Gross Assessment of Colonic Abnormalities, with Particular Focus on Diverticular Disease and Polyps:

An Autopsy Study

By

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A thesis
Submitted to the Faculty of Graduate Studies
University of Manitoba
In partial fulfillment of the requirements
For the degree of

MASTER OF SCIENCE

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Glossary of Terms

Abnormalities- Growths, diseases, conditions and symptoms within the colon.

cm- Centimeter

EBM- Evidence Based Medicine.

g- Grams

Incidence- Annual diagnosed rate, number of new cases each year.

More Likely- The percentage increase for the study population which increases cumulatively each year.

Polyps- Including both hyperplastic and non-hyperplastic lesions.

Prevalence- Estimated population of people with the disease.

WHO- World Health Organization

I-Abstract

I.1-Objectives-

To grossly evaluate colonic abnormalities within the Manitoba population by determining prevalence according to; age, gender, body mass index, body weight, body length, colon length, and fecal weight.

I.2-Methods-

A population study of 67 medico-legal autopsies from two major teaching hospitals was performed, examining the colon for abnormalities. The colon was resected just proximal to the ileocecal valve and just distal to the recto-sigmoid junction. Once the specimen was detached it was weighed with the autopsy scale opened and the feces removed. The specimen was patted dry and re-weighed. The specimen was laid on a blue specimen photograph board where the length was measured from the cecum to terminal sigmoid/rectum and photographed digitally. The specimen was then evaluated for any abnormalities. The specimen was then placed back into the body.

I.3-Results

Of the 67 colons assessed, 66% had an abnormality, of which 37.3% had diverticular disease and 24% had polyps, the two most common diseases. Age was the only significant factor ($p=0.004$) in this study affecting prevalence. The prevalence of multiple polyps was 63% with colon length being the only significant factor ($p=0.0265$) in this study affecting prevalence.

I.4-Conclusions

A progressive risk of increased abnormality formation is noted with age. Diverticular disease and polyps have similar prediction factors and disease prevalence. Many factors are suggested in the literature to influence the prediction of abnormalities, however only age was determined to be significant in this study. Multiplicity of polyps in the colon is significantly related to colon length.

II-Acknowledgements

Firstly, I would like to thank Dr. Charles Littman and Dr. John Younes for their supervision, direction, and assistance. I have learned much from them, both were instrumental in the completion of this project their support is greatly appreciated.

I would also like to thank Dr. Jerry Vriend, Dr. Jayantha Herath and Pathologist Assistants Ronald Brereton, Larry Bluhm and Lance Fuczek, for all they taught me, and for the time they took with me.

I would like to thank my parents (Andrew and Andria Blanchard) my grandparents (Pius and Jean Blanchard, and Charles and Donna Travis) and my siblings and their families (Jeremiah Blanchard and Sarah Bhagrath). Their love, support, and encouragement have helped make my goals and dreams a reality. I am thankful that they have always been there for me and blessed because I know I can always turn to them for guidance and support.

Lastly, I would like to thank the friends that I have met along the way, as they have made me focus on my dreams but not let me forget the importance of having fun.

III-Introduction

The gastrointestinal tract is made up of the esophagus, stomach, small intestine and large intestine. The large intestine (colon) consists of the cecum (with appendix), ascending colon, transverse colon, descending colon, and sigmoid colon (Martini, 2001). The large intestine differs from the small intestine in that it contains fatty appendages called appendices epiploicae and 3 longitudinal bands of muscle called taeniae coli, which form haustra or saccules (Moore, 2006). After digestion and absorption are completed in the stomach and small intestine the large intestine receives the residual liquid (chyme) consisting of approximately 75% water and 25% solids (Moore, 2006). The main functions of the large intestine are absorption and secretion. The large intestine absorbs water, sodium and chloride ions and secretes bicarbonate ions and mucin. The absorption rate within the large intestine helps form feces, with a range of consistencies from liquid to solid (Martini, 2001, Moore, 2006).

The mucus secreted by the intestine mixes with the chyme and bacteria (gut flora) producing feces. The flora and intestinal cells have a symbiotic relationship, the bacteria assists in the breakdown of fiber, creating nutritional requirements for its own nourishment and creates waste products for the intestinal cells nourishment (Colon, 2007). The flora in addition breaks down bilirubin, breakdown product of heme, which provides the yellow-brown pigment in feces (Martini, 2001). The movement of feces from the cecum to the rectum is a slow process that allow for water absorption, and results from muscular contractions.

The colon has three methods of fecal movement. Firstly, a contraction wave occurs along the longitudinal muscles as a result of spontaneous depolarization of the smooth muscle cells. The wave spreads along the entire length of the muscular sheet (Martini, 2001). Secondly, a contractive wave occurs along the circular muscles, creating an area of narrowing, propelling the feces (Martini, 2001, Robbins, 1994). Thirdly, areas undergo cycles of contraction that result in segmentation. Segmentation movements do not result in movement of the feces along the length (Martini, 2001). The first two movements together shorten the bowel and propel the feces forward, the third movement allows for churning and fecal formation. The mucus acts as a lubricant as the feces becomes more compact and less moist (Martini, 2001).

The ability for the intestinal epithelium to regenerate is remarkable (Robbins, 1994). The colonic epithelium is renewed every 3-8 days. The cells proliferate at such a rapid rate and are particularly susceptible to harmful agents, such as radiation, and chemical that cause mutations (Robbins, 1994). Diseases of the large intestine account for a large portion of the diseases that effect humans (Robbins, 1994), for example, congenital disorders, infections, diverticular disease, inflammatory bowel disease, vascular disease, polyps, and adenocarcinomas (Rubin, 2001). Of the many abnormalities that can occur within the colon, the most commonly noted in the literature are diverticular disease and polyps.

III.1 Diverticular Disease

Diverticular disease refers to two conditions, diverticulosis and diverticulitis. Diverticulosis is a disease process in which areas of weakening result in outward pouches in the mucosa (Martini, 2001), diverticulum being singular and diverticula being plural (Lester, 2006). Diverticular disease has an incidence of 1 in 906 and a prevalence of 1 in 136. The diverticula most often occur in parallel rows between the lateral taeniae and mesentery (Rubin, 2001). In European and western society, diverticula arise mainly in the sigmoid colon (distal disease), approximately 90% have distal disease, and 15% involving the cecum (right sided disease) and sigmoid colon (Stollman, 2004). However, to have the entire colon affected is not an uncommon finding (Ludeman, 2002). The taeniae shorten and the muscle wall becomes thickened, and deposits of elastin have been documented as being amplified by 200%, in comparison to uninvolved segments (Stollman, 2004, Ludeman, 2002). It has been documented that elastin deposits within the longitudinal muscle layers and thickens and shortens the segment causing a contracture (Ludeman, 2002). The diverticula range in size from 0.5-1.5 cm in diameter and can contain hardened fecal material (fecalith). The walls of the diverticula have epithelium and submucosa but lack the muscularis propria (Rubin, 2001).

Of individuals with this disease, approximately 75-80% will be asymptomatic (Stollman, 2004, Rubin, 2001). Diverticulitis presents at some point in the lives of the remaining 20%. Diverticulitis is inflammation at the base of a diverticulum, either as a result of irritation by the retained fecal material, or by an expanding floral population (Stollman, 2004, Ludeman, 2002). As inflammation continues, necrosis of the diverticular tissues results and fecal material and bacteria are released into the

peridiverticular tissues. The resulting abscess cavity is usually contained within the appendices epiploicae, but can rupture into the abdominal cavity causing peritonitis (Rubin, 2001).

Although explanations have been suggested as to the cause of diverticular disease, the pathogenesis remains unknown (Ludeman 2001). However, the literature widely accepts an inverse relationship between incidences of the disease and the fiber content in the diet (Stollman, 2004, Ludeman, 2002, Rubin 2001, Robbins, 1994). The disease was uncommon in the earlier part of the 20th century and is rapidly becoming a common clinical entity (Kang, 2004). In individuals in Western society, approximately 10% under the age of 40 are affected (Rubin, 2001). This percentage increases to 50% affected over the age of 60 (Robbins, 1994), and further increases to 50-66% in those over the age of 80 (Stollman, 2004). Kang (2004) notes the geographic and ethnic difference in the disease frequency and states the possible correlation to genetic or environmental influences. Although several possibilities have been put forth in the literature, no known etiology has been identified. This study aims to provide more insight into the etiology and assess the factors in prediction of the disease and the relationships between these factors.

III-2 Polyps

A polyp is defined as a mass projecting into the lumen of the gastrointestinal tract (Rubin, 2001). Polyps can be benign or malignant, form on the inside lining of the colon, and are pedunculated or sessile (Martini, 2001). Pedunculated polyps contain a stalk or pedicle, whereas, sessile polyps are flatted to the mucosal surface (Robbins, 1994). Polyps are formed as a result of abnormal mucosal maturation, inflammation, or architecture. Polyps arising as a result of these stimuli are non-neoplastic and are called hyperplastic. Polyps that arise as a result of proliferative dysplasia are termed adenomas (Robbins, 1994), are neoplastic and have an increased risk of developing colon cancer (Rubin, 2001). The term polyps from this point on will be defined as both non-neoplastic and neoplastic polyps unless otherwise indicated. Non-neoplastic polyps occur on a sporadic basis, increase with age, but tend to be more frequently seen in the left colon (Robbins, 1994, Thomson, 2000). Non-neoplastic polyps can occur singly, but usually occur in multiples (Bombi, 1988). Neoplastic polyps greater than 2 cm in size are reported to have a 50% incidence of cancer (Thomson, 2000). Clinically, routine colonoscopies are performed in individuals over 40 years of age with irritable bowel symptoms, such as constipation, diarrhea and occult blood. Polyps are identified and removed by snare polypectomy technique (Thomson, 2000). However polyps and cancers have a tendency to recur, and therefore individuals at risk are kept under colon surveillance and reassessed every three years (Thomson, 2000).

Although several studies have analyzed polyps very few predictors studies have been performed (Jass, 1992). It has been extensively documented that age is an important factor in the prediction of polyps; however no additional risk factors have been definitely

identified (Clark, 1985, Stemmermann, 1972, Paspatis, 2001). It has been proposed that polyps occur in response to western diet. A 1972 study examined individuals from South Africa Bantu and determined diet as a factor influencing the presence of polyps, as, neither polyps nor carcinoma of the colon were identified in this population (Stemmermann, 1972). Other factors influencing polyp prevalence have been suggested, such as size, multiplicity, and gender. This study intends to evaluate polyps and the factors affecting prevalence, in hopes of determining a relationship between prediction factors.

Colon disease accounts for approximately 15% of all deaths in North America. Of the 15%, 10% are colon/rectal cancers (WHO, 2004). Colon and rectal cancers are the 3rd leading cause of death in North America (Jermal, 2005, WHO, 2004). Cause of death is determined by an in-depth examination at time of autopsy. Goldman (1983) examined the validity of the autopsy and determined that 10% of autopsies studied have a finding that reveal a major diagnosis that if known could have prolonged life and 12% of autopsies revealed clinically missed disease, which would not have been affected with treatment. Goldman concludes that advances in technology have not reduced the validity of the autopsy but together form a standard of patient care. Each year cause of death information is collected and analyzed by the World Health Organization (WHO). The WHO produces a report based on demographics and incidence (Ruzicko, 1990). This information is then used and reported by several journals and is made available to clinicians in hopes of expanding the knowledge of diseases and assessing diagnostic and treatment techniques (Jermal, 2005). Evidence based medicine provides an example of such research.

Evidence based medicine (EBM), came about in hopes of improving patient care and as a cost control tool, which examines patient and diseases information with focus on efficiency of diagnostic tests and treatments (McQueen, 2001). EBM provides information on; the benefit of actions taken, and the evaluation and possible methods of improvement available to form a standard of care (McQueen, 2001). This study intends to briefly examine the benefits of examining the colon internally at autopsy with particular emphasis on understanding diseases, prevalence, health care funding, time required, and family views.

III.3 Hypothesis and Objectives

The objectives of this study are to examine the Manitoba population and to determine the prevalence of abnormalities within the colon: in addition to assess the prevalence of abnormalities in relation to the gender and age of the population and the significance of this finding. We hypothesize that approximately 40-50% of the colons examined will contain a minimum of one disease process. Of the colons examined an increase in pathology is expected with increased age. However, no definite significance is expected between gender and abnormalities, albeit the literature (Jass, 1992; Coode, 1985) has documented gender as a prediction factor. This study also aims to determine whether there is a statistically significant predictive value to factors (age, gender, body weight, body length, body mass index, colon weight, colon length, fecal weight, and colon plus fecal weight) which theoretically might contribute to colons containing multiple abnormalities and multiple disease processes. The relationships between these factors will also be examined relative to age, sex, and abnormalities.

We expect to find fecal weight as a significant factor in predicting abnormalities. However, no significant predictive effect is expected between body length/weight and abnormalities, or between colon length/weight and abnormalities.

IV-Literature Review

Necropsy studies of the colon have been performed and most are pre 1990's. Several studies have been set forth to determine the prevalence of abnormalities within the colon at post-mortem examination; however, they are limited and are described as having unsatisfactory results due to the considerable amount of interobserver variation (Jass, 1992). Several studies examine diverticular disease as an entity on its own and several compare polyps and diverticular disease as similar entities with similar prediction factors for prevalence. This study will examine these diseases as single entities.

Diverticular Disease

Diverticular disease has been examined since the early 20th century and many suggestions to the etiology have been put forth, including several contrary opinions (Hughes, 1969). Hughes (1969) determined an incidence of diverticular disease in 43% of all necropsies, and 48% in those over the age of 50 in a population of 200 necropsies in Brisbane. Hughes (1969) concluded increasing degrees of atheroma of the aorta as a possible etiology for this percentage, but discovered no association between obesity, length of the colon, gallstones or hypertension. Hughes (1969) does suggest a high residue diet as a protective factor in diverticular disease. In 1972, Stemmermann reported a frequency of 52% diverticular disease in a Japanese Hawaiian population. Stemmermann suggested an environmental factor in addition to age and genetics in the development of the disease. Coode postulated a dietary factor, as individuals who immigrated to Hawaii, had a higher intake of calories, particularly for beef and fat than those of Japan itself (Coode, 1985). In 1985 a necropsy study was performed in Hong Kong that revealed a low prevalence, 5% for both males and females (Coode, 1985). In a

Cretan population in 2001, the percentage of diverticular disease was reported as 22.9% (Paspatis, 2001), age and environmental factors were suggested as risk factors rather than genetics. In 2002, the prevalence of diverticular disease was determined to be 10% in the Western population over the age of 40 but states the true incidence will be much higher as most individuals are asymptomatic (Ludeman, 2002). Ludeman (2002) stated, "Although there is no conclusive evidence, it is widely accepted that there is an inverse relationship between the incidence of diverticular disease of the sigmoid colon and the fiber content of the diet" (Ludeman, 2002). In 2004, Kang concluded ethnic origin as a risk factor, as 4% of Asian males were identified having the disease, whereas, 22% of other ethnic groups were identified as having the disease (Kang, 2004). In addition, in a seminar on diverticular disease released in 2004, the prevalence of diverticular disease was said to increase with age from 10% in people less than 40, to 50-66% in people older then the age of 80, where incidence was also attributed to factors such as ethnic origin and dietary changes (Stollman, 2004).

The incidence and prevalence of diverticular disease ranges from 5% to 66% within the literature with age, gender, ethnicity and dietary factors being documented as causative agents. Of the information documented on diverticular disease, prevalence and morbidity, very little new information has been determined of its etiological factors (Hughes, 1969, Bassotti, 2005). Given the commonness of the disease, an important question to address is etiology.

