Richard D. Sontheimer: "inveterate curiosity" is the fundamental character trait of a "doctors' doctor"

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Editor's note

Despite occurrence over a small percentage of the population, rare diseases are often chronic and life-threatening. Around five new rare diseases are described in medical literature every week. The National Institutes of Health (NIH) Genetic and Rare Diseases Information Center lists autoimmune connective tissue diseases (CTD) such as lupus erythematosus (LE) and the idiopathic inflammatory myopathies as rare diseases. People with CTD will generally survive for at least 10 years after diagnosis, yet some can have a much worse prognosis (1). Attention to the special challenges faced by patients with rare diseases such as LE and dermatomyositis (DM) is constantly sought by the medical world. However, the market for new drug development for rare diseases is smaller than that for more common disorders. Thus, rare diseases as a rule attract considerably less research funding than more common medical conditions.

The skin is commonly targeted for autoimmune injury in both LE and DM. Psychosocially- and occupationally-disabling skin disease can result. Having been in the field of cutaneous manifestations of autoimmune CTD for over four decades, Dr. Richard D. Sontheimer from the Department of Dermatology at the University Utah Medical Center in Salt Lake City has been searching for ways to ease the pain of this particular group of patients, especially those with LE and DM. *Annals of Translational Medicine* (*ATM*) is honored to interview Dr. Sontheimer, acclaimed as one of the Best Doctors of America and America's Top Doctors, to share his insights into the current situation of his field, some recent breakthroughs in treating subacute cutaneous LE (SCLE), some recent research projects together with the findings and challenges encountered, and his genuine advice and encouragement to young physicians and researchers.

Figure 1 Dr. Richard D. Sontheimer.

Expert introduction

Richard D. Sontheimer, MD, is currently working as a full-time clinician educator in the Department of Dermatology at the University Utah Medical Center in Salt Lake City (*Figure 1*). He has been successfully involved in patient-oriented translational clinical investigation for 43 years at four USA academic medical centers starting at UT Southwestern Medical Center in Dallas in 1976. His career focus has been on the etiopathogenesis of the cutaneous manifestations of CTD, especially LE and DM. He was a member of the team lead by his mentor, Dr. James N. Gilliam, that first described in 1979 SCLE as a new photosensitive, non-scarring, immunologically-distinct cutaneous LE subset. Since then, Dr. Sontheimer has maintained a subspecialty academic focus on the evolving clinical concept of SCLE. This most recently has included clinical research contributions relating to drug-induced SCLE.

In addition, Dr. Sontheimer's work in the early 1990s focused needed attention on an orphaned cutaneous subset of DM—clinically-amyopathic DM (synonym DM sine myositis). That work has led to international recognition that patients with clinically-amyopathic DM are at

increased risk for developing potentially-fatal interstitial lung disease marked by the presence of circulating melanoma differentiation-associated protein 5 (MDA-5) autoantibodies.

Reflecting his long interest and focus in rheumatologic skin disease, Dr. Sontheimer was the lead co-editor of the first textbook dedicated exclusively to the skin manifestations of rheumatologic disease [*Cutaneous Manifestations of the Rheumatic Diseases*. First edition. Sontheimer RD and Provost TT, Editors. Williams and Wilkins, Baltimore, MD, 1996 (second edition published in 2004)].

In 2005, Dr. Sontheimer moved from the University of Iowa where he had served as the Chairman of the Department of Dermatology to the University of Oklahoma to continue his long-term research collaboration with the systemic LE research group led by Morris Reichlin at the Oklahoma Medical Research Foundation (OMRF). During his 5 years there, Dr. Sontheimer interacted with other members of this rheumatology research group including Drs. John Harley, Judith James, and Ira Targoff.

Dr. Sontheimer has previously held national and international professional leadership positions. Among these include: member of Board of Directors, society of Investigative Dermatology; medical advisory board member for the Lupus Foundation of America and The Myositis Association; Founding Presidents of the Medical Dermatology Society and the Rheumatologic Dermatology Society; International Organizing Committee member for the 1st–3rd International Conferences on Cutaneous LE and DM.

