The Promise of New Therapeutics in Sjögren’s Syndrome

by Elaine Alexander, MD, PhD
Chair, SSF Medical and Scientific Advisory Board, and Chair, SSF Clinical Trials Consortium

Recent clinical trial developments in the treatment of systemic manifestations of autoimmune diseases, such as systemic lupus erythematosus (SLE) and Sjögren’s syndrome (SS), both currently inadequately treated, have focused attention on promising novel B cell-related therapies. No effective therapy has been approved by the FDA for the treatment of SLE in more than fifty years. Recently, however, several late stage clinical trials in SLE have demonstrated considerable clinical promise. At the American College of Rheumatology (ACR) Annual Meeting in Philadelphia, Pennsylvania, on October 19, 2009, the Sjögren’s Syndrome Foundation (SSF) hosted a meeting to present and discuss the latest clinical trial developments in SLE and their ramifications for the potential therapy of systemic manifestations of SS. The meeting was attended by industry colleagues, international experts in the field of SS research and clinical medicine and physicians with an interest in SS.

The SSF meeting highlighted new data on two potential B cell-targeted biologics in SLE: Benlysta™ (belimumab, formerly LymphoStatB®) and epratuzumab. Belimumab is a fully humanized monoclonal anti-BAFF/BLYSS (B cell-activation factor of the TNF family/B-lymphocyte stimulator) antibody. During the meeting...

More than one dry eye

The entity Henrik Sjögren originally described is now defined as secondary Sjögren’s syndrome (SS), i.e., a dacrooadenitis (lacrimal gland inflammation) associated with signs and symptoms of keratoconjunctivitis sicca (KCS) that arises in patients with rheumatoid arthritis. Primary Sjögren’s syndrome (pSS) is defined as a similar immunopathology associated with KCS but independent of another underlying autoimmune disease. While mechanisms of pathogenesis may differ between primary and secondary SS dacrooadenitides, their common immunopathological feature is the accumulation of more- or less-organized lymphoid tissues that produce IgG directed against host tissue autoantigens.

Both Sjögren’s dacrooadenitides differ from other immunopathologies that also are associated with KCS. Of these, Wegener’s granulomatosis, sarcoidosis, graft-versus-host disease, diffuse infiltrative lymphocytosis associated with HIV infection, and human T lymphotropic virus infection are well-defined. However, many more cases of clinical dry eye disease are associated with processes that are so poorly understood that they typically are categorized as “non-Sjögren’s dry eye” and attributed to “primary lacrimal deficiency” or “chronic age-related dacrooadenitis.” The current concept of...
conducted in Europe with promising trial in SS with epratuzumab has been B cell proliferation. A small open-label receptor signaling which also may inhibit molecule involved in B cell antigen re-targets CD22, a B cell-specific surface Lymphoma (NHL). Epratuzumab is indications to UCB, epratuzumab was medicated and licensed for all autoimmune etiologists and Salvatore deVita, respectively.

During the ACR meeting, UCB reported Phase 2b trials results in SLE for epratuzumab. Developed by Immunomedics and licensed for all autoimmune indications to UCB, epratuzumab was initially used in non-Hodgkin’s B cell Lymphoma (NHL). Epratuzumab is a humanized monoclonal antibody that targets CD22, a B cell-specific surface molecule involved in B cell antigen receptor signaling which also may inhibit B cell proliferation. A small open-label trial in SS with epratuzumab has been conducted in Europe with promising preliminary results.

The successful clinical development by Genentech and FDA approval of rituximab, a chimeric anti-CD20 monoclonal antibody, in rheumatoid arthritis (RA) opened up the potential for B cell-directed therapy of rheumatic/autoimmune diseases. Rituximab is the only B cell-directed therapy approved to date for rheumatic/autoimmune diseases. Rituximab initially was developed to deplete malignant B cells in non-Hodgkin’s B cell lymphoma by virtue of CD20 expression on mature B cells but not B cell precursors or plasma cells. Although multiple case reports and open-label studies suggested a benefit of rituximab in SLE, the drug did not demonstrate clinical efficacy in the randomized Phase 2/3 EXPLORER trial. A Phase 3 study of rituximab in SLE nephritis did not meet primary endpoints. Based on clinical observations of improvement of signs and symptoms of SS in patients receiving rituximab B cell lymphoma therapy, a number of open-label trials have been initiated.

**Common Themes Unite SLE Clinical Trials and Longstanding SSF Goals**

HGS’s Benlysta™ Phase 3 clinical trial results support an FDA filing for registration and approval. If this biologic is approved for SLE, an extension to a second closely-related clinical indication (i.e. SS) would be logical and indicated. Although the fundamental etiopathogenesis of the diverse and numerous clinical manifestations and end organ involvement in SLE and SS in large part remains enigmatic, these disorders share a substantial number of immunologic similarities implicating B cell hyperactivity in pathogenesis, thus warranting further clinical development of B cell-targeted therapy in SS.

The SLE clinical development path elucidated over the last 10 years by industry leaders Genentech, UCB and HGS, and the experience and collective wisdom attained in the development of diagnostic criteria, disease activity indices, disease damage scores, outcome measures and clinical trial endpoints acceptable to the FDA for potential regulatory approval, all will enhance and expedite the clinical development path for SS. Clearly, meticulous clinical trial design and execution, with vigilant monitoring of safety and tolerability, can result in outcomes that support potential FDA registration. This is a major advance in the successful development of therapy for autoimmune diseases. SS is now poised for clinical trial development with one of more B cell-targeted therapies.

**Benlysta™ Clinical Trials in SLE: Primary Endpoint and Clinical Trial Design**

At the October SSF meeting, William Freimuth, MD, PhD, Vice President, Clinical Research, Immunology, Rheumatology and Infectious Diseases, Human Genome Sciences, described the development of a novel multi-dimensional evidence-based SLE Responder Index (SRI) at week 52 as the primary endpoint for use in the pivotal Phase 3 Benlysta™ clinical trials. While the HGS Phase 2 SLE study did not meet the co-primary endpoints, post hoc analysis demonstrated that a subset of serologically active SLE patients received clinical benefit. The subsequent pivotal Phase 3 trials employed restricted inclusion/exclusion criteria to enroll subjects most likely to benefit from Benlysta™ therapy and used the SLE responder index (SRI) as the primary clinical end point.

