

The Gene Scene

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Inside this Issue

- 1 Colorectal Cancer Risks
- 2 What is FAP?
- 2 Newsletter Staff
- 3 Message from the Editors
- 4 Research News
- 7 National FAP Registry Study
- 8 Questions and Answers
- 9 Kid's Corner

Colorectal Cancer Risks

By David Weinberg, M.D.

Considering the effect of family history on cancer risk is crucial. Nowhere is this more true than colorectal cancer (CRC). The lifetime chance to develop CRC for the average risk person (see Table 1) is less than 5%. However patients with a family history of CRC, and to a lesser degree polyps, are at substantially greater risk compared to the general population. This risk increases with the number and age of the affected relatives, as well as the relationship (1st degree, 2nd degree etc).

The most common clinical scenario for many patients is a 1st degree relative (mother, father, sibling) diagnosed with CRC around the age of 60-70. Most research studies have suggested that this relationship increases one's personal lifetime risk by approximately 3 fold. To place this in the context of other frequently discussed cancers, it means CRC risk may reach 15%, thereby exceeding most women's chance to develop breast cancer or of a man to develop prostate cancer. Risk is even greater if the relative developed CRC at a younger age, or multiple 1st degree relatives of any age are affected. The good news is that nearly all cases of CRC can be completely prevented by diligent use of CRC screening.

Unlike, for example, breast cancer where mammography is directed predominantly towards the early detection of disease, CRC screening attempts to prevent CRC through the detection and removal of polyps, a necessary first step to cancer, before CRC develops. While removal of any single polyp can not guarantee lifetime protection, a program of periodic polyp detection and removal can greatly reduce CRC risk over time.

At present, the most effective manner to perform CRC screening in persons at higher risk is periodic colonoscopy, an outpatient test that allows for the detection and removal of polyps. This is in contrast to CRC screening for persons at average risk, where a combination of annual testing of stool for occult blood combined with flexible sigmoidoscopy every 3-5 years is advocated. Alternatives include barium enema every 5 years or colonoscopy every 10 years.

Screening for persons at average risk should begin at age 50. In contrast, screening for persons at elevated risk should begin 10 years before their earliest affected relative, or age 40, whichever comes first.

Prevention of CRC is a real possibility. Reminding your primary care physician of your personal and family history is the best method to do so.

Table 1: Relative CRC Risk Evaluations Based on Family History

Average lifetime population risk is 2% - 5%
One 1 st Degree Relative affected = 3:1
One 1 st Degree and one 2 nd degree relative = 4:1
One affected relative under age 45 = 5:1
Two First Degree relatives = 8:1

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What is FAP?

By Lael Melchert, M.S., C.G.C.

The letters FAP stand for ***Familial Adenomatous Polyposis syndrome***. FAP is a rare condition where there is a tendency to develop many polyps mainly in the colon and rectum. But before we review FAP in detail, we should step back and describe some terminology so that everyone reading this article will have the same basic understanding.



Figure 1: A polyp is a mass of tissue that bulges upward or outward from a surface. In FAP, many polyps develop throughout the colon and rectum.

Stedman's Concise Medical Dictionary. Ed. McDonough, J.T., Williams & Wilkins, Philadelphia, PA 1994.

As you may already know, a ***polyp*** is a mass of tissue that bulges outward or upward from a surface (See figure 1). An ***adenomatous*** polyp is a polyp that is usually benign (non-cancerous), but has the potential to develop into a cancer. ***Cancer*** occurs when cells become abnormal and keep dividing and forming more cells without control or order. A ***syndrome*** is a collection of findings that together constitute a particular condition. In FAP, one common group of associated features includes: 100's-1000's of polyps in the colon and rectum, polyps in the stomach and duodenum, and freckle-like spots on the back of the eye. The colon, rectum, stomach, and duodenum are all part of the gastrointestinal system (figure 2). Other FAP-associated features may also be present, and will be described later. ***Familial***

