Elwood V. Jensen

A Conversation with Elwood Jensen

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VIDEO

Please visit **http://www.annualreviews.org/r/Jensen_interview** for a video of this interview.

INTRODUCTION

Elwood Jensen first described the estrogen receptor in 1958, opening up a new field of hormone action (1). This field expanded along with the nuclear receptor superfamily to incorporate the actions of a wide and still growing range of biological regulators that includes diverse steroids, thyroid hormone, retinoids, bile acids, and even heme. The 48 members of this nuclear receptor family encoded in the human genome impact nearly every facet of our biology. Elwood is a member of the National Academy of Sciences and was a recipient of the 2004 Albert Lasker Basic Medical Research Award "for the discovery of the superfamily of nuclear hormone receptors and elucidation of a unifying mechanism that regulates embryonic development and diverse metabolic pathways." At the Lasker Award ceremony, Nobel laureate Dr. Joseph L. Goldstein described Elwood as "the patriarch of the field" on the basis of "his pioneering work on the estrogen receptor, the matriarch of the superfamily" (2).

In the summer of 2010, the *Annual Review of Physiology* gave me the opportunity to discuss with Elwood this seminal discovery, along with other aspects of his long and illustrious career. As he describes in the interview, his work completely overturned the conventional view of hormone action at the time. It now seems quite odd that the prevailing "wisdom" was that estradiol acted as an enzyme cofactor in redox reactions that resulted in the transfer of hydrogen from NADH to NADPH (3). Elwood and his postdoctoral fellow Herbert Jacobson used tritium gas to reduce a double bond in an appropriate precursor, generating estradiol labeled to very high specific activity. When they administered physiological doses of this tracer to immature female rats, they found that it was not chemically altered, as predicted by the then current models, but was instead specifically retained in known estrogen target tissues such as the uterus. Elwood correctly deduced that the tracer was held there by a specific protein, which he termed estrophilin and which we now know as the estrogen receptor. Subsequent work by Elwood and colleagues (4) and also the late Jack Gorski and colleagues (5, 6) indicated that estrogen binds the estrogen receptor in the cytoplasm, and the complex then moves to the nucleus. Bert O'Malley was the first to clearly demonstrate that estrogen and progesterone act in the nucleus to induce specific messenger RNAs (7).

Elwood went on to purify the estrogen receptor and obtained polyclonal as well as monoclonal antibodies (8, 9). In collaboration with Pierre Chambon, Elwood used these antibodies to isolate the initial estrogen receptor cDNA clones (10), a major step in the elucidation of the nuclear receptor superfamily. Elwood and colleagues also contributed significantly to the development of diagnostic measurements of estrogen receptor in breast cancer specimens and to the development of estrogen receptor antagonist therapies (11). Thus, Elwood's discoveries not only unraveled fundamentals of molecular endocrinology but also led directly to major advances in breast cancer diagnosis and therapy.

As Elwood recounts in the interview, as well as in his Lasker Award essay (12) and other reminiscences, his pioneering studies benefited from what he terms an "alternative approach." He illustrates this with his remarkable story of ascending the Matterhorn, despite his complete lack of mountain climbing experience, and of only later learning that it had been the last major European peak to be scaled (**Figure 1**). In retrospect, as is often the case in science, what had appeared to be the most straightforward route was actually not simple at all, but insightful analysis revealed the feasibility of a seemingly intractable alternative. Edward Whymper was the English climber who deduced that the seemingly sheer northeast face could be climbed. Elwood's insightful alternative approaches to studying estrogen action allowed him to accurately follow the fate of the very small amounts of the hormone required for physiological responses and to identify, and later characterize, the estrogen receptor.

Figure 1

The genesis of "alternative approach": the Matterhorn ascent. In the top left of the photo, taken from Zermatt, the starting point of the final phase of Elwood Jensen's Matterhorn climb is the steep Swiss face, which meets the more moderate Italian side at the peak. Until the British climber Edward Whymper climbed the Matterhorn via the steep Swiss side, all previous ascents to the peak had been attempted from the Italian side. Jensen followed Edward Whymper's alternative route to the summit. This also inspired Jensen to try alternative approaches in his research that led to the discovery of the estrogen receptor. Shown in the picture are Mary, Jensen's late wife, who accompanied her husband up to this last section of the ascent, and Kyle Packer, the student from Colorado who had arranged for the guide, whom they were to join in Zermatt.