In the first Phase 3 clinical trial, the sample size was increased from 449 (Phase 2) to 865 patients and included 90 study centers in 13 countries, and patients continued with Standard of Care while on the randomized, double-blind placebo-controlled studies. The composite SRI included: 1) the Safety of Estrogens in Lupus Erythematosus National Assessment (SELENA) and SLE Disease Activity Index (SLEDAI); 2) the British Isles Lupus Assessment Group (BILAG); and 3) the Physicians’ Global Assessment (PGA). The study obtained a significant reduction in the primary endpoint, the SRI (SLE disease activity), flare rates and use of prednisone. In addition, Benlysta™ was safe and generally well-tolerated.

The results of the second Phase 3 trial, announced in November 2009, demonstrated similar clinical efficacy. Belimumab at 10 mg/kg plus Standard of Care demonstrated significant differences compared to Standard of Care alone in SRI response at 52 weeks. HGS also will analyze 76 week follow-up data.
Patients with serologically active disease who were included in over four years of follow-up study sustained improvement in disease activity and a decline in flares. FDA NDA regulatory filing is projected for early 2010.

**Epratuzumab Clinical Trials in SLE**

Top-line results reported in the fall of 2009 by UCB for Phase 2b trials of epratuzumab in SLE demonstrated a reduction in disease activity and steroid dosage observed at 12 weeks. Two hundred twenty-seven patients with moderate to severe SLE demonstrated a 24.9% advantage for those treated with epratuzumab compared to those receiving placebo. The frequency of adverse effects was similar for both groups. Endpoints included several SLE disease activity indices with a primary focus on BILAG. UCB now is proceeding with Phase 3 clinical trials.

**Outcome Measures in SS: Update**

The EULAR Steering Committee has made significant progress over the past several years in developing outcome measures in SS. Under the leadership of Claudio Vitali, MD, the initiative now involves approximately 40 international experts.

During the SSF event at the ACR meeting, Dr. Vitali and Steering Committee member Raphaèle Seror, MD, reported on two indices that deal with disease activity and damage, respectively. The EULAR Sjögren’s Syndrome Patient-Reported Index (ESSPRI) measures main symptomatic features such as dryness, fatigue and pain. The EULAR Sjögren’s Syndrome Activity Index (ESSDAI) covers systemic features such as vasculitis and synovitis and neurological, pulmonary, renal and hematological involvement and is based on physician evaluation. Having completed the definition and initial validation of ESSDAI and expecting similar completion of ESSPRI in Q4 2009, final validation of both indices is scheduled to start in Q1-2, 2010.

A website to facilitate communication and exchange on the SS activity index has been launched at www.eularsjogrenactivityindex.com.

**Conclusion**

The successful treatment of rheumatic/autoimmune disorders has entered into a new era with recent innovative clinical development efforts in SLE by several biotechnology/pharmaceutical companies. For the first time in more than 50 years, the possibility of the regulatory approval of one or more biologics for the treatment of SLE appears to be on the near horizon. These recent developments have potential positive implications for the development of more effective and safe therapies for SS. The SSF and the Clinical Trials Consortium is working closely with industry on further clinical trial development of B cell-related (and other) therapies for SS. In addition to the success of Benlysta™ and epratuzumab, other B cell biologics are in the pipeline for SLE and hold promise for SS. The SSF also will continue to support international development of a disease activity index, disease damage score, clinical outcome measures and clinical trial end points.

The SSF gratefully acknowledges HGS sponsorship of and participation in the October 19, 2009 SSF meeting at the ACR.
might be to identify molecular mechanisms that underlie the model-specific features and to determine how well each corresponds to the human. The process will be highly recursive, but it will lead to paradigms that describe the human. A recent publication by de Saint Jean et al1 illustrates the first two steps.

Features of lacrimal physiology that may be salient in SS pathogenesis reflect the role the lacrimal gland plays in the mucosal immune system. Thus, the lacrimal gland expresses homing receptor ligands and chemokines that recruit dimeric IgA (dIgA)-expressing plasmablasts; it provides signals that induce plasmablast differentiation and plasmacytes survival; and it expresses an epithelial cytophysiological apparatus that delivers secretory IgA (sIgA) to the fluid forming within the lumen of the acinus-duct system. Two model-specific features of this generic paradigm already are well-documented: (a) The organized mucosal immune inductive sites in the conjunctiva (CALT) are absent or poorly developed in rodents but well-developed in rabbits and humans; (b) The interacinar stromal spaces that provide the niche for plasmacyte survival are tightly constrained in the rodents but expansive in rabbits and humans.

A generic cytophysiological mechanism exposing autoantigens

The consistent finding that immunization with autoantigens and adoptive or autoadptive transfer of activated effector lymphocytes induce autoimmune dacrocyoadenitides in mouse, rat and rabbit models indicate that lymphocytes entering the lacrimal gland’s stromal spaces must encounter potentially pathogenic autoantigen epitopes presented by professional antigen-presenting cells. The cytophysiological apparatus that takes dIgA up from the stromal space and delivers sIgA to the nascent ocular surface fluid has been studied in rabbit models. As the authors have described elsewhere,10 an emerging paradigm posits that this transcytotic secretory apparatus also delivers a constant flow of potential autoantigens from the epithelial cell cytoplasm to the underlying stromal spaces.

Does a generic immunoregulatory regime enforce tolerance?