Prior recognition of his career contribution include: NIH Clinical Investigator Award, 1980–1983; NIH Research Career Development Award, 1987–1992; American Society of Clinical Investigation, 1989; American Dermatological Association, 1991; Best Doctors in America, 1992–2017; Association of American Physicians, 2001; Honorary Member, European Society of Cutaneous Lupus Erythematosus, 2004; Karolinska Institutet, Junior Faculty Nobel Forum Lecture, 2004; Arthur J. Rook Oration, 86th Meeting of the British Association of Dermatologists, 2006; Lifetime Achievement Award, Medical Dermatology Society, 2013. Lifetime Achievement Award, Lupus Foundation of America, Utah Chapter, 2016; Masters Recognition, American College of Rheumatology, 2017.

Interview

ATM: What do you think are the critical issues facing the field of cutaneous manifestations of autoimmune CTD?

Dr. Sontheimer: Inadequate research funding for rare/orphan diseases. The greatest challenge in this area has been the fact that all CTD having skin manifestations are uncommon enough to be classified as rare/orphan diseases. Rare disease status in the USA is a considerable deterrent to progress in better understanding the underlying causes of such disorders. Unless a clinical disorder affects ≥2% of the population, pharmaceutical companies typically do not focus attention on such diseases due to the high costs of drug development and approval in the USA. This results in delaying the identification of specific therapeutic strategies for minimizing the clinical impact of rare diseases.

An example is belimumab (Benlysta) which was the first new drug approved for the "rare" disorder, systemic LE, in the past 50 years. By comparison, nine systemic biologic drugs have been approved in the USA for the more common condition, psoriasis, over the same time frame. Without Food and Drug Administration (FDA) approval, commercial insurance companies in the USA often refuse to pay for expensive new targeted therapy for rare/orphan clinical disorders such as cutaneous LE, cutaneous DM and cutaneous manifestations of morphea/systemic sclerosis.

Complicating this is the fact that NIH research funding in the USA for common and rare clinical disorders has steadily fallen in real, inflation-adjusted terms over the past 50 years. This has presented considerable challenges to the patient-oriented translational clinical investigator career pathway, especially to those studying rare/orphan diseases.

High costs of modern medical therapy. During my four-decade career as a practicing physician, I have witnessed miraculous progress in the cellular, molecular and genetic understanding of the etiopathogenesis of human autoimmune diseases such as LE and DM. Such understanding has led to the development of targeted (and thus safer) drug therapies that focus on critical checkpoints in the cellular pathways of autoimmune tissue inflammation and damage. Also, during my career, the cost of health care in the USA, especially the cost of new targeted medications, has skyrocketed far beyond what inflation alone would justify.

The high production costs of modern recombinant biologic drugs seem to have triggered a reverse price war

within the USA pharma industry. This pharma feeding frenzy threatens to deprive many USA citizens of the clinical benefit of the past half-century of progress in federally-funded medical research. This is especially true for citizens who suffer from rare/orphan diseases. USA medical insurance companies often deny a physician's request to treat a rare/orphan disease patient with a biologic product that has not been FDA approved for the rare/orphan disease but has been FDA approved for a different more common, and thus, a more profitable disorder.

ATM: In a recent study, you hypothesized that long-term aminoquinoline antimalarial therapy with hydroxychloroquine (HCQ) could provide idiopathic inflammatory myopathy (IIM) patients with an atherosclerotic cardiovascular disease (ASCVD) comorbidity benefit. How did you come up with this hypothesis?

Dr. Sontheimer: A clinically efficient way to control LE skin inflammation is with oral aminoquinoline antimalarial therapy. Since intermittent/recurring skin changes are one of the most common clinical manifestations of systemic LE, such patients in the past were being treated with aminoquinoline antimalarial therapy for long periods of time for their skin changes. This led to the incidental clinical observation that long-term antimalarial therapy could have beneficial effects on some of the systemic manifestations of LE such as the rate and severity of systemic LE disease flares. As more systemic LE patients on long-term antimalarial therapy were observed, it was realized that the comorbidities of systemic LE such as premature ASCVD were mitigated compared to those systemic LE patients not on long-term antimalarial therapy.

