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A PROGRAM OF SUPPORT
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PEOPLE WITH ORAL
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HEAD AND NECK CANCER

GENETIC BASIS FOR HEAD AND NECK CANCER DEVELOPMENT AND TREATMENT

BHUVANESH SINGH, M.D.

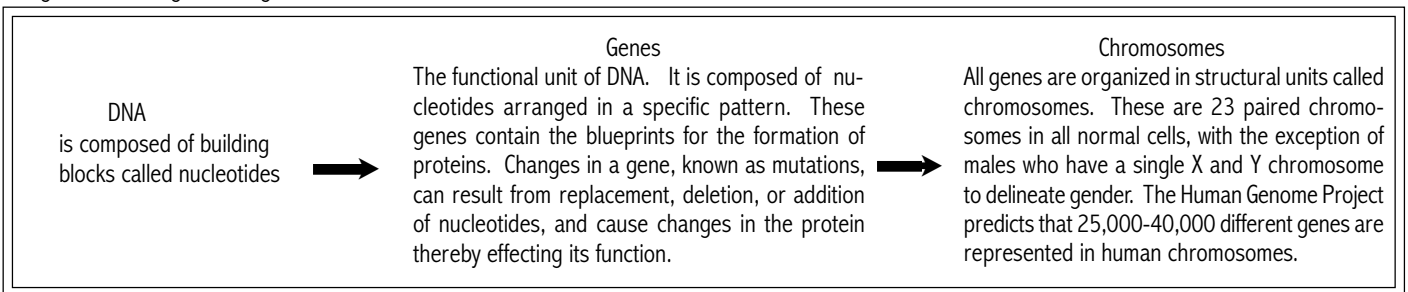
The blueprint for the development of all living organisms is contained within structures known as DNA (Figure 1). The DNA combine to make up functional units called genes, which dictate how individual cells appear or function. For example, our blood type, hair color and even gender is predetermined by the types of genes that we inherit from our parents. The genes that we contain are collectively known as a genotype and the appearance or function of the structures that result from the genotype is called the phenotype. Abnormalities in specific genes lead to several diseases. As examples, cystic fibrosis, some forms of diabetes, as well as many blood disorders result from an abnormality in a specific gene. Through the definition of the genotype, we can impact on an understanding of the biology of a disease process and its treatment.

Cancer, like other human diseases, results from abnormalities in DNA, which ultimately overcome normal growth regulation mechanisms. This unopposed growth is characteristic of cancer and is the

cause of local tumor development. The continued accumulation of DNA abnormalities in cancer cells allows them to invade into local structures, blood vessels and lymphatics, thereby promoting spread to other locations within the body. Clearly, if we can gain an understanding of the DNA abnormalities in a given cancer, we can predict its behavior and affect its treatment. Unfortunately, the current characterization of cancer genotypes remains elementary. Even with this limited knowledge, several new advances have significantly impacted on the management on many different cancers including leukemia and melanomas, as well as head and neck cancers. This review will try to provide a basic understanding of the genetics related to head and neck cancer development and how they may impact on current and future treatment.

A cell can acquire DNA abnormalities by several different mechanisms. It can inherit genetic abnormalities from its parents, it can be exposed to carcinogenic agents, or it simply can lack the ability to correct the abnormalities within the DNA structure that normally accumulate in all cells. In order for cancer to develop, specific abnormalities must occur in a given cell in a particular sequence and number. A cell typically requires years of accumulated damage before becoming malignant. It is therefore not surprising that the rate of cancer is highest in older patients whose cells have had life-long insults to their DNA. The role of inherited factors in the development of cancer is well documented and genes such as *APC* (a gene related to the development of colonic polyps) are known to impact on colon cancer development, and on the development of retinoblastomas (malignant tumors of the retina of the eye) and in mismatching repair genes in the development of several different forms of cancer. Abnormalities in individual genes alone are not sufficient to promulgate a cancer phenotype but rather they destabilize the genome and allow DNA abnormalities to accumulate, resulting in a more rapid rate for cancer development. The role of inherited factors can best be appreciated in the study of migrant populations. The rate of gastric cancer in Japan is extraordinarily high. If a Japanese individual migrates to the United States, his rates of can-

Figure 1. Basic genetic organization



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COMING IN MARCH, 2002

Cancer of the Thyroid

Sanziana Roman, M.D. and Robert Udelsman, M.D.

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cer remain quite high even though the environmental situation has changed.