If this paradigm explains how and why potentially pathogenic autoantigen epitopes come to be presented by antigen-presenting cells within the gland’s stromal spaces, the lacrimal gland’s role in the mucosal immune system also suggests an explanation for why potentially pathogenic epitopes should normally remain benign. dIgA-committed cells enter the mucosal immune effector tissues as immature cells (plasmablasts), and they must undergo a terminal differentiation process to mature as plasmacytes that secrete dIgA autonomously. One of the key chemical signals inducing plasmablast maturation is TGF-β. Notably, in the effector tissues that have been studied, as in tissues outside the mucosal immune system, TGF-β also directs antigen-presenting cells and T cells to up-regulate expression of TGF-β and, therefore, to function as immunoregulatory cells. Thus, just as TGF-β mediates oral tolerance in the gastrointestinal system, it might mediate self-tolerance in the lacrimal gland.

It has been known for some time that TGF-β is expressed in lacrimal glands of mice, rats, rabbits and humans. Evidence that it performs the predicted immunoregulatory function emerged when de Saint Jean et al unexpectedly encountered a striking difference between the ways epithelial cells from rabbit and rat lacrimal glands behave in primary culture. Cells from both models express MHC class II molecules. Acinar cells from rabbits gain the ability to function as surrogate antigen-presenting cells, stimulating pathogenic effector T cells from autologous peripheral blood to proliferate in ex vivo mixed cell reactions. When de Saint Jean et al tested rat lacrimal epithelial cells’ ability to act as surrogate antigen-presenting cells, they observed that, rather than stimulating, they suppressed proliferation of lymph node and spleen lymphocytes.

de Saint Jean et al then generated dendritic cells from rat bone marrow monocytes and stimulated them with TNF-α or LPS for 2 days. As expected, the activated dendritic cells stimulated lymphocytes to proliferate. However, when microporous inserts containing rat lacrimal epithelial cells were placed above the dendritic cell - lymphocyte mixed cell reaction, proliferation was suppressed to below baseline levels. The investigators went on to show that rat lacrimal epithelial cells release soluble mediators that prevent monocytes from up-regulating surface CD86 and MHC Class II expression and that induce mature dendritic cells to down-regulate surface CD86 expression. They also found that dendritic cells that had been exposed to the epithelial cell mediators suppressed, rather than stimulated, lymphocyte proliferation. They concluded that epithelial cells of the rat lacrimal gland secrete paracrine mediators which induce immature dendritic cells to mature as immunoregulatory antigen-presenting cells.

Additional experiments indicated that the critical paracrine mediators likely included TGF-β and IL-10. Notably, transcripts for both mediators were 100-fold more abundant in epithelial cells isolated from rats than in cells from rabbits. However, this disparity may reflect differences of lacrimal gland functional histoarchitecture rather than fundamentally different immunoregulatory principles. TGF-β and IL-10 immunoposivities are largely restricted to interlobular ducts in the rabbit.9, 11 In contrast, TGF-β immunopositivity is present in both acinar and ductal epithelial cells in the rat.12 Thus, TGF-β and IL-10 may mediate immunoregulatory regimes that enforce self-tolerance in the lacrimal glands of both rats and rabbits, but those regimes appear to be organized quite differently in the two models.

Immunophysiological role for a local inflammatory mediator

While epithelial cells in the lacrimal glands express effective immunoregulatory mediators, they also express at least one proliferation and survival factor, prolactin. Presumably, optimal prolactin levels help support plasmacyte, epithelial cell and T cell survival by abrogating TGF-β’s tendency to induce apoptosis.10 However, a preliminary report suggests that excessive prolactin levels abrogate TGF-β’s immunoregulatory functions and promote TH1 effector function, inducing a
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*Some patients may require twice-daily use for optimal results.
†Multicenter, 2-visit, 4-week, single-arm study conducted in moderate to severe Dry Eye patients who had previously been using ATs (N=520). Results are based on 418 patients who completed the study.
profuse, Sjögren’s-like lymphocytic infiltration.13 Interestingly, current evidence suggests that the histoarchitectural organization of prolactin expression parallels the organization of TGF-β expression. In rabbits, prolactin immunopositivity is concentrated in ductal epithelial cells.11 In rats, prolactin is present at appreciable levels in both acinar and ductal epithelial cells.14

**How good are the clues to pathogenesis as it occurs in natural settings?**

The rabbit model may provide clues to pathogenesis of the dacyrooadenitides. At least in rabbits, systemic levels of prolactin and of the reproductive steroid hormones influence the levels at which ductal epithelial cells express prolactin and TGF-β, and they also influence the extent to which these mediators are secreted to the stromal space versus the nascent ocular surface fluid.13 Thus, physiological and environmental factors that influence reproductive steroid and prolactin levels in different ways might be expected to disrupt the counterpoise between regulatory and pro-inflammatory signals to initiate different pathophysiological spirals. However, it is quite possible that the responses to physiological and environmental influences might differ importantly between the different models. Thus, while it seems reasonable to hope that studies addressing these mechanisms will advance understanding of pathogenesis and pathophysiology in directions that lead to better diagnosis and therapy, we should first consider which model is likely to be the most informative.

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**References:**


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**Contact Information:**

Aton Pharma
Lawrenceville, NJ 08648, USA

Rx Only

**LACRISERT®** (hydroxypropyl cellulose) **OPHTHALMIC INSERT**

**DESCRIPTION**

LACRISERT® Ophthalmic Insert is a sterile, translucent, rod-shaped, water soluble, ophthalmic insert made of hydroxypropyl cellulose, for administration into the inferior cul-de-sac of the eye.

Each LACRISERT is 5 mg of hydroxypropyl cellulose. LACRISERT contains no preservatives or other ingredients. It is about 1.27 mm in diameter by about 3.5 mm long. LACRISERT is supplied in packages of 60 units, together with illustrated instructions and a special applicator for removing LACRISERT from the unit dose blister and inserting it into the eye.

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LACRISERT is indicated in patients with moderate to severe dry eye syndromes, including keratoconjunctivitis sicca. LACRISERT is indicated especially in patients who remain symptomatic after an adequate trial of therapy with artificial tear solutions. LACRISERT is also indicated for patients with exposure keratitis, decreased corneal sensitivity, and recurrent corneal erosions.

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**WARNINGS**

Instructions for inserting and removing LACRISERT should be carefully followed.

**PRECAUTIONS**

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If improperly placed, LACRISERT may result in corneal abrasion.