Oral antimalarial therapy can also be of value for the inflammatory cutaneous manifestations of DM, although less so than for the cutaneous manifestations of LE. As an academic "immuno-dermatologist" based in tertiary academic referral medical centers in the USA throughout my career, I was frequently called upon to care for patients with cutaneous DM that had been unresponsive to first- and second-line dermatologic therapy. My initial treatment in such patients with respect to systemic therapy was the aminoquinoline antimalarial drugs since they do not significantly suppress the immune response with long term use. If I could not control the patient's skin inflammation with HCQ, a combination of HCQ and quinacrine might be successful. And when this first antimalarials combination did not help, a combination of chloroquine plus quinacrine might. It has been my experience that the majority of the otherwise treatment-refractory cutaneous DM patients that I have seen over the past 40 years could be helped with single agent or combination aminoquinoline antimalarial therapy.

Comorbidities such as premature ASCVD have become a major cause of morbidity and mortality in modern systemic LE patients. As previously mentioned, long-term oral HCQ therapy has been recognized to have a mitigating effect on systemic LE comorbidities. Recent evidence suggests that DM patients might be at risk for same comorbidities that systemic LE patients experience. This realization plus my long-term experience in using antimalarial therapy for skin inflammation in DM patients led me to the hypothesis that long-term antimalarial therapy might have a modulating effect on comorbidities such as ASCVD in DM patients (2).

ATM: What were the findings of this study? What insights does it bring to guide future studies in similar areas?

Dr. Sontheimer: The previous question did not relate to a hypothesis-driven research study resulting in specific findings. Instead, it was the formulation of a clinical hypothesis concerning one rheumatologic disorder, DM, based on an extrapolation from the existing literature on another rheumatologic disorder, systemic LE.

The hypothesis-driven scientific method of systemic research has served as the foundation upon which the modern Western health care enterprise has been built. However, the "medical hypothesis" article genre of medical/scientific publication has traditionally received far less attention. Full-time clinical practitioners in the USA have little opportunity to participate in systematic research within their areas of clinical interest. However, all of these individuals are intelligent and dedicated, and many have continued to nurture the character trait of curiosity that initially led them to pursue a career in science and medicine. Such individuals often keep abreast of what's going on scientifically in their various areas of clinical interest. Thus, being conversant in both the clinical and scientific worlds, they are in a unique position of being able to guide thought in their areas of focused clinical interest without having the ability to test such hypotheses in a systematic fashion.

Medical hypothesis publication gives such individuals an outlet for their creative thought. It has been said that the original thought of many breakthrough areas of science and medicine were initially presented in a medical hypothesis publication format. In addition, medical hypothesis

publication provides mid-career and late-career academic medical faculty who are no longer involved in NIH-supported research a platform for continuing to share the results of their intellect and experience with the broader scientific and medical communities.

ATM: Are there any breakthroughs in the treatment of SCLE over the past decade?

Dr. Sontheimer: There are two areas that I would consider significant in this respect.

(I) Identification of new classes of drugs that can induce SCLE. The only thing unique to SCLE has been the realization that the menu of drug classes that are now recognized to be capable of inducing the clinical expression of SCLE is much larger than we had initially imagined. Our more recent published literature review of drug-induced SCLE identified several new drug classes as triggers for SCLE including chemotherapeutics, protein pump inhibitors, antibiotics, recombinant biologics and other immunomodulators (3).

It is difficult to understand how a single pathomechanism might account for the structurally different drug classes that have been reported to trigger SCLE. One can argue that there are at least two pathogenetic subtypes of drug-induced SCLE.

A large percentage of the conventional, small-molecule drug classes that were initially recognized to be capable of triggering SCLE possess one or more unsaturated hydrocarbon ring structures. The designations "aromatic hydrocarbons" and "aryl hydrocarbons" are used interchangeably to refer to this class of chemical structures. Examples include calcium channel blockers, NSAIDs, ACE inhibitors, beta blockers, some antibiotics and thiazide diuretics, the original drug class reported to be capable of triggering SCLE. Photochemistry occurs in human skin when aromatic hydrocarbon ring structures absorb ultraviolet light. The pro-inflammatory results of such photochemical activation might possibly induce SCLE skin lesions via the isomorphic response of Koebner in an individual harboring underlying "sub-clinical" Ro/Sjögren's syndrome A antigen (Ro/SS-A) autoimmunity.

Large molecule recombinant biologic drugs and other immunomodulators might precipitate SCLE via different mechanisms such as disturbance of immune tolerance or direct activation/stimulation of pro-inflammatory signaling pathways not involving ultraviolet irradiation.

