INTERVIEWEE: Michelle Winn
INTERVIEWER: Jessica Roseberry
DATE: November 8, 2010
PLACE: Dr. Winn's office, GRSB-1

WINN INTERVIEW NO. 1

JESSICA ROSEBERRY: This is Jessica Roseberry. I'm here with Dr. Michelle Winn. She's an associate professor of medicine, and it's November 8, 2010, and we're here in her office in GRSB I [Snyderman Genome Research Science Building]. And I want to thank you so much, Dr. Winn, for agreeing to be interviewed today.

MICHELLE WINN: Thank you for having me.

ROSEBERRY: Let me ask you a little bit of background and just ask what got you interested in the field of nephrology.

WINN: Well, I came here to Duke in 1992 as a resident and was frequently interested in everything, and as I was—I actually started in Psych then went to Med Psych for two years and then eventually became a medicine resident, full medicine resident, and just enjoyed every rotation I did and thought that I needed to learn more about nephrology because I thought it was difficult at the time. And as a JAR [junior assistant resident] my first rotation I took was a renal consult month so I could learn more about medicine—I mean about nephrology—and just turned out to love it. It was just a lot of fun and exciting, and the kidneys seemed kind of complicated but doable. (*laughs*) It was a lot of fun. It was—I'm still excited about it. I had this great patient this weekend. I was rounding this last week, and I came in because it was very exciting, called the fellow in. We looked at the urine together that we spun and looked at it under the microscope

because it was a patient with potentially a pulmonary renal syndrome, which is a very acute situation that needs treatment right away with like cyclophosphamide and plasmapheresis and potentially dialysis and steroids, and so it was very—it was one of those acute situations that makes nephrology fun. It's not so much fun for the patient, but when you get situations like that where it's very acute and you need to make important decisions to save their kidneys, you want to go in and do all that you can, so it's a lot of fun. So it's—I just—I think nephrology is just interesting in so many aspects. There are lots of different things to do in nephrology like transplant nephrology, dialysis. When we round on the wards and see consults, we never know what we're going to get. It can be something very straightforward or something incredibly complex, so there's a lot of variety, and you feel like you're a complete doctor because you take care of the whole patient. The renal system itself encompasses everything including blood pressure and urine output and serum creatinine and cleaning your blood, and just everything you can imagine so it's—it involves the whole body.

ROSEBERRY: You mentioned that it seemed difficult and complex. Has that proven to be true?

WINN: It's—it is in some ways, and it's not as complex as it seemed when I started. It's still a very—well, the system itself is very complex. I think understanding it is a lot easier than I thought it would be. There's—it's—being in medicine is a lifelong learning process, so you never stop learning. There are always going to be new drugs, new things that come up that we need to learn about. So that's what makes medicine exciting in that it's forever, forever learning. But after doing it for a while I realized there are certain things that you just need to know the basics of and then you can work out anything else

pretty much from that, so it's a very complex system, yes, but understanding it was not as hard as I thought it was going to be when I first did my consult month as a JAR, as a resident.

ROSEBERRY: Can you tell me how nephrology at Duke has changed, maybe, since you first came?

WINN: Well, I'd start back at Medicine changing since 1992. When I started here in 1992 Joe Greenfield was the chair of Medicine. And he was extremely hands-on, was here every Friday night at midnight for pizza rounds for us so that we could talk to him, ask questions. He just seemed to know all and be all, (*laughs*) you know. When we had morning report we'd always—our goal was to try to stump him, and it never worked. That was our primary goal in life, you know. And I remember having this tube of pleural fluid that looked milky, and it was from a chylothorax, and he just got it right away. It's like, Oh, jeez. Anyway, and then Bart Haynes became chair, and he was just as bright. I mean, there's no question about it, but Medicine had started to change a little bit, and we started to have different work rules for residents, and things were starting to change. And then Pascal [Goldschmidt] and—well, Harvey [Cohen]—Pascal, Harvey, and then—and Mary Klotman now. And I think it's gotten harder to be a chair than it used to be. It's much more administrative now, instead it was more teaching I think when Joe Greenfield started. And I would say for Medicine residents, what I would tell them to do is take ownership of your patients, you know. Always feel like it's—they're your job, no one else's job. And it's gotten more difficult for them to have a relationship with patients because they have work-hour rules and they have to go home at a certain time, and they can't see a disease process from beginning to end because if their call day ends at noon—

