Giving Back—Profile of a DF Volunteer

Sarah C. Jackson, MD—Supporting the Future

"We are all very lucky in dermatology," Dr. Jackson says. "I've already been given so much in such a short time by the specialty that I feel it's essential to start giving back now." Dr. Jackson chooses to do this by supporting the specialty at the *Leaders Society* level as both a member and an active volunteer.

Sarah C. Jackson, MD

Dr. Jackson's private practice in New Orleans combines medical, cosmetic, and surgical dermatology. She is also a Clinical Assistant Professor at Louisiana State University. She became aware of the DF's impact on the specialty during her residency there, and now shares her enthusiasm for the DF with her fellow dermatologists.

In 2007, Dr. Jackson's first year of practice, she was invited by a colleague to become a member of the DF *Leaders Society,* and she eagerly joined. Three years later, she accepted an opportunity to participate in the *LS* volunteer campaign in her area and now leads the efforts to increase physician support throughout Louisiana. Not one to miss an opportunity to contribute to the specialty, Dr. Jackson is also the new President of the Louisiana Dermatology Society.

Dr. Jackson enjoys spreading the word about the Dermatology Foundation's critical mission, its success in fulfilling it—and most of all—the importance of giving now to support the specialty's future. She highlights the essential role of DF research funding in advancing the specialty by nurturing the early careers of those who will ensure scientific progress and continued teaching strength.

"I feel that giving back to the specialty is required of every young dermatologist. Everything that we contribute now will come back to us 10-fold. So why wait?"

The DF is exceptionally grateful to its many volunteers who work hard and give generously of their time to keep dermatology at the forefront of medicine.

several post-op days. Fortunately, adrenal crisis is rare and "in our concern for adrenal insufficiency we have likely been over-replacing many patients in the operative setting," Dr. Schlechte said. The size of the stress dose should be determined by the intensity and length of the surgical procedure. For minor and short procedures, it may not be necessary to give any supplemental steroid.

Guidelines. Schlechte also discussed the issue of tapering glucocorticoids after prolonged therapy with supraphysiologic doses in patients who no longer need the drug. While long-term steroid therapy cannot be abruptly discontinued, it is important to lower the dose rapidly to physiologic replacement and then begin a slow taper from that point. Clinicians should be familiar with daily physiologic doses of glucocorticoids in developing tapering regimens (5 mg of prednisone, 15–20 mg of HC, and 0.75 mg of dexamethasone). Schlechte also reviewed the serious metabolic and skeletal side effects of long-term glucocorticoid therapy.

Immunosenescence: Causes and Consequences

Janet A. Fairley, MD

Immunosenescence. This involution of the immune system with age, occurring in all mammals, involves a decrease in adaptive immunity that produces a chronic low-grade inflammatory state. There are many more CD8 (cytotoxic) T cells, fewer CD4 (helper) T cells, fewer CD19 (activation-capable) B cells, and fewer NK cells. This has multiple relevance for dermatology. Some skin diseases, e.g., CTCL, typically have worse outcomes in the elderly. Some skin diseases, e.g., bullous pemphigoid (BP), occur only in the elderly. The average age of onset in BP (a specific interest of Dr. Fairley's) is 75. And we will be seeing a great many more aging patients because this population segment is growing explosively. Possible causes of this immunosenescence include persistent antigenic stimulation, particularly from viruses—and this dovetails with some of Fairley's observations.

CMV and Immunosenescence. Infection with cytomegalovirus (CMV), a species-specific herpes virus, turns out to be ~50% overall in the U.S., increasing with age. After acute infection, which is typically asymptomatic, this extraordinarily immunogenic virus maintains a lifelong presence. In some, it remains dormant. In others, it reactivates, without symptoms, and provokes *T-cell memory inflation* and an *immune-risk profile.* CD8 T cells expand and predominate, with so many targeting the CMV virus that the reserve of naïve T cells becomes highly inadequate. These seropositive patients are also skewed from the more cell-mediated Th1-type of immune response to the more autoantibody-mediated Th-2 response. A mutation in the innate immune system receptor that recognizes CMV virus has been found in patients with autoimmune diseases. CMV has recently been associated with irritable bowel disease, dermatomyositis, and poor outcomes in CTCL, and significantly elevated anti-CMV titres have been found in autoimmune patients. Fairley described her experience, finding 95% of her BP patients seropositive for CMV compared to 66% in matched controls and 60% among pemphigus vulgarus patients. She also found that her BP patients react to a very common CMV protein that has some homology with the