Information for Patients

Patients should be advised to follow the instructions for using LACRISERT which accompany the package. Because this product may produce transient blurring of vision, patients should be instructed to exercise caution when operating hazardous machinery or driving a motor vehicle.

Concomiscurrence, Mutagenesis, Impairment of Fertility

Feeding of hydroxypropyl cellulose to rats at levels up to 5% of their diet produced no gross or histopathologic changes or other deleterious effects.

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No overall differences in safety or effectiveness have been observed between elderly and younger patients.

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Clinical experience with LACRISERT indicates that in some patients several weeks may be required before satisfactory improvement of symptoms is achieved.

**Issued June 2007**

**References:**

1. Koffler B; for the LAC-07-01 Study Group, Lacrisor (hydroxypropyl cellulose ophthalmic inserts) significantly improves symptoms of dry eye syndrome (DES) and patient quality of life. Ophthal Inquir 2007;41:258-263.


Dr. Snyder has been active in the field of physician health since 1990 and has delivered scholarly presentations at international physician health conferences in the United States, Canada, United Kingdom and Australia. Most recently, she presented at two workshops at the international physician health conference in Adelaide, Australia, and one for primary care physicians in Bendigo, Australia.

Patients with a life-altering chronic disease such as Sjögren’s present a special challenge to their treating rheumatologist or other physician leading their treatment team. In order to understand this more fully, we will examine a case study and then explore the interaction from the individual perspective of first the doctor and then the patient. Finally, we will discuss ways to make certain that you are building the best relationship with your patient to ensure open communication and a healthy partnership.

Case Study
Ms. W was a 40-year-old high-powered professional and a single mother of two. She had been active, enjoying weightlifting, running, skiing, dancing and bike riding prior to becoming ill. After two years of a gradual loss of muscle strength, development of clumsiness, loss of ability to work, crippling fatigue and work-ups by eight different neurologists, she was sent to Dr. X, a rheumatologist. He quickly made the diagnosis of CNS Sjögren’s and initiated treatment.

Over the last five years, Dr. X has had Ms. W on a variety of medications, many of which helped temporarily but lost efficacy. She was able to return to full-time work for four years. Ms. W was placed on Rituxan which worked well for one year. She then rapidly lost strength, could walk only with assistive devices, again developed extreme fatigue and went on medical disability. She was told no additional medications were available to treat her condition.

Ms. W came in for a three-month follow-up feeling upset and discouraged. When Dr. X entered the room, he looked at her chart and noticed she had not gotten the CBC he recommended. He was aware of some feelings of anger on the part of Ms. W and wanted to leave the room and get to the next patient. Both Dr. X and Ms. W sensed a breakdown in their relationship. Ms. W, therefore, did not tell Dr. X of her new symptom of shortness of breath.

The Physician Perspective
Physicians are educated to diagnose and treat, to be objective and to get it right. Most physicians come from a background of great academic success and overachievement, in which they are taught that studying hard and knowing the facts lead to success.

Furthermore, medical training tends to discourage display of emotion and instead emphasizes the need to be objective at all times. The result is that we as physicians become alienated from our own emotions and tend to feel awkward and uncomfortable when a patient displays strong emotion, especially negative emotion.

When Dr. X was preparing to enter the room with Ms. W, he was hoping to hear good news and see some improvement in Ms. W’s condition. When he saw no improvement and was greeted by her anger, sadness and multiple complaints, he felt helpless and frustrated. He was upset that Ms. W did not get her lab work done and did not respond to his interventions. At the same time, he felt shame and a sense of failure that he did not “get the right answer” and make Ms. W feel better.

Dr. X felt uncomfortable with Ms. W’s display of emotion and did not know how to respond. This made him anxious. He was already feeling rushed and burned out due to the decreasing insurance reimbursements for his services and the need to see more patients per day to keep his income steady. He, therefore, tried to make the visit as brief as possible. When he left the room, he felt relieved. He decided to have his PA follow Ms. W on future return visits.

The Patient Perspective
Ms. W idolizes Dr. X. He was the first doctor to diagnose her after two years of symptoms and eight doctors with no answers. During the first few years of treatment she always made sure to let him know how grateful she was for his expert professional care. Now that she is doing poorly, she feels ashamed that she has let him down.

For the patient, illness is a complex and multi-factorial problem. Ms. W, like any other patient with a chronic disease, has experienced a severe and complete disruption in lifestyle and sense of security due to her illness.

The patient with Sjögren’s suffers multiple losses which are often grieved like a death. The most obvious loss is that of good health and a sense of well-being. For Ms. W, the loss of physical function and the ability to walk had severe consequences. She lost the support of her colleagues when she could no longer work. The man to whom she was engaged called off their wedding and left her. She lost her career and her sense of security.
She also lost the ability to do many things that she loved such as dancing, running, skiing and hiking. She could no longer easily care for her children and manage her household. Her self-confidence was replaced with self-doubt and feelings of lack of control of her body and life. She feared what might happen next and felt alone in dealing with this new challenge.

The Result
When Ms. W entered the exam room for her 3-month appointment, she was in need of expressing her feelings about her illness and her problems with someone who would listen, understand and offer help, guidance and support.

When Dr. X entered the exam room, he was looking for facts, data, and answers to questions within a limited period of time which would help him assess the patient’s progress so that he could recommend safe and effective treatment.

These differing needs and expectations result in impaired communication and an erosion of the doctor-patient relationship.

Building a Strong Doctor-Patient Relationship
In order to connect with any patient, a physician must first understand and be present to his/her own emotions. Although this is not often taught in medical school or residency training, this type of self-awareness is teachable. For example, the author conducts such a program and may be contacted for more information.

It is important when you enter the exam room to greet the patient, make eye contact and sit down facing the patient. If the patient is emotional, acknowledge the patient’s distress in a calm and nonjudgmental voice. For example, you might say, “Good morning, Ms. W. You seem distressed.”