Polyps

Colorectal cancer is the 3rd leading case of death from cancer in North America and Europe (WHO, 2004). Carcinomas occur most often in sessile polyps and infrequently in pedunculated polyps (Cajucom, 1992). Several studies have documented the prevalence of polyps in the colon. Vatn (1982) identified 174 hyperplastic polyps, and 59 polyps (no histologic diagnosis could be made), and determined an increased incidence with age in men but independent of age in women (Vatn, 1982). In 1985 an evaluation of prevalence of polyps was performed; where males were determined to have a higher risk of bearing polyps. Age, region and gender were determined to be factors in predicting prevalence for polyps greater than 0.7 cm (Clark, 1985). Additionally in 1985, Coode determined the prevalence of hyperplastic polyps to be 22% in males and 15% in females (Coode, 1985). Coode notes a correlation between colorectal carcinoma and socio-economic class, in addition to high fiber diets having a protective effect against colonic cancer (Coode, 1985). In 1988, Bombi conducted an experiment and determined the prevalence of hyperplastic polyps to be 1.6% in Barcelona. This low incidence of hyperplastic polyps determined was described by Bombi as remarkable and he postulated that the Spanish diet of fish, vegetables, fewer milk derivatives and the use of vegetable oils in cooking might be of significance (Bombi, 1988). In a Filipinos study in 1992, 1.4%, 6 out of 416 individuals were determined to have polyps. This low prevalence was attributed to diet, as Filipino's diets are high in fiber and lower in fat, in addition, high calcium levels are implicated as chemopreventative agents due to its antiproliferative capabilities (Cajucom, 1992). In a New Zealand study in 1992, 251 hyperplastic polyps were identified in a population of 303 (Jass, 1992), of these gender was a significant

factor. Polyp prevalence was influenced by age, body mass and ethnic (European) origin (Jass, 1992). In 2001, McCashland determined that males have a greater risk of developing polyps or tumors than women, and that the incidence of identifying polyps increased with age (McCashland, 2001).

Of the information presented in the literature, polyps range from an incidence and prevalence of 1.4% to 26% with diet, age and gender being the most common causative agents. But no definite etiology has been identified.

V-Material and Methods

The population study was obtained from two major teaching hospitals, Health Science Center (HSC) and St. Boniface General Hospital, both located in Winnipeg, the largest city in Manitoba. Both serve remote areas of Canada. The large bowel, colons and partial rectums, were removed from 67 unselected cases, examined at the major teaching hospitals between March 2006 and February 2007. The autopsies were performed for investigation of sudden or violent or unexplained deaths.

The specimens were collected in a standard fashion: At the time of autopsy the intestine just proximal to the ileocecal valve and just distal to the sigmoid colon was removed. Once the specimen was detached it was weighed with an autopsy scale. The specimen was opened from the terminal end of the colon toward the cecum, along the taeniae coli, through the ileocecal valve. The specimen was then rinsed with cold water to remove feces, patted dry and then re-weighed. The specimen was then laid on a blue specimen photograph board, where the length was measured, from the cecum to terminal sigmoid/rectum and then photographed digitally. The specimen was then evaluated for any abnormalities. The specimen was then placed back into the body.

The following data was obtained in an excel spreadsheet for each specimen:

-Autopsy Number

-Date of Autopsy

-Age

-Gender

-Photographs Taken

-Absence of Appendix

-Body Length

-Body Weight

-Colon Length

-Colon/Fecal Weight

-Colon Weight

-Fecal Weight

-Type, Number, and Characteristics of Abnormalities

-Cause of Death

The data was collected, and examined using Microsoft Excel. Excel programming language was used and the main functions for analyzing the data were frequency tables, logistic regressions and Pearson product correlations. The regression models were used to examine the combined effect of multiple factors on the outcome variables. When there is a binary or ordinal outcome, linear regression is not appropriate and logistic regression is used to investigate the relationship between the probabilities of the outcomes.

The Pearson product correlation results for abnormalities, diverticular disease and polyps are shown in Appendix I.

Graphs depicting the mean were used, in which the mean is represented as a horizontal line and the 95% confidence of the mean depicted as triangles above and below the mean.

VI-Results

A population of 67 colons was assessed, 42 males and 25 females, ranging in age from 15 to 92 years, with a mean age of 58.3 years and a median age of 62 years . This study determined that 73% of individuals had a body mass index score above 23, ranging from 23-52, with a mean body mass index of 29. Of the 67 colons assessed, 42 colons, 66% were observed to have an abnormality. Of these, 54 abnormalities were identified shown in Table 1:6 cases with lymphoid nodules, 3 cases of melena (symptom of gastrointestinal bleed), 25 cases of diverticular disease (1 case of diverticulitis), 16 cases of polyps, 2 cases of lipomas of the ileocecal valve, and 1 case of stenosis.

Table 1 Number and Type of Abnormalities of the Large Intestine in 67 Autopsies.

Lymphoid Nodules	6
Diverticular Disease	25
Diverticulitis	1
Polyps	16
Lipoma of the ileocecal valve	2
Stenosis	1
Melena	3
Total	54

The relative frequencies of these abnormalities are shown in Table 2 and the percentages are shown in Figure 1, Figure 2 and Figure 3 shows the distribution of abnormalities by age and gender. Of the 67 colons examined, 37.3% had diverticular disease, 24% had polyps, 9% had lymphoid nodules, 4.5% had melena, 3% had lipomas, and 1.5% had diverticulitis and/or stenosis.

Table 2 Relative Frequencies of Abnormalities of the Large Intestine in 67 Autopsies.	
Lymphoid Nodules	0.089
Diverticular Disease	0.337
Diverticulitis	0.015
Polyps	0.238
Lipoma of the ileocecal valve	0.029
Stenosis	0.015
Melena	0.045

Figure 1 Percentage of Abnormalities of the Large Intestine in 67 Autopsies.

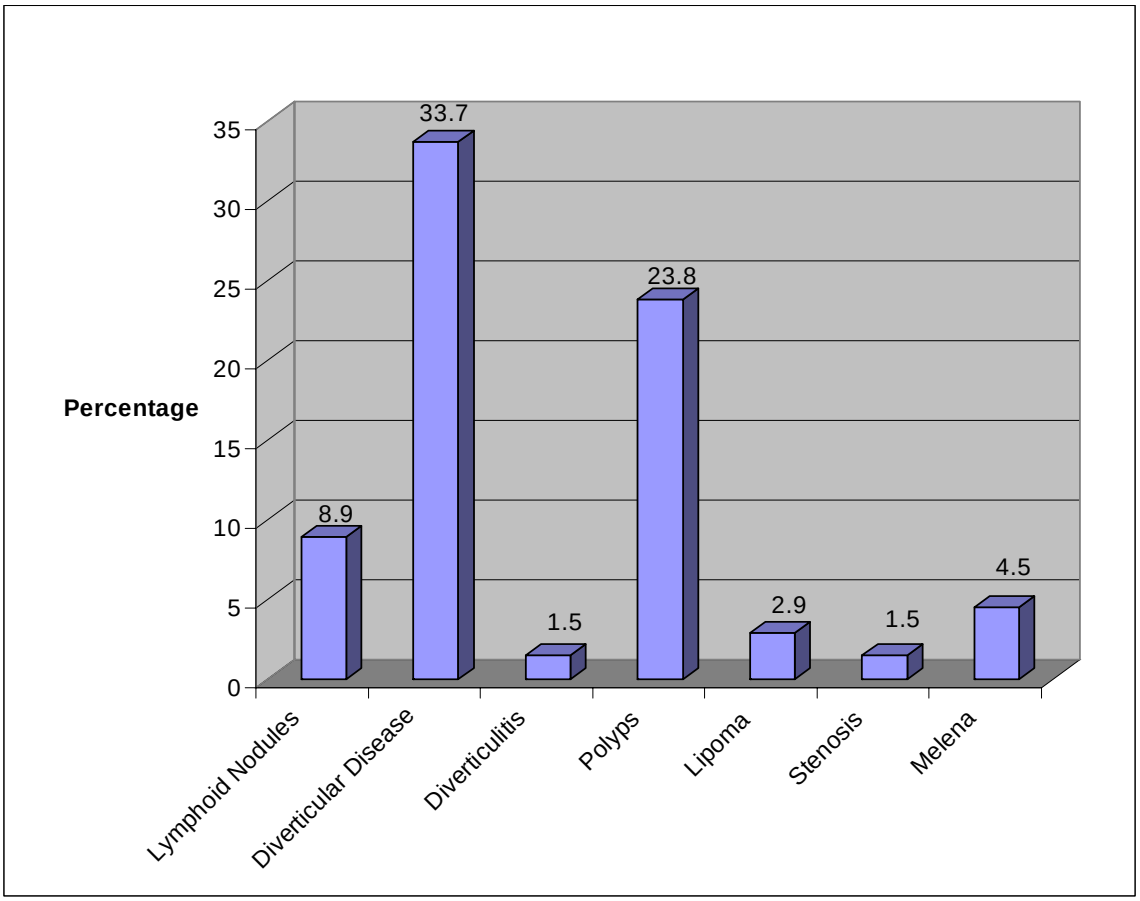


Figure 2 Distribution of Abnormalities by Age

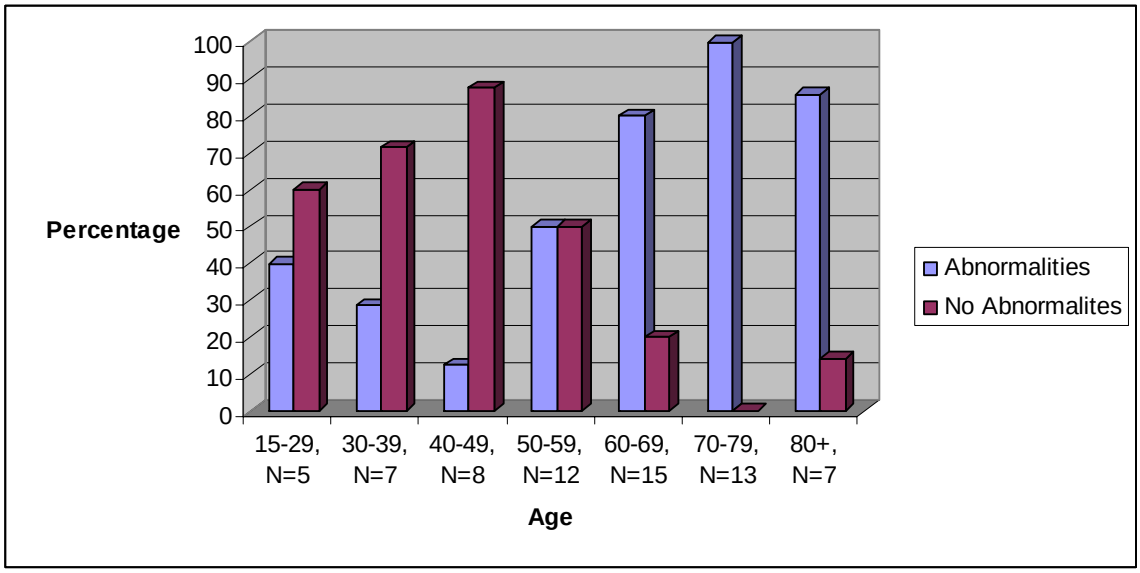
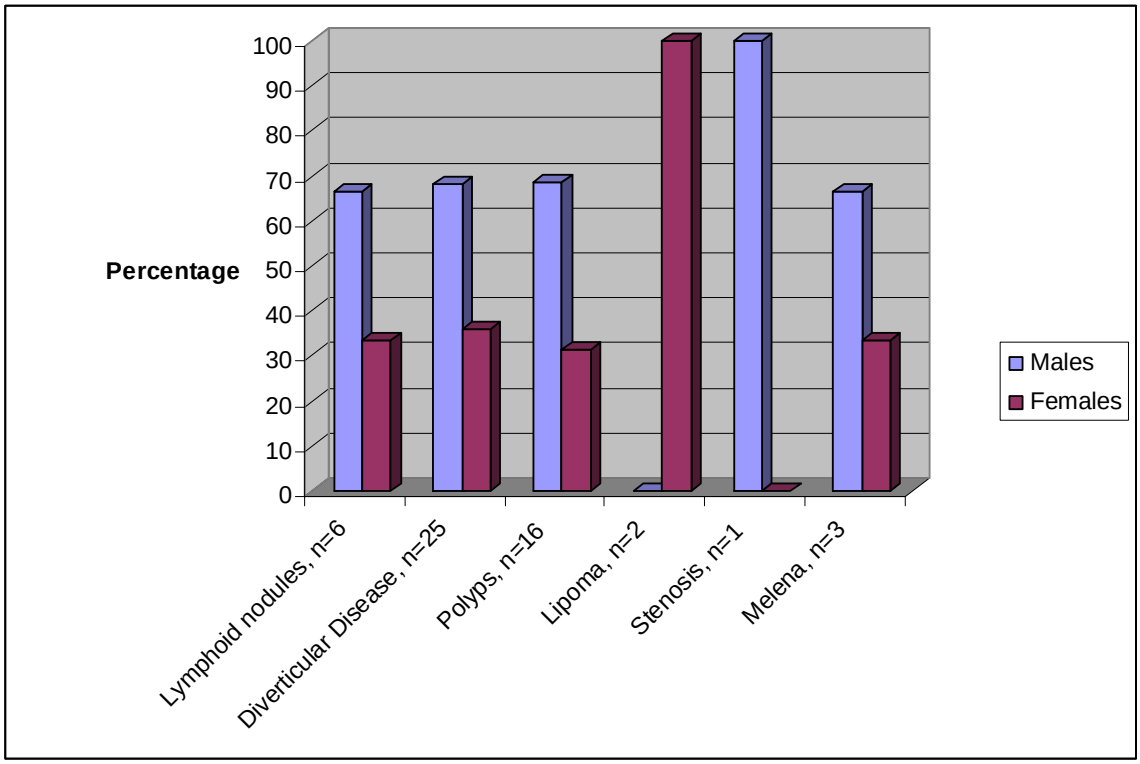


Figure 3 Distribution of Abnormalities by Gender.