With respect to head and neck cancer, inherited factors likely play a role, but the precise genes involved remain to be identified. Probably the most important factor in the development of head and neck cancer, as it is in the majority of human cancers, is exposure to environmental or carcinogenic agents. These agents can function in many different ways and ultimately result in DNA damage. Within head and neck cancer, the main carcinogenic agents are tobacco and alcohol. Tobacco contains several well-known carcinogens, whose exact mechanism of action remain to be defined. Similarly, alcohol is also potent as a carcinogen through several different metabolites. Interestingly, when tobacco and alcohol are combined, their affect is not additive but rather multiplicative, with the relative risk of developing a cancer being more than ten times higher when these agents are used in combination. Finally, our immune system plays a significant role in surveillance of our DNA. When an abnormality is recognized, it either kills the cell or initiates DNA repair mechanisms allowing it to stop the development of the cancer phenotype. Patients with abnormal immune systems, such as those with transplants who are immunosuppressed with medications, have fifty times higher rates for cancer development. Similarly, patients with HIV infection, with severe immunosuppression, have up to a five hundred times higher risk for the development of cancer. Although the understanding of each of the factors that result in DNA damage is quite difficult, it is even more complex to try to explain the interactions of all the processes that ultimately reflect a cancer phenotype.

Head and neck cancers progress from premalignant lesions, such as leukoplakia, to in situ carcinoma and finally invasive cancer. Genetic abnormalities underlie the tumor progression until the sufficient number and sequence of abnormalities are achieved. Mathematical models predict that 8-10 different genetic abnormalities are required for head and neck cancer to develop which is likely an under estimation of the actual number. Genes that contribute to the development of head and neck cancer can be characterized into three different types. Oncogenes are genes that give increased activity, growth, or survival ability to a given cell. Tumor suppressor genes, such as p53, block a cell that is abnormal from continuing to develop. Therefore, abnormalities in tumor suppressor genes themselves will allow an abnormal cell to continue to divide and multiply to form a cancer. Finally, the last category of genes, which can be broadly classified as surveillance genes, are those associated with the detection and repair of DNA abnormalities in a specific cell. These genes are also subject to abnormalities that can impair their functioning, leading to the increased accumulation of genetic damage and an accelerated rate of cancer development.

Many different tumor suppressor genes have been studied in head and neck cancer of which the most thoroughly investigated is p53. The p53 gene is found to be abnormal in almost all cancers that have been studied, and occurs in about 40-60% of head and neck cancers. This gene responds to damage in DNA by *either* stopping the cell's ability to divide and multiply or by causing cell death by several different mechanisms. Abnormalities in this gene are known to allow

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the development of cancers at an accelerated rate. Inherited abnormalities in p53 occur in patients with Li Fraumeni syndrome (Cancer syndrome resulting from inherited abnormalities in the p53 tumor suppressor gene). These patients are at risk not only of development of head and neck cancers, but also many other cancers throughout the body. The type of cancers that develop depend on the other DNA abnormalities that accompany the inherited abnormality in p53. This disorder is relatively rare, but studies on affected individuals have opened a window into the carcinogenic process and is allowing us to understand the intricate mechanisms associated with p53 function. Other tumor suppressor genes that have been investigated in head and neck cancer include Retinoblastoma gene and p16 gene, but the rates of abnormalities are not as prominent as that seen for p53.

The study of the oncogenes in head and neck cancer has lagged behind the studies of tumor-suppressor genes. The best characterized oncogene in head and neck cancer is cyclin D1, which is a gene that is involved in regulation of the cell cycle. It is the cell cycle that determines whether a cell will divide and multiply; therefore, an abnormality in the gene that regulates a cell cycle can lead to uncontrolled cell division and proliferation. Abnormalities in cyclin D1 occur in about 20-40% of patients and are associated with poor survival rates.

Finally, abnormalities in surveillance genes are the least well defined in head and neck cancer. Studies are looking at genes that affect repair of DNA in response to radiation or other forms of DNA damage, but their association with head and neck cancer remains limited. Based on a current study we have found an unusually high rate of head and neck cancer in patients with Fanconi's Anemia, (a disorder resulting from abnormalities in DNA repair genes) where there is an abnormality in DNA repair. We are currently investigating the role of Fanconi's Anemia genes in patients with head and neck cancer.

