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session

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Session I: Critical issues in the etiology of problem gambling

Problem gambling—Is it in your genes?

Presenter: Kamini Shah

(Introduction.) **Jon Grant**: Our next speaker is Dr. Kamini Shah, MHS, who is the project manager of several studies, including "Pathological gambling: Courses, consequences, and causes" at the Washington University School of Medicine, Department of Internal Medicine. She has a masters in health sciences from the Johns Hopkins University School of Hygiene and Public Health, and she's currently a doctorate of science candidate in the Public Health Policy and Management, Health Finance and Management, again, with the Johns Hopkins University of Hygiene and Public Health.

Kamini Shah: The question is "Problem gambling—is it in your genes?" And the answer is, "yes," sometimes it is. I've got the advantage over Jon in terms of the chicken and egg question because you do start out with your genes and so the temporal relationship is set there. Again, this is an area where there are probably more questions than there are answers.

There are a number of ways that we can study genetic effects on gambling. The simplest way or the most basic way is to look at family studies, where you're basically looking at the clustering of disease in relatives, and you're looking to see if there's a genetic effect: is there more clustering in the family members of affected individuals than in those who aren't affected?

In Walters's study they found a higher clustering with sons of problem gambling fathers than daughters of problem gambling mothers. In the more severe studies you can tell that there's a

familial effect. But a familial effect can be due to genes or can be due to shared-environment factors.

If you move into twin studies, you can do those in a number of ways. You can look at adoption studies for monozygotic twins—identical twins—who were reared apart, so one twin had his or her biological family and one had an adopted family. And then you look to see whether they're more like the adopted family or their biological family. Again, genes would be implicated if they're more like their biological family. Problems with that are that record-keeping for adoption studies can be very private. The studies are also difficult to conduct sometimes. So, it's nice if you have a twin registry or some way to access twins, and we're lucky enough to have one of those.

When you look at twins and carry out analyses with them, aside from those who were adopted, you can look at a co-twin control study, which essentially means that if you have monozygotic twins who are discordant for a behavior—again, one twin has the behavior, the other one doesn't—you can have the unaffected twin serve as a perfect genetic control for the other twin, and that allows you to eliminate some of the confounds and to get at your answers.

You can also look at concordance of disorder; basically, looking at this idea of one identical twin versus the other identical twin. Are they more likely to both have the disorder if they're identical twins as compared to nonidentical twins? And, again, if that correlation is greater for your identical twins, you've got that greater concordance, and that is termed the "classical twin design."

Keith Winters has a study, which is one of the studies that didn't really show a genetic effect, but they found with monozygotic twins greater participation in high-action games.

Then, if you take it a step further, you can actually estimate how great is this genetic influence. So, we've said, "OK. There is a genetic influence. Well, how great is it?" For this you need a very large sample of people, a large sample of twins, and this is hard to come by.

We have the Vietnam Era Twin Registry, a registry of over 7,400 twin pairs, approximately half monozygotic, half dizygotic. They're all males because of limitations—very few female twin pairs went into the military then—and they're middle-aged now because they were serving at that time, so there's a definite limitation to that study. A number of people have been involved with those studies. But many of the studies that estimate genetic influence come from this one sample, and from it we've found that about 64 percent of the variance in gambling behavior is due to genes.

We know that there is a pretty large genetic influence on gambling, but we don't know which genes are involved. Then you go to the next step of looking at molecular studies and there are two ways of doing that. One is to look at linkage analysis, where you basically look for a gene that is linked to a disorder and it'll be present more in affected family members than nonaffected family members, and then you follow a pedigree. That hasn't been done yet for pathological gambling and one of the reasons is that to follow that kind of a linkage you need a clear mode of inheritance for a disorder. And we just don't see that with gambling or with a lot of behavioral issues.

So then, you can do association studies, which look at affected individuals versus nonaffected individuals, and ask, "Are certain genes more present in those affected versus those not affected?" Those are called "association studies" and much of this work has also come from two groups. One uses a population in Spain, and one uses a population here in the United States, with smaller samples than the twin registry, and I believe both are Caucasian samples.

Gambling and many behavioral issues and psychiatric disorders are "polygenic," which means that you don't have one gene that's driving the whole situation. A lot of different genes act in little ways and maybe in conjunction with each other, and so we have to tease out what's really going on.