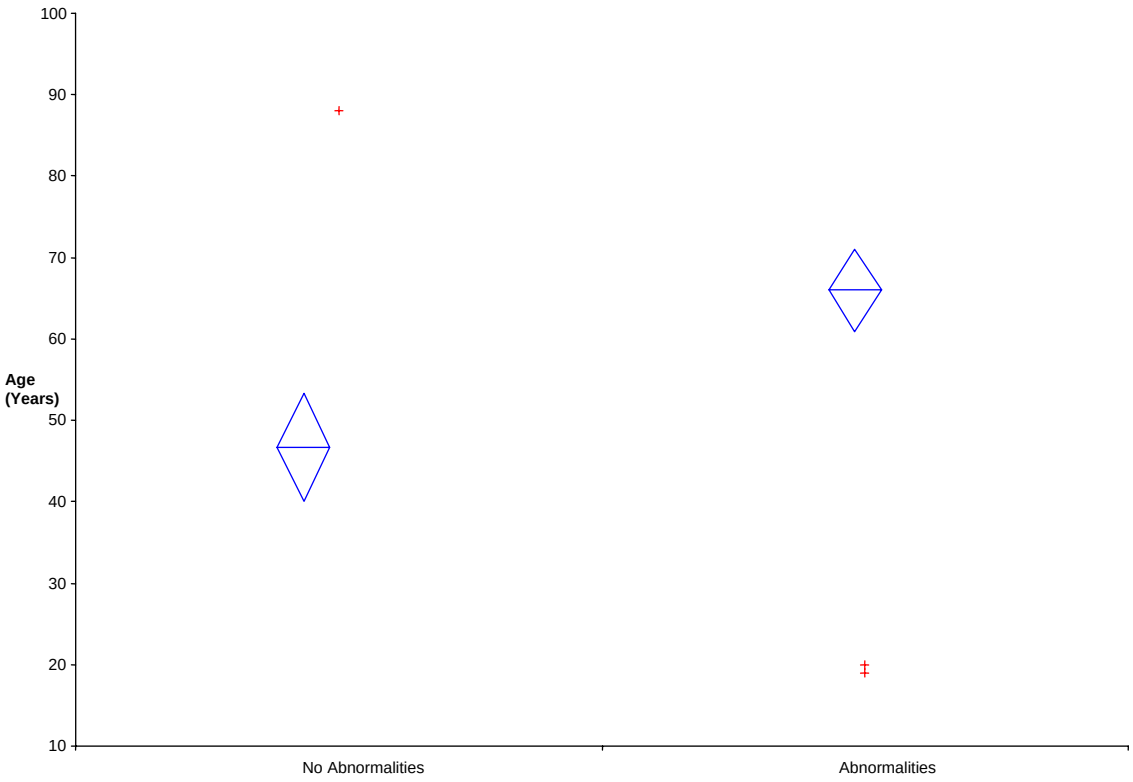
To determine the factors influencing the presence of abnormalities within the colon, a logistic regression was used, with abnormalities as the dependent variable and age, gender, body mass index, body weight, body length, colon plus feces weight, colon weight, fecal weight, and colon length as predictor variables. The sample size was 67. Age ($p=0.0004$) was shown to make a significant contribution ($p<0.05$) to predicting abnormalities. Body weight ($p=0.1536$), Gender ($p=0.1658$), body length ($p=0.1837$), colon plus feces weight ($p=0.2311$), body mass index ($p=0.3131$), and colon weight ($p=0.3869$), fecal weight ($p=0.3590$), and colon length ($p=0.9214$) were shown to have no significant effect on predicting abnormalities ($p > 0.05$). The statistical results from the logistic regression are shown in Table 3. For every year of age increase (from age 15-92), the individual was 6.8% more likely to have an abnormality.

Table 3 Logistic Regression Output for Abnormalities and Predicting Factors.

Factor	Variable	Coefficient	Standard Error	p=	Odds Ratio	Low	High	Intercept
Age	1	0.0662	0.0188	0.0004	1.0685	1.0298	1.1086	-3.2306
Body Weight	1	-0.0163	0.0114	0.1536	0.9839	0.9621	1.0061	1.8159
Gender	1	0.7223	0.5212	0.1658	2.0592	0.7414	5.7195	0.0800
Body Length	1	-0.0363	0.0273	0.1837	0.9643	0.9141	1.0174	6.6856
Colon Plus Feces Weight	1	-0.0003	0.0003	0.2311	0.9997	0.9991	1.0002	1.0827
Body Mass	1	-0.0353	0.0350	0.3131	0.9654	0.9014	1.0338	1.4680
Feces Weight	1	-0.0006	0.0007	0.3590	0.9994	0.9980	1.0007	0.6804
Colon Weight	1	-0.0002	0.0003	0.3869	0.9998	0.9992	1.0003	0.8678
Colon Length	1	-0.0009	0.0009	0.9214	0.9991	0.9816	1.0169	0.6608

The mean age of individuals without abnormalities was determined to be 46.9 years with standard error of 3.21 years; the mean age of individuals with abnormalities was determined to be 65.2 years with a standard error of 2.51 years. The distribution of age with respect to individuals with abnormalities and those without abnormalities is shown in Figure 4, depicting the mean and 95% confidence of the mean.

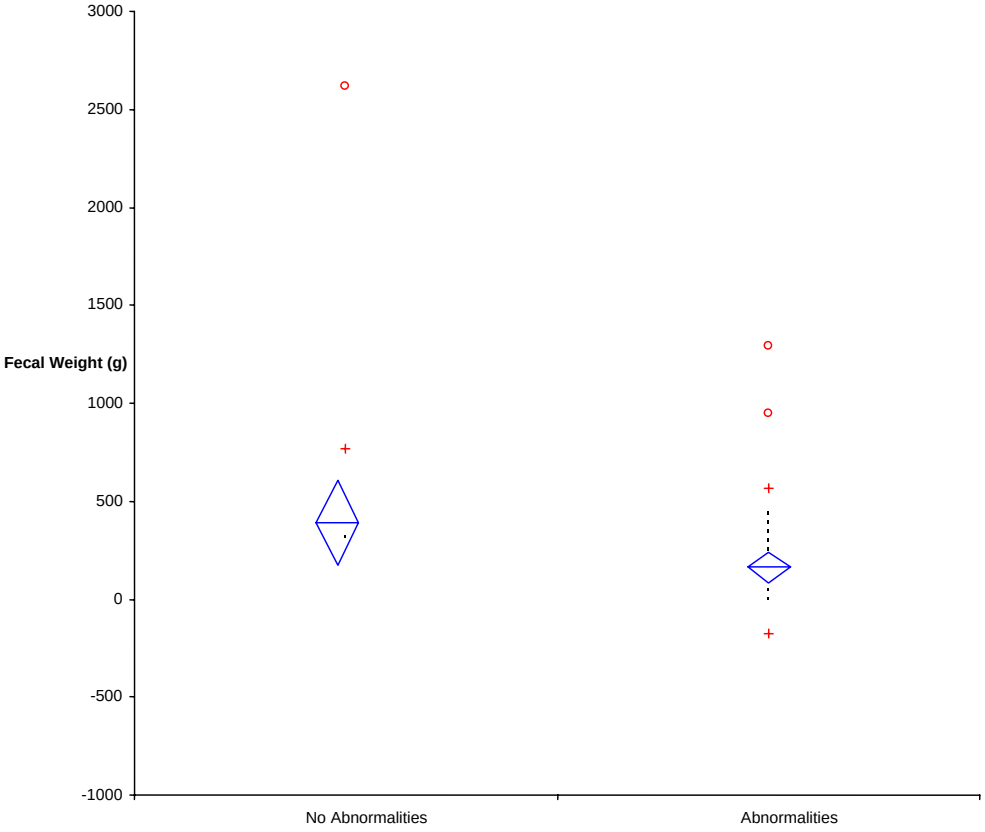
Figure 4 Distribution of Mean Age in Individuals with and without Abnormalities



	n	Mean	SD	SE	95% CI of Mean	
No Abnormalities	25.00	46.92	16.07	3.21	40.29	53.55
Abnormalities	42.00	65.21	16.27	2.51	60.15	70.28

Although fecal weight was hypothesized to be significant, no significant effect was identified (p=0.3590). Figure 5 shows, the mean fecal weight for individuals without abnormalities was determined to be 303.20 g with a standard error of 106 g; the mean fecal weight for individuals with abnormalities was determined to be 211.02 g with a standard error of 38.6g.

Figure 5 Mean Fecal Weight Distribution in Individuals with and without Abnormalities



	n	Mean	SD	SE	95% CI of Mean	
No Abnormalities	25	303.20	530.24	106.04	84.32	to 522.07
Abnormalities	42	211.02	250.08	38.58	133.09	to 288.95

A Pearson product correlation was performed in which a significant relationship ($r=0.63$, $n=13$, $p<0.05$) was identified between age and colon length in females with an abnormality. No significant relationship was noted in females without an abnormality ($r=0.05$, $n=12$, $p>0.05$). Figure 6 shows that older females with long colons are more likely to have abnormalities. A significant Pearson product correlation ($r=0.66$, $n=13$, $p>0.05$) was identified between colon length and age in males without abnormalities. No significant relationship was noted in males with abnormalities ($r=-0.14$, $n=17$, $p<0.05$). Figure 7 shows that older males with longer colons are less likely to have abnormalities.

Figure 6 Linear Relationship between Age and Colon Length in Females.

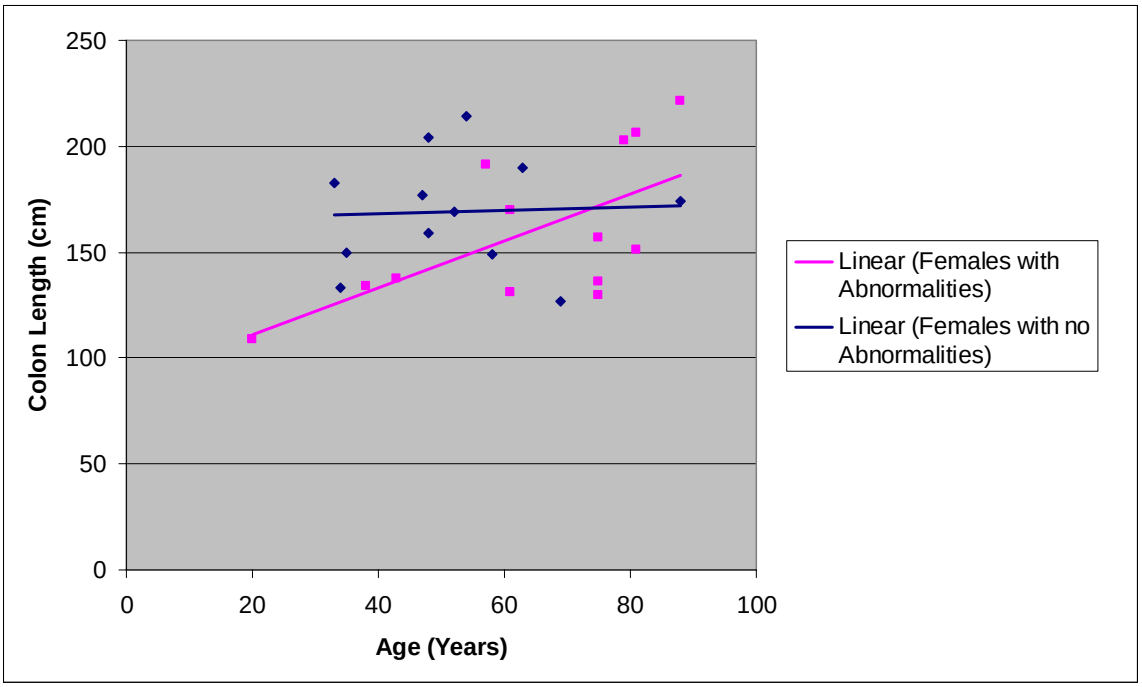
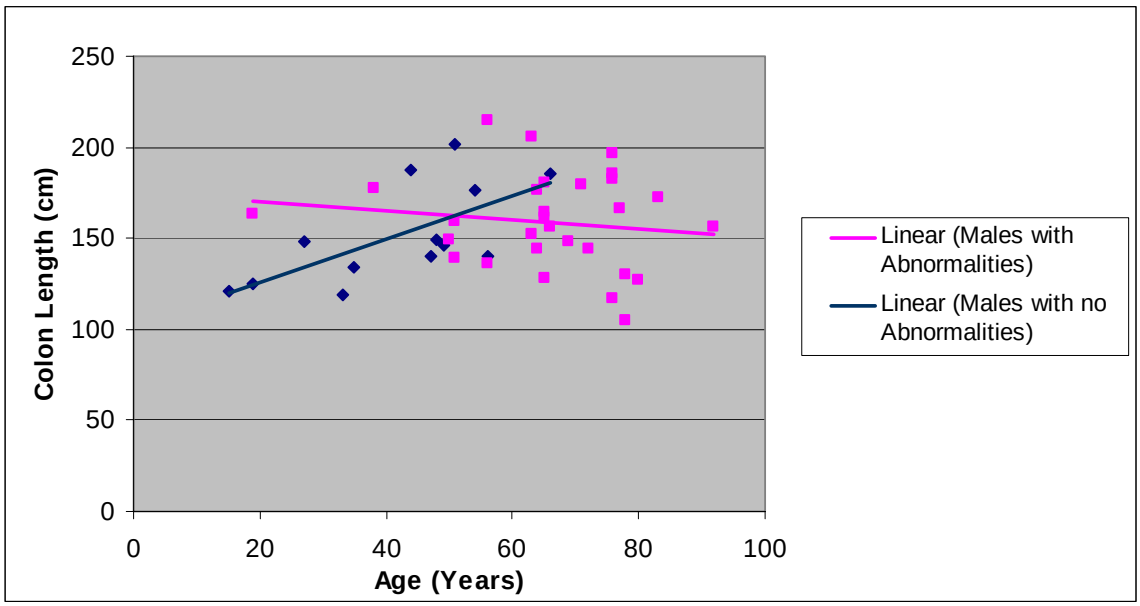


Figure 7 Linear Relationship between Age and Colon Length in Males.



Incidence of Diverticular Disease

Of the abnormalities observed in the colon diverticular disease is the most common. The distribution of diverticular disease based on gender and age are given in Table 4, Table 5, and Table 6 and shown in Figure 8, and Figure 9. Diverticular disease occurred in 37.3% of this autopsy population, with 40.5% of males and 32% of females being affected. Diverticular disease increases with age from 12-33.3% under the age of 60 to 46.7-71% over the age of 60.

Table 4 Distribution of Diverticular Disease in Males.			
Age	***Specimen***	***Diverticular Disease***	***Percentage***
15-29	4	0	0
30-39	3	1	33.33
40-49	4	0	0.00
50-59	8	3	37.5
60-69	11	7	63.63
70-79	9	4	44.44
80+	3	2	66.66

Table 5 Distribution of Diverticular Disease in Females.			
Age	***Specimen***	***Diverticular Disease***	***Percentage***
15-29	1	0	0
30-39	4	0	0.00
40-49	4	1	25.00
50-59	4	1	25.00
60-69	4	1	25.00
70-79	4	3	75.00
80+	4	2	50.00

Figure 8 Distribution of Diverticular Disease in Males and Females by Age

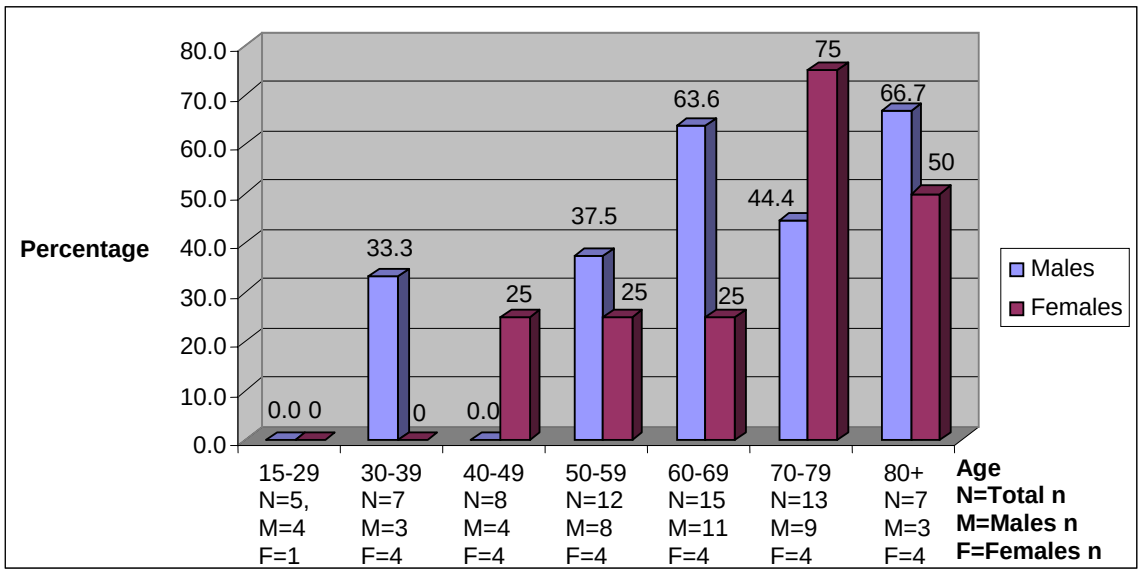
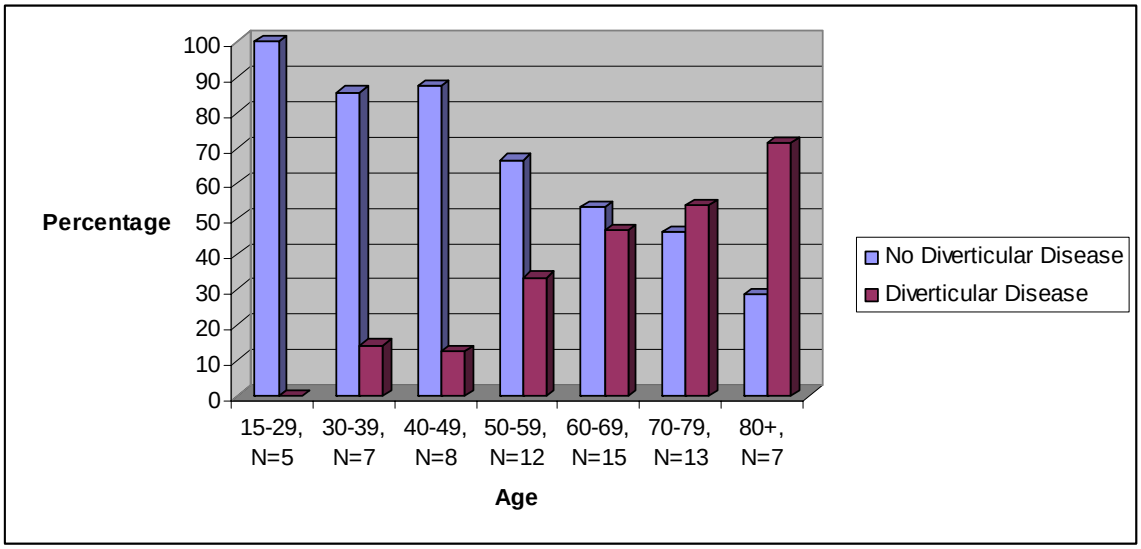


