Alice Shaw: Hello. I'm Dr. Alice Shaw, a medical oncologist at the Massachusetts General Hospital Cancer Center. I'd like to welcome you to Improving Outcomes for Patients with Non–Small Cell Lung Cancer: A Six-Part Virtual Tumor Board Integrating Best Practices and Emerging Evidence to Enhance Care, brought to you by the publishers of *The ASCO Post* and Harborside Medical Education.

Alice Shaw: Today we'll focus on the newest diagnostic and therapeutic paradigms in the field of non–small cell lung cancer. For decades the mainstay of therapy for metastatic non–small cell lung cancer, was platinum-based chemotherapy. For over a decade, genetic alterations and targeted therapies have become part of the treatment schema, with new targets and treatments discovered and approved rapidly.

Alice Shaw: Most recently, immunotherapy has become part of the treatment paradigm for non–small cell lung cancer as first-line monotherapy, first-line treatment in combination with chemotherapy, and second-line therapy. Immunotherapy has even transformed the treatment paradigm in locally advanced disease.

Alice Shaw: Included in each tumor board discussion will be one to two case studies illustrating key aspects of the topic at hand. Here to discuss new diagnostic and paradigms for non–small cell lung cancer, are two expert clinicians from Massachusetts General Hospital. Can you please introduce yourselves?

Lecia Sequist: I'm Lecia Sequist. I'm a medical oncologist at Mass General. I focus on lung cancer and I do research on EGFR lung cancer as well as novel methods of early cancer detection.

Justin Gainor: My name's Justin Gainor. I'm also a medical oncologist in the Center for Thoracic Cancers at Massachusetts General Hospital. My area of focus is on cancer immunotherapy and I'm an active investigator studying new immunotherapies in trying to identify novel biomarkers of response in resistance to immunotherapy.

Alice Shaw: Here are our financial disclosures.

Alice Shaw: In this module, we'll be discussing strategies for evaluating biomarkers such as EGFR and PD-L1 in patients with non–small cell lung cancer using a case study of a 61-year-old female patient. The learning objectives for this module are to evaluate best practices and interpret the clinical significance of emerging data regarding the role of biomarkers for diagnosing and treating patients with non–small cell lung cancer, to plan strategies to incorporate best practices and emerging data regarding biomarkers into practice, and to use biomarkers effectively to guide treatment selection.

Alice Shaw: So for our first case, SD, and I'm sure this is a patient who you know, is a 61-year-old woman with a 15 pack/year smoking history, who developed cough and shortness of breath last spring and was evaluated by her primary care

physician. Chest x-ray demonstrated a 3.6-cm left lung mass. She then presented to a Boston hospital for further evaluation. Chest CT confirmed a 3.3-cm left lower lung mass. Staging PET scan showed that the left lower lung mass was FDG avid and revealed extensive FDG-avid lymphadenopathy, including left supraclavicular, left prevascular, left paratracheal, and left hilar nodes. Brain MRI demonstrated a 2-cm right parietal mass with associated edema and a 6-mm left occipital lesion. She underwent ultrasound-guided FNA of the left supraclavicular lymph node, and pathology was positive for malignant cells. These were TTF-1 positive p40 negative, consistent with lung adenocarcinoma. ALK IHC was negative and ROS1 IHC was negative. Her PD-L1 using E1L3N was 20%. Tumor tissue was submitted for multiplex next-generation sequencing testing including for mutations within EGFR, BRAF, and other genes.

Alice Shaw: So Dr. Sequist, if we can start, maybe you can comment first on this patient's molecular testing so far. In general, what molecular test should be offered to patients with metastatic lung adenocarcinoma? And what do you think is the most efficient way to do this testing?

Lecia Sequist: Well, this is a changing landscape, as more markers are always being developed and more drugs are becoming approved. It does become crucial for us to know the status for every patient. Currently for adenocarcinoma patients, it's really important to test for EGFR mutations, ALK and ROS translocations, BRAF mutations, as those are the approved indications. But in practice, I think it's also very important to test for MET exon 14, skip mutations, and even HER2 mutations or amplification of MET or HER2 because we have drugs that are very active for all of those. And an emerging one is also RET translocation. So really every few months the paradigm is changing.

Alice Shaw: And what are your thoughts about doing this testing all up front as multiplex testing versus doing it one test at a time, which is what we used to do?