whereas our call day didn't end. It—whenever we were post-call we stayed until the work was done, whether that's eight or nine at night or earlier, that just depended on when your work was done. I think there—we've got to find a happy balance between just saying, You go home at 11:00 versus 9:00 p.m. the next night after being on all night, but interns won't be on more than sixteen hours this coming year at a time, whereas we could be on, I don't know, you know, (*laughs*) forever. It seemed like forever. So I would just say Medicine has changed in that it's harder for Medicine residents—all residents, Surgery residents, everyone, to get a good relationship with patients so that they have that: This is my patient and I do everything I can to take care of them. It's harder for them, so they have to work harder at it, I think. And also they have less exposure to patients and disease processes so they have to read more than we did because we were always here taking care of the patient. And for them, they go home post-call, they get a day off a week. I remember as a SAR in six months—my last six months as a SAR, senior assistant resident, I had one day off, and we had days like—if I did gen med [general medicine], and we did two months at a time with gen med and I was the leader, the SAR of the group, on Sundays we were allowed one day off a month. But on that Sunday when you had your time off you usually come in and rounded because it was your patients and you didn't want anybody telling you—anybody doing anything to your patients but you, so you would go in, see your patients and leave around one in the afternoon, and that was a day off. That was great. (*laughs*) And then you'd expect the intern to call you with questions about your patients. Even though you weren't technically on, you felt this ownership like, My patients. And I won't—it was just—and I—it was a lot of fun. It was very—it was difficult. It was a very difficult three years.

Honestly, I mean there's nothing to say that—I'm not trying to say we were any better or worse than things are now; I'm just saying it's very different, and what I do feel proud about is that when we finished as—when we graduated from Duke Med as a generalist, we knew how to take care of patients. And that was the one thing you could be confident in. If somebody was walking across the street and just all of sudden they're on the ground, you don't panic, you don't get upset, you don't scream and—it's just complete calm. You just go over there and take care of it, and that I really liked. I really liked feeling in control of the situation, because I'd had some much experience in ICUs [intensive care units] and do—be on a code team, and you just feel comfortable taking care of patients, which is the best, you know, so—. Then your question was about nephrology? How has it changed? Let's see. Well, it changes a lot being a fellow to being (*laughs*) an attending and then running your own lab. When I first started in nephrology which I think was around '95 or '96 Bill Yarger was our chief, and then within a couple years Tom Coffman became our chief. I always looked at Bill Yarger and said, If I had just the physiology he knows in his little pinkie, if I had that, I'd be brilliant. He just—he's one of these old-school, been around forever—. Before people were even called nephrologists he'd been around and he was doing these single-nephron GFRs [glomerular filtration rates] with these needles and these little glomer—I mean, just doing things that were amazing, and he just had a lot of knowledge. Tom came along, and he is the same. I mean, he is incredibly bright, and he's—he's been an incredible mentor for me. (*telephone ringing*) Sorry. Okay. So he's been an incredible mentor for me. He—he's like everything a physician-scientist should be in that he runs his lab, he has this brilliant academic career, he was just—he's the past president of the American

Society of Nephrology, never seems to lose his cool, (*laughs*) you know, and he has—you know, he's provided me with a lot of mentorship in regards to grantsmanship, how to write grants, how to be a good doctor, you know, in so many ways. He's got a balanced life which is good, but he's—he's always working, too. I mean that, you know. I say balanced and always working, but (*laughter*) he's able to balance things he likes to do with work, which he does a lot. And I think he and Laura Svetkey who—I don't know if you know her, but she's our like vice chair for faculty affairs or something like that. She's been my other major mentor.

