



**Name:** Fatou Jallow

**Email:** fjallow@wisc.edu

**Major Professor:** Dr. Linda A. Schuler

**Degree Objective:** Ph.D. Endocrinology and Reproductive Physiology

**Background:** BS Biology - Dillard University New Orleans, LA

**Current Research Project:**

The protein hormone prolactin (PRL) is the key mediator of lobuloalveolar proliferation and differentiation during pregnancy. When PRL binds to its receptor, it activates multiple signaling cascades including Jak2 and Signal transducer and activator of transcription 5 (Stat5). Stat5 has two highly conserved isoforms, Stat5a and Stat5b. Although encoded by separate genes, there is nearly 97% similarity in amino acid sequence between the two proteins. When Stat5a and Stat5b are activated by phosphorylation (within the C-terminal domain) at tyrosine 694 and 699 respectively, they homodimerize or heterodimerize, and translocate to the nucleus where they regulate transcription. Stat5a mediates most of the physiologic actions of PRL in normal mammary epithelia. Recent studies have indicated that Stat5a and Stat5b have opposite roles in breast cancer cell migration and invasion. Stat5a inhibits migration and invasion in aggressive breast cancer cell lines *in vitro*, whereas Stat5b has the opposite effect. Only recently have potential differences in the activities of these two isoforms in response to activation by PRL been compared. Many reports suggest that the relative levels of Stat5a and Stat5b will determine the outcome of PRL signals, however, the differential control of Stat5a and Stat5b expression, particularly within mammary epithelium, is poorly understood.

We recently published that chronic post-pubertal treatment of PRL/TGF $\alpha$  transgenic mice with the anti-estrogen, ICI 182,780 (ICI), resulted in mammary tumors with significantly higher Stat5b transcripts and nuclear Stat5b protein levels. Nuclear Stat5a protein was significantly lower with ICI compared to non-treated females, while Stat5a transcripts trended lower compared to non-treated female mice. It has also been reported that acute *in vivo* treatment of pubertal females with estrogen for 3h elevated mRNA and nuclear localization of Stat5a. These data suggest that estrogen alters the balance between Stat5a and Stat5b, altering the outcome of PRL signals. However, this has not been directly tested. Understanding the crosstalk between PRL, estrogen and Stat5a/b will narrow the gap in the literature and further elucidate the role of Stat5b in mammary tumor progression.

**Honors:**

1999–2001 Dillard University Institutional Academic Scholarship

2011-2013 Predoctoral trainee, R25 GM083252, TEAM Science program, University of Wisconsin



- 2013-2015    Predoctoral trainee, T32 HD041921, Endocrinology/ Reproductive Physiology training grant, University of Wisconsin
- 2013        Platform presentation: *Mammary Gland Gordon Conference, June 8-14, 2013. PRL/TGF $\alpha$  induce ER $\alpha$ -dependent proliferation of mammary epithelial cells and tumorigenesis in the absence of estrogenic ligand.* UW Endocrinology and Reproductive Physiology Program Symposium, July 8, 2013. Best of Session Talk
- 2013        Carl Storm Underrepresented Minority (CSURM) Fellowship
- 2015        Predoctoral trainee, R01CA179556, Comparative Biosciences, University of Wisconsin
- 2015        Travel Award, FASEB

## Grants Received:

- September 2011- August 2013    NIH R25 Scholar; National Institute of General Medical Sciences, IMSD Institutional Research Education Grant, "Training & Education to Advance Minorities in Science" (TEAM-Science) NIH R25 GM083252;
- September 2013 – August 2015    NIH T32 Trainee; NIH Ruth L. Kirschstein National Research Service Award 5T32HD041921-10
- September 2015 – Present        NIH R01CA179556, Comparative Biosciences, University of Wisconsin

## Publications:

O'Leary KA, Jallow F, Rugowski DE, Sullivan R, Sinkevicius KW, Greene GL, Schuler LA. Prolactin Activates ER $\alpha$  in the Absence of Ligand in Female Mammary Development and Carcinogenesis in Vivo. *Endocrinology*. 2013;154(12):4483-92.

## National Presentations:

Fatou Jallow, Kathleen A. O'Leary, Debra E. Rugowski, Ruth Sullivan, Kerstin W. Sinkevicius, Geoffrey L. Greene and Linda A. Schuler. "PRL/TGF $\alpha$  induce ER $\alpha$ -dependent proliferation of mammary epithelial cells and tumorigenesis in the absence of estrogenic ligand". Mammary Gland Gordon Seminar/ Conference, June 8-14, 2013.

## Other Presentations:

Fatou Jallow, Kathleen A. O'Leary, Debra E. Rugowski, Ruth Sullivan, Kerstin W. Sinkevicius, Geoffrey L. Greene and Linda A. Schuler. "PRL/TGF $\alpha$  induce ER $\alpha$ -dependent proliferation of mammary epithelial cells and tumorigenesis in the absence of estrogenic ligand". ERP- 2013 Annual Research Symposium    **Award:** Best of Session Oral Presentation



Fatou Jallow, Debra E. Rugowski and Linda A. Schuler. "Estrogen increases the ratio of Stat5a to Stat5b in mammary epithelia, which influences the outcome of prolactin signaling". ERP – 2014 Annual Research Symposium. Poster presentation

Fatou Jallow and Linda A. Schuler. "Multiple receptors mediate the actions of estrogen and ICI 182,780 in prolactin-induced ER $\alpha$ + breast cancer". ERP- 2015 Annual Research Symposium. Poster presentation

Fatou Jallow and Linda A. Schuler. "Multiple receptors mediate the actions of estrogen and ICI 182,780 in prolactin-induced ER $\alpha$ + breast cancer". 2015 FASEB, THE GROWTH HORMONE/PROLACTIN FAMILY IN BIOLOGY AND DISEASE. Poster presentation

## **ERP Service:**

2011 – 2012 Seminar Committee

2013 – 2014 Student committee member

2013 – 2014 Symposium organizing committee