(II) Upregulation of class I interferon signaling in lichenoid skin disorders including lupus-specific skin disease. The most important new insight in this area has been the confirmation that the genetic signature of lupus-specific skin disease (acute cutaneous LE, SCLE and classical discoid LE) is the same as that for the systemic manifestations of LE—upregulation of class I interferon signaling. In addition, recent studies have suggested that interferon-beta may be the key pro-inflammatory driver of both cutaneous and systemic LE inflammation. This has led to ongoing examination of targeted therapy as a new therapeutic approach for refractory lupus-specific skin disease. Clinical trials are currently underway examining a recombinant antibody that specifically neutralizes interferon-beta activity as well as a different recombinant antibody that binds to and blocks the class I interferon receptor.

ATM: Would you introduce us to a recent NIH-funded research project that you are involved in (e.g., your role in it, scope, objective, research direction and current status)?

Dr. Sontheimer: I received a NIH Clinical Investigator Award, and following that, a NIH Research Career Development Award, in support of my early investigative career work focusing on the etiopathogenesis of lichenoid skin disorders. At the time of death of my mentor, Dr. James N. Gilliam in 1984, I was the Co-Investigator on his long-running NIH R01 grant entitled "*Mechanisms of Cutaneous Injury in Lupus Erythematosus.*" After his untimely death, I assumed the role as Principal Investigator on this grant and successfully renewed it on a competitive basis for several five-year funding cycles ending in 2004. I have not been actively involved in NIH-funded research since then.

Mid- and late-career patient-oriented translational clinician investigators in the USA can have difficulty maintaining their NIH research grant funding. Because of the success in their early career research endeavors, they are expected to volunteer their time reviewing NIH grant applications submitted by other investigators. An individual's commitment to an NIH R01 Study Section can include attending Study Section grant review sessions held in the Washington, DC area three times per year for a four-year term. There is a considerable amount of grant

review preparatory work at home required before each of these committee meetings.

In addition, mid-career clinical investigators are often called upon to assume other academic roles within their home institutions such as administrative and teaching leadership positions. Successful clinical research faculty often become clinical champions within their area of subspecialty clinical interests. Along with this goes the demands of increasing patient care and post-graduate teaching responsibilities. Six challenging years trying to restructure a struggling, near-bankrupt USA academic dermatology department as its new chairman contributed to the loss of my NIH-research funding in the early 2000s.

These distractions from their research interests along with the fact that physician investigators are required to compete with fulltime PhD scientists head-to-head for NIH research grant funding, account for the winnowing of research productivity of patient-oriented translational clinical investigators during their mid- and late-careers.

During my career, an alternative training pathway was developed for students who were interested in pursuing both medical and scientific research careers—The Medical Scientist Training Program. Formalized by the NIH in 1964, this program supports a training tract resulting in both MD and PhD degrees that typically requires 8 years to complete. It takes a rather special individual to add the extra four years required to complete a PhD training to the time required to complete medical school (four years) and the time to complete a clinical residency (typically a minimum of four years). The Medical Scientist Training Program has produced some truly outstanding medical scientists. However, to remain competitive for NIH funding such individuals typically have to devote 80% of their time and effort to research leaving relatively little time for patient care and teaching interests.

Caveat: it should be noted that my responses in this interview should be viewed through the lens of a curious, retirement-aged clinician investigator who some would say only dabbled in true, reductionist science during his career. My patient-oriented translational clinical investigation research training occurred during a two-year research fellowship based within a clinical department of dermatology rather than through a formal PhD. training program. Today, it would be very difficult for someone with my research training background to successfully compete for NIH reductionist research funding with those having completed MD and PhD degrees in a Medical Scientist Training Program.

ATM: Did you come across any bottleneck in your career/ research? What has been the driving force for you to overcome these challenges and move forward?

Dr. Sontheimer: I encountered three bottleneck issues during the various phases of my academic research career, some of which could be resolved and others not.

(I) Early career—lack of experimental animal models of lupus specific skin disease. When I started my research career on the immunopathogenesis lupus-specific skin disease, there were no viable experimental animal models for this pattern of inflammatory skin injury. We were limited to experimental work on clinical samples taken from human patients with cutaneous LE. This was a major roadblock for us as working experimentally with human tissue samples alone has many drawbacks. It was only when gene knock-out and knock-in technology became generally available in the 1990s that reliable experimental animal models of the lichenoid/interface dermatitis pattern of human cutaneous LE inflammation could be developed.