Then stop talking and allow the patient time to verbalize his/her feelings without interruption. When the patient is done, it is important to acknowledge his/her distress and indicate understanding. The more detached the physician appears to be, the greater the tendency for the patient’s emotions to escalate and vice versa. Although this will only take a few short minutes, you will learn much about the patient’s concerns and will earn his/her trust and cooperation. Working together toward the same identified goal also strengthens the doctor-patient bond. By asking the patient to identify his/her goals early in treatment and expressing them in the form of mutual goals, both doctor and patient can work together to control the common “enemy,” the illness.

It is important to realize that a multi-disciplinary approach is essential in order to address all of the Sjögren’s patient’s needs and that you, yourself, cannot solve all of the patient’s problems. For example, mental health professionals who understand the effects of Sjögren’s can offer your patients individual and/or group support in dealing with loss, life change, and depression should it develop.

Keeping your patient informed and educated also strengthens the doctor-patient bond. Allow the patient easy access to your office by phone or email should questions arise and provide prompt feedback through your qualified office staff.

Lastly, trust is essential to a healthy doctor/patient relationship. You must be honest and straightforward with your patient within a context of empathy and respect.

Physicians: A Strong Doctor-Patient Relationship Starts With You!

• Become self-aware.
• Listen to your patient. Don’t interrupt.
• Encourage your patient to talk about how she/he is feeling physically and emotionally.
• Facilitate a multi-disciplinary approach to treatment, including the involvement of mental health professionals as needed.
• Ensure your patients are well-informed.
• Develop trust through honest and respectful dialogue.

4th International Conference on “B Cells and Autoimmunity”
August 19-22, 2010, Nara, Japan
Studies of B cell physiology recently have contributed to new and surprising insights into major questions about autoimmunity and potential immuno-intervention strategies. This meeting will bring together basic scientists and clinicians with research interests in a range of autoimmune diseases to discuss advances in B cell biology, its role in autoimmunity and the prospect of designing effective therapeutics. For more information, visit http://web.rcai.riken.jp/bcellevent/index.html.

7th International Congress on Autoimmunity
May 5-9, 2010, Ljubljana, Slovenia
A wide range of hot topics in autoimmunity will be covered including diagnostics, therapeutics, infections and vaccinations. For more information, visit www.kenes.com/autoimmunity2010.
SSF Outstanding Abstract Award Announced at ACR

The recipient of this year’s SSF Outstanding Abstract Award at the American College of Rheumatology (ACR) annual meeting is Seshagiri Rao Nandula, PhD, of the Division of Nephrology, University of Virginia, Charlottesville, Virginia. Dr. Nandula was recognized for his abstract, “Activation of Innate Immunity Leads to Accelerated Development of Sjögren’s Syndrome-Like Disorder in NZB/W F1 Mice.” The senior author on the study is SSF 2008 and 2009 Innovative Concept Grant recipient Umesh Deshmukh, PhD.

Does Sjögren’s (SS) develop as a result of an adaptive immune response to chronic activation of innate immunity in genetically susceptible individuals? Dr. Nandula and colleagues set out to answer this question. Examining NZB/W F1 mice, their data supported this theory and also demonstrated, for the first time, a potential role for the IL-21 pathway in the development of SS. The latter finding might provide a new future target for immunotherapy in SS.

The Foundation recognizes excellence in research by new and/or young investigators who present outstanding abstracts on Sjögren’s syndrome research at professional meetings.

SSF Student Fellowship Available Through ACR

The SSF and the American College of Rheumatology (ACR) are calling for applications from students who want to focus on a project in Sjögren’s (SS). Students enrolled or accepted at a U.S. medical school or other graduate school program may apply. One such award will be provided through the ACR Research Education Foundation (REF) Preceptorship Program and will be offered through the ACR REF/Abbott Medical Student Research Preceptorship and the Health Professional Graduate Student Research Preceptorship.

Applications may be uploaded to the ACR website starting February 1, 2010, for the first quarterly deadline of May 1. If an ACR-SSF Student Fellowship is awarded during Cycle 1, no further awards will be made in the award year. If an award is not made, applications will be accepted for the next cycle. More information is available at www.sjogrens.org/research, www.rheumatology.org/ref/awards/hpprecep.asp, and www.rheumatology.org/ref/awards/summerresearch.asp.

The SSF is partnering with several professional societies for its Student Fellowships for FY2010-2011. Guidelines will vary depending on the organization through which the award is given; additional fellowships will be announced in the spring Sjögren’s Quarterly and on the SSF website.

The latest expansion of the SSF research program is made possible by The Bannon Humphery Foundation of Charleston, South Carolina, which joins the SSF for a second year in its goal to foster future scientists and clinicians in Sjögren’s.

Continued on page 10

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News From SSF Research Grantees and Student Fellowship Recipients

Olga Baker, DDS, PhD, has left the University of Missouri, Columbia, Missouri, to become an Assistant Professor in Oral Biology, School of Dental Medicine, University at Buffalo, State University of New York (SUNY). Dr. Baker received an SSF grant in 2006 and 2007 for her project, “Effects of pro-inflammatory cytokines on polarized salivary epithelium.” She has been an author on at least four articles in professional journals since receiving her SSF grant and is Principal Investigator for NIH grants in 2007 and 2008 for her project, “P2Y2R Mediated Immune Responses in Salivary Gland Dysfunction.”

Patrizio Catugelgi, MD, of the Johns Hopkins School of Medicine, has published two journal articles based on his research supported by an SSF grant awarded in 2007 and 2008. They are:


Dr. Catugelgi’s SSF research grant project was entitled, “Role of IL-12 in the inducible BALT found in Sjögren’s lungs.”

Fortunato Battaglia, MD, PhD, has been named Assistant Professor and Director of Neuroscience, New York College of Podiatric Medicine and Columbia University. He will carry out his Sjögren’s research in the Department of Psychiatry at Columbia. Formerly, he taught at the City University of New York Medical School.

SSF research reviewers noted at the time of Dr. Battaglia’s 2009 SSF Research Grant award that Dr. Battaglia was an exceptionally innovative researcher who was poised to rise in his profession and that an SSF grant might help ensure that he make Sjögren’s a focus in his future career. This hope is coming to fruition as he starts work on his SSF-funded grant project, “Depression and anxiety in a mouse model of Sjögren’s syndrome.” Dr. Battaglia’s grant is funded by the Galewood Foundation.