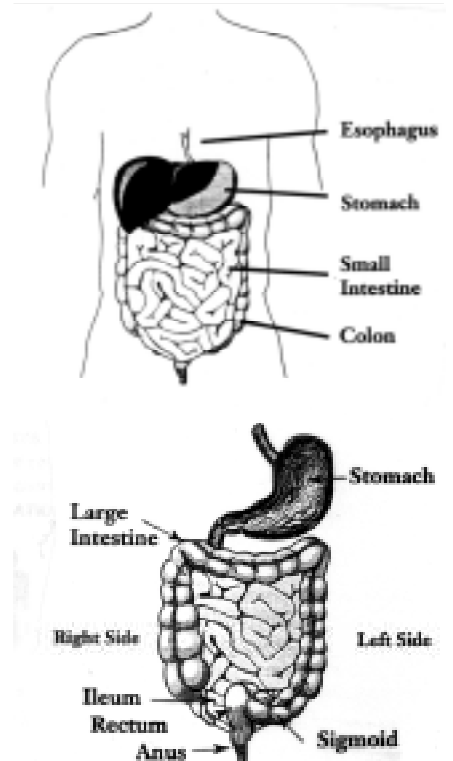


Figure 2: The gastrointestinal (GI) tract. The colon and rectum are part of the GI tract. Together they form a tube, 5 to 6 feet long, called the large intestine (also called the large bowel). The rectum is the last 6 to 8 inches of the large intestine. The duodenum connects the stomach to the small intestine.

Cancer Genetic Counseling, A Guide for Communicating Genetic Information. Rubinstein, W.S, Gettig, E.A., Stadler, M.P., Milliron, K.J., University of Pittsburgh Cancer Institute and Magee-Women's Hospital. 1999.

means that FAP is a condition that "runs in families", and is caused by a change in a particular gene which can be passed from generation to generation. ***Genes*** are segments of DNA that contain chemical information to make proteins, control bodily traits, or influence the activity of other genes. Genes are housed on larger structures called ***chromosomes***, and most people have 23 pairs of chromosomes (46 total), with one pair that came from the mother and the other pair from (continued on page 3)

(continued from page 2)

the father (figure 3). Chromosomes are numbered 1 through 22, with the 23rd pair being called the sex chromosomes since they determine whether a person is male or female. The chromosomes are found in almost every cell in the body, but are so small that they can only be seen using a microscope. Because the genes are housed inside the chromosomes, they can't be seen by a microscope, but can be analyzed by using special "molecular" genetic tests.

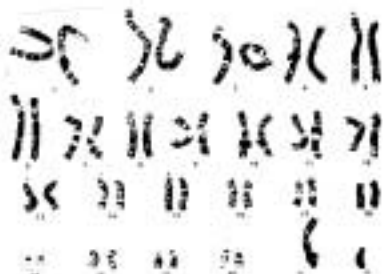


Figure 3: Almost every human cell contains 23 pairs of chromosomes (46 total) which can be distinguished by their size and banding patterns. This set of chromosomes is from a male. The APC gene responsible for FAP is found on chromosome number 5.

A permanent change in a gene, called a *mutation*, can alter or destroy the function of a protein that can predispose to disease. FAP is caused by a mutation in the adenomatous polyposis coli gene, or APC gene, which is found on chromosome number 5. Molecular tests can be done using a person's blood sample to find out if they have a mutation in the APC gene causing FAP.

So, what is FAP?

FAP is a syndrome that predisposes individuals to colon cancer. It occurs in approximately one in every 5,000-10,000 individuals. The main feature of FAP is the appearance of hundreds to thousands of pre-cancerous polyps in the colon and rectum. The