ROSEBERRY: In the Department of Medicine?

WINN: In the Department of Medicine, right. So Tom has been like my academic mentor, and Laura's been like my life mentor in that just very savvy with conflicting situations that arise that you need to work your way out of in a very delicate way, and just very savvy. And she actually got me very much interested in diversity, which I've always had an interest in, but she was one of the founding members of the Minority Recruitment and Retention Committee for the Department of Medicine, she and others. And seeing their commitment to diversity really helped me say, I need to take a lead in this at some point and do what I can do. I look at the difference with diversity between when I came in '92 and now, and it's just incredible. There might have been two minority residents when I was a Medicine resident throughout the whole three years—I mean, just not many at all, maybe three, and now it's upwards of 15 percent or so, it might even be more. It's just really big difference. And a group of around 150 or so, from two to three to fifteen to twenty is a huge difference. And after I went on faculty in '99 I eventually became the chair of the Minority Recruitment and Retention Committee,

and we were able to really, really go after stellar minority applicants. I mean, you know, I've always said, being a minority myself, that I am not interested in anyone who's not as good if not better than me coming to Duke. I always want them to be better than me and excel and be great, and I think we've done a really good job of attracting that type of person to Duke. That's what we want for all of our residents, to be better than we are, you know, so—

ROSEBERRY: So how does that committee do that?

WINN: Well, we meet once a month, sometimes more during the recruitment season, sometimes less in the slow season, but we put into place early special ways to try to recruit minorities who are really good. So we'll—we—one of our committee members meets with every minority who comes to interview for internship and residency here, and then we will decide who we really want to go after, and then we will just torture them. (*laughs*) No, I'm just kidding. We will just really spend a lot of time letting them know how interested we are, we bring them back for a second-look weekend. They usually have a dinner with minority residents who are here now, and then we just spend a lot of time trying to get them to come back. And it's really paid off. I think—you know, it's been challenging getting people to come to Duke for a lot of reasons, but things have changed a lot. And I think first of all geography is one thing; people just don't think much about Raleigh-Durham, and they don't know it's such a great area. It's a fantastic area for kids. I don't have any kids, but my colleagues (*laughs*) have them. It's a great area for raising children. And so that's one of our first issues, they want to go to a bigger metropolitan area. And then secondly there is this old myth about Duke being toxic for residents, and it—I think—I don't know where this myth started, but I think when I was a

resident here, we were all very proud to be here and very proud to get through the program and be very good doctors. So those are the two challenges I think with bringing people here. (*pause in recording*)

ROSEBERRY: (*unintelligible*).

WINN: Yeah, well, okay. Okay. So I've never—I've never thought it was toxic when I was here; I just thought it was challenging, and when you leave you're just—you're ready, you're ready for the world, and I'm proud of that. I think it's just your mindset and where you want to go to.

ROSEBERRY: Can I follow up just a little bit on that?

WINN: Sure. Sure.

ROSEBERRY: What did—what was the rumor? (*laughter*)

WINN: Well, it was just that Duke was really hard on its residents, and you work all the time, you're never at home.

ROSEBERRY: Sort of the work-life balance thing?

WINN: Right, and I guess when we became residents we never expected it to be (*laughs*) easy, you know. And it—I think there were some things that could have been better. Honestly, it just wasn't something we thought about, you just did what you had to do. And you got through your three years or four years, whatever you were doing—with me I did the extended time because of Med Psych—you just did what you had to do. Now things have changed quite a bit where I think people are trying to be more considerate of personal time, and then the ACGME [Acreditation Council for Graduate Medical Education] has changed a lot of rules so you don't have a choice (*laughs*) but to be more considerate of people's personal time. But Duke was already moving in that direction

before all of these rules. But it's still a matter of wanting to put out the best people you can into the atmosphere so—.

ROSEBERRY: Do you feel like you've been able to mentor residents?