(II) Middle career—human calreticulin's relationship to the Ro/SS-A autoantigenic ribonucleoprotein particle system. In the mid-1980s, our research work examining the molecular identity of the Ro/SS-A autoantigenic ribonucleoprotein particle system led to the cloning and sequencing of human calreticulin. Calreticulin had previously been identified by other workers in non-human mammalian species as being a high-affinity calcium-binding molecular chaperone that was thought to reside exclusively within the lumen of the endoplasmic reticulum. However, our studies showed that human calreticulin could be precipitated from extracts of a human lymphoblastoid B cell line (Wil-2) by autoantibodies present in systemic LE patient serum specimens that were known to be mono-specific for Ro/SS-A autoantibodies by Ouchterlony double–immunodiffusion. This form of human calreticulin had an apparent molecular weight of 60 kDa on SDS-page analysis.

Thus, at that time we assumed that we had identified the major polypeptide component of the human Ro/SS-A ribonucleoprotein particle system which was previously known to be a 60 kDa polypeptide. This led to a controversy in the field concerning the relationship between calreticulin

and the Ro/SS-A ribonucleoprotein particle system, the basis of which has never been fully been resolved. The interested reader is referred to a recent, more in-depth earlier discussion of this issue by the author in this journal (4). I feel that this bit of scientific controversy negatively impacted my ability to continue to be successfully in competing for NIH research funding.

(III) Later career—academic clinical department leadership. Always lurking in the back of my mind was the thought that at some point clinical department leadership might be in my destiny. After looking at several earlier opportunities to move in that direction, I accepted an appointment as the Chairperson of the Department of Dermatology at the University of Iowa School of Medicine in 1998.

However, the next five years would prove to be the most difficult, frustrating and challenging years of my professional life. Not everyone has that rare combination of talents needed to successfully manage the diverse aspects of that middle management job within a large healthcare system. I found out through experience that I was not one of those individuals. However, other patient-oriented translational clinical investigators have gone on to successfully lead USA academic clinical training programs over several decades while remaining productive in their areas of research interest.

ATM: Having been involved in the study of cutaneous manifestations of autoimmune CTD for decades, how did you become involved in this line of research at the very beginning?

Dr. Sontheimer: As a medical student at the University of Texas Southwestern Medical School in Dallas, Texas in the late 1960s, I became intrigued by the potential application of the new insight that was being gained at that time into the human immune response in treating autoimmune inflammatory diseases such as LE and DM. After completing an internal medicine residency at the University of Utah Medical Center in Salt Lake City in 1976, I returned to UT Southwestern in Dallas for a Dermatology Foundation-supported Immunodermatology Research Fellowship in Dr. James N. Gilliam's lab in the Division of Dermatology.

Not having had prior laboratory research experience, it was with some trepidation that I began working on a project examining the clinical significance of the non-lesional lupus band in the New Zealand Black (NZB)/New Zealand White (NZW) F1 hybrid mouse model of systemic LE disease activity and damage. This and several other projects that I became involved in over the following two years yielded interesting, publishable results, including the initial description of the "SCLE" subset. At that time, I transferred to the UT Southwestern Dermatology Residency Program for my clinical training with the intent of pursuing an academic dermatology career once I had become board-certified.

I was invited to join the UT Southwestern Dermatology Faculty in 1979 with the intent of continuing my research interests in collaboration with Dr. James Gilliam. Following his untimely death from cancer five years later, I became the principal investigator on his NIH R01 grant entitled, "*Mechanisms of Cutaneous Injury in Lupus Erythematosus*."

ATM: As one of the best doctors of America and America's Top Doctors, what makes a good doctor in your opinion? What would be your advice to younger doctors/researchers who would like to become successful in your field?

Dr. Sontheimer: I believe that various aspects of character need to come together either by design or chance for a young person to ultimately become recognized as a "doctor's doctor" by their peers. But first, we should discuss the different ways that the designations "best doctor"/"top doctor" are currently used in the USA.

Over the past three decades, a number of physician-rating agencies have appeared in the USA, two of which you have referred to in your question above. It has recently been reported that there are at least ten such agencies now based in the USA.