average age at which polyps develop is 16 years. (By the age of 10 years, 15% of people with FAP will have polyps; by the age of 20 years, 75% will have developed polyps; by 30 years, 90% will have polyps). Inevitably, at least one of these polyps progresses to cancer by the age of 60 years or sooner, unless the colon is removed. The average age that colon cancer develops is 39 years in an untreated person with FAP. Therefore, it is very important that people who have FAP are identified at a young age, so that the colon can be removed to prevent a cancer from developing. Some, but not all, people with FAP also have features that occur outside of the colon. Some of these include: polyps in the stomach and duodenum, freckle-like spots on the back of the eye (called congenital hypertrophy of the retinal pigment epithelium, or CHRPE), osteomas, dental abnormalities (late-erupting, missing, extra teeth, or cysts), soft tissue tumors (epidermoid cysts and fibromas), and desmoid tumors. Polyps in the stomach, often called gastric fundic gland polyps, are polyps with little potential to become cancerous. Polyps of the duodenum are seen in at least 50% of people with FAP, and have a 4-12% lifetime risk to develop into a cancer. Polyps of the small intestine are also seen in people with FAP, but they rarely become malignant. About 10% of persons with FAP develop desmoid tumors. Desmoid tumors are benign fibrous tissue tumors that most often form in the abdomen or in the abdominal wall and may occur spontaneously, or may be stimulated by abdominal surgery. Desmoid tumors may compress abdominal organs or complicate surgery, but typically do not become malignant. Other cancers that occur infrequently, but with a higher incidence in people with FAP than in the general

(continued on page 4)

Message from the Editors

We are pleased to welcome you to the first issue of *The Gene Scene*, the official newsletter of the Jefferson Familial Colorectal Cancer Registry. One of our main goals in creating this newsletter is to provide information to individuals and families who are at increased risk of developing colon and rectal cancer. This newsletter will be a reliable and convenient way to keep you informed of the very latest research and treatment in hereditary colorectal cancer.

We have all witnessed or experienced first hand the challenge of colorectal cancer. We feel that education about colorectal cancer is an essential step in preventing the disease. By knowing your risk, you and your physicians can devise an appropriate cancer screening and surveillance strategy.

The Gene Scene will be published twice a year. In each issue we will highlight a specific hereditary colorectal cancer syndrome, provide relevant research news, and provide a list of resources of interest to individuals with an increased risk of developing colorectal cancer. This newsletter is for you and your family. Therefore, in each issue we will answer patient questions and devote a special section to children.

We appreciate and look forward to hearing from our readers. If you have questions or an idea for an upcoming newsletter please feel free to contact Deborah Rose at (215) 955-0026 or e-mail at Deborah.Rose@mail.tju.edu.

Research News:

The Kimmel Cancer Center's mission is to improve upon the survival and quality of life of cancer patients by translating basic research discoveries into new strategies to prevent, diagnose, monitor and cure human cancer. In keeping with this mission, scientists and physicians work together to study why some individuals may develop cancer while others do not. Richard Fishel, Ph.D., Professor of Microbiology and Immunology, is part of the multidisciplinary team at Jefferson studying the genetic basis of colorectal cancer. Dr. Fishel is a world-renowned scientist in the field of colorectal cancer genetics. In 1994, Fishel and his colleagues discovered two of the genes responsible for an inherited form of colorectal cancer called Hereditary Nonpolyposis Colorectal Cancer (HNPCC).

Aspirin May Reduce Susceptibility to Hereditary Colorectal Cancers

A new study suggests that aspirin may interfere with the cancerous process. It may prevent the development of a particular type of hereditary colorectal cancer in those at high risk for the disease.

Scientists at Jefferson Medical College believe they've uncovered a potential biochemical mechanism by which aspirin interferes with colorectal cancer development in those individuals who carry a particular gene mutation that makes them very likely to get the disease.

(continued on page 5)

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population include: hepatoblastoma (liver cancer), and cancer of the thyroid, pancreas, adrenal glands, gallbladder, and brain (particularly a type called medulloblastoma).

Are there different types of FAP?

Three categories of FAP exist in addition to classic FAP including: Attenuated FAP (AFAP), Gardner syndrome, and Turcot syndrome. Each used to be thought of as a distinct condition, but now are all recognized as being part of the overall spectrum of FAP.

Attenuated FAP (AFAP) is a "milder" form of classic FAP characterized by fewer colon polyps (usually 1-50). Polyps in AFAP are typically found in the right side of the colon (also called the proximal or ascending colon). Despite there being fewer polyps, AFAP still has a high lifetime risk for colorectal cancer with a tendency to develop stomach and duodenal polyps. The average age to develop a colon or rectal cancer in AFAP is approximately 55 years, (compared to 39 years as in classic FAP). There is a risk for duodenal cancer as in classic FAP, but other cancers have not yet been seen. The freckle-like spots on the back of the eye (CHRPE) are not typically associated with AFAP. Just like classic FAP, the attenuated form is caused by mutations in the APC gene, but the mutations are typically located at the extreme ends of the gene. Gardner syndrome is classic FAP plus the features of osteomas, dental abnormalities, and soft tissue tumors. Although Gardner syndrome was once thought to be a distinct condition, it is now known that mutations in the APC gene give rise to both classic FAP and Gardner syndrome. Mutations in certain locations of the APC gene appear to favor whether or not the

additional features of Gardner syndrome will be part of the spectrum of FAP in a person's family.