To do association studies you have to have somewhere to start. There are a lot of genes out there. You can't just go studying them all, so what has happened is that because of the similarities, I believe, with addictive disorders, and the research on alcohol and all that came before the research on gambling, a logical place to start are the genes that have been shown to be involved with other addictive disorders. These types of genes, dopaminergic genes, serotonergic genes, and noradrenergic genes, have all been found to be related to gambling. And Jon did a wonderful job earlier, so I won't go into the details there, but there are small samples in this work. And that limits your ability to test for these effects, and when the effects are there, they tend to be small.

Dr. Cummings has a nice analysis wherein he looked at the effect of a number of different genes and found that 15 of them were related to gambling, but if you looked at the effect of any one of those genes, it was only about two percent of the variance. That's very little and it is very hard to detect a specific gene being involved with gambling.

Now, let's go back to the twin registry for estimates of genetic influence. Remember that this is with middle-aged males in an old study, based on data from 1992, so it does use the old DSM-III-R

criteria. But what we found was that, in the range of 50 percent of the variance in reporting, the first five symptoms had a genetic influence. There were limitations in our modeling procedures, often due to low prevalence of certain items. But, typically, when you have familial effects, you assume that they're largely genetic unless there's some very strong relationship between an environmental factor and a disorder. You can assume that much of that familial chunk is actually genetic. Again, limitations in our modeling prevented us from looking at the other four symptoms [*unclear*].

In a later study we used a bivariant model and could actually estimate the genetic, as opposed to the familial, influence on problem gambling, and it was 64 percent.

Something to throw out there for interest is that there are a lot of good reasons why we look at gambling as an addiction. We model treatments after what we've learned about other addictive behaviors to see how well they work. You do have to start somewhere and there are certainly many, many, many similarities between gambling and other addictions, whether it's just the phenomenological symptoms that look like withdrawal and tolerance and things like that, or whether it's the neurotransmitters that are involved, for instance, dopamine and that whole reward pathway, things like impaired decision making that affect both types of [*unclear*], similar types of comorbidities, antisocial personality, and then similar gene effects.

It's interesting to look at the overlap between pathological gambling and other psychiatric disorders in terms of "Does gene A affect pathological gambling, and does gene A also affect antisocial personality disorder, major depression, nicotine dependence, alcohol dependence, and drug dependence?" Then you can see that there definitely are some genes that are shared, but it's a relatively small percentage of them. The drug dependence estimate is a little funky because we had a confidence interval that went from here to there, but especially if you look at those first four, you do see that the overlap is significant.

But there is yet another story out there, and one of our questions is, "What are the other genes that affect pathological gambling?" Now, I should probably say that if you look at comorbidity of gambling with some of these other disorders, the genetic influence on that comorbidity is greater, and ask, "Is that same gene affecting both disorders?"

Where does this go? A question was raised earlier asking how close we are to being able to do something. Well, I think the future is here. The models that we use (statistical issues, various sampling designs) are expanding and developing rapidly, and it is almost as if by the time you finish the manuscript you are

developing, that your analysis is already somewhat old because now there's a new and better model for it. We're definitely learning as we go and you have to imagine what you could do with this genetic information. Genes are immutable, so to speak, but that doesn't mean they're irrelevant because genetic information can be applied.

Whenever I talk about these things, someone always tells me that I'm blaming the victim, and I'm not. I'm not, because a genetic influence is there, but it is not blaming the victim. There is certainly no one gene that determines that you will have gambling problems, and so that's not the focus here at all.

Instead, what you might want to think about is how you can use this genetic information. Gene effects are relative. When you look at our estimation models, the environmental effects and the genetic effects have to equal one, so there may be times in a person's life, say, youth, adolescence, when the environmental effects on your behaviors are greater. At that time your genetic effects will be lower, because just by definition it has to equal one. There may be some cohort effects that weigh in in terms of how big the genetic influence is. Remember, we're looking at a sample of middle-aged men.

You can also look at tailored treatments. People have certain genetic patterns. Maybe certain treatments will be more effective with some than with others. Maybe some people are more likely to have treatment failure. You could also look at correct outcomes. We know the question asking whether abstinence is the only way to go, or is controlled gambling possible after you've had a pathological gambling experience? And, again, there may be a genetic effect in determining which one of those is possible for an individual.