WINN: I do. I mean, I think I've spent a substantial amount of my time mentoring. I'm meeting with someone on Friday who's a resident right now who wants to know what they do. I've always had a renal fellow in my lab pretty much who's been doing research. I think I'm on speed dial for some people and (*laughs*) not for others, but, you know, I think they know I'm a resource here if they need to talk, and I've helped people with everything from interpersonal relationships, relationships with attendings that they don't get along with and they can't understand why, to speaking to attendings who might have some misconceptions about a resident, to helping someone along their career path or talking to them about their new job at Duke as an attending and how they structure that job and what's going to be best for them versus—balancing that with what's best for Duke, that kind of thing, so yeah, I would say so, and I enjoy it. It's been eleven years now that I've been on faculty so I've been here a while.

ROSEBERRY: I'd like to ask you a little bit about your research.

WINN: Okay.

ROSEBERRY: If you could kind of tell me about what you're doing.

WINN: Sure. Sure. So that started in 1995 actually. I was going to do research with Laura Svetkey and Peter Conlon—who is from Ireland and who's since gone back to Ireland—said he'd found this interesting family in his clinic and I should look at it because no one's really studying this very much, and it was a family with hereditary FSGS which is focal segmental glomerulosclerosis, and it's a disease—it really affects

anyone. And if you've ever heard of Alonzo Mourning or Sean Elliott, they're both NBA basketball players, completely healthy, they both have FSGS, both have had kidney transplants because of it. It causes—about 20 percent of the people who are on dialysis in the country have FSGS, up to 20 percent or so. So by studying hereditary FSGS you can apply what you learn about the pathogenesis and the pathophysiology to the more common idiopathic kind which is what you would see more commonly in the dialysis unit. And so Peter had this family, and it turned out to be this large family with FSGS. A lot of the members—sisters, brothers, aunts, had kidney disease, a lot of them had had biopsies. So we gathered a pedigree, and then it was decided I would come to the Center for Human Genetics to learn human genetics with Margaret Pericak-Vance and Jeff Vance. So Peggy Vance was a statistical epidemiologist, Jeff Vance was a molecular geneticist: husband-and-wife team. Margaret was the chief of the center. And this is before we moved into this big, beautiful building. We were in like five or six different buildings because there were so many of us, and we couldn't all fit in one place. And so I came into the lab. Well, first in 1996 I went to New Zealand because we found another large family and we more fully ascertained them. And I went to New Zealand as a fellow and my chief, Tom Coffman, said, Don't ask for anything else (*laughs*) for quite a few years, because it was an expensive trip, especially in the nineties. And I collected blood on 100 individuals from a family with FSGS—huge family, one of the largest in the literature, if not the largest, with hereditary FSGS in the literature. And I brought their blood back and started working on doing linkage analysis. I started doing genetic studies on them including linkage analysis to look for the gene that caused FSGS in their family, and I think it was 1999 that we got the linkage. And I started out by myself in the lab

working in Jeff's lab but by myself because he was—he did neurologist things and I'm a nephrologist, but I learned from the people in this lab what to do. And then eventually there was grant money to support this, and I got a K08 which is a mentored-research scientist grant. So '99 we found the linkage to chromosome 11q21 to 23 and the LOD [logarithm of the odds] score was almost 10, D11 S 2000 which is a microsatellite marker, my favorite microsatellite of all times. (*laughs*) And then we spent the next few years looking through the region trying to find the gene. So we linked it to an area on a chromosome, then you have to look for the gene within that area on the chromosome. I'm talking millions and millions of bases. When I first started I think the gene was somewhere in 18 million bases and then over time I was able to narrow this down to 2.8 million bases.

ROSEBERRY: How did you do that?