The George and Elizabeth Wile Chair for Cancer Research, Elwood was 90 years old when I interviewed him at the Vontz Center for Molecular Studies at the University of Cincinnati College of Medicine on July 19, 2010. He was recruited there in 2002, remaining scientifically active, as demonstrated by his description, with collaborators Sohaib Khan and Thomas Burris, that the estrogen antagonist tamoxifen can bind to two distinct sites on the receptor (13, 14). Elwood had suffered some health problems, including being hit by a truck, but was getting around on an electric scooter and was full of energy and very sharp during the interview. In fact, after my somewhat

garbled first question, I barely got a word in for the rest of the 38-min interview. It is appropriate to clarify some of the events that he referred to in the period around his discovery of the estrogen receptor, however. The meeting in Vienna where Elwood first presented his major discovery on the estrogen receptor to the three other speakers plus two additional attendees was in 1958. The progesterone receptor work by O'Malley and colleagues (15) that he mentions was published in 1970. John Baxter and colleagues' 1975 description (16) of chromatin binding properties of the thyroid hormone receptor was based on earlier studies by Oppenheimer and colleagues (17) demonstrating the existence of such a receptor. Ron Evans, who shared the Lasker Award with Elwood and Chambon, began his postdoctoral fellowship with James Darnell in 1975. But in 1986 Evans and colleagues (18), along with Bjorn Vennstrom and colleagues (19), showed that the cellular proto-oncogene c-*erbA* encodes the thyroid hormone receptor.

It was a thrill and a privilege to be able to talk with Elwood about his work and his life and to be able to share it with you. I hope that you enjoy the interview, too.

INTERVIEW AT THE VONTZ CENTER FOR MOLECULAR STUDIES AT THE UNIVERSITY OF CINCINNATI COLLEGE OF MEDICINE, JULY 19, 2010

Moore: So, Elwood, it's a great pleasure for me to have a chance to have a chat with you today. I thought that maybe we would get started if you could tell us how things were in terms of hormone action, what people were thinking about estrogen and other hormones as to how they worked, and the problems that were being addressed and your point of view towards them.

Jensen: Well, in the 1950s, that was the big era of enzymes. Biochemistry was largely endocrinology, and transcription, RNA synthesis, and that didn't come in for another ten years. So the current thinking of steroid hormones, estradiol, the principal one, was that it had to be reacting with enzymes. And the conventional model that they had is that the 17-hydroxy group of estradiol was oxidized by enzymatic oxidation using one coenzyme, and then with that it was oxidized to a ketone group to estrone, and then it was reduced by the oxidized form of another coenzyme, therefore—thereby transmitting or translocating a reduced hydrogen from one form to a reduced form of what was then called NADP. And that was known to be an important factor in many biosynthetic reactions.

And coming into the field and having joined the Department of Surgery at the University of Chicago, and Dr. Huggins, who had formed that in the middle '60s and brought in a couple of, two or three, young people to join it. He was an urologist who won the Nobel Prize for work on prostate cancer, but he was interested in breast cancer and estrogens too. And he showed me how very tiny amounts of estradiol—say, in a test animal, such as the rat, you could make the female reproductive tract grow to six times or so its size in the immature animal or the castrated animal. Then everybody thought it had to be enzymes.

So that fascinated me. How could such a tiny amount of an organic compound cause such a remarkable growth response? And as I said, the prevailing theory then was that it was oxidized by one coenzyme reduced by another, but then on the other hand, you had a synthetic estrogen that was diethylstilbestrol, which could not be oxidized. It had no aliphatic hydroxyl groups. That's a perfectly good hormone.

So we said, well, maybe we should attack this from a different angle. A little background for all of this was my experience there in 1947 in Zurich learning about steroid chemistry with Professor Ruzicka, who won the Nobel Prize and was head of biochemistry there in the so-called ETH, Eidgenössische Technische Hochschule, in Zurich. While I was there, I promised my late wife of 41 years that I would not try to climb any mountains because I had no experience. So when I

came to Switzerland, I saw the Matterhorn standing all alone. And most peaks are just a little bit of elevation along a ridge of mountains, but the Matterhorn stands all by itself regally there, with the northeast face facing Switzerland and looking like a sheer wall of rock. It was the last major mountain in Europe to be climbed.

So I saw it, and I was approached by a student from Colorado, a young man who was getting his PhD there in Zurich, and he was an expert climber. He had lined up a guide to accompany him. You're allowed to have two guides to climb there now. Used to be you didn't have [to]—and in the first climb finally when someone climbed the Matterhorn, four out of seven people were killed.