Clearly, this is work in progress and we're showing some genetic effects even in small, preliminary samples for genes and gender in terms of pathological gambling. You might ask, "Could genes be affecting that telescoped progression that you see in women in gambling?" Natural recovery, age of onset, again, like with many disorders—breast cancer, Alzheimer's disease—the stronger genetic effects are with the more severe forms of the disease. And we're seeing that, as I've shown with the data, with gambling as well.

Finally, we haven't been able to do much with our models in looking at that environmental genetic interaction. Someone isn't just going to be sitting in their home and out of the blue develop a gambling problem. But because of some statistical modeling issues—and, again, it involves power and sample size—we have some limitations in terms of how much we can model that process.

What do we need to do to improve our models and our estimation techniques?

The first question is this issue of clinically versus genetically informative phenotypes. Now, a phenotype represents expressed behavior or whatever happens as a result of the genes. It's what you observe. And within the clinical realm with the DSM you've got a threshold model of four or five plus symptoms, depending on which set of criteria you use. Either you have the disease or below the criteria you don't. Obviously there is a sense of a continuum with subthreshold gamblers versus pathological gamblers, but there's still a dichotomous view of the disorder. It may be that when looking at genes this is not the most clinically informative way to go. For instance, the genes may be more related to more biological forms of the disease, so what we're working on is defining phenotypes that might be more informative from a genetic point of view. And you can use multiple things to define a phenotype—the more you can narrow that phenotype down, the more likely you're going to be able to detect these teeny tiny genetic effects.

An endophenotype is a biological marker for a disease, and, usually, these markers have continuous values, like blood pressure or serum cholesterol. With them you get greater power for your studies, as with studies of, say, heart disease. We haven't found something like that yet for gambling, but if it is out there, it will help us.

And finally, we need to look at gender issues and, particularly, at racial issues, which we haven't been able to study much in the two samples that I've been talking about. And, clearly, there's reason to think that genetic frequency would be different with different races.

Alex Blaszczynski: Thanks, Kamini. We'll have time for questions after lunch.

[After lunch session.]

Alex Blaszczynski: We have an hour and a half to continue our discussions.

I think this is a key issue that we're talking about, in terms of the fundamentals of neurobiology, of genetics. It has implications, as Kamini has mentioned, in terms of blaming the victim. Questions such as, "If you do have a genetic predisposition to gambling, is it inevitable that you're going to develop problems? If not, what are the protective factors? What is the implication for relapse?" So there's a whole range of questions that we're going to cover.

Kamini Shah: A gentleman asked me after you all left for lunch for

a clarification about the size estimates that I gave earlier. And the issue is this: when you say that there's a 64 percent effect, a gene effect on pathological gambling, it's a little different than just talking about a pure correlation. And without getting into all the statistics of how it happens, you base what you're doing on these correlations and concordances.

But then there's modeling that gets you to those estimates. And if any of you are at all interested, we have an article that came out in this last *Journal of Gambling Studies*, that talks through some of that modeling in a nonstatistical manner. So I would refer you to that.

Getting back to issues and implications, I was trying to make some notes about things that I might have missed. Something important in these studies, and the reason why we haven't been able to do as much as we would like, involves the need to identify big samples. I can't stress that enough. It is a challenge for a number of different reasons. You can look at clinical samples where you get the higher proportion of pathological gamblers, per se, but then you end up with generalizability problems. You look in the community and it's a relatively rare disorder. So how do you get enough people?

These are the kinds of things that we have to grapple with before we can do more. I keep talking about increasing the power. And that means we'll be able to identify these differences. And you have to understand that just because we're not identifying an effect, it doesn't mean that it's not there. It just may be that we aren't able to detect it given what we've got.

And that's where you get into some of these issues like phenotypes and such. Because, if you can do things within modeling to help you identify effects, you can do it with the same size sample. You could take a phenotype, or an observed behavior. Or you could even think of Alex's typology of different types of gamblers, and instead of looking at all gamblers together, try to break it down so there's a meaningful grouping, in particular, a grouping that might be more related to a genetic load.

For instance, with the third category that was mentioned this morning—with the biological group—or even the second category with the emotional vulnerability, there was an issue of psychiatric disorder. And we know there's a genetic link with psychiatric disorders. So you may try to pull a group of people like that out and look at them. Perhaps you've got a narrow phenotype. Plus, genetically, you also want to try to get people who are a little more similar, so you don't have a vast heterogeneity with just a very small ability to detect changes.