Several new therapeutic approaches have been derived from the increasing understanding of the genetic basis for head and neck cancer. The most prominent of these therapeutic approaches is the manipulation of the p53 gene to try and modulate the cancer phenotype. The

onyx 15 gene-therapeutic virus has received much attention in the media. This virus targets cells with p53 mutations and results in their destruction. The combination of onyx 15 and standard chemotherapy has shown very promising early results in human studies, highlighting an approach of therapeutic augmentation with the use of biologic agents.

The investigators at the M.D. Anderson Cancer Center have used an alternative approach to restoration of p53 function using an adenovirus as part of their gene therapeutic approach. Although the work has shown some promising results, there has been significant controversy regarding the use of an adenovirus due to a death directly attributed to the adenovirus therapy.

Biologic therapy has also targeted angiogenesis and growth factors such as the epidermal growth factor. C225 is an investigational agent that blocks the receptor responsible for transmitting the epidermal growth factor signal. Several investigators have obtained promising results in the treatment of head and neck cancer by blocking the epidermal growth factor receptor. The results are most promising when C225 is combined with radiation, but again the final data regarding the efficacy of this agent is not yet available. Within our own institution, we are quite excited about the application of an anti-angiogenesis agent in the treatment of patients with refractory cancers. In our very early studies, we have seen some promising results and are actively pursuing further investigations in this field. Although several other biologic therapies are on the horizon, none have achieved the early successes of the above-mentioned approaches.

The work that we are doing within our own laboratory stems from the realization that our understanding of the DNA abnormalities in head and neck cancer remains quite elementary. Accordingly, we are using several new techniques to screen the head and neck cancer genes to identify abnormalities that may be relevant to the carcinogenic process. These techniques include comparative genomic hybridization (CGH), spectral karyotyping (SKY), cDNA array, tissue array, and real time PCR. Using a combination of these studies, we have identified several candidates that may function as oncogenes or tumor suppressor

genes in head and neck cancer. Our attention has focused on an abnormality on chromosome 3, which we have found to be abnormal in over 50% of cases. What is more interesting, is that this abnormality is related to exposure to tobacco carcinogens and is present in all tobacco-related malignancies, including lung and esophageal cancers, in addition to head and neck cancer. We have found that when this abnormality is present, the patient's response to treatment is poor and survival is significantly lower. Through a series of analytic maneuvers, we have shown that two genes are involved in this abnormality. The first gene is named PIK3CA, and is an integral part in signaling from growth receptors, including those associated with growth and head neck cancer. We have shown that this oncogene interacts with p53 gene to define a balance between cell death and survival and determination of the cell's ultimate fate. We are targeting gene therapeutic strategy to try to manipulate this gene and allow its clinic utilization. The second gene that we have identified is a new gene, which we have tentatively named squamous carcinoma related oncogene (SCRO) We know that abnormalities in this gene allow cancer to develop in mice and we are actively investigating its function to try to determine its implications in head and neck, lung and esophageal cancers.

In addition, through cDNA array technology (a method for screening thousands of genes at one time) we have been able to screen 22,000 different genes and have identified several genes which may be relevant to head and neck cancer and are actively investigating each of these individually to determine their significance. Over the next 2-3 years we anticipate a development of an understanding of the DNA abnormalities within head and neck cancers and utilization of this information not only to predict cancer course, but also to impact on its treatment. We actively anticipate the completion of these projects and their impact on the management of our patients.

Editor's Note: Bhuvanesh Singh, M.D., is Director of the Laboratory of Epithelial Cancer Biology and Assistant Attending Surgeon at Memorial Sloan Kettering Cancer Center, NY. Dr. Singh is also Assistant Professor of Surgery at Weill Medical College of Cornell University, NY. For more information on this subject, please contact Dr. Singh's office at 212-639-2024.

A TIME FOR SHARING The Story of an Ongoing Search

In September of 1997, I was diagnosed with squamous cell carcinoma of the right tonsil; a time I shall not soon forget. Never in my life had I ever experienced anything like that verdict. You all know the feeling.

Radiation was the preferred treatment. I was very thankful there was a means of solving my problem and although informed by oncology medical staff of some of the potential detrimental side effects of radiation treatment, I had a rather nonchalant attitude about it all. In my mind, it would be about the same as getting a chest x-ray twice a day for a few weeks. As to any side effects, I recalled some Marine Corps experience where physical stamina was a necessity and I figured this would be a "walk in the park." No problem with this. Well, as you can guess, my arrogance was swiftly rewarded. After only a few doses of radiation, I became a very humble person, no taste, dry mouth, sore throat, discontinued eating solid food, and fatigue. It was a rude awakening that really got my attention.