Lecia Sequist: Yeah. Well, it's certainly much more efficient, as far as the tissue is concerned and as far as time is concerned, to do them in a multiplex fashion. But at the same time, when someone is newly diagnosed and, of course, anxious to get started on treatment, you also want to try and test some of the things rapidly. And depending on your practice setting, you may not be able to get all of those done rapidly. So I would say that ALK, ROS, and EGFR are probably the ones to try and turn around the quickest.

Alice Shaw: And perhaps, Dr. Gainor, you could comment on the ALK and ROS testing in this case. As I mentioned she had IHC for ALK and ROS, so not an NGS test. What are your thoughts on using IHC for ALK and ROS1?

Lecia Sequist: Yes. IHC is a useful test because it can be performed very quickly. Also, a lot of our pathologists are very familiar with doing IHC testing. In particular for ALK, IHC is a very good test, and that's because there's not a lot of background staining. We know that ALK usually isn't expressed in the lung. But in the setting of an ALK rearrangement, it's topically expressed. It's very clear cut when there's

an ALK rearrangement based on IHC testing. And that is an approved test right now. With respect to ROS1, there is higher background there. So you do want to follow it up with a confirmatory test.

Alice Shaw: So would you be comfortable acting on a positive ALK IHC assay?

Justin Gainor: I would be comfortable.

Alice Shaw: Now unfortunately, for this case, there wasn't sufficient tissue from that FNA of her lymph node to do next-generation sequencing testing. So while another tumor biopsy was being arranged, the patient was referred to radiation oncology. She actually underwent stereotactic radiosurgery to both of the brain metastases. After stereotactic radiosurgery, she then underwent an additional biopsy. This is now a CT-guided biopsy of the lung mass itself, and tissue was submitted for NGS testing. So Dr. Sequist, is there data to support potentially thinking about a liquid biopsy in this case? Would you have acted on liquid biopsy results?

Lecia Sequist: Potentially. I think in a situation like this, where the tissue has been exhausted, and you have to go in and do another biopsy, that might be a perfect scenario to try and do a liquid biopsy, especially because you have this key missing test for EGFR. I would personally still try and get the tissue biopsy so that you can be very confident in your full panel of results. But EGFR, in particular, is one where there is an FDA-approved plasma test and the plasma is pretty accurate at detecting EGFR mutation.

Alice Shaw: So had this patient had a negative liquid biopsy result, though, you likely still would have pursued a tissue biopsy?

Lecia Sequist: Exactly, because we know that about 30% of patients don't shed DNA. It depends on their tumor biology and to some extent their tumor burden. So a negative test is less helpful when it comes to liquid biopsies. A positive test, you can feel comfortable treating patients with a positive test. But if it's negative, especially if it's negative for any tumor associated mutations, it could just be that you're not detecting any circulating DNA.

Alice Shaw: Dr. Gainor, what about for rearrangements? ALK, ROS, and RET? How comfortable would you be using a liquid biopsy to try and detect one of these rearrangements?

Justin Gainor: I think that's a good question because we know that the ability to detect certain genetic alterations is different in the plasma and that things like point mutations, which are activating and EGFR are easier to detect them more complex genetic alterations like rearrangements. Just because I didn't see an ALK rearrangement or ROS1 rearrangement in the plasma, the same rules apply in that it could just be that this is not a shedding tumor. But if it were there,

then I would have more confidence, in that these plasma tests tend to be more specific. The presence of it there would give me confidence to treat the patient.

Alice Shaw: I think rearrangements are more complicated than mutations. We know that oftentimes the breakpoints can occur throughout a number of different introns, depending on the target gene. And that can be very complicated then for these liquid biopsy assays. But we have had those cases where a tumor tissue was insufficient, as in this case, and we detected a rearrangement in plasma and we were able to act on that.

Alice Shaw: So in this case, she did undergo the tissue testing though again, and as during the time that the NGS test was cooking, the patient was initiated on first-line therapy with carboplatin, pemetrexed, and pembrolizumab.

Alice Shaw: So Dr. Gainor, I mentioned that this patient did have PD-L1 testing on that first diagnostic specimen. And that showed 20% positivity using the PD-L1 antibody E1L3N. And I'm wondering, this is kind of a confusing area for a lot of us. I'm wondering if you could start by reviewing first the different PD-L1 assays and how they're used in clinical practice.