Each agency employs its own proprietary rating systems for physicians. Some agencies employ peer-physician polling to identify their "best doctors"/"top doctors". In the peer-physician polling approach, physicians are randomly contacted in a community by the agency and asked that if they or a family member developed a specific medical problem, which doctors in that community would they want to have treat them. Other agencies allow physicians to nominate themselves for inclusion in best doctors/top doctors categories. Such physicians can then use this "award" distinction to market their practices to new patients. Each type of these agencies appears to monetize the results of their physician rating system on a for-profit basis either directly or indirectly.

For a more objective approach to identifying "doctors' doctors", one can look for physicians who have received awards from their non-profit professional societies. Such awards that I have received during my career for which I am most grateful include: election to Membership in the American Society of Clinical Investigation, the American Dermatological Association and the Association of American Physicians; Career Achievement Awards from the Medical Dermatology Society and the Utah Chapter of the Lupus Foundation of America; and "Master" designation by the American College of Rheumatology.

Perhaps the most fundamental character trait that a young person destined to become a doctors' doctor should have is what I would refer to as "inveterate curiosity". A child who is curious and who stays curious as they move through life will be better able to master the many educational challenges that a physician must continually overcome. And, the curious practicing physician who relishes (rather than just tolerates) the ongoing requirement for continuing medical education throughout their career will be able to better provide their patients with the most up-to-date medical care. In addition, a curious practicing physician is in an ideal position to recognize and call attention to new disease states, a previously unrecognized disease variant or a novel new treatment approach for an existing disease. And, it is my belief that ongoing curiosity about disease states and the optimal delivery of medical care can somewhat insulate a practicing physician from the monotony of the day-by-day aspects of medical care delivery. Ongoing clinical curiosity might provide some degree of protection from physician burnout. While curiosity might have killed the proverbial cat, it could save a medical career.

Dr. Faith T. Fitzgerald made some very interesting points on curiosity, physicians and medicine in her essay entitled "Curiosity" that appeared in the *Annals of Internal Medicine* in 1999 (5). In this essay she relays a wonderful quotation by a colleague, "*Dr. Erich Loewy, in an unpublished paper, points out that curiosity, the primal 'wonderment' that stimulates exploration, engages both imagination (conceiving the alternative explanations of new phenomena) and intelligence (mapping out the best way to delineate which explanation is likeliest). Both imagination and intelligence are integral to humanities, science and the synthesis of the two, which is clinical medicine.*" Dr. Erich H. Lowey has commented separately on the importance of curiosity, imagination and compassion in both science and ethics (6).

Dr. Fitzgerald went on in her essay to write, "*What does curiosity have to do with the humanistic practice of medicine? Couldn't it just convert patients into objects of analysis? I believe that it is curiosity that converts strangers (the objects of analysis) into people we can empathize with. To participate in the feelings and ideas of one's patients—to empathize—one must be curious enough to know the patients: their characters, cultures, spiritual and physical responses, hopes, past, and social surroundings. Truly curious people go beyond science into art, history, literature, and language as part of the practice of medicine. Both the science and the art of medicine are advanced by curiosity.*"

As a dermatology residency program director at three USA medical schools during my career, I often struggled to distinguish between numerous outstanding medical students and young physicians who wanted to pursue career training in dermatology. I often thought that a quantitative index of curiosity might help me to better select applicants for such training positions. There has been some discussion about the need for an objective measure of curiosity as a personality trait (i.e., a "curiosity quotient"). However, to my knowledge the "curiosity quotient" concept remains factitious. It is my understanding that there is currently no existing accredited psychological testing mechanism that can quantify innate curiosity.

No matter how brilliant, creative and capable a doctor is, she/he will not be recognized as a doctor's doctor if they have poor interpersonal skills. The ability to put patients at ease will always make the medicine go down easier. Kindness in a relationship can be contagious. The doctor's doctor must be a good listener and be willing to find the time to listen. The doctor's doctor is able to convey honest empathy and caring. This all boils down to the Golden Rule. How would the doctor want to be treated if their relationship with the patient was reversed?