Another 2009 SSF Research Grantee, Helen P. Makarenkova, PhD, authored an article published this fall in *Science Signaling*, a journal of the American Association for the Advancement of Science. The article is entitled, “Differential Interactions of FGFs with Heparin Sulfate Control Gradient Formation and Branching Morphogenesis” and credits the SSF for supporting her research.

Dr. Makarenkova is an Associate Fellow in Experimental Neurobiology at the Neurosciences Research Foundation, San Diego, California. She is an excellent scientist who is using her innovative work in neurobiology to focus for the first time on Sjögren’s. Makarenkova’s grant also is funded by the Galewood Foundation.

Aileen Chang, MD-candidate, was invited to deliver an oral presentation on her SSF-supported Student Fellowship project at the 10th International Symposium on Sjögren’s Syndrome in Brest, France, in October 2009. Chang recently completed her 2008 Student Fellowship on “The Integration of Traditional Chinese Medicine (TCM) in the Diagnosis and Treatment of Sjögren’s Syndrome” at the Penn Presbyterian Medical Center, University of Pennsylvania.

Amirala Pasha, DO-candidate, is serving as the Co-President of The Student Osteopathic Internal Medicine Association, College of Osteopathic Medicine, University of New England, for the 2009-2010 academic year. The Association provides special opportunities for medical students who are interested in Internal Medicine including trips to national/regional Internal Medicine conventions, lectures by practicing internists and hands-on activities that expose medical students to clinical/practical procedures performed by physicians.

Pasha is a current SSF Student Fellow focusing on a project entitled, “The effects of corneal cooling on tear production, blink rate and sensation.” He hopes to present his project results at the 2010 Association for Research in Vision and Ophthalmology (ARVO) meeting. His SSF Student Fellowship is supported by the Bannon Humphery Foundation.
HOW ARE YOUR SJÖGREN’S PATIENTS BALANCING THEIR DRY-MOUTH SYMPTOMS?

EVOXAC® CAN HELP.

Dry-mouth symptoms of Sjögren’s aren’t always recognized as a medical problem until they start disrupting daily lives.1,2 Yet adequate salivary flow is essential for good oral health, initiating enzymatic food digestion, facilitating taste bud sensation, and more.3,4 If your patients are having trouble with their dry-mouth symptoms due to Sjögren’s syndrome, learn how EVOXAC (cevimeline HCl) can help.5-7

Learn how EVOXAC can help increase salivary flow in patients with dry-mouth symptoms associated with Sjögren’s syndrome at EVOXAC.com.

IMPORTANT SAFETY INFORMATION

EVOXAC (cevimeline HCl) is indicated to treat the symptoms of dry mouth in patients with Sjögren’s syndrome.

- Cevimeline HCl is contraindicated in patients with uncontrolled asthma, known hypersensitivity to the drug, and when miosis is undesirable, e.g., in acute iritis and narrow-angle (angle-closure) glaucoma
- Cevimeline HCl can potentially alter cardiac conduction and heart rate and produce transient changes in hemodynamics. Cevimeline HCl should be administered with caution and under close medical supervision to patients with a history of cardiac disease, controlled asthma, chronic bronchitis, or chronic obstructive pulmonary disease
- Cevimeline HCl should be administered with caution to patients taking beta-adrenergic antagonists because of the possibility of conduction disturbances and to patients with a history of nephrolithiasis or cholelithiasis
- If a patient sweats excessively while taking cevimeline HCl, dehydration may develop
- Caution should be advised while driving at night or performing hazardous activities in reduced lighting
- Safety and effectiveness in pediatric patients have not been established

• Cevimeline HCl is metabolized by the P-450 isozymes CYP2D6 and CYP3A4. Thus, there may be potential for interaction between cevimeline HCl and other compounds
• Special care should be exercised when cevimeline HCl is taken by geriatric patients, considering the greater frequency of decreased hepatic, renal, or cardiac function
• The most frequently reported adverse events associated with the pharmacologic action of a muscarinic agonist (>10% incidence) in clinical trials of cevimeline HCl were: excessive sweating, nausea, rhinitis, and diarrhea. Consult the full Prescribing Information for other adverse events

References:

Please see next page for brief summary of full Prescribing Information for EVOXAC.
When the American Recovery and Reinvestment Act of 2009 (ARRA) was signed into law this year, the Sjögren’s Syndrome Foundation (SSF) was excited about the unprecedented stimulus funding it would provide for all medical and scientific research and especially for Sjögren’s (SS). In response to the law, the National Institutes of Health (NIH) created “The NIH Challenge Grants” offering specific topics that the NIH deemed as priority areas for funding. These grants are provided for a 2-year period to jumpstart research into areas that the NIH considers to be particularly timely and/or that need attention.

The SSF identified more than half a dozen Challenge Grant priority areas for which Sjögren’s investigators might apply, contacted researchers about these opportunities and worked with many of them to support their submissions. In addition, the SSF solicited Sjögren’s experts as reviewers for NIH in response to the overwhelming number of applications – more than 20,000 applications in all. While the SSF shared the disappointment on the part of many applicants in Sjögren’s who could not be funded, it was pleased with the number of projects that were indeed awarded.

The NIH has launched a new grants database called the Research Portfolio Online Reporting Tool (RePORT). Sjögren’s is included as a search term, and we also have found additional projects relevant to Sjögren’s using search terms for key symptoms closely associated with SS. Some projects funded by stimulus monies, consequently, may have been missed. With that caveat, we follow with a list of grants that are either Sjögren’s-specific or cite relevance to areas such as dry eye or dry mouth and will increase our knowledge of Sjögren’s-related symptoms. We also should note that in addition to the Challenge Grants in Sjögren’s, Non-ARRA NIH Grants awarded in Sjögren’s in 2009 number more than thirty. Details about all projects can be found at http://projectreporter.nih.gov.