Fortunately with the wonderful attention of the staff of Shands Cancer Clinic at the University of Florida, Gainesville, the radiation treatment was gratefully concluded. A week later, with an overnight in the hospital, a right neck dissection to remove one node was successfully completed, also. I returned home in high spirits figuring I was in the final turn and determined to make a speedy recover...get the staples out of the incision and be on my way. Things did go quite well for a while. I began to supplement the liquid diet of "Boost Plus" with blender treated cold cuts mixed with soup into a smooth mixture. I drank a lot of cool tea with a "meal" to get over the dry mouth. Finally the big day arrived and I ate some sliced steak. Things were really looking up.

However, the euphoria lasted only a few weeks before the onset of my current problems. I started to experience tightness

in the masseter and diaphragic muscles on the side of the face that work the jaw, coupled with intermittent muscle spasms that locked the jaw closed. Also any effort to exert a force on the jaw as in chewing, triggered locking spasms. No solid food was possible. Jaw opening was reduced down to a five mm. opening. It was time to get a move on and do something. And so started the search for relief.

First stop was a full check up for any TMJ jaw dysfunction. The exam revealed no anomalies in the structure of the jaw. It was plain to see the muscles were out of

....I may have reached the end of rehabilitation in the opinion of some; however, my intention is to keep the search active, as I do strongly believe that eventually something will turn up out there. Just call me an eternal optimist.

order for some reason. I consulted with the ENT doctor who had done the neck surgery. The conclusion indicated some evidence of fibrosis resulting from radiation collateral damage which in turn might be causing the trismus and jaw locking. Several medications were tried to relax the muscles, but were not satisfactory. It was decided to get into some Physical Therapy without delay. I began a physical therapy program; twice weekly sessions of ultra sonic message, heat application, and physical manipulation of the jaw to increase the opening. The hands of the therapist got a real work-out and so did my jaw, The opening increased to 18 mm. This went on for over a year until a limiting plateau was

reached of opening of the jaw to a maximum of 18mm. Physical therapy could not get beyond this point. Face muscles still experienced inadvertent spasms accompanied by the jaw locking. It was still impossible to chew. At that point PT was discontinued.

I attempted to use a "Therabite" machine to exercise the jaw. The spasms and locking jaw made it impossible to go forward. Next stop was a consultation with a neurologist. I was advised there was nothing available in the field of neurology that would be helpful to me. The options for treatment seemed to rapidly diminish. I read about BOTOX on the Internet. It appeared it might be helpful to relieve the muscle spasms. I consulted with my doctor and he agreed to go ahead with BOTOX injections into the jaw muscles. Preliminary to injections, an EMO test exam was conducted by a specialist to determine face muscle condition. Results indicated damage to the masseter and diaphragic muscles. For several months I received EMG (Electro Myography Guided) BOTOX injections specifically directed into the areas of concern. The BOTOX was not successful: no tangible improvement in the condition.

What to do next? Maybe Alternative Medicine could be the answer? The search directed me to acupuncture as another option. I located an MD specializing in acupuncture and rehabilitative medicine who also had extensive experience in throat surgery. It was a good combination to follow.

The treatment selected was electrical stimulated acupuncture; one session every four days. A pulsating low power electrical current is directed through various acupuncture needles inserted into the muscles which causes them to react to restore circulation. It is a painless procedure. Acupuncture was helpful to the extent that the inadvertent occurrence of spasms decreased, but not the intensity of clamping. Any attempt to chew, triggered a locking spasm. The maximum

jaw opening remained at 18mm. Recently I resumed a very gentle exercise regimen with "Therabite" to expand the jaw opening. I need to be careful not to trigger spasms and jaw locking. It is too early to evaluate any results. I plan to continue with the acupuncture as right now it seems to be the only option available.

Overall my condition is not deteriorating and that is considered progress. Life style is somewhat limited due to muscle spasms and jaw locking. My diet consists of 6 cans of "Boost Plus" each day supplemented with foods put through the blender including ice cream and chocolate cookies...plenty of calories. I have no problem retaining my weight. I am able to drink

Coke, Gatorade, and cool clear tea. These liquids along with "Salivart" spray (artificial saliva), help with the dry mouth problems. However, water seems to be a problem for me.

Conscientious daily attention to dental care has been helped with "Biotene" dry mouth tooth paste and "Biotine" mouth wash, plus brushing with "Prevident" 1% Sodium Fluoride tooth paste which is very effective. I continue to swim, ride my bike, and lift weights daily to maintain fitness. I have been cancer free for almost four years and have so many reasons to be very thankful. I also owe so very much to my patient wife, who has been a gentle pillar of support, keeping me going in the right direc-

tion when frustration sets in.