I think a lot of work needs to be done on phenotyping. And it encourages me to see the literature now going much more in the direction of looking at types of gamblers, at subtypes, and that all gambling isn't equal. I think that will help this field out tremendously.

The effect of diagnostic reliability is another important approach. Because one of the issues with looking at genetic effects is this idea of how you classify a person as being disordered and having the disease or the illness versus not having it; this is critical to your estimates. How do we categorize these people? Based on the DSM? Are we getting a lot of false positives or false negatives? Do we have people that we're saying have the disorder, but really don't? Things like that affect the modeling. So I think as progress is made in classifying gambling and gambling disorders, that will help move this field along as well.

I think we need to look at how gamblers are different and to give that as least as much thought as we're giving now to looking at how gamblers are like other addicted individuals. I don't know that that's the magic bullet, or anything like that. But I certainly think it's an interesting place to go. I certainly think the evidence suggests that that might be useful. And certainly we, who are doing the research and the modeling, are also dependent on folks who are out in the field doing the clinical work and all, to help us define some of these things so that we can do better modeling.

Alex Blaszczynski: Are there any questions from the audience?

Carlos Blanco: I have two questions. When we talk about subtyping, we seem to consider problem and pathological gambling as two different entities, but research suggests that they might be a unitary construct. So, the first question is, how do you interpret this subtyping? Do you think it's environmental? Or do you think there are certain genes that provide a general vulnerability for pathological gambling, and then other genes that specify the subtype?

Kamini Shah: Some of our studies do show that continuum of gambling and that single liability throughout, and when you look at the idea of subtypes, there could well be some genetic differences. And you might be able to more clearly identify them by looking at a slightly different phenotype.

One of the advantages with phenotypes is that you may not be just looking at it based on genetics. You might be pulling other things into it, like personality and other things that also have a genetic load. And by looking at more of these variables that all are correlated, you might be able to increase your likelihood of finding

that gene that actually affects all of those things.

Carlos Blanco: But you think that the subtypes are really genetically based? Or do you think that they're more environmentally based? I realize you don't have the answer, I just want your thoughts.

Kamini Shah: I don't know. I find right now that the literature on subtypes is a little confusing. It's all over the place. And I think one of the things that has happened in the literature is that a lot of the studies of subtypes look only at pathological gamblers, at clinical samples, at people who have sought treatment. And clearly those folks are different; there's not as much work being done pulling in problem gamblers and recreational gamblers.

One issue with genetic studies involves looking for controls for our cases. And we don't do association studies. But what is the correct control? Who do you include in your study? One of the issues with only having pathological gamblers, versus your general population control, is that if you identify a genetic effect, are you identifying a gene for someone having an interest in gambling, or are you identifying a gene for someone who has a problem with gambling? So unless you have that middle block of people you can't tell what you're finding.

So, I'm not quite sure how to answer that, because when I look at the literature and see things like motivation to gamble, and risk taking, and impulse seeking, and psychiatric comorbidity, and a lot of the things that seem to be used right now to define these different types… right now my bias would be to say that there is a large genetic load.

Carlos Blanco: These studies suggest that there's a lot of genetic load, which would suggest biological treatment for these disorders. But my impression is that psychotherapy works better than medication right now.

I'd be happy if you wanted to answer this as well, Jon. What are your thoughts? Do you think that if it's a biologically based disorder, it should be treated with medication? Or is there room for psychotherapy? That we don't have the right medications, but eventually the medications will be better than the psychotherapy?

Kamini Shah: While he's fiddling with his microphone… one of the things that we've got an interest in looking at right now is cognitive distortions, and how these affect gambling. There is a literature out there about these. For instance, cognitive behavioral therapies are focused on dealing with these distortions and helping gamblers to understand that what they're thinking is not necessarily correct nor

does it reflect reality. But the issue is that perhaps there's actually a genetic influence on things like cognitive distortion and your likelihood of processing information that way. So yes, I still think there is room for the genetics in that kind of therapy. But certainly, more directly perhaps with the pharmacology, because there you can more directly tailor it.