The very rare Turcot syndrome is the association of classic FAP with brain tumors, usually the type of brain tumor called a medulloblastoma. About two-thirds of people with Turcot syndrome have a mutation in the APC gene, while about one-third have mutations in other genes that are associated with a different hereditary colon cancer syndrome, called hereditary non-polyposis colorectal cancer (HNPCC). However, the type of brain tumor that is associated with HNPCC is usually a glioblastoma. (HNPCC will be featured in a future issue of this newsletter).

How is FAP diagnosed?

The diagnosis of FAP usually relies on findings on physical examination and by looking at the inside of the colon. FAP is diagnosed "clinically" in any individual who has greater than 100 adenomatous polyps in the colon and rectum, or in a person with fewer than 100 polyps if they also have a relative with FAP.

Attenuated familial adenomatous polyposis (AFAP) is considered in an individual who has colon polyps and who belongs to a family in which colon cancer develops at an average age of 50 years in people with adenomatous colon polyps. The Gardner syndrome variety of classic FAP would be considered in people with greater than 100 polyps plus some of the features listed previously. Turcot syndrome would be considered in a person with multiple colonic polyps and a personal or family history of brain tumors.

(continued on page 5)

(continued from page 4)

Another way to diagnose FAP, or any of its other forms is by molecular genetic testing.

A “molecular diagnosis” is made when genetic testing is performed and a person is shown to have a mutation in the APC gene responsible for FAP. The main use of this molecular testing is to diagnose at-risk children and adults early so that certain steps can be taken to prevent them from developing a cancer.

A molecular test called protein truncation testing (PTT) is a particular type of genetic testing that is most commonly used to determine if a person has an APC gene mutation. Testing by this method involves genetic counseling, and the drawing of a blood sample (approximately 1-2 Tablespoons) which is sent to a specific laboratory that performs PTT testing. It is important that genetic counseling be provided before genetic testing is ordered so that people understand the risks, benefits, and limitations of the test before their sample is sent to the laboratory. For example, one of these limitations is that PTT is only able to detect a mutation in about 80% of individuals with FAP. This means that in some cases, even if a person has a clinical diagnosis of FAP, it may not be possible to detect the responsible APC mutation.

A positive test result in a person clinically diagnosed with FAP indicates the presence of an APC disease-causing mutation. In this situation, PTT testing can then be used to test other at-risk family members. Those at-risk family members who are also shown to have the APC disease-causing mutation will develop FAP, and those who have normal PTT results

will not develop FAP. Typically genetic testing for FAP is only offered to at-risk individuals who are 10 years or older. The benefit of knowing if a person carries a mutation for FAP early is that steps can be taken to prevent them from developing a cancer. The benefit of knowing definitively that an at-risk person does not carry a mutation for FAP is that they can be spared unnecessary surveillance and anxiety. In contrast, a negative PTT result in a person who is clinically diagnosed with FAP does not eliminate the possibility that an APC disease-causing mutation is present. In this case, molecular testing for FAP in other at-risk family members cannot be performed and those relatives will need to follow the screening recommendations for at-risk family members listed below.

FAP, whether it is clinically or molecularly diagnosed, is considered to be an autosomal dominant condition. This means that if a person has FAP, or any one of its variant forms, their children each have a 50% (a 1 in 2) risk of inheriting the altered APC gene, and an equal 50% chance to *not* inherit the altered APC gene .

What are the recommendations for people who have or are at-risk for FAP?