Searching for a way to stop the jaw spasms and improve the jaw function to chew solid food still is my top priority. I plan to continue to investigate every source of information that comes to my attention. In this regard SPOHNC has been extremely helpful. I may have reached the end of rehabilitation in the opinion of some; however, my intention is to keep the search active, as I do strongly believe that eventually something will turn up out there. Just call me an eternal optimist.ⁿ

H. J. Hart
Hobe Sound, FL



New Members to SPOHNC's Medical Advisory Board

Support for People with Oral and Head and Neck Cancer is pleased to welcome Drs. David W. Eisele and Jesus E. Medina to its Medical Advisory Board.

David W. Eisele, M.D., is Professor and Chairman of the Department of Otolaryngology-Head and Neck Surgery at the University of California, San Francisco. Prior to accepting this position in July of 2001, Dr. Eisele served as director of the Division of Head and Neck Surgery Department of Otolaryngology-Head and Neck Surgery at the Johns Hopkins Hospital in Baltimore, Maryland. He was also the Director of the Johns Hopkins Head and Neck Cancer Center and Associate Professor of Oncology at Johns Hopkins University School of Medicine.

Dr. Eisele received his Medical degree from Cornell University Medical College in New York and completed his Post-Doctoral training at the University of Washington in

Seattle, Washington. He is the recipient of many honors and awards including "America's Top Doctors" in 2001.

Dr. Eisele is presently an Associate Editor of "Laryngoscope", the Journal of the American Laryngological Rhinological and Otolological Society. His scientific publications include numerous articles and book chapters and he is co-editor of several books. In addition, Dr. Eisele has lectured extensively on topics related to head and neck cancer.

Jesus E. Medina, M.D., F.A.C.S. was born in Arequipa, Peru, where he attended medical school at the National University of San Agustin. He came to the United States in 1974 and trained in otorhinolaryngology at Wayne State University School of Medicine. He then did a one-year fellowship in Head & Neck Surgery at the M.D. Anderson Hospital and Tumor Institute in Hous-

ton, Texas. Following completion of his training in 1981, he became an Assistant Professor in the Department of Head and Neck Surgery at M.D. Anderson. In 1984 he accepted a position as an Associate Professor and Director of the Head and Neck Cancer Program in the Department of Otorhinolaryngology at the University of Oklahoma. He became Chairman of the Department in 1991, and he currently holds the Paul & Ruth Jonas Chair in Cancer Treatment and Research.

Dr. Medina has devoted his career to the care of patients with head and neck cancer, and has authored numerous scientific publications and book chapters on a variety of topics in head and neck oncology. He serves on the editorial board of "Head and Neck Surgery", "Otolaryngology-Head and Neck Surgery" and other scientific publications.ⁿ

Acupuncture to Treat Xerostomia (Dry Mouth) in Head and Neck Cancer Patients Following Radiation Therapy

Richard C. Niemtow, MD, PHD., MPH

Xerostomia or dry mouth is experienced by approximately 70% of patients after receiving radiotherapy for treatment of oral and head and neck cancer. In the majority of cases, existing saliva may no longer be useful. Eating may become a chore as it is often difficult to swallow and taste. Patients may not be able to chew gum as it falls apart in the mouth. Candy may not dissolve. Talking may require the use of frequent sips of water. This lack of saliva also leads to an increase in tooth decay and gum disease. If saliva is present, it may be very sticky and thick. This is a very uncomfortable situation for the patient. There are several drugs on the market designed to stimulate and maintain the flow of saliva. However, at the present time, the success rate using these drugs varies and some side effects of the drugs may be bothersome.

What happens to the salivary glands during radiation? First, let us briefly review the anatomy and the physiology of the salivary glands. The salivary glands secrete saliva, a somewhat alkaline fluid that moistens the mouth, softens food, aids in digestion and is extremely important for maintaining and preserving good oral health. The submaxillary glands are located around the mouth under the lower jaw, the sublingual glands are located beneath the tongue, and the parotid glands are found in front of each ear. The buccal glands, in the cheeks near the front of the mouth, also secrete saliva. The saliva of the parotid gland contains enzymes called amylases, one of which, known as ptyalin, aids in the digestion of carbohydrates.