Justin Gainor: Sure. As you mentioned, this was a point of considerable confusion. That was in part due to the fact that all of the different PD-1 or PD-L1 inhibitors in the clinic had different diagnostic tests using different antibodies, and many of them had different scoring algorithms. Some of them were focused on PD-L1 expression on the tumors, some tumors and immune cells, and each with different cut points. And I think it's important to point out that in contrast to the genetic alterations, which we've been discussing where it's really either positive or negative, in the setting of PD-L1, it's really a continuous variable. So defining a cut point is important.

Justin Gainor: Thankfully, over the last couple years, we've had some clarity added to this issue, thanks to a number of harmonization efforts, which compared the various diagnostic PD-L1 assays in a set of primary lung tumors, where we can really look at the concordance of these tests. One of the important efforts to harmonize was the Blueprint Project and another collaboration with the NCCN and Bristol-Myers Squibb. In one of these pivotal studies, it looked like the concordance of all these antibodies was quite good. There is one antibody, the SP-142, that looked like it showed lower rates of PD-L1 expression compared to the others. But that was really the outlier. This patient had the E1L3N antibody, which was highly concordant with the other antibodies that are used in clinical practice.

Alice Shaw: What do you make of this patient's PD-L1 score, which was 20%, in terms of her treatment options?

Justin Gainor: I think the most clinically relevant cut point right now is a cut point of 50%. That was really established by the KEYNOTE-24 study, which showed that among

patients who have PD-L1 expression of 50% or greater, first-line pembrolizumab was superior to platinum doublet chemotherapy. We've seen with subsequent studies testing lower cut points, that most of those studies were negative and it really has shown that 50% really defines a particular subgroup of patients who are most likely to benefit from single-agent PD-1 therapy.

Alice Shaw: So, Dr. Sequist, we'll be reviewing, actually in detail, the data on frontline carboplatin, pemetrexed, and pembrolizumab in one of our later modules, but I'm wondering if you can just briefly discuss, since this is a patient we both know and have treated, the selection of this triplet regimen for her, given what you know about her so far, including her PD-L1 staining.

Lecia Sequist: Well, this is becoming a very common scenario, because the PD-L1 staining comes back so fast, in most cases, much faster than the genetic testing. I think in the era prior to PD-1 availability, PD-1 drug availability, people had gotten very comfortable with this notion of if you have a high suspicion of a genetic driver, then you would try and hold off on starting chemotherapy.

Lecia Sequist: But also if you really needed to start therapy for symptomatic burden, there was not a downside. Things are a little bit different now that immune therapy has been added to frontline chemotherapy for many patients, including this patient. And the reason is because if you then find out a short while after you started on treatment that they have a driver mutation and you want to think about a targeted therapy, then things can get a little bit trickier.

Lecia Sequist: When I've been in the situation with patients where I need to get started, I don't have the genetic results back, and I have a high suspicion that they may have a driver mutation based on their history, I try to keep it to chemotherapy alone and not add in pembrolizumab just because of concerns of toxicity, which I'm sure we're about to talk about.

Alice Shaw: We hadn't met this patient yet because she was seen on the outside, and she did have a 15 pack/year smoking history. I think it was probably quite anxiety provoking for both her and her provider to be waiting. They already had one biopsy done without sufficient tissue. Then they had to do the stereotactic radiosurgery for the brain lesions, set up another biopsy, wait for the testing. So I think oftentimes patients and their oncologist get into this situation where they feel like they should start therapy before molecular testing comes back. And so I hear you. I think in this case though, she went ahead with the three-drug regimen. She received two cycles of carboplatin, pemetrexed, and pembrolizumab, and then during, I believe, the second cycle, the results of her NGS testing returned. This NGS testing showed she had an EGFR exon 19 deletion along with other mutations including a P53 mutation, an ARAF mutation, CDKN1B, PDGFR, a RET mutation, quite a few actually, and her TMB actually came back at about 10.6.

Alice Shaw: I'm wondering actually, Dr. Sequist, if you can first comment on the NGS reports that we see these days. You know, we didn't simply see an EGFR exon 19

deletion, we actually saw a lot of different mutations, and how do you sort through these mutations? And do you feel confident saying that the EGFR exon 19 deletion is the main driver for this patient?