Table 6 Distribution of Diverticular Disease by Age.			
Age	***Specimen***	***Diverticular Disease***	***Percentage***
15-29	5	0	0
30-39	7	1	14.28
40-49	8	1	12.50
50-59	12	4	33.33
60-69	15	7	46.66
70-79	13	7	53.84
80+	7	5	71.40

Figure 9 Distribution of Diverticular Disease by Age.



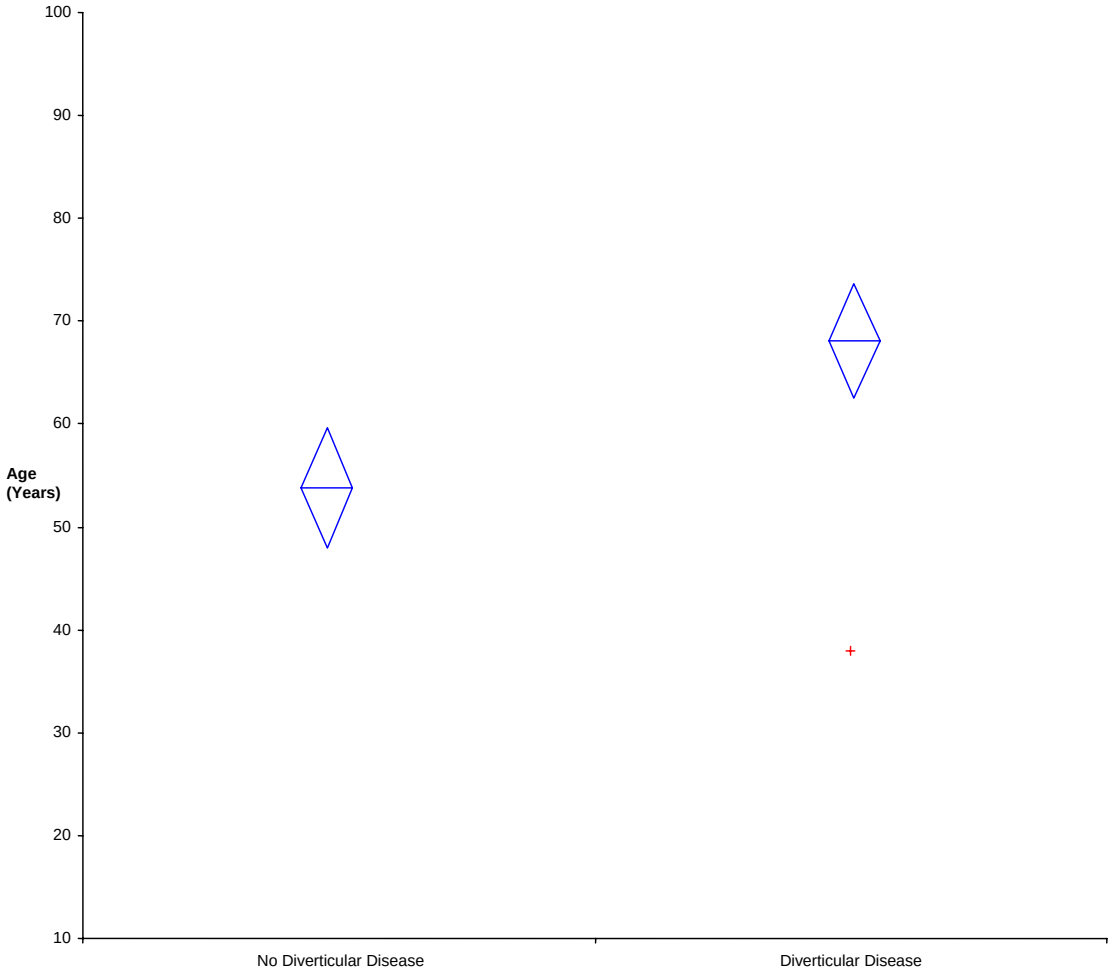
To determine the factors influencing the presence of diverticular disease, a logistic regression was used, with the presence of diverticular disease as the dependent variable and age, gender, body mass index, body weight, body length, colon plus feces weight, colon weight, fecal weight, and colon length as predicting variables. Age ($p=0.0030$) was shown to make a significant contribution ($p<0.05$) to predicting the presence of diverticular disease. Feces weight ($p=0.2084$), body length ($p=0.2289$), and gender ($p=0.4887$), colon weight ($p=0.5506$), body mass index ($p=0.8250$), colon length ($p=0.8857$), body weight ($p=0.8761$) and colon plus feces weight ($p=0.9970$) were shown to have no significant effect on predicting the presence of diverticular disease ($p > 0.05$). Table 7 shows the logistic regression results for diverticular disease. With each year increase in age (from age 15-92) individuals are 5.6% more likely than an individual younger than them to have diverticular disease. Figure 10 shows, the mean age of individuals without diverticular disease was determined to be 52.9 years with a standard error of 2.91 years; the mean age of individuals with diverticular disease was determined to be 67.6 years with a standard error of 2.69 years.

Feces weight was hypothesized to be a significant predictor however no significance effect was identified ($p=0.2084$). Figure 11 shows the mean fecal weight of individuals without diverticular disease was determined to be 292.8 g with a standard error of 69.4 g; the mean fecal weight of individuals with diverticular disease was determined to be 165.8 g with a standard error of 38.8 g.

Table 7 Logistic Regression Output for Diverticular Disease and Predicting Factors.

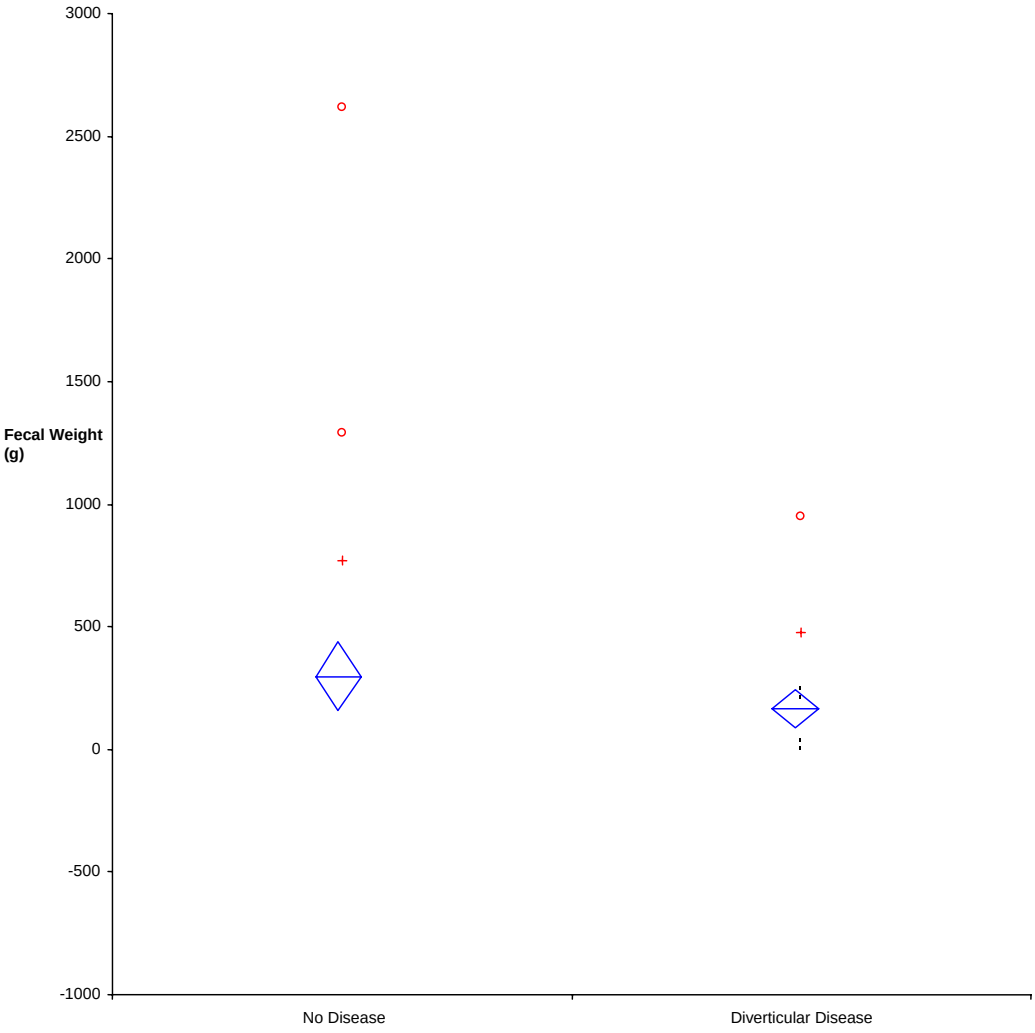
Factor	Variable	Coefficient	Standard Error	P=	Odds Ratio	Low	High	Intercept
Age	1	0.0545	0.0184	0.0030	1.0560	1.0186	1.0947	-3.8319
Feces Weight	1	-0.0015	0.0012	0.2084	0.9985	0.9962	1.0008	-0.2006
Body Length	1	-0.0355	0.0279	0.2289	0.9670	0.9157	1.0213	5.1938
Gender	1	0.3681	0.5316	0.4887	1.4450	0.5097	4.0965	-0.7504
Colon Weight	1	0.0002	0.0003	0.5506	1.0002	0.9996	1.0007	-0.7504
Body Mass Index	1	0.0077	0.0349	0.8250	1.0077	0.9412	1.0790	-0.7065
Colon Length	1	0.0013	0.0090	0.8857	1.0013	0.9838	1.0191	-0.7256
Body Weight	1	-0.0018	0.0114	0.8761	0.9982	0.9762	1.0207	-0.3789
Colon Plus Feces Weight	1	0.0000	0.0003	0.9970	1.0000	0.9995	1.0005	-0.4965

Figure 10 Distribution of Mean Age in Individuals with and without Diverticular Disease



	n	Mean	SD	SE	95% CI of Mean	
No Diverticular Disease	42	52.90	18.85	2.91	47.03	58.78
Diverticular Disease	25	67.60	13.45	2.69	62.05	73.15

Figure 11Mean Fecal Weight Distribution with and without Diverticular Disease



	n	Mean	SD	SE	95% CI of Mean	
No Disease	42	292.810	449.6968	69.3897	152.674	to 432.945
Diverticular Disease	25	165.800	193.7642	38.7528	85.818	to 245.782

Pearson product correlation were performed in which a significance relationship ($r=-0.46$, $n=17$, $p<0.05$) was identified between body weight and fecal weight in females without disease, but not with disease ($r=0.03$, $n=8$, $p>0.05$). As body weight increased in females with diverticular disease feces weight remains the same. Figure 12 shows a negative relationship in females without disease, but no significant relationship in females with diverticular disease. A similar significant Pearson product correlation ($r=-0.45$, $n=17$, $p<0.05$) was identified for body mass index to fecal weight in females without diverticular disease. Figure 13 shows that as body mass index increases fecal weight decreases in females without diverticular disease and fecal weight remains the same in females with disease. A significant Pearson product correlation ($r=0.63$, $n=8$, $p<0.05$) was identified between colon length and fecal weight in females with diverticular disease, but not in females without diverticular disease ($r=0.09$, $n=8$, $p>0.05$). Figure 14 and Figure 15 show the relationship between colon length and fecal weight in males and females with diverticular disease.

Figure 12 Linear Relationship between Body Weight and Fecal Weight in Females.

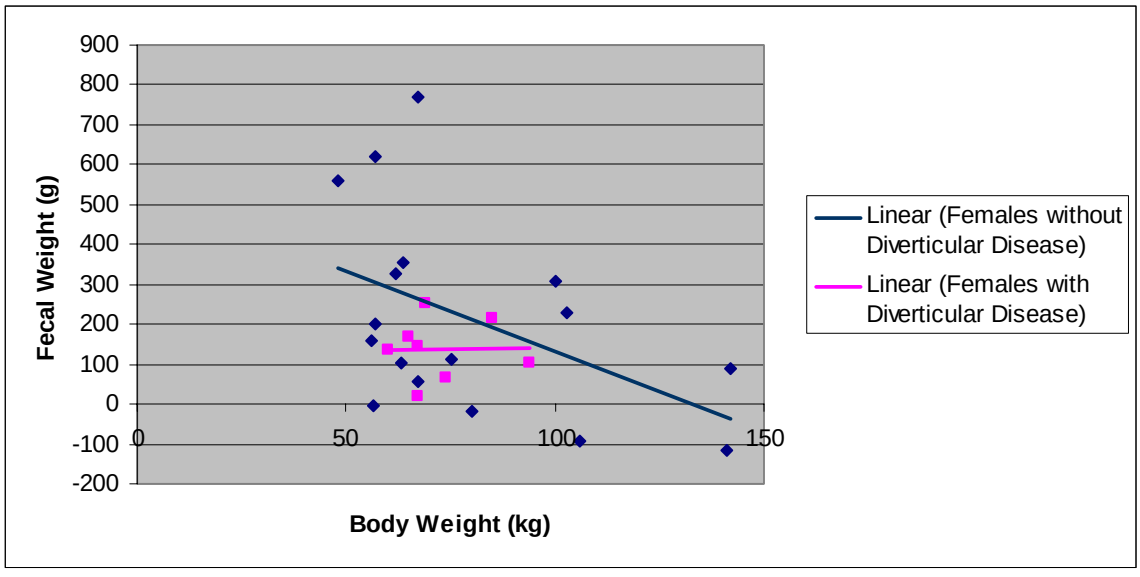


Figure 13 Linear Relationship between Body Mass Index and Fecal Weight in Females.

