS2)) \\
\% \text{Corpus Histamine} \\
\text{dHdSc} &= E\_cell*(\text{MM}(\text{Ne},k\_NH,a\_NH) + k\_GH)*\text{dM2}(\text{Sc},k\_SH) \\
\text{dHdNe} &= (E\_cell*/(\text{M2}(\text{Sc},k\_SH)))*(\text{dMM}(\text{Ne},k\_NH,a\_NH)) \\
\% \text{Corpus Acetylcholine} \\
\text{dAdSc} &= P\_cell*(\text{MM}(\text{Nc},k\_NA,a\_NA) + k\_GA + k\_HA)*\text{dM2}(\text{Sc},k\_SA) \\
\text{dAdNC} &= (P\_cell/\text{M2}(\text{Sc},k\_SA))*(\text{dMM}(\text{Nc},k\_NA,a\_NA)) \\
\text{dAdAc} &= -h\_b\_Bc - \text{MM}(\text{Fd},k\_Fd,a\_FA) - \text{beta}_a \\
\text{dAdFd} &= -\text{Ac}*\text{dMM}(\text{Fd},k\_Fd,a\_FA) \\
\text{dAdBC} &= -h\_b\_Bc \\
\% \text{Corpus Bicarbonate} \\
\text{dBcdNC} &= \text{dMM}(\text{Nc},k\_Bc,a\_NB) \\
\text{dBcdAc} &= -h\_b\_Bc \\
\text{dBcdBC} &= -h\_b\_Bc - \text{beta}_a \\
\% \text{Antral Bicarbonate} \\
\text{dBadNC} &= \text{dMM}(\text{Nc},k\_Ba,a\_NB)
\end{align*}
\]
% Central Neural Activity

dBadAa = -h*b*Ba
dBadBa = -h*b*Aa - kappa_b

% Enteric Neural Activity

dNcdFd = dMM(Fd,N_max1,k1_fd)
dNcdNc = k_Nc

dNedFd = dMM(Fd,N_max2,k2_fd)
dNedNe = -k_Ne

% Transfer Functions and Bode Analysis

s = tf('s'); % defining the transfer function variable

% X(s): transfer function for Bas1

X_num = dAdSc*dScdNc*(s+dBcdBc) + dAdNc*(s+dBcdBc)*(s+dScSc) + dAdBc*dBcdNc*(s+dScSc);
X_den = (s+dScSc)*(s+dNcdNc)*(s+dAdAc)*(s+dBcdBc) - dAdBc*dBcdAc);

Xs = X_num/X_den
figure; bode(Xs)
title('Bode Plot: X(s) = Ac(s)/Bas1(s)')

% Y(s): transfer function for Bas2

Y_num = dAdSc*dScdNe*(s+dBcdBc);
Y_den = X_den;

Ys = Y_num/Y_den
figure; bode(Ys)
title('Bode Plot: Y(s) = Ac(s)/Bas2(s)')

% Z(s): transfer function for Food

Z_num = (dAdSc*dScdNc*dNcdFd + dAdSc*dScdNe*dNedFd)*(s+dBcdBc) + dAdNc*dNcdFd*(s+dAdAc)*(s+dScSc) + dAdBc*dBcdNc*dNcdFd*(s+dScSc);
Z_den = (s+dAdAc)*(s+dBcdBc) - dAdBc*dBcdAc)*(s+dScSc)*(s+dNcdNc);

Zs = Z_num/Z_den
figure; bode(Zs)
title('Bode Plot: Z(s) = Ac(s)/Food(s)')

AA = [Xs,Ys,Zs];