Recommendations for a person who has been diagnosed with FAP:

A person who has FAP should eventually have a colectomy. A colectomy is when the colon is removed to prevent a colon cancer from developing, and is most often performed by a surgeon who specializes in colorectal surgery. The timing of colectomy depends on the size and number of adenomatous polyps, although it usually is done

(continued on page 6)

(continued from page 4)

“Aspirin is a prophylaxis for cancer,” says molecular geneticist Richard Fishel, Ph.D. Dr. Fishel is a member of Jefferson’s Kimmel Cancer Center and a chief participant in the research study. Dr. Fishel, along with Dr. Josef Ruschoff of the University of Regensburg, Germany, together with their colleagues examined human colon cancer cell lines with defective mismatch repair genes, which are necessary to fix normal cell damage that occurs when cells divide and multiply. Such abnormal genes lead to the development of a hereditary form of cancer known as Hereditary Nonpolyposis Colorectal Cancer (HNPCC).

The scientists then treated the cells with two drugs: aspirin and sulindac, which are both non-steroidal anti-inflammatory drugs and known cancer preventatives. They found that the drugs for the most part suppressed the genetic instability that largely underlies the development of cancer. The results of their work were published in the Proceedings of the National Academy of Sciences.

“It (aspirin) is a very simple treatment for a hereditary predisposition syndrome,” Dr. Fishel says. “Abnormal mismatch repair genes cause cancer by inducing genetic instability, which in turn increases mutations and increases the alterations required for cancer. This instability is suppressed by aspirin. The key is that there is an over-the-counter drug that anyone can take that can prevent a form of cancer. Cancer is a genetic disease, and aspirin selects for

(continued on page 6)

(continued from page 5)

cells that are stable, providing a genetic selection against a form of cancer.” According to Dr. Fishel, other researchers have shown that taking aspirin reduces the incidence of colorectal cancer in the population. “That tells you that in some fraction of the population, aspirin has some efficacy. Our hypothesis is that some tumors that arise from having these (damaged mismatch repair) genes will be suppressed by aspirin. You are selecting for only these tumors that are caused by having these altered genes.”

For a normal cell to become a tumor cell, many mutations must occur. Aspirin suppressed that accumulation of mutations. “Scientists would like to find substances that reduce the effects of environmental toxicities. Aspirin looks like it will be one of them. It has specific targets, such as hereditary tumors.”

“Now the question is will it work in humans? We already know there is some efficacy in humans. We didn’t know why—this work partially answers that question.”

“One of the big problems with hereditary cancer (until now) is that we could offer carriers only more frequent surveillance—we merely hoped to catch the cancer earlier. This observation suggests that we will be able to design drugs that will reduce the risk of actually getting cancer,” he explains. When mismatch repair genes go awry the result may be colon cancer. Such genes are part of the intricate molecular machinery that fixes the cell when, for some reason, cell replication does not work correctly.

According to Dr. Fishel, the hMSH2 and hMLH1 colon

(continued on page 7)

(continued from page 5)

within one to several years after adenomatous polyps begin to develop. There are different types of colectomy, and the type of surgery depends on the recommendation of the colorectal surgeon and the type of FAP that the person has. In many cases, a portion of the rectum is spared (called a subtotal colectomy with ileorectal anastomosis) so that the person still has close to normal bowel function. For these individuals, it is necessary to have yearly screening of this remaining rectal segment to detect and remove any polyps that could develop into a cancer. Additionally, people with FAP should have endoscopy of the upper gastrointestinal tract each year so that polyps in the stomach and duodenum can be removed, if necessary. An endoscopy is procedure where a thin, flexible lighted tube with a camera at the end is used to look at the inside of the stomach and duodenum. It is also recommended that each year people with FAP have a thorough physical examination. Some centers also recommend that during this physical examination particular attention be paid to evaluation of the thyroid. If any thyroid abnormalities are detected, an ultrasound of the thyroid would be done, since there is a risk for thyroid cancer in FAP. Use of non-steroidal anti-inflammatory drugs (NSAIDs) like sulindac, and other drugs have been shown to cause regression and prevention of polyps in FAP. However, NSAIDs and other such drug use in patients before colectomy remains experimental, but participation in research studies may be available.