Doctors' doctors must be trustworthy. There are a few simple things that one can do when first interacting with a new patient to facilitate putting them at ease and gaining their trust. Consider washing your hands in a manner visible to the patient upon entering the exam room. Introduce yourself by name and professional title to the patient. Make an attempt to pronounce the patient's name. Acknowledge the presence of any family member(s), friend(s), personal medical assistants or translators who have accompanied the patient. If for only a brief time, find a way to sit down so that you can interact with the patient on the same eye-to-eye level. This can magnify in the patient's mind the amount of time you have spent with them. And always find a way to touch the patient. This can logically be done during a physical examination or simply as a gesture of

respect by bumping fists or shaking hands with the patient at the end of the clinical interaction. These simple measures can serve to put patients at ease and set the stage for a more humanistic interaction. I have always been an adherent to the Golden Rule philosophy of treating others as you would want to be treated as being a highly effective way of interacting with anyone I have met for the first time.

Another fundamental character trait of doctors' doctors is the willingness to serve as the "physician-of-last-resort" for a sick human being. A doctor's doctor is one who must be perceived as being the patient's advocate within the bureaucratic morass of the of the modern Western approach to health care delivery. However, this has become increasingly challenging for modern physicians in the USA who now often work within the framework of large healthcare systems in which non-physicians are often in positions of power over how health care is delivered.

I feel that another character trait of someone on a "doctor's doctor" career pathway is the need to be recognized for their contributions. It has been said that the need to be recognized is the cornerstone of self-esteem. All human beings need to be recognized for their inherent value. However, some might need to be recognized more than others. And still others might need recurring recognition to the point of obsession.

Those affected by a "recognition" obsession are unlikely to have the tools for successfully overcoming the rigors of professional training for a life in medicine. So perhaps it is those curious practicing physicians who have a strong but manageable need for recognition that go on to become doctors' doctors. Those who distinguish their accomplishments by publication in peer-reviewed medical journals become the most widely recognized and acknowledged as doctors' doctors.

All learning dies if held in silence. The passing of accumulated knowledge and wisdom from one generation of physicians to the next is a primal organizing principle of medicine. Therefore, it is not surprising that the word "doctor" is derived from the Latin word "docere" meaning to instruct, teach or point out. In addition to bedside teaching, medical teaching can be distributed widely through publication. Thus, it is not difficult to understand why many of the acknowledged doctors' doctors are based within academic medical institutions.

The doctor's doctor must have the ultimate respect for the scientific process but learn to cast a critical eye on undocumented claims of miracles. While not perfect, the scientific process is the only light we have burning in that dark sea of guesswork, anecdotes, snake oil and charlatanism. And doctors' doctors should have a firm understanding of recognizing and minimizing medical errors.

I would also say that the curious young doctor should do her/his best not to become financially overcommitted whenever possible. Large debts accrued early in one's professional life will lessen one's options as one's career matures. And it is important to remember that there are different ways of getting paid professionally. For some it is money alone. For others, it can include protected time to think, explore, learn, teach, travel, write.

And, the nascent doctor's doctor should not ignore her/his personal life. A supportive partner and family will always be a cornerstone component of the foundation of a successful professional life.

My advice to the young physician who might want to pursue a career of medical curiosity is to get the very best foundation in clinical knowledge and research training possible in one's area of medical interest. Ideas still come from individuals. However, idea-supporting or idea-opposing scientific research is carried out these days by teams, not by individuals. Team work is essential to moderate medical discovery.

And finally, seek out a compatible and supportive Mentor early in one's career. It is not surprising that many doctors' doctors have studied under the preceding generation of doctors' doctors. My Mentor, Dr. James N. Gilliam, who was the consummate doctor's doctor had an enormous impact on my personal career direction in medicine and the later success of that career path (4).

The career path of a doctor's doctor can be a long and arduous one. If one were to set the goal of becoming a doctor's doctor at the very beginning of medical school, it could be very daunting. I certainly did not start my medical career that way. I was more of a "short-term-goal" kind of person.

As an insecure youth, my first goal was to try to successfully complete a University pre-med degree. After achieving that, with increasing confidence I set my next goal of getting into medical school. Next it was to complete an internal medicine residency. After that, it was to pursue a specialty career path in dermatology. This "follow-your-nose" philosophy plus a bit of good fortune and a dash of growing confidence led me to pursue a doctor's doctor career path. Always remember that hard-work and good fortune tend to travel together.

"*Diligence is the mother of good luck*."—Benjamin Franklin.

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Footnote

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