So the law requires no more than two climbers to a guide. They wanted someone else to share the cost of the guide. I was in good physical shape from tennis and boxing and judo. I was fascinated by the mountain, so my wife released me from my promise, and we started out at 6:00 in the morning to get to the top before the sun melted the snow and made landslides.

It was the hardest physical thing I ever did in my life because I had no experience—although I was in good physical shape, but the atmosphere was rather oxygen free up there. So we got to the top about 8:00 in the morning, starting out early when it was still dark. I was going to stay—I had to stay up there for a while, and no one was there. And after a half-hour, the guide said: No, we have to start back. I'm glad we did because on the way down, where we'd been about an hour before, there was a landslide, and two days later two people were killed in such a landslide. So a long-winded background here. This idea of—I got ahead of myself a little bit. So it was the—when we got back I started reading a little bit about the history of the Matterhorn, and I learned that it was the last mountain to be climbed.

I wondered how it could be the last mountain to be climbed if even a novice could do it with the help of a guide. Everybody tried to go up on the Italian side, which looks—the Italian border goes right over the head of the top of the Matterhorn, the Swiss on one side, and Italy in one, and the French border coming in almost there. So it's right at the corner of three countries. And everybody tried to come up from the Italian side because that looks more gradual. It turns out there's one place in there where it was not—only a very few experts have ever made it up there, and people hadn't—as I said, it was the last one to be climbed. And an Englishman engraver named Edward Whymper who had tried the Italian side a couple of times—his hobby was mountain climbing—failed.

He came with binoculars and studied that northeast face of the Matterhorn, the different faces, and said maybe it isn't as difficult as one thinks. So they started out to try the northeast face, which looks like a sheer wall of rock, but there are crevasses and things in there. And they made the first climb, 1865, this actually was, and that was the first climb of the Matterhorn on the descent. He went up with six colleagues all roped together. On the descent the rope broke between number three and four, and four, five, six, and seven fell to their deaths. So now, you can have two people to a guide.

Anyway, this idea of alternative approach, try to do it differently than people are—the conventional way, that didn't seem to be working. So when we looked at the estradiol and making the uterus grow this way, everybody thought that it was being oxidized and reduced, but no one could really prove it well. It didn't explain diethylstilbestrol. So we decided to take an alternative approach. My graduate student Herbert Jacobson, now a professor at Albany Medical School, and I, we decided to try to determine not what the hormone does to the tissue, which everybody was looking at—what results from giving the hormone, what about the enzyme—but what happens to the hormone itself. Well, the reason probably people hadn't tried this so much was that its very low dose, as I mentioned, that to form—to find the actually physiological dose and to give a hyperphysiological dose wouldn't really be significant. Luckily, we were at the University of

Chicago then, and the Fermilab, where the plutonium project was developed and everything, had the facilities to handle carrier-free tritium.

If you tried to bring that on campus, they wouldn't allow it. But we were able to go out to Fermilab and take 6-dehydroestradiol and developed an apparatus that we could measure the uptake of tritium by a catalytic reduction of a double bond. We wanted to be sure that it was complete because we thought maybe there'd be an adverse catalytic effect of the higher isotope—tritium, three times as heavy as hydrogen. Actually, it went a little bit faster with tritium than with hydrogen itself. So with this apparatus, we were able to reduce—get 6-dehydroestradiol, or we could actually detect a billionth of a gram—a trillionth of a gram.

Really, you need about a billionth of a gram to be able to study it well. We could find a trillionth of a gram, so we came back, administered this to immature rats or castrated rats, and find out to our surprise nothing happens to it. It's not changed chemically.

Moore: So I think that you started the synthesis with 30 curies of carrier-free tritium; is that right?

Jensen: Well, about 60, I think, is what it took in our apparatus. We used up all of that and then pumped the rest back again for the next time.

Moore: Not millicuries, curies?

Jensen: Curies, yeah. So they had the facilities in the Fermilab. A side effect I should say [is] that later in Chicago I had the pleasure of living next door to the Fermis for a couple of years and going to their daughter's wedding. Wonderful gentleman and deserving of all the plutonium work he did.

So in any case, what we could do is to find what happened to the hormone in the tissue—the answer being nothing—but it did bind to a hitherto unrecognized protein there, an extranuclear protein there in its target tissues, and caused it to move to the nucleus and act as a transcription factor for synthesis of specific RNAs. But people—biochemists didn't like that. They said, where were the enzymes? How dare you call it a receptor when it isn't in a membrane? People thought receptors were in membranes.