Lecia Sequist: I think historically, a lot of the reason we didn't appreciate all these other mutations is we weren't looking for them. As our panels have gotten larger, we've picked up the ability to see more and more passenger mutations. It turns out that a lot of the classic tumors that we've been studying and the cases that have brought these drugs, the targeted drugs into the forefront, a lot of those patients did have a wide range of passenger mutations as well. And I think when you see a classic driver mutation, in this case, a deletion 19 EGFR, you can be pretty confident that they will still respond to therapy, even though there are a lot of passenger mutations.

Lecia Sequist: It is a little bit unusual for an EGFR patient to have this many, and to have a tumor mutation burden that's that high, but we certainly see patients with that background.

Alice Shaw: I do find those reports can be very confusing to many of us in academia, as well as in the community, because they often do have this whole list of mutations in sometimes known drivers. I think in this case, as you said, a classical driver, EGFR exon 19, would be certainly reasonable to act on this like your typical EGFR mutation-positive patient.

Alice Shaw: Dr. Gainor, as Lecia mentioned, this case was notable because she actually had a reasonably high tumor mutation burden, or TMB, at 10.6 mutations per megabase. I'm wondering if you can summarize a little bit for us, what exactly is TMB. What are the thresholds? What are the clinical implications, especially for this patient?

Justin Gainor: Yeah. I think this is an evolving diagnostic, really. Initially, the first time we started hearing about TMB was work by Naiyer Rizvi, where they looked at about 30-odd patients with lung cancer and they were treated with PD-1 inhibitors, and they did whole-exome sequencing. There they saw that, just a higher number of nonsynonymous mutations, that is mutations that change the amino acid sequence, was associated with response. I think there are some at the intuitive level that makes sense. That the more mutations that cancer has, the more it may be foreign to the immune system, and the more likely it is to be recognized and eradicated.

Justin Gainor: Since that initial seminal work, there have been efforts to try to standardize reporting of TMB, and right now, many of these reports are reporting both synonymous and nonsynonymous mutations in filtering for germline mutations. Many define more than 5% allelic frequency is a common metric for TMB and they report mutations per megabase. I think that that reflects the fact that it's just impractical to do whole-exome sequencing on standard of care patients. Many times what we're relying on is using these smaller panels of targeted next-generation sequencing. Thankfully, it looks like there's high concordance

between a TMB calculated by the gold standard, which is whole-exome sequencing, versus a smaller targeted NGS panel.

Alice Shaw: Although, that smaller panel probably has to have a minimum of maybe 150, 200 ...

Justin Gainor: Yeah. I would say at least. And I think what you're getting at is that you want to have sufficient dynamic range to really pull apart differences and mutation levels. But because you're using a smaller number of genes, that's why you're normalizing it based on the coverage. That's why the reports are typically mutations per megabase.

Justin Gainor: The data that's really guided certain cut points, I would say that the data that we have from clinical trials, the best data is from CheckMate 568 and CheckMate 227. There it was exploring the combination of nivolumab and ipilimumab, and it looked like at least for the combination, the cut point of around 10 mutations per megabase, was defined as high TMB.

Justin Gainor: I think it's worth also mentioning that's a pretty high percentage of our patients, so about 44% of patients will actually have a high TMB defined by that metric.

Alice Shaw: So this patient did actually have, just squeaked above that a threshold for high. She was already on carboplatin, pemetrexed, and pembro as I mentioned and she had her first set of restaging scans after two cycles. Actually there was a pretty significant reduction in the size of her left lung mass and the lymphadenopathy had also significantly improved. Importantly, there were no new or progressive lesions. The brain MRI that was done also showed a response to the treatment including probably the SRS. So she continued on carbo, pem, and pembro for another two cycles. At this point is when she came to Mass General and met us. At that time, we reviewed her new restaging scans, now having completed four cycles of the triplets, and the scans continue to show an ongoing response.

Alice Shaw: In general, she had tolerated the chemo and the immunotherapy quite well, but she was clearly developing cumulative side effects such as worsening fatigue and anemia. Also some anorexia, kind of worsening with each cycle.

Alice Shaw: So before we go on to what happened with her, I thought Dr. Gainor, could you briefly say a word about what is the typical maintenance regimen for a patient like this who's already received four cycles of the triplet regimen?

Justin Gainor: Right. I think it's worth mentioning that before the introduction of pembrolizumab with chemotherapy, our typical maintenance had been pemetrexed, and that has been shown to produce an overall survival benefit. In the KEYNOTE-189 study, that really established the role for the triplet of carboplatin, pemetrexed, and pembrolizumab. In that study, after four cycles,

patients then went on to receive the combination of pemetrexed plus pembrolizumab for up to 35 cycles.