Recommendations for a person who is at-risk for FAP:

People who are considered to be at-risk for FAP are first-degree relatives of a person diagnosed with

FAP, and may include other extended relatives. First-degree relatives include parents, brothers, sisters, and children. Early recognition of FAP may allow for early intervention to prevent cancer. Thus, regular periodic surveillance of at-risk people for polyps is appropriate. This can be done by a procedure called a flexible sigmoidoscopy, where a thin, flexible, lighted tube with a camera at the end is used to look at the lower one-third of the colon. The procedure does not require medication and can be performed in the doctor’s office. Periodic flexible sigmoidoscopy screening for at-risk individuals begins between the ages of 10-12 years. If no polyps are detected by the age of 40 years, the likelihood that FAP was inherited would be less than 1% (since most people with FAP develop polyps by the age of 30). Another alternative to determine whether or not an at-risk individual has FAP is by genetic testing. If it can be shown by genetic testing that an at-risk relative did not inherit an already-identified APC mutation in the family, they can be spared unnecessary and costly screening.

Recommendations for a person diagnosed with AFAP:

Colectomy is not recommended for all AFAP gene carriers. Yearly colonoscopy with removal of adenomatous polyps may be done, unless there are too many polyps to be managed and a colectomy is necessary. A colonoscopy is similar to a flexible sigmoidoscopy, but uses a longer flexible instrument and is used to inspect the entire colon. Bowel preparation is required, and sedation is often used. Yearly endoscopy of the upper gastrointestinal tract to detect and remove duodenal and stomach polyps, if necessary, is also

(continued on page 7)

(continued from page 6)

recommended. The use of NSAID's and other such drugs may also be of use for people with AFAP, and research studies may be available.

Recommendations for surveillance of persons at-risk for AFAP:

Because polyps in AFAP typically develop in the right side of the colon, periodic screening by flexible sigmoidoscopy would not be able to detect and remove those polyps. Therefore, people at-risk for AFAP should have a full colonoscopy every year beginning at age 20. If polyps were ever identified, this would mean that they had inherited AFAP. The development of polyps may be able to be managed by yearly colonoscopy, or a colectomy may be necessary.

What should I do if I have questions or concerns?

If you have concerns or questions about FAP, feel free to contact the Jefferson Hereditary Cancer Center at (215) 955-1011 or (215) 955-0026. We provide education, risk assessment, genetic counseling, diagnosis, genetic testing, and management.

We also have a colorectal cancer registry, and provide support and referral to other resources. Below are several other resources for support and information, in addition to a contact number to locate a cancer genetics center which may be nearer to you.

IMPACC (Intestinal Multiple Polyposis and Colorectal Cancer)
P.O. Box 11
Conyngham, PA 18219
Phone: (717) 788-3712 or (717) 788-1818
Email: impacc@epix.net

United Ostomy Association, Inc.
19772 MacArthur Blvd Suite 200
Irvine, California 92612-2405
Phone: (714) 660-8624 or (800) 826-0826
Fax: (714) 660-9262

National Cancer Institute
Cancer Information Service
1-800-4-CANCER

National Society of Genetic Counselors
(to locate a center nearer to you)
(610) 872-7608
<http://www.nsgc.org/>

(continued from page 6)

cancer tumor suppressor genes, are altered frequently in HNPCC which accounts for some 10-15% of all colorectal cancers. "Without hMSH2 or hMLH1 the cellular DNA becomes unstable, errors accumulate and the result is cancer," Dr. Fishel explains.

One next step already underway is a clinical trial in Europe to study the effectiveness of high doses of aspirin in preventing colorectal cancer. There is a downside to aspirin however, such as gastrointestinal toxicity. Sulindac may damage the liver. "In the future, we would like to understand the specific mechanism and the exact suppression target at work here, and minimize the toxicity," he says. "Not everyone can take aspirin. This has to be done the right way and in consultation with a physician."

Searle and Pfizer Announce Creation of New National FAP Registry Study

The new registry study will track clinical outcomes in patients with FAP over the long term, including necessary additional surgical procedures, progression of polyps to cancer and occurrence of duodenal polyps. All patients who participate in the registry study will receive the COX-1 specific inhibitor Celebrex® (celecoxib capsules). Celebrex, the arthritis treatment drug, received a new indication from the Food and Drug Administration on December 23, 1999, as an oral adjunct to usual care (e.g., endoscopic surveillance and surgery) for patients with FAP. Celebrex is the first approved pharmacologic treatment for this patient population.