During treatment for head and neck cancer, the salivary glands are frequently included in the fields of radiation. Unfortunately, these glands may become severely damaged or destroyed during the radiation treatment. Consequently, the patient may experience different degrees of dry mouth for his/her lifetime. At best, the remaining saliva function may be scant and is often thick andropy.

The author of this article has developed an acupuncture technique to help overcome dry mouth in patients who have been irradiated for oral and head and neck cancer. This

treatment was developed in November 1999 at the Navy Medical Center at San Diego, California. More than 70 patients have been treated since 1999 to the present time with various degrees of saliva restoration without complications. As in all medical treatments there is a small number of failures. For the most part, those patients treated with acupuncture have found that it is superior to costly drug therapy. Other physicians have also reported success following this specific protocol. For the patient, this means that the discomforts of a dry mouth may no longer be an acceptable complication. Although the resulting production and quality of saliva may not be completely normal, it has permitted many patients to enjoy a better quality of life: Gum can be chewed, candy dissolves, food can be swallowed with less difficulty and talking is not constantly interrupted by frequent sips of water.

The treatment consists of three small needles placed in each ear and one needle placed near the tip of both index fingers. The patient produces saliva in approximately 20-30 minutes. The quantity of saliva is impressive. Several follow up treatments are required and in the majority of cases the saliva flow may be permanently reestablished. Follow up treatments are tailored to the needs of the patients. It is not unusual for patients to go for periods extending over 30 days without needing additional acupuncture.

Acupuncture using the above protocol may contribute to temporary relief of dry mouth for some patients with xerostomia after radiotherapy. Longer follow-up, optimization of technique and further prospective objective measurement of saliva response continue in our clinic. Further research is imperative to optimize acupuncture techniques for head and neck cancer patients.

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Editor's Note: Richard Niemtow, MD, PHD., MPH is a Colonel in the United States Air Force (USAF), on loan to the U.S. Navy for a special acupuncture project. Dr Niemtow is the first physician practicing acupuncture full-time at the Naval Medical Center in San Diego, California, with a special interest in oncology.

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from PAT'S PANTRY
PROVENÇAL



Gratin Daupinois (French scalloped potatoes)

- | | |
|---|--|
| 4 or 5 potatoes, peeled and sliced thinly | 2 egg yolks |
| 3 tbsp olive oil | 1/3 cup heavy cream or sour cream |
| Milk | 1/2 cup grated Swiss or Gruyere cheese |
| | Salt to taste |

Preheat oven to 375 degrees. Put thinly sliced potatoes in a pyrex baking dish which has been greased with the olive oil. Sprinkle with salt. Add milk to cover. Bake until potatoes are almost done, about 30 to 40 minutes. Beat egg yolks and add cream. Pour into the potato mixture, stirring rapidly. Top with the grated cheese and bake about 10 -15 minutes longer, until the cheese melts and the mixture is bubbling. Put into blender and blend, adding milk as needed.

February Tip: You can add onions if you like. Use this as a side dish with one of your other prepared vegetables or protein dishes. Always be aware of varying your nutrients every day—the more the better!

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 SPOHNC-BOCA RATON, FL
 SPOHNC-BOSTON, MA
 SPOHNC-BRIDGewater, NJ
 SPOHNC-DALLAS, TX
 SPOHNC-FAIRFAX, VA-Heads Up
 SPOHNC-HOUSTON, TX
 SPOHNC-LONG ISLAND, NY
 SPOHNC-LONG ISLAND, NY-East (now forming)
 SPOHNC-MANHATTAN, NY (now forming)
 SPOHNC-MAYWOOD, IL-Loyola (now forming)
 SPOHNC-MIAMI, FL
 SPOHNC-MIAMI, FL-Mort Silverblatt Head and Neck
 SPOHNC-MORRISTOWN, NJ
 SPOHNC-NJ-PA
 SPOHNC-OMAHA, NE-UNMC
 SPOHNC-PITTSBURGH, PA
 SPOHNC-SAN DIEGO, CA
 SPOHNC-WASHINGTON, DC-LCC
 Froedert Cancer Center (Milwaukee, WI)
 Good Samaritan Hospital (Portland, OR)
 Greater Baltimore Medical Center (Baltimore, MD)
 James Cancer Hospital (Columbus, OH)
 L.I. College Hospital (Brooklyn, NY)
 Memorial Sloan-Kettering Cancer Center (New York, NY)
 Temple Cancer Center (Philadelphia, PA)
 University of Chicago Hospital (Chicago, IL)
 University of Michigan Hospitals (Ann Arbor, MI)

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