Alice Shaw: About two years.

Justin Gainor: For about two years, right.

Alice Shaw: So that was one of the options for this patient now that she had completed four cycles. So now switch over to maintenance, possibly the doublet that you recommended, but Dr. Sequist, she was particularly interested in switching to an EGFR targeted therapy given the discovery of the EGFR exon 19 deletion, and of course, we now know that there are multiple first and next-generation EGFR inhibitors available. And so, for this patient with advanced EGFR-mutant in lung cancer, what would be the recommended first-line EGFR targeted therapy for her?

Lecia Sequist: So as of about a year ago, we're now recommending osimertinib as a frontline therapy, or the first EGFR therapy for someone who has started chemo in the setting of a known EGFR mutation. This is based on the FLAURA study, which was osimertinib randomized to first-generation TKI of the provider's choice, so either erlotinib or gefitinib. They called this the standard of care arm. So osimertinib compared to the standard of care arm improved progression-free survival, almost doubling it from around 9 to close to 19 months. It's now FDA approved as a frontline therapy for that significant improvement in PFS. And the other advantages to starting with osimertinib are that the side effect profile is milder and also the CNS penetration is higher, so everything lines up in the direction of frontline osimertinib.

Alice Shaw: She had an EGFR exon 19 deletion. Do you feel like the type of EGFR mutation, particularly EGFR exon 19 vs L858R would impact your selection of osimertinib?

Lecia Sequist: I don't think so. A few years ago, we talked a lot as a field about whether we should choose different drugs for different mutations, particularly between afatinib vs erlotinib, but in the era now of osimertinib, in the FLAURA study, they stratified by mutation type and looked specifically at whether one mutation did better or worse with the osimertinib compared to standard of care, and they didn't see a difference.

Lecia Sequist: Exon 19 deletion tumors tend to perform a little bit better overall, and that carried through in FLAURA, but there was no differential performance based on the drug that the patients were treated with by mutation.

Alice Shaw: So really, when we met the patient, the main sort of discussion was, should she continue on maintenance pem/pembro versus switching over to osimertinib as her first-line targeted therapy. And Lecia, you already kind of referred to this a little bit earlier about the potential for switching directly from an immunotherapy potentially to a targeted therapy. So I'm wondering what both

of you think about her situation now, especially because she's very motivated to switch to a targeted therapy. Would you be comfortable switching her now to osimertinib or would you propose something else?

Lecia Sequist: So because all of these drugs are new, we're still learning about this and a lot of the data is maturing. But we know from a couple of different sources that there's some concern for toxicity when thinking about an EGFR TKI and a checkpoint inhibitor together. There were some clinical trials that actually looked at the combination of these regimens, and they had to be aborted for excess toxicity.

Lecia Sequist: I think one of the more compelling things, at least in my practice and how I think about patients, is that there was a post-marketing search of the FDA database looking at reported toxicities from patients being treated with commercial drugs. They looked at the rate of new pneumonitis and saw that there was an expected rate of pneumonitis for patients receiving, it was nivolumab, because at that time, that was the only FDA-approved checkpoint inhibitor. There was an expected and small rate of pneumonitis for patients receiving EGFR TKIs, but when they looked at the subset of patients who had received, in the commercial setting, both a checkpoint inhibitor followed by a TKI or vice versa, that the rate of pneumonitis was several fold higher—as much as 26%—and that is definitely something that I've seen in practice as well.

Alice Shaw: So this was the publication by Oshima and colleagues. And I think it was pretty striking because like you said, the majority of the patients actually had received these therapies not concurrently but actually sequentially. And oftentimes nivolumab followed by an EGFR TKI, it could have been any of them actually, then there was this higher incidence of pneumonitis.

Alice Shaw: Dr. Gainor, what do you think? Are you also concerned about, for example in this patient, moving her directly from her chemo immunotherapy regimen to osimertinib?