WINN: You use more microsatellites, and we luckily had twenty-five people in this family who were affected, so we could narrow the region down. But if you have very few people who are affected, it's hard to narrow the region down because they're so alike genetically. These people—these twenty-five affected individuals were between 700 people apart, this family was so large at either end of the pedigree, so—(*pause in recording*). Okay. So then around 2003 we finally found the gene. This was during a time, late nineties, early 2000s the Human Genome Project was nowhere near finished, so often when we looked in one area or another it would change with each new freeze of the NCBI or the golden path. It's a dataset that shows you basically all the genes in an area, all the nucleotides in an area, and because they were still working out all the regions of all the chromosomes things would change with each freeze. So we sequenced a lot of

genes we didn't need to sequence because they were no longer in our area when we got the next freeze out like in eight months. It'd be a completely different area or things would be different, different genes in an area. Now it's very much worked out, much easier than it used to be. So we found this gene, TRPC6 which is transient receptor potential cation channel 6 which is an—it's a cation channel that allows sodium and calcium into cells. It makes a pore with four of them together. It can be TRPC3,- 6, or -7, and it inserts into the plasma membrane and allows calcium entry after it's somehow excited to do this, and that may be through angiotensin II or some other hormone or some G protein-coupled receptor. And this allows calcium entry to the cell. And what we think is wrong with this family's TRPC6 is that they have a mutation of a proline to a glutamine. Proline's important for protein folding. And so this proline is gone, it's highly conserved in evolution from guinea pig, dog, C. elegans, all the way down. It's a highly conserved proline, so that means it's very important, it needs to be there, and this family doesn't have that proline; they have a glutamine because of one nucleotide change. So what we believe is that this family's TRPC6 allows too much calcium into the cell. We're not talking serum calcium that is in your veins, you know, like I take a calcium tablet; we're not talking about that. We're talking about intracellular calcium which is very local to cells, and it regulates many, many cellular functions. We believe that their TRPC6 allows too much calcium entry, and we believe that in turn causes maybe apoptosis of the podocyte cell which is my favorite cell in the whole body, it's a beautiful cell. I'll show you a picture when we're done. (*laughs*) Causes apoptosis of the podocyte cells, or something that causes them to move or retract from the capillary and then thus they have proteinuria. Proteinuria sets up for other things, and they eventually

get end-stage kidney disease; they need to go on dialysis. So since that time—since that—and that was published in *Science* in 2005—I've been very, very lucky. I got the PECASE award, which is the Presidential Early Career Award for Scientists and Engineers, so got to meet President [George W.] Bush, and that was exciting going to the White House; my husband got to come, and I got the ASN [American Society of Nephrology] Young Investigator Award for this work, which was very much appreciated. And since finding TRPC6, we're now doing mouse models with TRPC6, so we're doing mouse models of human disease, trying to work out exactly how TRPC6 causes FSGS in this family. And now other investigators have found TRPC6 to be mutated in about eleven—nine, ten other families, so about eleven families total including ours have mutations in TRPC6, so it's becoming more common to find it now, and—

ROSEBERRY: Now that you know what to look for, is that—?

WINN: Yeah, well, you know to look at TRPC6 and then you just look for changes in the nucleotides to see if there are mutations in there. And then what's exciting about it is that it's the first gene of its kind to cause FSGS, whereas other genes that have been found to cause FSGS were cytoskeletal kind of genes like podocin, alpha-actinin-4, nephrin, PCLE1, all of the—all—I mean, well, PCLE1 less so, but these were all genes that—TRPC6 was the first of its kind, to be a cation channel to cause FSGS, so the good thing about that is it's druggable. You can find a molecular drug target and try to treat it. And it's also found to be upregulated in other diseases like diabetic nephropathy, which is actually much more common than FSGS, the like one or two cause of end-stage renal disease in the US.

ROSEBERRY: So this gene also—

WINN: Is upregulated, so if we try to block it maybe we can help people with diabetic nephropathy. Still a long way from any of this, you know, it's going to take a lot of time to understand—to work this out. And then the—and some other kidney disorders it's upregulated in so that you want to kind of beat it down, back into submission (*laughs*) so there's a lot of work to be done, but it's exciting work so—.

ROSEBERRY: Can you tell me about those possibilities for treatment?