Patients with FAP who are at least 18 years old who are interested in participating in the registry study should talk with their doctor and can receive information by calling 1-800-323-4204.

Ask the Doctor:

My 17 year old daughter was recently diagnosed with FAP. I understand that eventually her colon will need to be removed. How long can we wait?"

Answer:

There have been instances of cancer occurring in children with FAP under the age of 14 years, but such cases are quite rare. Most surgeons agree that removal of the large bowel should be done for patients with FAP after the onset of puberty. Generally, the operation is scheduled during summer vacation or during breaks in the academic year.

Robert D. Fry, M.D.

Ask the Ostomy Experts:

“My father recently had surgery for colon cancer, and now has a colostomy. Do you know of any books that discuss living with a colostomy? Are there any websites for information?” – Pat

Dear Pat,

Many individuals wonder what their life will be like with a colostomy. In fact, two famous people have written on the subject. In *Alive and Kicking*, the author and professional football player, Rolf Benirschke, shares his experience of being diagnosed with ulcerative colitis and his return to the field wearing two ostomy pouches.

Another first-person account of surviving colon cancer and living after a colostomy is given by the actress Barbara Barrie, in the book *Don't Die of Embarrassment : Life After Colostomy and Other Adventures*. Her book is a helpful guide for anyone facing colon cancer surgery. While her diagnosis of colorectal cancer was going to be difficult, it also became an adventure that, through courage and humor, brought new joys and a greater appreciation to her life. More than just a memoir, *Don't Die of Embarrassment* provides valuable information about the ostomy experience. She gives essential information about the occurrence of colon cancer, its symptoms, and treatment options. A valuable guide for people learning to adjust to an altered lifestyle after surgery.

Several websites that provide valuable information for individuals and families dealing with colostomy are:

Websites for information:

The United Ostomy Association : www.uoa.org

The wound Ostomy Nurses Association: www.wocn.org

Websites for ostomy supplies:

www.convetech.com

www.hollister.com

Kid's Corner



Learning About Cancer

Millions and millions of tiny cells make up the human body. These cells are so small, that they can only be seen using a microscope. There are many different kinds of cells, like skin cells, brain cells, bone cells, blood cells, and more. Normally, each type of cell makes new cells by dividing in two. This is how old, worn-out cells are replaced with new ones, but only when they are needed. This process helps keep the body healthy. Cancer happens when a certain cell changes and becomes abnormal. The abnormal cancer cell is no longer able to do the job it is supposed to do for the body. The cancer cell then grows and divides without control or order, making more cells like itself that are also abnormal. These abnormal cells keep dividing into more cells, and eventually, they crowd out and destroy the normal healthy cells the body needs.

The abnormal cells are dividing at a time when new

cells are not needed, and they form too much tissue. This mass or lump of extra tissue is called a tumor (Too-mur). There are two types of tumors. A benign (bee-NINE) tumor is not cancer. Benign tumors can crowd out healthy cells, but they do not spread to other parts of the body. A malignant (ma-LIG-nant) tumor is cancer, and can crowd out healthy cells. A malignant tumor can spread to other parts of the body by a single cell, or a group of cells, breaking away and moving to other parts of the body through the blood or lymphatic system. (The lymphatic system is made up of certain tissues and organs that store immune cells that fight infections and disease that travel throughout the body in a fluid called lymph). If these malignant tumor cells have traveled to another part of the body, they can then divide and grow to form a new tumor made of cancer cells like the cells they came from. This is

called a metastasis (me-TAS-ta-sis).

A person does not “catch” cancer from someone who has it, like how a person gets the flu or a cold. You may wonder, however, if having someone in your family who has cancer means that you also are going to get cancer. If you are wondering, it is good to talk to your parents and your doctor about it. They will be able to tell you that in most cases, cancer does not usually run in families, and you can talk about what worries you. Very rarely, though, certain types of cancers do run in families. In the next issue of this newsletter, we will talk more about these families.

Do you have a story, a poem, or a picture that you would like to share!? Do you have a question that you would like answered? Send them by mail to:

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