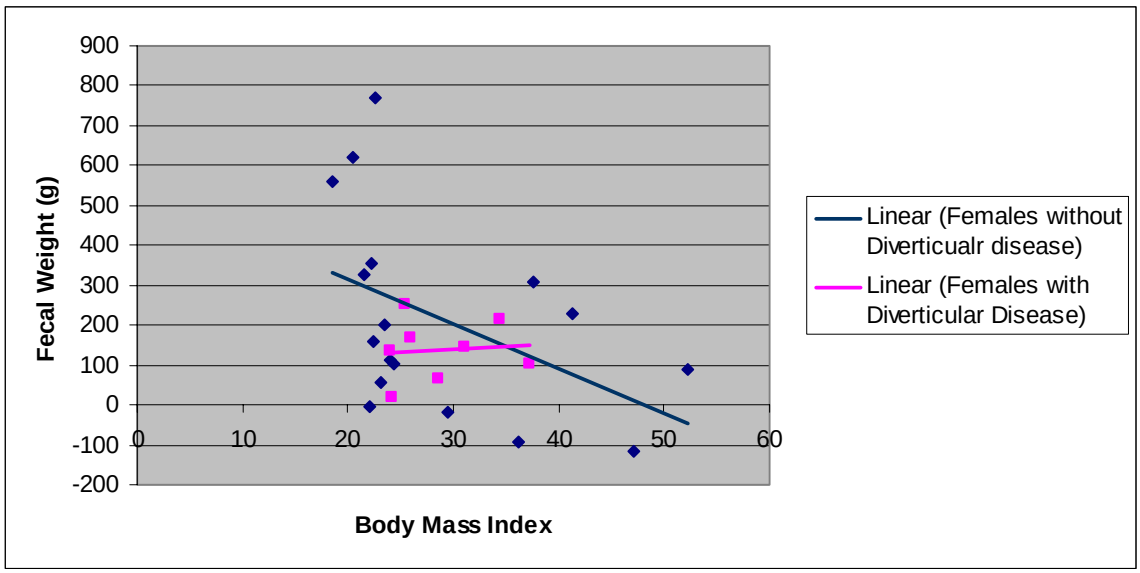


Figure 14 Linear Relationship between Colon Length and Fecal Weight in Females.

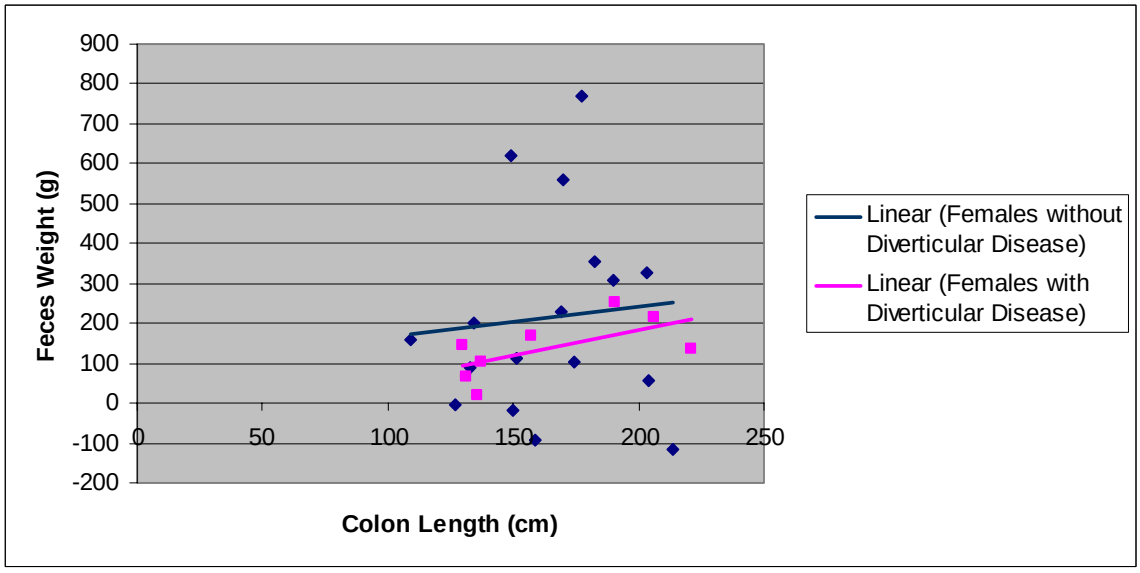
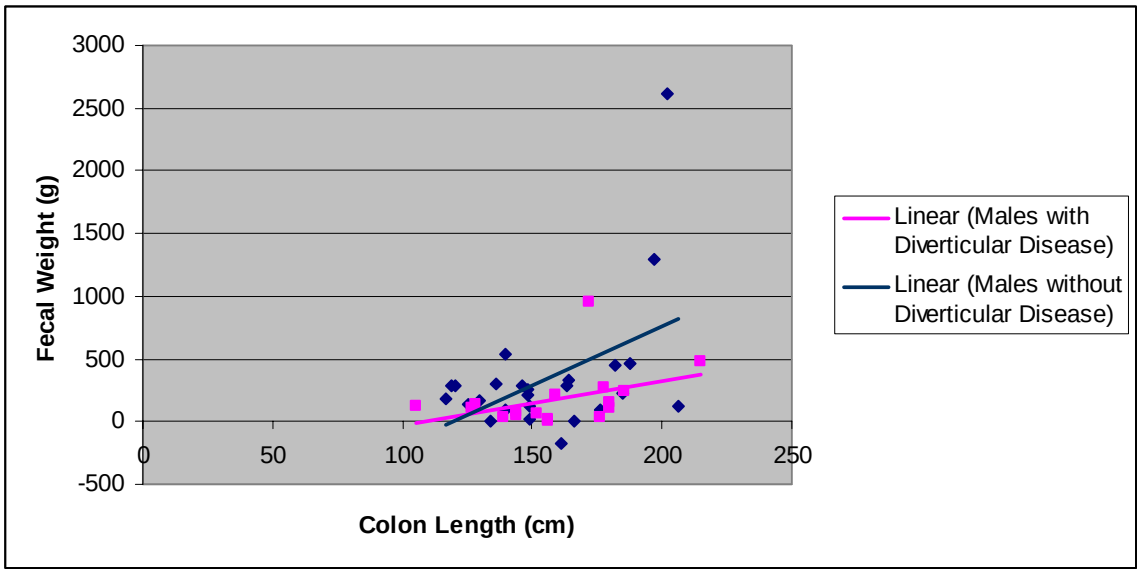


Figure 15 Linear Relationship between Colon Length and Fecal Weight in Males.



Incidence of Polyps

Polyps were determined to be the second most common disease in the colon. Distribution of polyps based on gender and age are shown in Table 8, Table 9, Table 10 and Figure 16, and Figure 17. Polyps occurred in 24% of individuals in this population, 31% of males and 20% of females. The occurrence of polyps increased from no prevalence under the age of 40 to 12-16% between ages 40 to 60 and further increased to 33-46% over the age of 60.

Table 8 Distribution of Polyps in Males.

Age	***Specimen***	***Polyps***	***Percentage***
15-29	4	0	0.00
30-39	3	0	0.00
40-49	4	2	50.00
50-59	8	2	25.00
60-69	11	4	36.36
70-79	9	5	55.55
80+	3	0	0.00

Table 9 Distribution of Polyps in Females.			
Age	***Specimen***	***Polyps***	***Percentage***
15-29	1	0	0.00
30-39	4	0	0.00
40-49	4	1	25.00
50-59	4	0	0.00
60-69	4	1	25.00
70-79	4	1	25.00
80+	4	2	50.00

Figure 16 Distribution of Polyps by Age and Gender

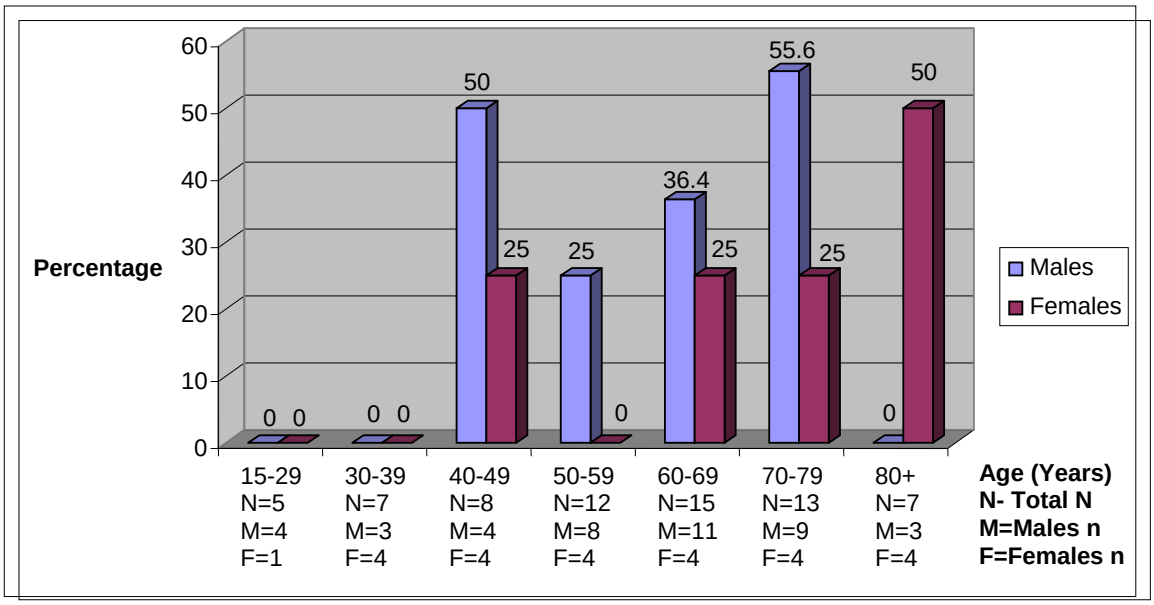
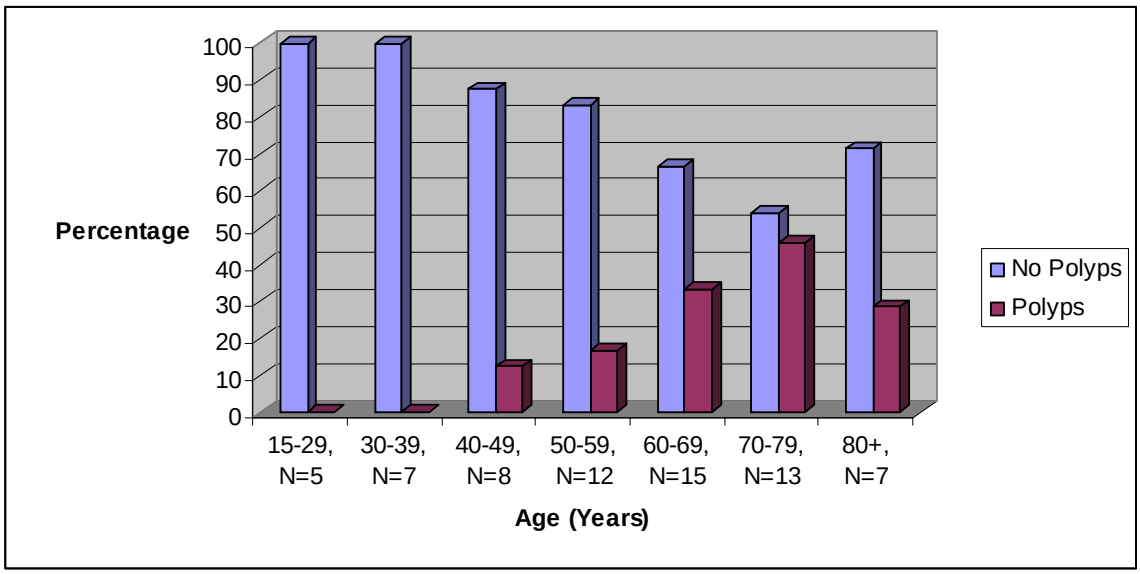


Table 10 Distribution of Polyps by Age.			
Age	***Specimen***	***Polyps***	***Percentage***
15-29	5	0	0.00
30-39	7	0	0.00
40-49	8	1	12.50
50-59	12	2	16.66
60-69	15	5	33.33
70-79	13	6	46.15
80+	7	2	28.57

Figure 17 Distribution of Polyps by Age



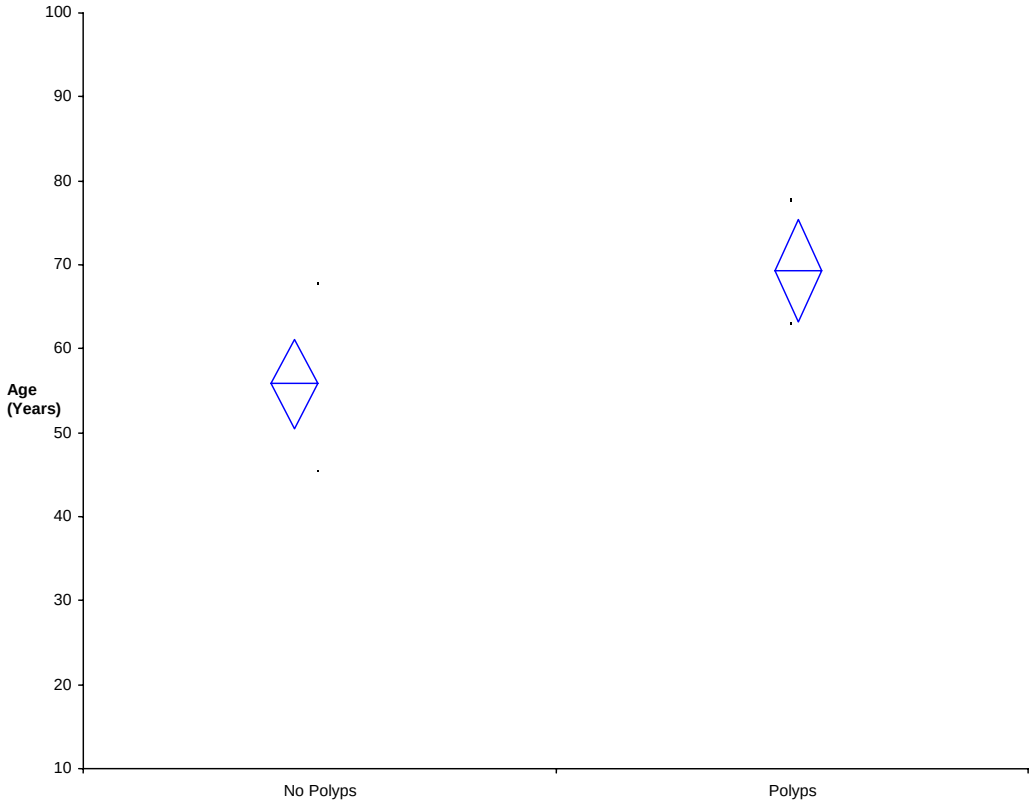
To determine the factors influencing the presence of polyps, a logistic regression was used, with the presence of polyps as the dependent variable and age, gender, body mass index, body weight, body length, colon plus feces weight, colon weight, fecal weight and colon length as predicting variables. Age was shown to make a significant ($p=0.0100$) contribution to the presence of polyps. Colon plus feces weight ($p=0.2499$), colon length ($p=0.2860$), colon weight ($p=0.3138$), gender ($p=0.5665$), feces weight ($p=0.6119$), body mass ($p=0.8040$), body weight ($p=0.9109$) and body length ($p=0.9112$) were shown to have no significant effect on the presence of polyps ($p > 0.05$). Table 11 shows the logistic regression for polyps and age. For every year increase in age (from age 15-92) the individual was 5.6% more likely to have polyps.