### SS and SS-Related ARRA-Funded NIH Grants

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<tr>
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<th>Organization</th>
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<td>Mathematical Model of Parotid Acinar Differentiation</td>
<td>Douglas S. Darling</td>
<td>University of Louisville</td>
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<td>Mechanism of Lacrimal Gland Secretion</td>
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<td>Control Of Conjunctival Goblet Cell Mucin Production</td>
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<td>Modulation Of Conjunctival Goblet Cell Differentiation by Immunoregulatory Cells</td>
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<td>T Cell Tolerance &amp; Autoimmunity to Nuclear Antigen LA</td>
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<td>Oklahoma Medical Research Foundation</td>
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<td>Mucins of the Ocular Surface</td>
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<td>Schepsen Eye Research Institute</td>
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<td>ADS Fiber Entry and Trafficking in Lacrimal Acini</td>
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<td>University of Southern California</td>
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<td>EGCG Intervention in a Sjögren’s Syndrome Model Prior to Disease Onset</td>
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<td>The Relative Role of Sex and Genes During Development of Systemic Autoimmunity</td>
<td>Trine N. Jorgensen</td>
<td>Cleveland Clinic Lerner Col/Med-CWRU</td>
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<td>Regulation of SMG Development by Adhesion Receptors</td>
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<td>Boston University Medical Campus</td>
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<td>A High-Resolution in Situ Proteomics Atlas Of Salivary Gland Development</td>
<td>Melinda Larsen</td>
<td>State University of New York at Albany</td>
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<td>1RC1DE020402-01</td>
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<td>Autophagy &amp; Functional Restoration Of Irradiated Salivary Glands</td>
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<td>University of Arizona</td>
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<td>Modulating WNT and Hedgehog Pathways for Functional Restoration of Salivary Gland</td>
<td>Fei Liu</td>
<td>Texas A&amp;M University Health Science Ctr.</td>
<td>NIDCR</td>
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Participants Solicited for Study on Sjögren’s and the Perception of Symptoms

Sjögren’s (SS) patients are wanted for a study on the impact of personality traits on the perception of symptoms in SS. The investigator will focus on resiliency, which is defined as the ability to think positively and make the most of a disadvantageous situation. An individual’s symptoms and thought patterns surrounding their experience with SS will be analyzed to determine whether resiliency determines one’s perception of symptoms.

Participants will be asked a series of questions via the internet regarding their health, level of functioning, coping skills and social support. All personal information will remain confidential. No costs will be incurred by participants, and participants may receive a copy of the final study.

The investigator, Wanda Ronda, is a graduate student in psychology and also an SS patient, and is making this topic the focus of her Master’s thesis at the Gordon F. Derner Institute of Advanced Psychological Studies at Adelphi University in Garden City, New York. She wants to bring greater awareness in her professional field of SS and the impact this disease has on patients. For more information, contact Ronda via email at wandaronda@mail.adelphi.edu.
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Have you or someone you know ever thought about running a marathon?

Do you have a desire to challenge yourself?

Do you want to make a difference for Sjögren’s syndrome?

Then join Team Sjögren’s and train to participate in the 2010 Country Music Marathon & Half-Marathon in Nashville on April 24, 2010.

We are looking for 20 inspired individuals to join us as we begin to train for this challenge. We understand that not all Sjögren’s patients are able to participate in a marathon, so we hope you will extend this invitation to family members as well as friends who may be interested in participating in this challenge!

To sign up, contact Elyse Jordan directly at (800) 475-6473 ext. 217 or ejordan@sjogrens.org


Publication on Sjögren’s Available

Already known in Sjögren’s (SS) professional circles for her research and publications in SS, Jiska M. Meijer, MD, DMD, of the University Medical Center Groningen, the Netherlands, is publishing her thesis for an additional advanced degree in oral and maxillofacial surgery. Entitled “Sjögren’s Syndrome: Treatment and Treatment Evaluation,” the book describes the impact of SS on the socioeconomic status of patients, new treatment options in SS (including the use of rituximab) and progression and treatment evaluation (including salivary proteomic and genomic biomarkers).

Copies are available at cost from Dr. Meijer. She may be contacted at j.m.meijer@kchir.umcg.nl.

International Association for Dental Research (IADR)

General Session
July 14-17, 2010, Barcelona, Spain

Watch www.iadr.org for information on abstracts and program submission and schedule. The mission of the IADR is to advance research and increase knowledge for the improvement of oral health worldwide; support and represent the oral health research community; and facilitate the communication and application of research findings.

6th International Conference on the Tear Film and Ocular Surface: Basic Science and Clinical Relevance
September 22-25, 2010, Florence, Italy

Abstracts submitted by young investigators for consideration of a Travel Award are due no later than April 15, 2010; all other abstracts are due by June 15, 2010. In addition to reports from the International Dry Eye Workshop and the International Workshop on Meibomian Gland Dysfunction, a session is schedule on Late Breaking News in Sjögren’s Syndrome. For information on all topic sessions, abstract submission and conference registration, visit www.tearfilm.org/Tfos_conferences.html.
2009 SSF National Patient Conference Audio CDs are Now Available!

Six of our most popular talks from the 2009 National Patient Conference held in Arlington, Virginia, are available for purchase as audio CDs. Each talk is 30-40 minutes long and each CD comes enclosed with the handouts and visual aids used by the presenter. Buy just the talks you want to hear or purchase the whole set! Whether you attended the conference or not, these audio CDs are an excellent way to have a permanent resource with some of the most vital information available to Sjögren’s patients.

These CDs may be purchased using the order form below, online at the SSF Store, or by calling the SSF office at 800-475-6473.

Dry Eye and Sjögren’s – Gary N. Foulks, MD, FACS: Professor of Ophthalmology and Director of the Cornea and External Disease Service at the University of Louisville School of Medicine, Dr. Foulks discusses your dry eye symptoms and complications and informs you about the latest dry eye treatments, covering the extensive range of help available from artificial tears to silicone plugs to systemic drugs.