But about six years later, Bert O'Malley down in Houston actually tried—got interested in this and tried similar experiments with progesterone and found out that its target tissues had a receptor protein which the hormone caused to move to the nucleus and synthesized specific RNAs. About the same time, Ron Evans—and John Baxter a little bit later—showed with thyroid hormone a similar receptor protein being caused by using the hormone to move to the nucleus and act as a transcription factor. So then about ten years after our first report at a meeting in Vienna, where only five people were present because everybody was there to learn how estradiol worked in a different—our session conflicted with a plenary session where 1,000 people were [there] to hear how estradiol now we know doesn't work—and five, three of whom were other speakers were there to learn about estrogen receptor. So that's the background of estrogen receptor.

And it stuck in my mind this idea of alternative approach: If it isn't working by conventional methods, try something differently.

Moore: An alternative approach—

Jensen: An alternative approach, yeah. That founded the field of nuclear receptors, as it's known, estrogen being the first, progesterone the second. Now there are about 49 was the last figure I've seen of biochemical regulators that all follow the same pattern, so it did pay off.

Moore: Forty-nine in mice, but humans only have 48. Mice have one extra.

Jensen: [Laughter] Anyway, then we had other challenges. One had the receptor protein, one tried to make antibodies to it, so one could use immunochemical methods to study the receptor and measure it a lot better than the binding technique—and without any success. People also have actually proposed—these are immunochemists—it was proposed maybe estradiol was so

ubiquitous of a hormone that binds to its receptor and—but the receptor is not recognized as a foreign protein because it's present to some extent in so many tissues.

But to us then coming in with a little background in alternative approach and what the tissue did to the hormone, we decided we would use an unconventional method. We thought if we had the receptor protein and added what we thought might be the antibody preparation from the immune response and then we had the radioactive hormone bind to the receptor as a marker, if you had an antibody, it would make it move faster then because the antibody [was] about the same size as a receptor protein.

Moore: So this was in a sucrose gradient?

Jensen: Yes, sucrose gradient in an ultracentrifuge, and it worked just like a charm. We could show that you could detect the antibodies there. You could see that different antibodies could react, different preparations of antibody could react, with different places in the receptor protein because if you could saturate it with one, you could add the second antibody, and then you would get further movement on the gradient. So we were able to get the first antibodies to the receptor and use those then to measure the receptor, study it, and determine its amount in biological methods, including those from humans.

And the third alternative approach we had then was for studying the response of breast cancer to hormonal-type therapy. So it was known that there was some connection as early as the 1890s. Sir George Beatson took the ovaries out of—he was struck by the fact that breast cancer patients with large tumors because then they didn't find it as early enough, and you had very large tumors, that during menopause, the tumor would grow and then retract again. So there must be some connection, something between the ovary and the breast cancer. They didn't know about steroid hormones, of course, until about 1920.

So back in the late 1890s, the idea of something, some connection between the ovary and breast cancer was established by Beatson, who was knighted for this, Sir George Beatson, then. There are places that have Beatson Institutes. So anyway, people then started taking out the ovaries for breast cancer, in the younger woman taking them out, and in older woman taking out their adrenal glands because that's a source of estrogens after menopause, or taking out then the pituitary gland, which controls all of these hormone factories. But the only problem is only about 1/3 or a little bit less—1/3 of the patients will respond to this kind of therapy. And you're taking out these tissues, organs out of [ten] patients, and about three would respond. And all that time when they didn't respond, you'd have to take about eight months to see if they did.

The cancer would be growing and growing all the time. So one had some idea to identify which are the ones that are going to respond because then the ones that aren't going to respond, if you predict this, then you can use chemotherapy right away when the [tumor] is still much smaller. So we decided there's an alternative approach. Let's measure the receptor content of the breast tumors of patients who are going to undergo adrenalelectomy or a hypophysectomy or ovariectomy in a younger patient and see if there's any correlation there.

And it was again quite clear by this approach. You could see that those that didn't, that had escaped from the hormone dependency of the original breast tissue, they didn't need the hormone anymore. So they didn't make the receptor, and they did not respond to taking away the receptor because they didn't need it anymore. They had gotten an escape. But those that responded, they still had the hormone because the hormone was making them grow, and taking it away, they went away. So this gave you a good way to predict which [patients] should have adrenalectomy or hypophysectomy and which should be put on chemotherapy right away before the tumor had a chance to grow anymore after it was first detected.