Figure 18 shows, the mean age of individuals without polyps was determined to be 54.9 years with a standard error of 2.63 years; the mean age of individuals with polyps was determined to be 69.3 years with a standard error of 2.89 years.

Table 11 Logistic Regression Output Between Polyps and Predicting Factors.

Factor	Variable	Coefficient	Standard Error	p=	Odds Ratio	Low	High	Intercept
Age	1	0.0550	0.0214	0.0100	1.0566	1.0133	1.107	-4.6134
Colon Plus Feces Weight	1	0.0003	0.0003	0.2499	1.0003	0.9998	1.0009	-1.7631
Colon Length	1	0.0109	0.0203	0.2860	1.0110	0.9909	1.0315	-2.9299
Colon Weight	1	0.0003	0.0003	0.3181	1.0003	0.9997	1.0009	-1.6186
Gender	1	0.3502	0.6109	0.5665	1.4194	0.4287	4.6998	-1.3863
Fecal weight	1	0.0004	0.0007	0.6119	1.0004	0.9990	1.0017	-1.2493
Body Mass Index	1	0.0099	0.0397	0.8040	1.0099	0.9343	1.0916	-1.4955
Body Weight	1	0.0004	0.0127	0.9109	1.0014	0.9768	1.0266	-1.2717
Body Length	1	-0.0004	0.0308	0.9884	0.9996	0.9409	1.0618	-1.1473

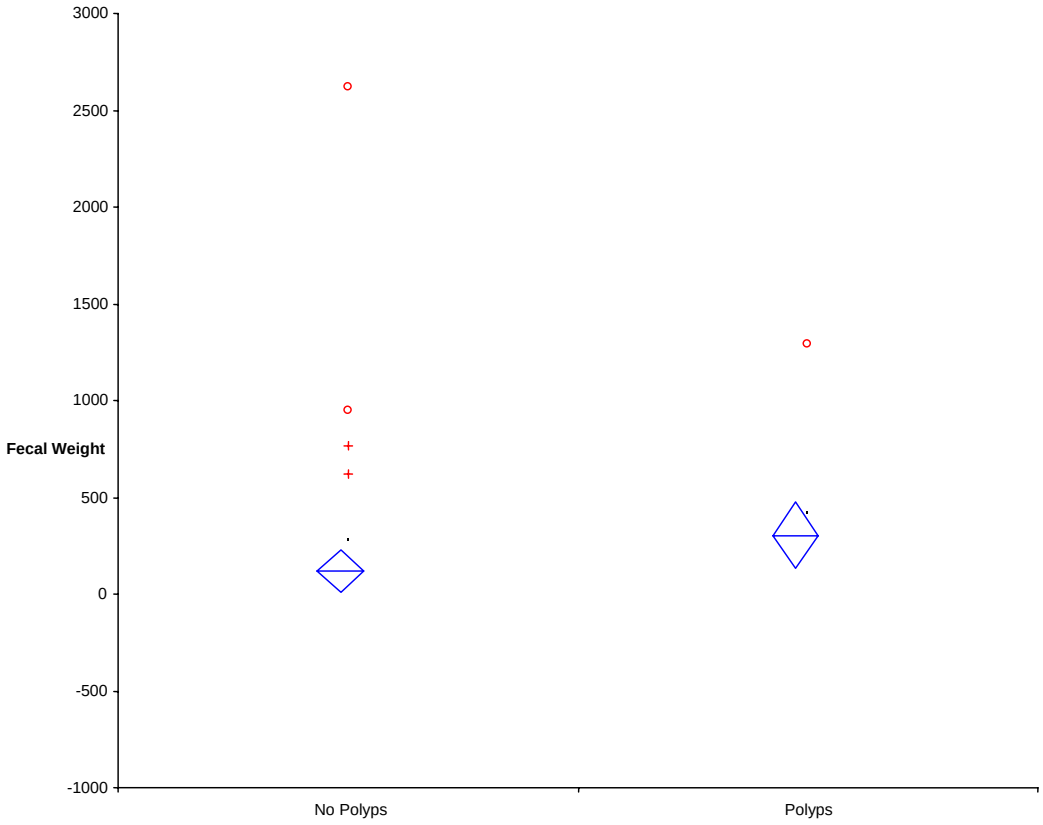
Figure 18 Distribution of Age in Individuals with and without Polyps



	n	Mean	SD	SE	95% CI of Mean	
No Polyps	51	54.961	18.8499	2.6395	49.659	to 60.262
Polyps	16	69.313	11.5915	2.8979	63.136	to 75.489

Although fecal weight was hypothesized to be a significant predictor no significant effect was identified ($p=0.6119$). Figure 19 shows, the mean fecal weight of individuals without polyps was determined to be 233.3 g with a standard error of 55.48 g; the mean fecal weight of individuals with polyps was determined to be 287.6 g with a standard error of 80.6 g.

Figure 19 Mean Fecal Weight in Individuals with and without Polyps



	n	Mean	SD	SE	95% CI of Mean	
No Polyps	51	232.157	396.1992	55.4790	120.724	to 343.590
Polyps	16	287.688	322.3815	80.5954	115.903	to 459.472

Pearson product correlation were performed in which a significant relationship ($r=-0.62$, $n=11$, $p<0.05$) was identified between age and body weight in males with polyps, but not in males without polyps ($r=-0.08$, $n=31$, $p>0.05$). Figure 20 shows that as age increased body weight decreased in males with polyps, and age increased as body weight remained the same in individuals without polyps. A significant Pearson product correlation ($r=-0.76$, $n=5$, $p<0.05$) was identified between body weight and fecal weight in females with polyps, but not in females without polyps ($r=-0.35$, $n=20$, $p>0.05$). Figure 21 shows an inverse relationship between fecal weight and body weight in females with polyps, but not in females without polyps. In this study all females with polyps were less than 100 kilograms where females without polyps were greater than 100 kilograms. A significant Pearson product correlation ($r=0.46$, $n=20$, $p<0.05$) was identified between body length and colon length in females without polyps, but not in females with polyps ($r=-0.19$, $n=5$, $p>0.05$). A similar significant Pearson product correlation ($r=0.49$, $n=11$, $p<0.05$) was identified between body length and colon length in males with polyps, but not in males without polyps ($r=-0.03$, $n=31$, $p>0.05$). Figure 22 and Figure 23 show the relationship of colon length and body length in males and females with and without polyps.

Figure 20 Linear Relationship between Age and Body Weight in Males.

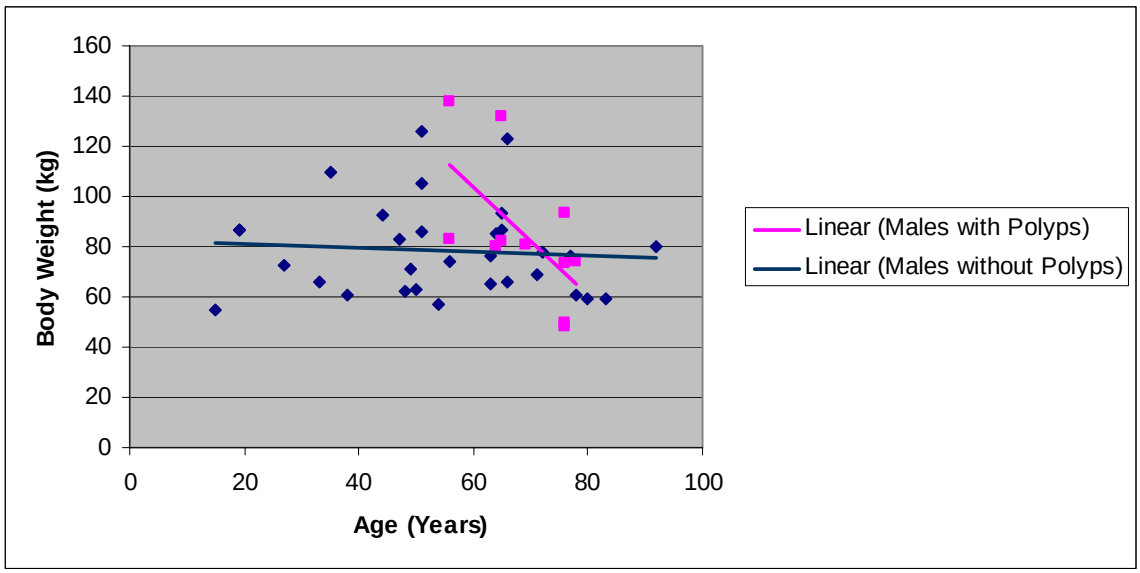


Figure 21 Linear Relationship between Body Weight and Fecal Weight in Females.

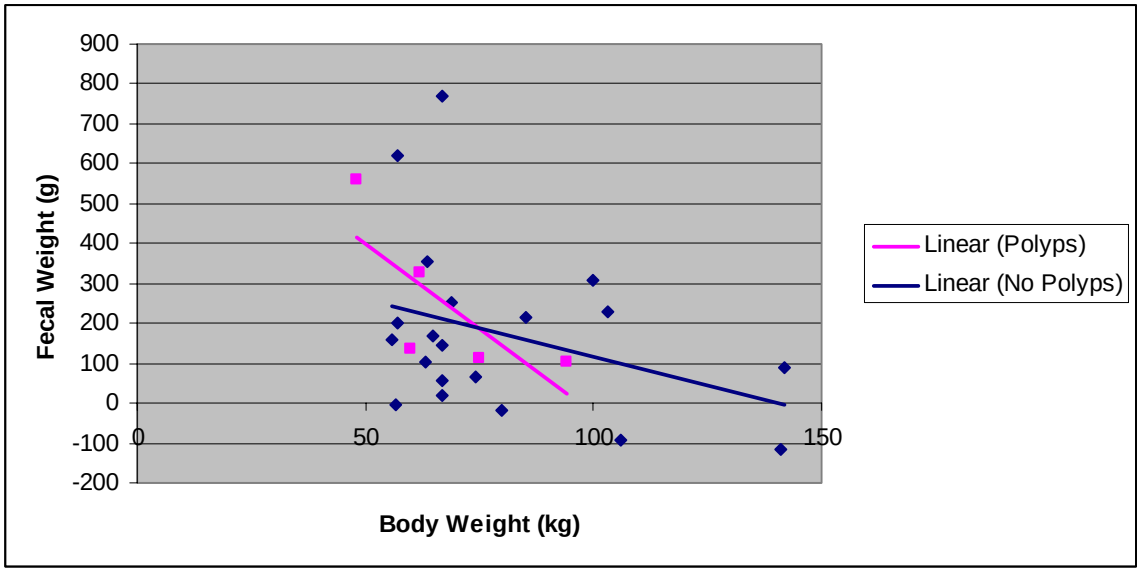


Figure 22 Linear Relationship between Colon Length and Body Length in Females.

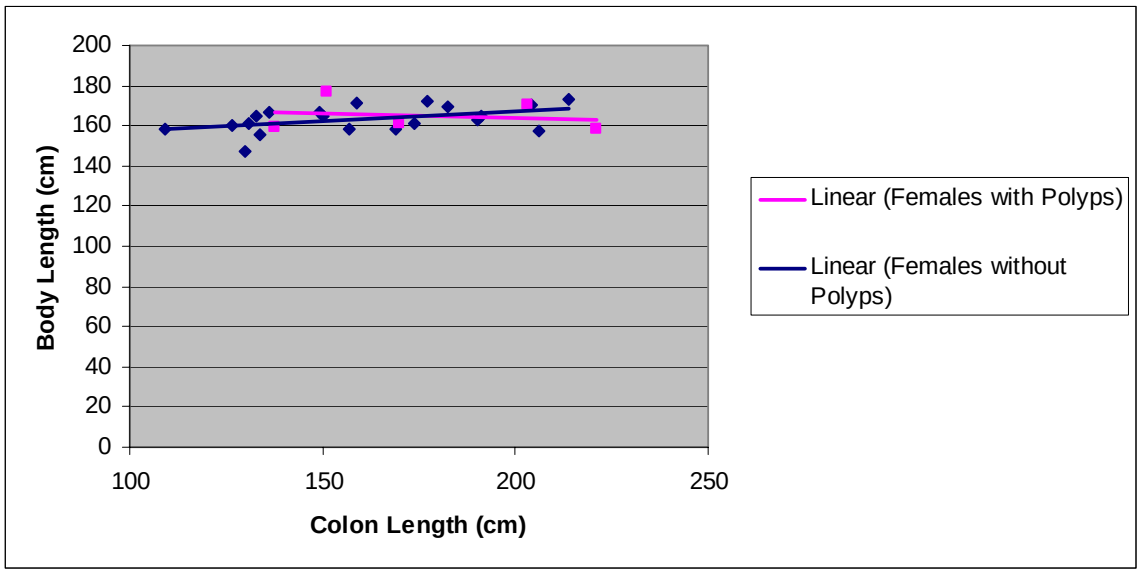
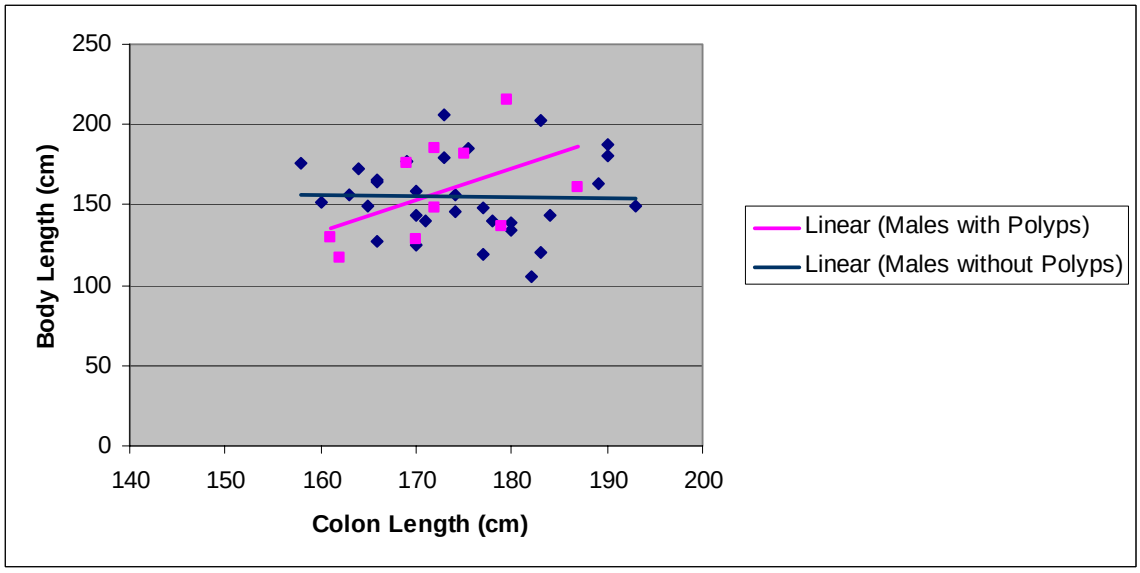


Figure 23 Linear Relationship between Colon Length and Body Length in Males.



Incidence of multiple abnormalities

Of the 67 colons analyzed 9, 13.4% had multiple abnormalities. To predict the factors influencing the presence of multiple abnormalities, a logistic regression was used, with the presence of multiple abnormalities as the dependent variable and age, gender, body mass index, body weight, body length, colon plus feces weight, colon weight, fecal weight, and colon length as predictor variables. Colon weight (p=0.0993), colon length (p=0.1827), colon plus feces weight (p=0.1858), body length (p=0.2311), age (p=0.2344), body mass (p=0.3293), feces (p=0.4748) body weight (p=0.5571) and gender (p=0.6356) were shown to have no significant effect on predicting multiple abnormalities ($p > 0.05$). Table 12 shows the logistic output for multiple abnormalities and predicting factors.