Dry Mouth and Sjögren’s – Andres Pinto, DDS, DMD: An Assistant Professor in Oral Medicine and Director of the Oral Medicine Clinical Center at the University of Pennsylvania School of Dental Medicine, Dr. Pinto, who has more than eight years of clinical experience with Sjögren’s and its oral manifestations, answers your questions about your teeth, gums, saliva, swallowing and more.

Gastrointestinal Manifestations of Sjögren’s – Matthew Nichols, MD: The GI manifestations of Sjögren’s are multiple, and gastrointestinal involvement is common. Dr. Nichols will enhance your understanding of how the esophagus, stomach, liver, and intestines are affected by Sjögren’s.

Managing Patients with Immunosuppressive Treatments – Frederick B. Vivino, MD, FACR: Chief of Rheumatology, PENN Presbyterian Medical Center; Director, University of Pennsylvania Sjögren’s Center. Dr. Vivino discusses the organ involvement and various complications as well as the possible immunosuppressive drug treatments available.

Non-Hodgkin’s B Cell Lymphoma and Sjögren’s – Elaine L. Alexander, MD, PhD: A world-renowned rheumatologist and immunologist, Dr. Alexander clears away the confusion surrounding this potential complication of Sjögren’s. This lymphoma expert will define the risks to Sjögren’s patients and will outline the symptoms.

Neurological Manifestations of Sjögren’s – Julius Birnbaum, MD: Uniquely trained in internal medicine, rheumatology and neurology, Dr. Birnbaum recently pioneered a new clinic devoted to the care of patients with neurological complications of Sjögren’s and other autoimmune diseases.

All of these audio CDs can be purchased using the order form below, online at www.sjogrens.org or by contacting the Sjögren’s Syndrome Foundation office at 800-475-6473.

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Patients with Sjögren’s frequently suffer from decreased mucus/nasal secretions and dryness of the nose and sinuses.

Please note that to ensure easier understanding on the part of the patient, we use common lay terms for the following tips instead of more precise medical and surgical terminology.

- Monitor the humidity in your home with a simple humidistat. For Sjögren’s patients, an optimal range of humidity is between 55% and 60% regardless of the ambient temperature.

- Use a humidification system built into a furnace that pushes forced hot air through one’s home.

- Try a bedroom humidifier, which generally comes in two types. While more expensive, a self-sterilizing unit is ideal in that it continuously sterilizes and cleans the steam prior to admitting it into the air. A more modestly-priced humidifier is adequate but must be cleaned at least twice a week to limit the possibility of circulating fungus in the air.

- Avoid dry environments, such as automobiles with closed heating systems and airplanes. Baseboard heating in the winter can contribute significantly to decreased humidity. Obvious places to avoid are the sauna at your health club and the hot desert.

- Enjoy high humidity environments, such as a steam bath, although remember that hot and long baths can dry out the skin.

- Avoid medications that increase dryness when possible. Many medications used to treat the upper respiratory tract such as decongestants and antihistamines are drying. Many other medication classes also may contribute to nasal/sinus drying. When in doubt, check with your physician.

- Note that immunosuppressant drugs particularly may exacerbate drying of the nasal cavity and lead to attendant crusting, bleeding, foul smell and discharge. Discuss all potential side effects of your medications with your physician.

- Practice good oral and nasal hygiene and avoid toxic agents. Remember that alcohol and smoke have a drying effect. Even secondhand smoke has now been shown to contribute to nasal irritation.

- Consider using an over-the-counter emollient such as Ponaris® to cleanse the nose, particularly if large crusts and debris are present.

- Use over-the-counter nasal drops and buffered saline sprays regularly (as often as every hour) to lubricate the nasal passages and nasopharynx. Additionally, over-the-counter gels such as Rhinaris® and AYR® work like sprays but last longer and are recommended particularly at night prior to going to sleep.

- Discuss the prescription medications Salagen® and Evoxac® with your physician. These have been shown to help Sjögren’s patients with dry mouth, and potential added benefits for dry nose, sinuses and nasopharynx should be considered.

For more information on Sjögren’s syndrome, visit the SSF Web site at www.sjogrens.org, call 800-475-6473, e-mail ssf@sjogrens.org or write to the Sjögren’s Syndrome Foundation, 6707 Democracy Blvd, Suite 325, Bethesda, MD 20817.

Clinicians: Please make multiple copies of this Patient Education Sheet and distribute to your patients. If you have an idea for a topic or want to author a Patient Education Sheet, contact us at sq@sjogrens.org.
The Oklahoma Medical Research Foundation (OMRF) has announced the launching of a major international collaborative genetics research study in Sjögren’s – the Sjögren’s Genetics Network (SGENE).

**Purpose**

SGENE is a collaborative group of researchers and clinicians supporting genetic studies in Sjögren’s (SS). Through coordination of a large international effort, OMRF seeks to organize DNA samples and associated clinical data for well-designed genetic studies. Thousands of samples are needed.

Collaborators and contributors are sought who can assist in identifying patients who meet the American-European Consensus Group criteria for classification of SS. OMRF will work with clinicians to arrange for patient recruitment, collect DNA samples and obtain clinical data relevant to SS.

**Background**

Modern genetics approaches are revolutionizing our understanding of numerous complex diseases. Through studies involving thousands of cases and controls, association with over 30 genes now has been confirmed for autoimmune diseases such as lupus and Crohn’s disease. More genes are expected to be confirmed for these disorders in the near future, underscoring a complexity we also expect to find in SS. Large cohorts of SS patients will be needed to firmly and comprehensively characterize the genetic architecture of SS.

**Participation in SGENE**

A Sjögren’s Genetics Coordinating Center based at OMRF is collecting and organizing DNA samples and clinical data for large-scale genetic studies. Over 13 SGENE collaborating sites have contributed DNA samples and clinical information with additional collaborators continually welcome to join.

For more information and if interested in participating, please contact:
Kathy Moser, PhD
Oklahoma Medical Research Foundation
825 N.E. 13th Street
Oklahoma City, OK 73120
Tel: (405) 271-2534
E-mail: moserk@omrf.org