Moore: So this led to tamoxifen, right?

Jensen: This was taking out the tissues.

Moore: That was before, right?

Jensen: Yeah. And there were some antiestrogens that were known, tamoxifen being one of them, but not much study was done. Then a man named Craig Jordan and I—and he was at Northwestern University at that time before he went to New York—we thought maybe we could try and see if tamoxifen wouldn't do the same thing as taking out the tissue, taking out the hormone-producing tissue. And sure enough, there was a pretty good correlation between the amount of receptor that was there and their response. So just as it was by taking out the hormone, you didn't have to do that. You just give them the antiestrogen tamoxifen. Craig and I used to delight the audiences when we would give talks by a little jingle that [went]:

A lady with growth neoplastic
thought castration was just a bit drastic.
She preferred that her ill could be cured with a pill.
Today it's no longer fantastic.

Anyway, that's the background of tamoxifen, and that does put the—it may prevent the estrogen from acting. Then along came other people studying this and found that getting something to prevent the synthesis of the estrogen in the first place rather than its action on the tissue might have some effect. And along came the so-called aromatase inhibitors preventing the synthesis of the hormone, putting the factory out of business rather than the action of the product at the target level. So today, some patients do better on tamoxifen. Some do better on [the aromatase inhibitor] Arimidex, but most do a little better on Arimidex, and people are taking it more and more there.

My wife, who you just met, is a hormone responder. She had her breast cancer removed, and it was receptor positive. Every breast cancer now has [levels of] hormone receptor, estrogen receptor, determined on it, and she had it. It was hormone receptor positive, and they then gave her—they took it off [and checked] with a mammogram, but to make sure if there were any metastases that were already there when they took out the primary tumor, they put her on Arimidex. So she's finishing her fourth year of Arimidex, and the receptor is gone.

So this is in a way an alternative approach to do with a pill instead of put the surgeon out of business and use a chemical to inhibit the action of estrogen by keeping estrogen from being there to act. So this then was what I like to say was made one especially for my wife's benefit from it. It makes one feel good.

And it did bring joy to my heart in Shanghai when the 50th anniversary of our presentation in Vienna where five people were present. The 50th anniversary of that was 2008, and Shanghai had a celebration of this as a surprise for me. They did not tell me. They asked me please to come and give a lecture and bring your slides along and bring some photos along if you can too and a CV. So when I got there, there was this beautiful brochure dedicated to the 50th anniversary here. And on this one they had to figure, they said 100,000 ladies every year are spared, prevented from dying due to our work. So those made me feel especially good.

Moore: I would think so. That's spectacular, really.

Jensen: Especially when my wife had the same. This was 52 years now, and I think a lot has been learned since then, but we do still have a problem with cancer. Early detection is important, but in many of the patients, we can do well with hormonal therapy. Unfortunately, those that have escaped the chemotherapy, we're getting better agents too in chemotherapy, but those remissions are not quite as good, and the chemotherapeutic agents are pretty toxic.

Moore: So I think that you and Sohaib [Khan] are doing some more on tamoxifen now, right?

Jensen: Right. That's one of the things that we're studying right now since I came here. He's the one that came when I had retired from Chicago because they had retirement age back when I

reached 70—that went up when I was 73. When I was 70, you had to retire at age 70. Of course, that isn't as old as it was 30 years ago.

Moore: Right.

Jensen: Anyway, I had to retire, but we spent a year, my present wife, in New York at Memorial Sloan Kettering, which is interesting. I couldn't have lived 35 years like I did in Chicago in New York, I think, but it was interesting for a year being right there with Memorial Sloan Kettering and Rockefeller University right on this corner there in Manhattan. Then they got a job in Hamburg ready for me, and [the] Albert [von] Humboldt Foundation set up a professorship for me to go to Hamburg for a while. I spent about seven and a half years in Hamburg. And then I was going to retire, but they wanted me to come to the Karolinska for a year because Jan-Åke Gustafsson [now] from Houston had discovered—he had discovered a second estrogen receptor, namely, now, the ER-β is what it's called. And what we had was ER-α. So ER-β and ER-α they were studying quite a lot there. And I went there for a year and ended up spending three years and had about four publications with Jan-Åke on ER-β. And knowing that both of the two different ER receptors, you can get a better picture than you could with one receptor alone, so it all adds up.