Jon Grant: It's an interesting question, Carlos. But I'm always of the opinion that both medication and therapy will still do something fundamentally different to the brain. What groups are going to benefit most from medication versus therapy, or from a combination? We don't know. I think the genetics may lead us to some understanding of what people may benefit from something. When all is said and done, it's all biological. It's just a matter of how you are able to understand it. It'd be ideal, I guess, if you could look at a gambler and say, "Well look, based on this gene, and based on your subtype, you will benefit from eight weeks of CBT [cognitive behavioral therapy]. However, the person next to you will benefit from Paxil only." That would be the ideal world.

But I think either way they're going to benefit from it because it's going to change their brain. We're obviously just not there yet. But that would be the ideal, I think, of combining the genetics. Also imaging, which I talked about, with treatment options. That would be the perfect world. Yet I don't know of a disorder or a medical condition that can actually do that at all. So, holding gambling to that standard may be aiming a little too high at this point, given the fact that nobody else seems to do it.

Kamini Shah: There has been, I think, some work done with pharmacogenetics. And in terms of how definitive it is or not, I'm not sure. But with issues such as dosing, for instance, that some individuals may need a higher dose, or may react badly to a higher dose, it's all at an early stage.

We had looked at trying to do a study with that approach with gambling and we're not there yet. Because until we know a little bit more about what's going on biologically, we can't take the next step of trying to figure out how that interacts with genetics. [*Unclear.*] So in a way, some parts of the field have to wait a little while sometimes for other parts to catch up. And, as Jon said, we are so new at this. And even with disorders that have been out there for eons, they haven't gotten there yet with this.

Alex Blaszczynski: I'm just wondering whether there'll be any advance in identifying the antigambling gene. Are there any questions? There must be some questions. Otherwise, I'll have a panic anxiety. *(Laughter.)* I'd like to ask the panel members about samples. I think it's something we probably don't pay enough attention to. I'm raising the question of the potential for certain

agencies, certain institutions, certain people with research interests, to attract certain types of clients to their particular facilities.

And whether some of the genetics, some of the fMRI [functional magnetic resonance imaging] studies, some of the OCD [obsessive compulsive disorder] spectrum disorder studies, our own studies, cognitive therapy studies, suffer from the fact that some people filter through certain types of individuals to certain facilities. I would argue that within a psychiatric facility, you're probably more likely to get the more severe end of the spectrum, to get people with impulsivity-type disorders. If there is a known interest in particular fMRI studies on dual diagnoses, or on certain types of individuals, there's going to be a particular funneling effect, or filtering effect, leading to those particular institutions. Is this my fantasy? Jet lag? Or is it reality?

Rachel Volberg: We're still working on technology here. I've almost exclusively done general population and patron surveys. And the challenge there, of course, is that there are so few problem pathological gamblers in the population, that you have to have large samples in there to be able to identify enough people to have anything meaningful.

The issue that you've raised is a somewhat different one, I think, and speaks to the question, "Are particular types of problem gamblers attracted to particular types of treatment?" And they therefore end up in your research sample, because of their belief, or their feeling, that that type of treatment is going to be effective with them. I have to say, it's an intriguing possibility. Maybe some of the other research folks in the audience might have some ideas about how you would control for that, or how you would address that, in doing that kind of work.

Jon Grant: In my experience, I think that people who have gambling addictions are so desperate for treatment that they will go wherever they can find somebody who will give treatment. Yet it's always intrigued me that we live in this information age. The possibilities for people to know where to go are not difficult. People call me from all over the country saying, "Is there anybody in my area?" Why don't people know? How can we make that information available? And I think, "Yeah, there's somebody 30 miles from you." But why don't they know that?

When people sign up for treatment studies, for fMRI studies, I have found that it's almost like *Field of Dreams*; if you build it, they will come. And if you let people know that there's something out there that will help them get more information about the illness, or have it treated, they will flock to it. But I just don't think we necessarily do a good job.

We have so many different options—newspapers, Internet, all these things that only hit slices of populations—that I don't think we do a good job of letting people know about all these things. And that's the selection bias that I see; people just randomly find something, or just randomly don't.

Kamini Shah: My two cents here is that I think that there are definitely issues about clinical populations being different from nonclinical populations, just by the fact that they were seeking treatment. Even before you get into the issue of "did they seek this treatment versus that treatment?", there is the fact that they sought treatment, because such a small proportion of problem gamblers do seek treatment. But there's also a volunteer bias because you have people that are in a clinical situation and they have more awareness of their illness.