Table 12 Logistic Regression Output between Multiple Abnormalities and Predicting Factors

Factor	Variable	Coefficient	Standard Error	P=	Odds ratio	Low	High	Intercept
Colon Weight	1	0.0006	0.0004	0.0993	1.0006	0.9999	1.0012	-2.8374
Colon Length	1	0.0127	0.0129	0.1827	1.0174	0.9919	1.0434	-4.6951
Colon Plus Feces	1	0.0005	0.0004	0.1838	1.0005	0.9998	1.00012	-2.7266
Body Length	1	-0.0497	0.0415	0.2311	0.9516	0.8773	1.0321	6.5229
Age	1	0.0265	0.0222	0.2344	1.0268	0.9830	1.0726	-3.4886
Body Mass Index	1	0.0433	0.0444	0.3293	1.0443	0.9572	1.1393	-3.0718
Fecal Weight	1	0.0013	0.0018	0.4748	0.9987	0.9953	1.0022	-1.6121
Body Weight	1	0.0087	0.0149	0.5571	1.0088	0.9798	1.0386	-2.5682
Gender	1	-0.3443	0.7243	0.6356	0.7095	0.1715	2.9342	-1.6582

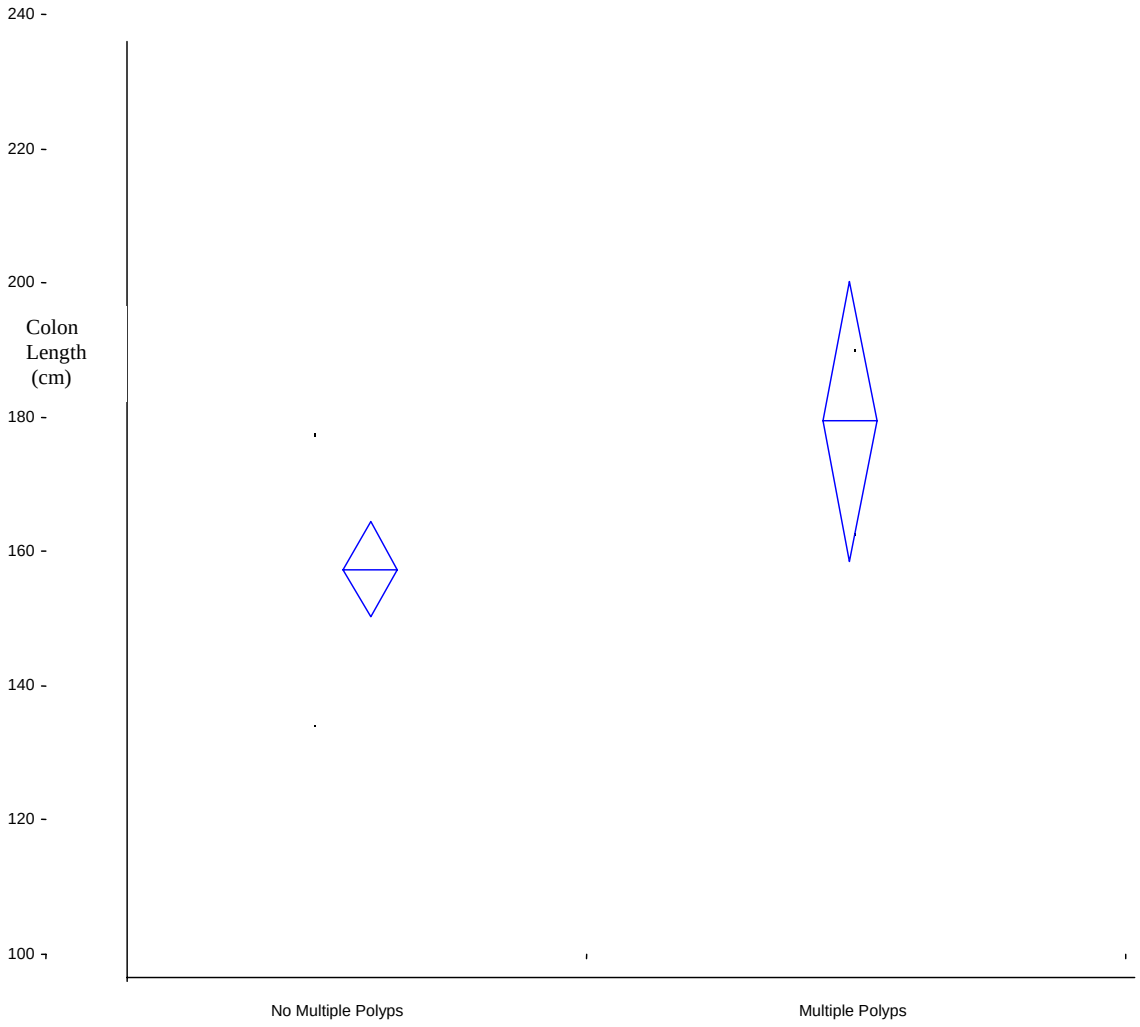
Incidence of multiple polyps

Of the 16 cases of polyps identified 10, 63% had two or more polyps. Of these colons analyzed the number of polyps in a single colon did not exceed 10. To determine the predicting factors for presence of multiple polyps, a logistic regression was used, with multiple polyps as the dependent factor and age, gender, body mass index, body weight, body length, colon plus feces weight, colon weight, fecal weight, and colon length as predicting variables. Shown in Table 13, colon length was determined to be a significant ($p=0.0265$) factor in predicting multiple polyps. Age ($p=0.1412$), colon weight ($p=0.1419$), colon plus feces weight ($p=0.1588$), body weight ($p=0.2724$), body mass index ($p=0.4312$), body length ($p=0.7700$), gender ($p=0.8490$), and feces weight ($p=0.9692$) were shown to have no significant effect on predicting multiple polyps ($p > 0.05$). For every centimeter increase in length (from 105-221cm) an individual is 3.01% more likely to have multiple polyps. Figure 24 shows, the mean colon length of individual without multiple polyps being 156.29 cm with a standard error of 3.56 cm; the mean colon length of individuals with multiple polyps being 178.75 cm with a standard error of 9.23 cm.

Table 13 Logistic Regression Output between Multiple Polyps and Predicting Factors

Factor	Variable	Coefficient	Standard Error	p=	Odds Ratio	Low	High	Intercept
Colon Length	1	0.0297	0.0134	0.0265	1.0301	1.0035	1.0575	-6.7109
Age	1	0.0325	0.0221	0.1412	1.0330	0.9893	1.0786	-3.7510
Colon Weight	1	0.0005	0.0003	0.1419	1.0005	0.9998	1.0012	-2.5626
Colon Plus Feces	1	0.0005	0.0003	0.1588	1.0005	0.9998	1.0011	-2.6268
Body Weight	1	0.0152	0.0139	0.2724	1.0153	0.9881	1.0433	-2.9842
Body Mass Index	1	0.0342	0.0435	0.4312	1.0348	0.9503	1.1268	-2.6832
Body Length	1	0.0104	0.8357	0.7700	1.0105	0.9422	1.0838	-3.5028
Gender	1	-0.1335	0.7015	0.8490	0.8750	0.2213	3.4603	-1.6582
Feces Weight	1	0.0000	0.0009	0.9692	1.0000	0.9981	1.0018	-1.7139

Figure 24 Mean Distribution of Colon Length by Individuals with and without Multiple Polyps



	n	Mean	SD	SE	95% CI of Mean	
No Multiple Polyps	57	156.219	26.9308	3.5671	149.074	to 163.365
Multiple Polyps	10	178.750	29.1912	9.2311	157.868	to 199.632

VII-Discussion

This study has reviewed an autopsy population of 67 in Manitoba and determined that 66% of individuals had some type of abnormality. This result is higher than the hypothesized result of 40-50%. Of the abnormalities identified the following prevalence's were determined; 37.3% diverticular disease, 24% polyps, 9% lymphoid nodules, 4.5% melena (symptom of gastrointestinal bleed), 3% lipomas and 1.5% diverticulitis and/or stenosis. Age was shown to make a significant ($p=0.0004$) contribution to predicting prevalence. The biological basis of aging is still not completely understood; however it is known that each species has a life span, which is determined by genetic and environmental factors. Each cell has a programmed molecular clock that reaches senescence after a specified number of replications. Cells sustain a variety of injuries throughout their life span and are exposed to molecular damage (Rubin, 2001). This study shows that with each year increase in age (from age 15-92) an individual is 6.8% more likely to develop an abnormality. In this study the mean age was 46.9 years for individuals without abnormalities and the mean age was 65.2 years with abnormalities. However age doesn't seem to be the only predicting factor. This study does not identify any other significant factors in predicting abnormalities but significant relationships between the factors tested provides some insight. This study shows a significant relationship ($r=0.63$, $n=13$, $p<0.05$) between age and colon length in females with abnormalities, but not in females without abnormalities ($r=0.05$, $n=12$, $p>0.05$). However, it was also determined that a significant relationship ($r=0.66$, $n=13$, $p<0.05$) existed between age and colon length in males without abnormalities, but not in males with

abnormalities ($r=-0.14$, $n=17$, $p>0.05$). Which raises the question, why is their opposite trends between males and females?

This study shows no significant relationship between gender and prevalence of abnormalities (see specific abnormalities below). Although gender differences have been noted in the literature (Jass, 1992); gender has not been studied extensively. Several studies document males as having greater risk, and higher prevalence (McCashland, 2001). Although this study showed no significant relationship, gender cannot be excluded as a factor; further studies into gender as a prediction factor should be performed, examining the affect of colon length and gender in the prediction of abnormalities. Colon length as a predicting factor in abnormalities has not been documented within the literature. Studies have been performed evaluating dietary changes in relation to colon length during colon tumorgenesis in rats, as the rats aged there colons remained the same length, and no carcinogenic relationship was determine for colon length (Chang, 1997).

Fecal weight was hypothesized to be significant in predicting abnormalities; however was not determined to be significant. This study showed the mean fecal weight of individuals without abnormalities was 303.20 g and the mean fecal weight for individuals with abnormalities was 211.02 g; interesting, however not statistically significant. A similar result was shown in 2003, in which no significant difference was determined between stool volumes and disease (Stollman, 2004).

Of the abnormalities identified, diverticular disease was the most common at 37.3%. As shown in the literature review, the incidence of the disease varies greatly between the sources. There appears to be numerous implications as to the etiology, with age, diet, racial variation being attributed the most. This study determined an incidence of

the disease in 12-33% of those under the age of 60 and 46-71% of those over the age of 60. A seminar in 2004 published the following results, in which 10% of individuals under the age of 40 were determined to have diverticular disease and 50-66% of those over the age of 80 were determine to have diverticular disease (Stollman, 2004). The results from this study are slightly higher than the literature values; this could be attributed to the small sample size, or possibly an unidentified prediction factor specific to the Manitoba population.

In this study age was determined to be significant ($p=0.0030$) in predicting diverticular disease. Age as a predicting factor has been documented in several of the studies examined (Hughes, 1969, Stemmermann, 1972, Paspatis, 2001, Kang, 2004, Stollman, 2004). The disease presents usually between age 40-50 and increases progressively with age (Hughes, 1969), peaking in the 8^{th} decade (Stemmermann, 1972). This study shows that the mean age of individuals without diverticular disease was 52.9 years, and 67.6 years in individuals with diverticular disease. Many speculate a supplementary factor other than age alone in predicting the progression of the disease; however this study does not determine any other single factor then age in predicting diverticular disease.

A significant Pearson product correlation ($r=-0.45$, $n=17$, $p<0.05$) was determined between body weight and fecal weight in females without diverticular disease. A similar result was identified with body mass index and fecal weight ($r=-0.46$, $n=17$, $p<0.05$). As females without diverticular disease increased in weight their feces weight decreased, but not in females with diverticular disease ($r=0.03$, $n=8$, $p>0.05$), ($r=0.10$, $n=8$, $p>0.05$) respectively. However, this study determined individuals without diverticular disease

have a mean fecal weight of 292.81 g; those with diverticular disease have a mean fecal weight of 165.80 g. Although feces weight is lower in individuals in this population with diverticular disease no significant difference was identified, but the relationship of body weight and fecal weight in diverticular disease should be examined in future studies. As the literature states that smaller stool volumes are thought to increase luminal pressure, this increase in pressure is thought to cause discrete 'little bladders' known as diverticula. A future study with a larger sample size might determine a relationship between pressure, stool volume and diet. The literature also states that it is not known if changes in the motility and the increased luminal pressure are causes of the disease or if they are symptoms (Stollman 2004). It has also been documented that diet effects stool volumes. Cellulose has been shown to increase the bulk of stool volume, as the plant cell walls bind water and salts, this increase bulk prevents hyper-segmentation (Ludeman, 2002). It has been shown that diets high in fiber from fruits but not cereals have been beneficial in reducing the risk of diverticular disease (Aldoori, 1997). As a result of these diet guidelines, diverticular disease has been labeled a disease of western diet (Kang, 2004).

The western diet consists mostly of dairy products, cereals, refined sugars, refined vegetable oils and alcohol, which accounts for 72% of the daily energy (Cordain, 2005). The recommended daily fiber intake is 20-30g/day, in western society the average intake is 15.1g/day (Cordain 2005). Individuals with lower fiber diets were determined to have longer transit times and smaller stool volumes (Stollman, 2004). This study shows no statistical relationship between fecal weight and disease ($p=0.2084$). A significant correlation is identified between body mass index and fecal weight in females without diverticular disease ($r=-0.45$, $n=17$, $p<0.05$), but not in females with disease ($r=0.10$, $n=8$,

$p>0.05$). Fecal weight cannot be excluded as a factor in predicting prevalence. Further studies should explore diet and obesity in the prediction of diverticular disease. Statistics show that 34.2% of adults in Manitoba are classified as obese by their body mass index (DHHW, 1988). Based on the criteria determine by the Department of National Health and Welfare, 73% of individuals in this autopsy study were determined to be obese, having body mass index score above 23, ranging from 23-52, with a mean body mass index of 29. Obesity and diet may be important factors in prevalence in a larger study population.

Colon weight and fecal weight were determined to have a significant relationship ($r=-0.53$, $n=17$, $p<0.05$) in females without diverticular disease, but not in females with diverticular disease ($r=-0.02$, $n=8$, $p>0.05$). However the literature has not identified colon length or fecal weight as prediction factors (Hughes, 1969, Stollman 2004), the literature does identify fecal relationships in rat models (Stollman, 2004). Although an inverse relationship between diet and disease is assumed, this study is unable to provide statistics support, as no information regarding diet can be determined from fecal weight.

Diverticular disease is an incidental finding at autopsy, and is not a common cause of death (Hughes, 1969). The literature states that 20% of all individuals who have diverticular disease will have diverticulitis at some point in their lives (Stollman 2004, Ludeman, 2002); this study identified one case in 67 as having diverticulitis, in which a 1.3 cm in diameter abscess cavity was identified in the overlying peridiverticular tissue and was unrelated to cause of death. Although examining the colon at time of autopsy is a good method of determining prevalence of diverticular disease, it is rarely a contributing factor in cause of death. Further studies of diverticular disease are required

as the literature has no new theories on determining etiology than were present in the early literature.

Polyps were the second most common disease at 24%, 16 of the 67 cases, were identified. This study determined only age to have a significant statistical contribution ($p=0.01$) to determining prevalence, no polyps were identified in individuals under 40 years of age, individuals between 40 and 60 had an incidence rate of 12-16%, increasing to 28-46% over the age of 60. The statistical significance of age has been established in many studies (Stemmermann, 1972, Vatn, 1982, Clark, 1985, Bombi, 1988, Coode, 1988, Cajucom, 1992, Jass, 1992, McCashland, 2001, Paspatis, 2001). This study shows that for every year increase in age (from 15-92) an individual is 5.6% more likely to have polyps. Individuals without polyps had a mean age of 54.9 years; individuals with polyps had a mean age of 69.3 years. Our result of 24% was consistent with that of the literature wherein the incidence ranged from 1.6% to 26% (Bombi, 1988).