Then when I was in—that's in Stockholm, Sohaib Khan came up to—was in Stockholm for some other reason. He came over and said, when you get back, won't you come to Cincinnati for a while? That was very tempting because I grew up in Springfield, Ohio. I was born in North Dakota but moved to Springfield when I was four, and up there Cincinnati was the big city. I knew it pretty well coming down to watch the Reds play baseball or come to the opera, symphony. So I took tennis lessons at Camargo Country Club in college and in high school, actually, at the Hyde Park Country Club. So I said, I'll come for seven months. And I'm here eight years now and not smart enough to know when to leave.

But in any case, that's a little bit of how I got here and how I got to interact with other people like Jan-Åke Gustafsson, who's another Texan—who had to retire at age 65 in Stockholm. I tried to get him to come here to the University of Cincinnati, and he was going to think about coming here. But somehow, our administration got him mixed up and thought that he had to come right away or they couldn't have him. We weren't ready to have him right away. No, he had until another three-quarters of a year, and so they told him no. Then when they found out that he could come, they already had someone else. So Jan-Åke went to—Jan-Åke had already decided to come to Texas. He's happy there.

Moore: He seems to be happy there.

Jensen: That's a little background of the whole history of estrogens, estrogen receptor, and breast cancer. And still a lot of women do—are not found early enough or do not get the proper therapy. But more and more they are doing this, and many are alive today. It would not have been [the case] even ten years ago.

Moore: That's really spectacular. So I think that the readers and the viewers of the Web site of the *Annual Review of Physiology* will be thrilled to have had this opportunity to hear your stories. Do you have any other stories for them? Any other things that you'd like to pass along?

Jensen: Let me think.

Moore: I'm thinking about boxing.

Jensen: About what?

Moore: About boxing. When you were much younger, of course, you, I understand, were in the golden gloves.

Jensen: Oh! Yeah. I was. My mother was a grade school teacher. I was in Minnesota until I was four, born in North Dakota. She taught me to read and write when I was four. I came to start school in Springfield—that first year, that's what you learned, how to read and write.

So they put me ahead right away and to the end of the second year, so a year and a half ahead of time, and that made me much younger than everybody else, and I was kind of tall, skinny, and socially immature. So this was a handicap. I'm thinking how I got started on boxing. Anyway, I guess it was—

Moore: At Wittenberg?

Jensen: It must've been at when I was in Wittenberg, and they had an intramural boxing tournament there—that was how it was. And being tall and skinny gave me an advantage. I fought in the 140-pound class of welterweight. That gave me an advantage of reach [over] some of the more stocky individuals.

So in the Memorial Hall in Springfield, they would have each spring—about three or four weeks in a row, they would have what they called the golden gloves for three counties, not only Springfield, Ohio, but they had a big what's called then a CCC camp. They had a lot of pretty rough people working—during the Depression, this was. So they had people coming up in Memorial Hall every Friday night for a while. So I do remember that first Friday night. Here I came out, this skinny, young Norwegian Dane from North Dakota originally. And the fellow, he dropped his guard a little bit, and I caught him right on the chin, and he went down for the count.

I raised my hand there with all this—I guess 3,000 people out there Friday night. That sort of changed things. The next Friday night, they came up against another pretty tough-looking guy, but he raised his guard a bit, and I caught him in his solar plexus. He went down to the count. So two knockouts in a row.

Actually my late wife, Mary, whose father was a boxing fan, she was in the audience there in Memorial Hall. I didn't know her, of course, at that time. She was somewhat older, five years older than I, but she was there to see me win.

In the finals of the three-county tournament, I somewhat fortunately, I think, came down with the flu epidemic of 1939. They did have a flu epidemic then, and I recovered enough to go down and fight, but I did not recover enough. I lost the decision, and my opponent went on and won. He was really quite good. He went on and won the state and the national [tournaments and] turned professional.

So anyway, it was this boxing that you reminded me of that helped me a lot with self-confidence that I had because of being younger than other people. So I did have a good career at Wittenberg. I had no problem going into intramural boxing there, and I played on the tennis team there. I did enjoy Wittenberg and was president at my fraternity there. It all kind of—a lot of it went back to the boxing and all of that.

Moore: It all worked out very well.

Jensen: It all worked out very well, right.

Moore: Well, thank you, Elwood.

Jensen: You're welcome.

Moore: I really enjoyed talking with you, and I hope that you have many more years of success as things continue to move forward.

DISCLOSURE STATEMENT

David D. Moore is not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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