WINN: Well, because it's a cation channel there are lots of things you can do, but you can put—you can make a drug that actually kind of stops up that channel and doesn't let calcium come in as much, or it causes the channel not to work properly and therefore you have less TRPC6 activation, less function I should say, and that may help decrease the amount of apoptosis or cell death that you would find. But like I said that's a long time away, and we're still trying to exactly understand how TRPC6 works, this mutation works. And others are trying to understand how their mutations work. We do know that it is a gain-of-function mutation and that it does cause an increase in intracellular calcium, but how that happens were still working on.

ROSEBERRY: Is that what your lab is doing currently?

WINN: Part. That and we're looking at how if you don't have TRPC6, if it affects you. So like if you try to block TRPC6 and you don't have it, is that going to hurt you as well as having too much or having it work too well? So we're working on a lot of things, a lot of different things—transcriptional regulation of TRPC6, a knockout model of TRPC6, you know, a transgenic overexpression model of TRPC6, and we're trying to make a knockin mouse model of TRPC6, so—

ROSEBERRY: Can you tell me—I'm not a scientist, so I just—

WINN: So the knockout mouse model which we've worked with most of all—and we're also looking at it in an Akita model, which is diabetes model as well right now but—. So a knockout model is, say if you were to stop it from working, does that hurt you, like I just said. So that's what the knockout model tells us, and we do various things to the mice like make them have proteinuria and see if you don't have TRPC6 is that beneficial as compared to wild-type mice. A transgenic overexpression model, what that does is we're overexpressing TRPC6 only in podocytes, and so only in this one cell, and we want to see if it causes proteinuria, if it's sufficient to cause proteinuria, which is what we measure how bad or good your kidney function is especially in regards to FSGS. The third knockin model is an exact recapitulation of the human disease, so exactly that one mutation is knocked in, in that one allele and the other allele is normal, and see if that is sufficient to cause proteinuria. And then lastly we're looking at this diabetes model, the Akita mouse model, and—because we found that TRPC6 is upregulated in diabetes. We didn't find, it but it's been found; it's in the literature now that it's been upregulated in diabetes. We want to know if not having TRPC6 and having the Akita diabetic model, if that's protected as compared to those that are wild type for that. So there's lots going on. We're also—I didn't even tell you that we have about 150 families with FSGS, so we're also looking for other genes that cause FSGS. We know that TRPC6 doesn't cause disease in those families so we're doing other linkage studies like I started out doing in '96, and we have idiopathic, so eventually one day we'll be able to do an association study with that. We also have—these were dominant—autosomal-dominant families but we also have recessive families so we're looking at them, so there's a lot going on. So I still do the human genetics part but also the mouse-model part.

ROSEBERRY: So tell me about an MD as a researcher.

WINN: Well, after just being on for a week (*laughs*) of rounding, it is difficult to juggle, honestly, but you know after all is said and done when I go to the hospital it seems like I dread it when it's coming up for me to round, but then when I go do it I love it. So like this weekend I was so excited about this patient. I was like, Oh my God, this is so exciting. I mean, it's not every day you get a pulmonary renal syndrome and get to try to work out if that's what it is, and it turns out they didn't have dysmorphic red cells when we spun their urine down, and it probably wasn't a pulmonary renal syndrome but still quite exiting; they need a biopsy today, a kidney biopsy. So it's difficult to juggle the lab and rounding, you know, and hopefully your chief protects you enough so you have enough time to work in the lab, because you really can't do more than 25 percent, 20, 25 percent clinical if you're going to be a physician-scientist, and especially if you're doing translational research like I'm doing, it's just impossible to spend enough time writing grants, writing papers to stay academically viable, you know. And my chief has been good about protecting me in that way. But it's busy, and you have to be very efficient when you're not rounding to make sure you get all of your work done. There are lots of things that are expected of you including not only writing grants and papers but reviewing papers. I'm going to be at the ASN this year moderating session, I'm doing the World Congress on Nephrology where I'm giving a talk, but I'm also the chair of the Abstract Committee for Molecular Genetics for the World Congress, and so I have to find people to review the abstracts along with me, and then I'll decide which abstracts get to stay in. So it's—there are lots of other things in addition to rounding and your lab that

you do as an academician but it's also the way that you learn and stay up to date in your craft with what other people are doing.