The literature concludes that men have a higher prevalence of colonic polyps then women (McCashland, 2001), however this study showed no significant relationship between gender and polyps. Gender should be further explored in a larger population size.

Each of the other factors tested (body weight/length, colon weight/length) were shown not to be significant ($p > 0.05$). The factor hypothesized to be significant was fecal weight; however no statistical relationship was identified. Individuals without polyps have a mean fecal weight of 233.3 g; individuals with polyps had a mean fecal weight of 287.6 g. Jass in a 1992 study examined feces as a factor exposing the colon to harmful elements and injury. Although diet is attributed to playing a major role, no statistical

information can be evaluated from this population. It is theorized that diet is an important factor; Bombi postulates that diets rich in fish, vegetables, fewer milk derivatives and the use of vegetable oils in cooking might be of significance (Bombi, 1988). Although fecal weight alone was not a significant factor in predicting polyps, a significant inverse relationship (Figure 21) between body weight and fecal weight was identified in females with polyps ($r=-0.75$, $n=5$, $p<0.05$) but not in females without polyps ($r=-0.35$, $n=20$, $p>0.05$) . Body weights of females with polyps were all below 100 kilograms. As female body weight increased, female fecal weight decreased. Once again this leads us to speculate diet, as lower fecal weights are attributed to low fiber diets. Feces weight changes have been documented in rats feed specific diets with known fiber contents. Chang (1997) determined that dietary fiber was a significant factor ($p<0.0001$) in fecal output. Chang also notes that fish oil diets had a protective affect against colon adenocarcinomas. This study is unable to provide any information on the dietary demographics of the population studied, but suggests that further studies assessing diet in the prediction of polyps be performed. Another explanation could be attributed to a sample size, as only 5 females with polyps were identified. Of the polyps identified in this study all were unrelated to cause of death.

Of the 67 colons examined 9, 13.4% contained multiple abnormalities. Of the diseases most commonly associated together were diverticular disease and polyps. This is attributed to both diseases having the same age distribution in this autopsy study. This was also shown by Stemmermann in 1972, in which he stated that diverticula and polyps are likely to coexist in populations with high risks of cancer. He bases this on his conclusions that age, genetics and environmental factors influence both diseases, and that

no definite link is attributed to the coexistence of these disease (Stemmermann, 1972). Although no statistical significant relationship was identified ($p > 0.05$), each factor assessed provides insight into trends in the influence of predicting multiple abnormalities. Multiplicity and/or multiple abnormalities may reflect injury to the mucosal (Jass, 1992). Although it is evident that abnormalities occur in multiples, no information as to prediction factors was identified.

Of the 16 cases of polyps identified 10, 63% contain multiple polyps. The total number of polyps in a single specimen did not exceed 10. The literature identifies multiplicity as an indicator of mutagenic activity, as multiplicity is related to malignancy risk (Jass, 1992). Colon length was not hypothesized to be significant however a statistical significance relationship ($p=0.0265$) was identified. For every centimeter increase in length (from 105-221cm) the individual is 3.01% more likely to have multiple polyps. The mean length of the colon in individuals without multiple polyps was determined to be 156.29 cm; the mean colon length in individuals with multiple polyps was determined to be 178.75 cm. The longer the colon the greater the surface area the more likely the mucosal will be exposed to harmful agents and induce mucosal damage (Jass, 1992). It should be noted that colon length measurements were not standardized for time since death, and therefore do not reflect muscles relaxation, and factors of autolysis.

Jass concludes that age, race and body mass index have little significance in the prediction of multiplicity. This study confirms this finding as no statistical relationships were identified in the prediction of multiple polyps ($p > 0.05$).

Locating abnormalities such as these within the colon at autopsy is, more often than not, an incidental finding. A study examining 106 autopsies was performed in

Winnipeg, Manitoba, 8% had a major diagnosis of missed clinical significance identified (Gough, 1985). Of the 8%, colonic abnormalities were not identified as a cause of death. Autopsies are performed as a method of understanding an illness, co-existing conditions and determining cause of death (CAP, 2001). Autopsies are beneficial to a community in that they increase the knowledge of cause and course of an illness as well as the effects of different treatment methods (CAP, 2001). There has been a decline in autopsy requests as a result of family concerns (Baron, 2000). In addition, costs of autopsies are an increasing issue, as the cost can reach upwards of $3500 per case (Baron, 2000). An autopsy takes on average two to four hours to perform (CAP, 2001). The external examination of the colon is performed routinely; and several diseases including diverticular disease are frequently identified as a result of out-pouching in the colon wall (Stollman, 2004). Internal examinations are performed when indications of disease are evident, for example, adhesions, congestion, and possible gastrointestinal bleed. The distal 10 cm of the intestine is removed and examined in all autopsies. This portion of the intestine has the highest potential of malignancy as colorectal cancer is the 3rd leading cause of death (McCashland, 2001). This area is also the most common area examined clinically as there is no need for x-rays or barium studies, and abnormalities can be identified by digital rectal examination (Moore, 2006).

On average the removal and internal examination of the colon in this study took approximately 10-15 minutes, in which, only one of the 67 cases contained an abnormality (melena) which was related clinically to the cause of death (gastrointestinal bleed). Therefore, examining the colon seems unnecessary when considering family views, time and cost required to examine the colon when the signs of disease and/or

abnormality are absent. On the other hand, this study identified 66%, 42 of the 67 colons examined to have some clinical finding of interest. Therefore to provide an accurate and complete autopsy report, colon abnormalities should be documented and for that reason the colon must be examined in each autopsy to facilitate this standard of care.

VIII-Conclusions

Meticulous examination of the colon at autopsy in the Manitoba population yielded a frequency of 66% abnormality, slightly higher than the hypothesized figure. Frequencies of 37.3% for diverticular disease, 24% for polyps, 9% for lymphoid nodules, 4.5% melena, 3% for lipomas, and 1.5% for diverticulitis and/or stenosis were identified. As hypothesized, there is strong statistical evidence to indicate that age plays an important role in the prediction of prevalence of these diseases, as diverticular disease increased from a prevalence of 12-33% under the age of 60, to 46-71% over the age of 60, and polyps increased from a prevalence of 12-16% under the age of 60 to 33-46% over the age of 60. We hypothesized that fecal weight would play a significant role in the prediction of abnormalities; however, no significant statistical relationship was identified. There are an increasing number of literary reviews on the importance of diets high in vegetable fibers, and low in refined foods in lowering the risk of abnormalities. The literature identifies lower fecal weight with lower fiber diets resulting in an increased risk of disease; however no direct connection can be assessed in our population. Colon length was not hypothesized to be significant in the prediction of abnormalities, however proved to be statistically important in predicting the presence of multiple polyps, possibly attributed to large surface area providing greater opportunity for mucosal injury and proliferative damage. Although several factors when considered together were determined to affect prevalence and prediction factors, none other than age and colon length were determined to be a factors on its own.

This study is limited in that a sample size of only 67 autopsies was assessed. Therefore a true representation of the abnormalities in the Manitoba population can not

be determined. As a result of the small sample size 42 males and 25 females no gender relationships could be identified. In future studies a larger sample size, with an equal male to female ratio may be more reflective of the population's afflictions.

This study is also limited with respect to patient demographics. Further exploration of ethnic origins and dietary habits may provide more insight into the prediction patterns of disease and abnormalities.

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Appendix

Pearson product correlation for females with no Abnormalities, n=12, p=0.05

Females with no Abnormalities	*Age*	*Body Weight*	*Body Length*	*Body Mass Index*	*Colon plus feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.30	1.00						
Body Length	-0.44	0.15	1.00					
Body Mass Index	-0.25	0.98	-0.02	1.00				
Colon plus feces Weight	-0.07	0.81	0.14	0.78	1.00			
Colon Weight	-0.05	0.82	0.10	0.80	0.96	1.00		
Feces Weight	-0.04	-0.44	0.10	-0.43	-0.34	-0.58	1.00	
Colon Length	0.05	0.14	0.47	0.05	0.28	0.24	0.01	1.00

Pearson product correlation for females with Abnormalities, n=13, p=0.05

Females with Abnormalities	*Age*	*Body Weight*	*Body Length*	*Body Mass Index*	*Colon plus feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	0.13	1.00						
Body Length	0.24	0.06	1.00					
Body Mass Index	0.03	0.89	-0.40	1.00				
Colon plus feces Weight	0.29	0.69	-0.16	0.71	1.00			
Colon Weight	0.29	0.78	-0.16	0.78	0.97	1.00		
Feces Weight	-0.03	-0.51	0.02	-0.46	-0.10	-0.32	1.00	
Colon Length	0.63	0.00	0.20	-0.08	0.47	0.36	0.38	1.00

Pearson product correlation for males with No Abnormalities, n=13, p=0.05

Males with no Abnormalities	*Age*	*Body Weight*	*Body Length*	*Body Mass Index*	*Colon plus feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	0.34	1.00						
Body Length	-0.13	0.15	1.00					
Body Mass Index	0.38	0.94	-0.19	1.00				
Colon plus feces Weight	0.35	0.82	0.02	0.79	1.00			
Colon Weight	0.30	0.73	-0.11	0.76	0.84	1.00		
Feces Weight	0.17	0.33	0.22	0.23	0.49	-0.07	1.00	
Colon Length	0.66	0.47	0.08	0.42	0.39	0.09	0.57	1.00

Pearson product correlation for males with Abnormalities, n=17, p=0.05

Males with Abnormalities	*Age*	*Body Weight*	*Body Length*	*Body Mass Index*	*Colon plus feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.29	1.00						
Body Length	-0.34	0.51	1.00					
Body Mass Index	-0.17	0.93	0.17	1.00				
Colon plus feces Weight	0.11	0.62	0.20	0.71	1.00			
Colon Weight	0.07	0.74	0.24	0.75	0.95	1.00		
Feces Weight	0.09	-0.25	-0.14	-0.04	0.32	0.00	1.00	
Colon Length	-0.14	0.31	0.16	0.40	0.65	0.55	0.39	1.00

Pearson product correlation for females with no Diverticular Disease, n=17, p=0.05

Females with No Diverticular Disease	*Age*	*Body Weight*	*Body Length*	*Body Mass Index*	*Colon plus Feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.16	1.00						
Body Length	0.22	0.25	1.00					
Body Mass Index	-0.20	0.98	0.06	1.00				
Colon plus Feces Weight	-0.01	0.83	0.20	0.80	1.00			
Colon Weight	-0.02	0.86	0.18	0.83	0.97	1.00		
Feces Weight	0.04	-0.46	-0.02	-0.45	-0.31	-0.53	1.00	
Colon Length	0.36	0.21	0.50	0.12	0.40	0.33	0.09	1.00

Pearson product correlation for females with Diverticular Disease, n=8, p=0.05

Females with Diverticular Disease	*Age*	*Body Weight*	*Body Length*	*Body Mass Index*	*Colon plus Feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.63	1.00						
Body Length	-0.26	-0.01	1.00					
Body Mass Index	-0.45	0.91	-0.43	1.00				
Colon plus Feces Weight	-0.06	0.73	-0.08	0.68	1.00			
Colon Weight	-0.07	0.73	-0.06	0.67	0.99	1.00		
Feces Weight	0.06	0.03	-0.19	0.10	0.10	-0.02	1.00	
Colon Length	0.48	-0.17	0.11	-0.21	0.39	0.31	0.63	1.00

Pearson product correlation for males with no Diverticular Disease, n=25, p=0.05

Males with No Diverticular Disease	*Age*	*Body Weight*	*Body Length*	*Body Mass Index*	*Colon plus Feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.02	1.00						
Body Length	-0.46	0.40	1.00					
Body Mass Index	0.25	0.91	0.00	1.00				
Colon plus Feces Weight	0.23	0.66	0.15	0.76	1.00			
Colon Weight	0.20	0.69	0.06	0.75	0.86	1.00		
Feces Weight	0.05	0.05	0.20	0.10	0.40	-0.13	1.00	
Colon Length	0.35	0.25	0.16	0.35	0.44	0.20	0.46	1.00

Pearson product correlation for males with Diverticular Disease, n=17, p=0.05

Males with Diverticular Disease	*Age*	*Body Weight*	*Body Length*	*Body Mass Index*	*Colon plus Feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.35	1.00						
Body Length	-0.08	0.39	1.00					
Body Mass Index	-0.08	0.38	1.00	1.00				
Colon plus Feces Weight	-0.34	0.95	0.08	0.07	1.00			
Colon Weight	-0.07	0.72	0.14	0.14	0.71	1.00		
Feces Weight	-0.10	0.80	0.21	0.21	0.77	0.98	1.00	
Colon Length	0.08	0.07	-0.17	-0.17	0.11	0.59	0.39	1.00

Pearson product correlation for females with no Polyps, n=20, p=0.05

Females No Polyps	*Age*	*Body Weight*	*Body Length*	*Body mass Index*	*Colon plus Feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.15	1.00						
Body Length	-0.31	0.31	1.00					
Body mass Index	-0.08	0.97	0.07	1.00				
Colon plus feces Weight	0.05	0.83	0.34	0.78	1.00			
Colon weight	0.06	0.84	0.30	0.80	0.97	1.00		
Feces Weight	-0.08	-0.35	0.08	-0.36	-0.21	-0.43	1.00	
Colon Length	0.20	0.31	0.46	0.22	0.50	0.43	0.11	1.00

Pearson product correlation for females with Polyps, n=5, p=0.05

Females with Polyps	*Age*	*Body Weight*	*Body Length*	*Body mass Index*	*Colon plus Feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.50	1.00						
Body Length	0.42	0.05	1.00					
Body mass Index	-0.64	0.93	-0.31	1.00				
Colon plus feces Weight	-0.50	0.58	-0.75	0.82	1.00			
Colon weight	-0.38	0.70	-0.59	0.88	0.97	1.00		
Feces Weight	-0.13	-0.76	-0.13	-0.67	-0.46	-0.67	1.00	
Colon Length	0.73	-0.64	-0.19	-0.54	-0.07	-0.10	0.15	1.00

Pearson product correlation for males with no Polyps, n=31, p=0.05

Males with no Polyps	*Age*	*Body Weight*	*Body Length*	*Body mass Index*	*Colon plus Feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.08	1.00						
Body Length	-0.35	0.29	1.00					
Body mass Index	0.06	0.91	-0.13	1.00				
Colon plus feces Weight	0.13	0.64	0.05	0.62	1.00			
Colon weight	0.18	0.62	-0.02	0.63	0.87	1.00		
Feces Weight	-0.07	0.22	0.15	0.13	0.50	0.00	1.00	
Colon Length	0.21	0.30	-0.03	0.30	0.43	0.27	0.39	1.00

Pearson product correlation for males with Polyps, n=11, p=0.05

Males with Polyps	*Age*	*Body Weight*	*Body Length*	*Body mass Index*	*Colon plus Feces Weight*	*Colon Weight*	*Feces Weight*	*Colon Length*
Age	1.00							
Body Weight	-0.62	1.00						
Body Length	-0.62	0.79	1.00					
Body mass Index	-0.51	0.97	0.63	1.00				
Colon plus feces Weight	-0.39	0.76	0.54	0.95	1.00			
Colon weight	-0.46	0.89	0.55	0.95	0.94	1.00		
Feces Weight	0.24	-0.40	-0.08	-0.06	0.13	-0.22	1.00	
Colon Length	-0.13	0.40	0.50	0.66	0.71	0.54	0.48	1.00