But there are also issues around community surveys which use advertisements, as opposed to direct-digit dialing, or random-digit dialing, where you've got someone who is volunteering, who has looked at an ad, has said, "Oh, that sounds interesting." Or, "Oh, that applies to me." Someone has taken the initiative to call you and wants to participate. That person is also different from the person who looked at that ad, but chose not to do anything about it.

Issues of sampling are beyond just the clinical realm. And as an aside, to get back to our genetic models, we actually did a study looking at the genetic effect on treatment seeking for alcoholism and found a 41 percent genetic load. So these guys are different.

Alex Blaszczynski: Is there a gene for treatment seeking? Is that the implication? *(Laughter.)*

Could I have some feedback from the audience in respect of do you believe that there is a filtering effect of certain types of severity, or certain characteristics of clients attending different types of centers? Because you have community centers. You have hospital-based centers. Veterans Administration [VA]. Private practice. Talking amongst yourselves, do you detect that there is any difference between subtypes? Could we go for the microphone please? We like to give people the limelight.

Joanna Franklin: My sense is that much as Dr. Grant's saying, the gamblers will go wherever they can find help. Phoenix is an interesting example of several different centers in one metropolitan area. And though it's not an entire spectrum of possible treatment venues, it's a selection of venues. Folks are clumping and clustering based on preferences that I certainly don't understand. But they're interesting to look at. If you go to Flagstaff, if you go to Tucson, you don't see that.

I think for the counselor something like the pathway model that we talked about this morning is much more helpful because you can get any number of different folks. Trying to understand which one is which, and who is who… lots of counselors who are relatively new to the field bring the shotgun with them and figure, "Let's try a little bit of… and see what's going to work." We don't have enough in the way of assessment information that lets you categorize. At least not in the hands of us, the folks in the trenches, so to speak, that lets you categorize: "I think you'll do best with this, and you'll do best with that." We're not there yet.

Some counselors are somewhat resistant even to the medication trials, with some medications that have been suggested in studies. It's almost like, "When all else fails, we'll think about a pill, but not until we've tried everything else under the sun." So availability has a lot to do with it. And in some areas there's ample GA, as opposed to no GA, and where there are ample state-funded treatment programs and regardless of income you have access to care. Louisiana is a great example of that.

In other states, you have to have job income, maybe insurance, or forfeit your first-born child in order to find some access to care. So it's a mixed bag. *(Laughter.)*

Rachel Volberg: I just have to reinforce what Joanna said, that resources for pathological gamblers are spread very thin. I would say that like any number of people addicted to other things, they may present at a mental health clinic, or have some episode, in which they have a 72-hour commitment at a state mental hospital, or even go through the private psychiatric network, if they have medical insurance. A lot of that probably depends upon whether they actually know what ails them.

And of course, we have a help line for Delaware and several other places. And by definition, they have some idea of what's wrong, or they wouldn't be calling a problem gambling help line. They come through us because we're the only game in town. And we see every conceivable variant, all the subtypes, the genders. We just don't see many young people.

Rachel Volberg: [… responding to a comment about the low numbers of people seeking treatment…] There's so much that can be said on this. A couple of things. When we had a treatment program in Las Vegas and were running a pharmacological study at the same time, the people who came in for the study were not the people who came in for treatment. And when they finished the study, they didn't say, "Okay, now I want treatment." They just finished the study and went off someplace. So what the differences were between those who came in for treatment, and didn't want to volunteer for the study, and those who came in for the study, and

then didn't want treatment, I'm not sure. Superficially on several measures they looked much the same. But still, there must have been something different about how they self-selected, how they chose to deal with their problem.

With data from the VA and comparable data we've collected in the private sector we see some significant differences in those populations of gamblers. On cognitive variables. On personality factors. So when we've done our brain scans on veterans, where you may have significantly higher rates of attention deficit disorder and cognitive deficits of various kinds, you're going to get different results.

So yes, you do have to be careful to describe the population you're dealing with. If you don't have comparable data for other populations, you're not sure what you're looking at. So I think that's a significant concern. You can have a population like veterans, where you may have a whole different genetic loading than nonveteran populations. It's an interesting question.

Jon Grant: The one comment that I have in response is this. I think the idea of stigma is still obviously quite huge with gambling addiction, as much as education has tried to suggest that it's more common than is thought. People will tell me, "You know, I really want to get help. But I don't want this on my insurance. Can you offer me something so nobody has to know I have this?"