ROSEBERRY: Do you work with the DTMI [Duke Translational Medicine Institute]?

WINN: I have. Not so much directly, but they helped me with some statistics on a paper I just got published, so they've been very helpful, actually, yeah. I hope they stay around for a long time.

ROSEBERRY: Well, let me ask you about being a female in medicine. We talked a little bit about diversity, but I also wanted to ask about that form of diversity as well.

WINN: Hmm, you know, I don't see it as much of an issue. Frankly, diversity and being a female—being a minority and being a female has never been an issue for me. I mean, I just grew up in an environment where it just—I expect to be treated equally and therefore I am. I mean, you know if you don't, you will find out my wrath. I mean, I'm just telling you I just don't—I expect to be treated equally. And people understand—if they understand you take yourself seriously and you expect to be taken seriously, it's no big deal. And you will have the occasional person who's not nice, say something that's not nice to you. I've had patients call me names that aren't very nice. I don't take it— I'm not going to go and cry in a corner about it; it's just not my nature. You know, I mean, I just do something about it. What I do depends on the situation. I'm more likely than not if it's a colleague let them know exactly where I'm coming from, and then we're fine and we can move on and go about our business and take care of patients, and we're fine. If it's a patient that's a little more complicated, and I just say, You know, I just—you have to speak to me respectfully, and I will speak to you respectfully and we can go on from there. And frequently it's okay. Sometimes you have to get someone else involved

because they—you—they don't want you taking care of them, but it's so rare that this happens. It's really not a big part of my life, and I just don't let it become a big part of my life. My father was a marine, and he was in Korea and Vietnam; he was a lifelong marine. My mother was a nurse at a naval hospital. I'm from Camp LeJeune. And in our house growing up we had every ethnicity you can imagine in our house. It was more a matter of rank than ethnicity in our house. And I just didn't grow up in an environment where it mattered what you were, but then I went to—I went to the North Carolina School of Science and Mathematics and then Chapel Hill, UNC-Chapel Hill for undergrad. I just never experienced very much of it because I just didn't let it become a part of my life and it didn't get me down. That being said, then you go into some environments where you do see that there's a tendency not to be as accepting of other people, and so you just try to change it if you can. I mean, it—but— as far as women go, I think when I started medical school we were close to 50 percent women, and I've got to say I think there might have been two women in all of Nephrology before I started as far as fellows go. I think I was the second woman I think—no, the third, maybe; maybe the third. I was the first I think underrepresented minority to get the ASN Young Investigator Award in all of twenty-something years. There are lots of barriers still to be broken, but things have changed so dramatically since 1992, since 1988 when I went to medical school; things have just changed dramatically, but like I said I just don't see it—I just don't let it become a big part of my life. I just handle it and then move on, and if you let stuff like that drag you down you'll be sitting back and never going anywhere and you'll be sitting back looking at some situation where you're still steaming and fuming over it and you have a lot more to do, you know. And I think that's probably what some

people would like for you to do is sit there and steam over it instead of just move on and they're like, Hmm, well, that didn't affect her like I thought (*laughs*) it was going to. And you're like, No, it sure didn't. So—my parents always told me I was as good if not better than anyone else, I was as equal as anyone else, so that's what I went in expecting, and we just had a household like that where like I said, if you were a private you may not get a whole lot of respect, but if you're a staff sergeant or gunnery sergeant or whatever you might get more, you know; it's just different, so—.

ROSEBERRY: Well, what question did I not ask you today that you'd like to address or that I should have asked you?

WINN: Hmm, that's a good question. I guess I would just say the most defining influence on my life has been my parents, that they were the ones who were always, always there for me, and I was very lucky to have them, I'd say. I certainly wouldn't be where I am or who I am without them. I've lost both of them now but—but they did their job for me, and I've been very lucky about that. That's about it.

ROSEBERRY: Thank you so much.

(*end of interview*)