Lecia Sequist: I share Lecia's concerns, and I think this applies not just to EGFR inhibitors. I think as a class, I have concerns about moving from a PD-1 inhibitor to a TKI as we've seen similar concerns for toxicity even among ALK inhibitors. Although the spectrum of that toxicity differs there, it's more hepatotoxicity, but still seeing unexpected toxicities when you move from a PD-1 inhibitor to a TKI. And part of this is due to the fact that the PD-1 inhibitors last in your system a lot longer. The half-life of pembrolizumab is 22 days. So, even though this patient, just got a cycle three weeks ago, it's still very much in their system. Not to mention the T cells are primed. I would share the concerns of jumping straight to osimertinib in this patient.

Alice Shaw: So is there an optimal washout then from a checkpoint inhibitor before switching to TKI?

Justin Gainor: I don't think we know yet. I completely agree with Lecia that this is an evolving question but I wouldn't want to jump straight away. One consideration that I'd have is to actually go to pemetrexed maintenance by itself, dropping the pembro for a few cycles and then come in with the osimertinib. So get a few cycles under our belt and trying to get as much distance from the pembro as possible while at the same time recognizing that this patient has the lung cancer, and we want to give them the most effective therapy. So trying to achieve some balance there.

Alice Shaw: So this patient did exactly that actually. We recommended that she transition to osimertinib but with sort of a bridge of single-agent pemetrexed to try and offset any potential sort of overlap of the immunotherapy with osimertinib. She's completing her chemo now, and we'll start with osimertinib hopefully in the next month or so. So Dr. Sequist, assuming she's responsive to osimertinib—we of course expect that as she's an EGFR mutation-positive patient, we expect her to respond, but unfortunately, we also expect that at some point in her future she'll also develop resistance.

Alice Shaw: Maybe the last minute or so maybe just a brief summary in terms of where we are in our understanding of osimertinib resistance.

Lecia Sequist: So a lot of what we know about osimertinib resistance is in the setting of giving it as a second TKI after T790M positivity. But with that caveat, it does look like osimertinib can cause a point mutation at the spot where the drug binds EGFR called C797S. That's one of the more common things that we see. We also see MET amplification and both of those occur in about a quarter of patients. So together they make up about half of the patients.

Lecia Sequist: The MET amplification looks like it may be an actionable resistance mechanism. There are several MET inhibitors out there, and there are several publications of activity when you give someone a combined MET inhibitor and continue to osimertinib in the setting of osimertinib-acquired MET resistance, MET amplification.

Alice Shaw: C797S is a little trickier. We don't yet have a drug that effectively targets that mutation. So that's something that hopefully will be coming in the next couple of years.

Lecia Sequist: Then there's other smaller, less common resistance mechanisms that we see.

Alice Shaw: I think you're about to present and actual publish on one of those less common but still very important targetable mechanisms. Maybe a word about that.

Lecia Sequist: Yeah. Sure. We have found, and others have found in a small subset of EGFR patients, that they can actually acquire a RET translocation at the time of resistance. And since there are some new exciting specific RET inhibitors that have been showing a lot of activity in the clinic, we decided to try combining

osimertinib with one of these specific RET inhibitors, called BLU-667, and we treated two patients with that regimen and both had a tremendous response. So this is early data, it needs a lot more verification and larger numbers of patients, but that could potentially be an actionable resistance mechanism that's coming out of osimertinib.

Alice Shaw: Pretty exciting that RET rearrangement can be de novo, a de novo driver, but also now turn up as an actionable resistance mechanism as well.

Lecia Sequist: Absolutely.

Alice Shaw: So to summarize this module, all patients with metastatic nonsquamous, non–small cell lung cancer should undergo multiplex testing at diagnosis. And to date we've talked about a number of these validated targeted therapies for, actually I think seven, now molecular subsets which include EGFR, ALK, ROS1, BRAF, RET, MET exon 14 skipping, and actually TRK, which we didn't speak too much about. And likely there are more to come.

Alice Shaw: All patients with metastatic non–small cell lung cancer should undergo PD-L1 testing, PD-L1 expression levels, Justin, as you mentioned, 50% or higher. That's a positive test result for first-line pembrolizumab monotherapy.

Alice Shaw: And finally, tumor mutation burden, or TMB, is emerging as an important molecular biomarker that can predict response to checkpoint blockade including dual nivo and ipilimumab in non–small cell lung cancer.

Alice Shaw: Thank you both for joining me today. We hope you enjoyed this discussion. Be sure to check out the other modules in this virtual tumor board series on Improving Outcomes for Patients with Non–Small Cell Lung Cancer. For more information please visit educate.ASCOpost.com.