Because I think more and more people are suspicious over the privacy of their records, and what insurance companies know and don't know. And I don't think it's paranoia. I actually just think it means well-informed people. And so when people come in for treatment, they have to have a certain different perspective and confidence, I think, in some way. Because they're saying, "Okay. This is going to go through insurance. You're going to bill me. Somebody might write down a little code that says pathological gambling."

I think a lot of people are aware of that stigma and how it may affect their work, their future insurance, all of these. And this is also why I like the option of being able to offer some types of studies for people and being aware that some people simply won't go for any treatment if it means having to give too much personal information.

Rachel Volberg: I wanted to comment on that too because this idea of insurance coverage for treatment for problem gambling, or for other disorders, is a singularly U.S. one. And let's not lose sight of the fact that in order to get coverage for problem gambling treatment of a professional kind, in the U.S., for the most part—with some few exceptions—people have to meet diagnosis. And they

have to have insurance. And the insurance company has to know what they're paying for.

In other countries, that's not the case. And so I think it's important not to be parochial, and just think about this in terms of U.S. issues, but to understand that there are a lot of different ways of doing this and that that may also impact people's willingness to access treatment services and participate in studies.

Kamini Shah: In terms of the different treatment programs, clearly, a lot of these people with gambling problems, as you point out, may actually show up in a drug treatment center, or somewhere like that.

And particularly in that instance, you then have to deal with the comorbidity issue where you've got a gambler who is also addicted to alcohol, drugs, et cetera. And how do you deal with that when you want to study gambling? And so there is an issue around whether you exclude people from studies who have comorbid disorders like that. Which means how many pure gamblers do we really have out there? Or do you keep them in the study?

Because then what you're really finding in your genetic study of gambling is the effect of genes on gambling over and above the effect on these other things. So I think the issue of comorbidities might also become important, in terms of which gambling treatment center you were at.

Alex Blaszczynski: [… in response to a question…] But in raising the question of EMDR [eye movement desensitization and reprocessing] and other treatment paradigms, I think the important element is to provide some degree of evidence enhancement that these interventions are quite effective.

It concerns me that when we did a review in Australia of some of the counseling services and looked at the particular methods of treatment, it became quite depressing and quite worrying from a clinical perspective that you have counselors who don't use any particular diagnostic criteria, or any particular measure to assess the problem of pathological gamblers in their particular clinics. But then they run a range of esoteric treatment interventions of unknown effectiveness with that particular population, with the assumption that it works over there, so therefore I can do it over here. And a high percentage of people are doing reflective listening without using elements of interventions that have been empirically validated to some extent.

But all of that, I think, is in the treatment domain. Again, addressing the audience to try to stimulate you, post-lunch: is it the genetics?

Is the neurotransmitter element really that important for counseling interventions? Does it really matter whether someone has the genetic makeup, immutable or not?

We know from the research presented here that there's a 60/40 percent split, genetics versus environment. But that can change, depending on certain circumstances. So it's not immutable. We can't change the genetic component, so do we need to worry about it from a treatment-intervention perspective? Or do we just focus basically on what we can modulate or modify, and assume that there is some genetic component? Do we ignore it? Or do we take it into account in modifying our treatment interventions? Any comments on that? From the audience, preferably.

Loreen Rugle: One thing I do with my clients is to give them a lecture on the biological, on the psychological. Believing that information is power, and empowering patients and clients, is giving them that information. And while at times they think, "Oh, immutable gene. I'm doomed," this gives you power to decide how your treatment should progress. And they come in with a question of "Why? Why do I keep doing this? What's wrong with me?" And helping them have that understanding and awareness of what puts you at risk, where your vulnerabilities are, is important in empowering them to make informed treatment decisions, and be part of that treatment-planning process. So I think it's important.

And to understand, for me, what I'm working with, and all the domains, and whether treatment is likely to be longer or shorter, and how to triage, and get a medication referral, is critical in relapse prevention.

Alex Blaszczynski: Or is it perhaps, Lori, too premature to raise those particular issues, since there are inconsistencies in responses, small effect sizes, biased samples? Is it worth it?

[End of taping for this presentation.]

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contents | intro | keynote address | session I | session II | session III | session IV

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