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Expert Answers to Common Questions on *MET* exon 14 Skipping Mutations in NSCLC

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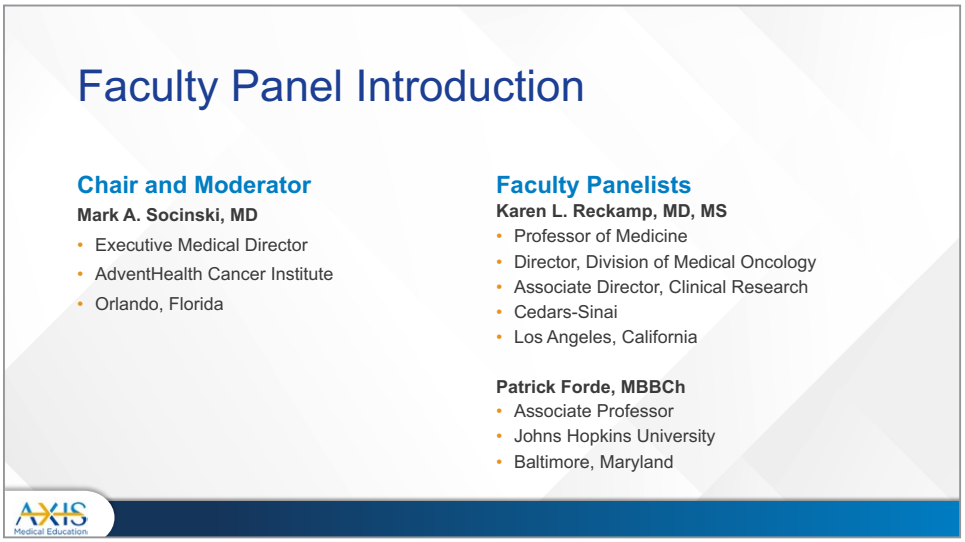
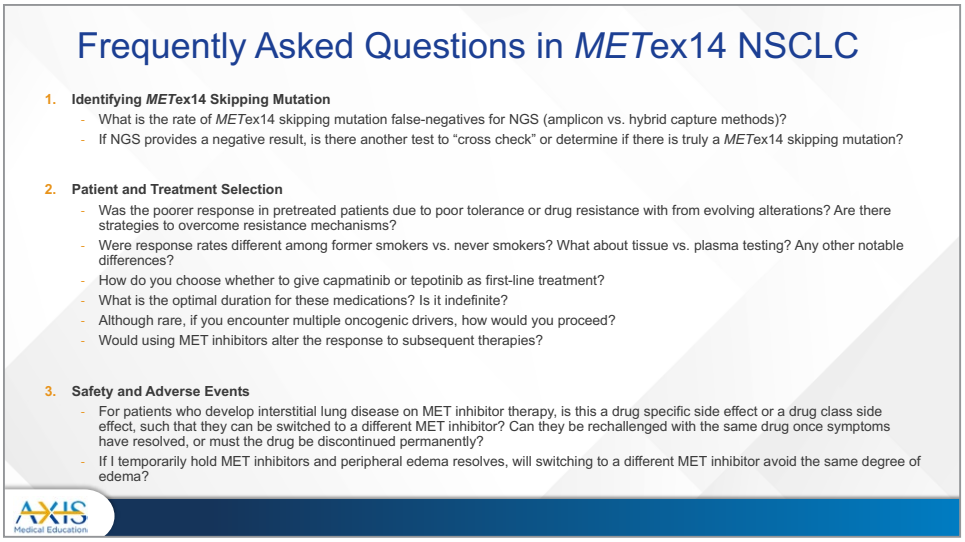
Expert Answers to Common Questions on *MET* exon 14 Skipping Mutations in NSCLC

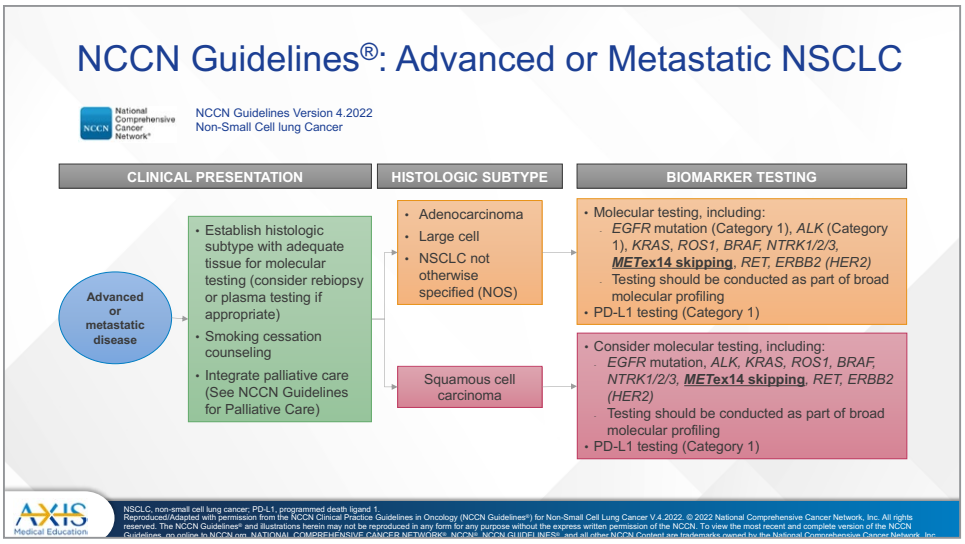
Mark A. Socinski, MD; Karen L. Reckamp, MD, MS; and Patrick Forde, MBBCh

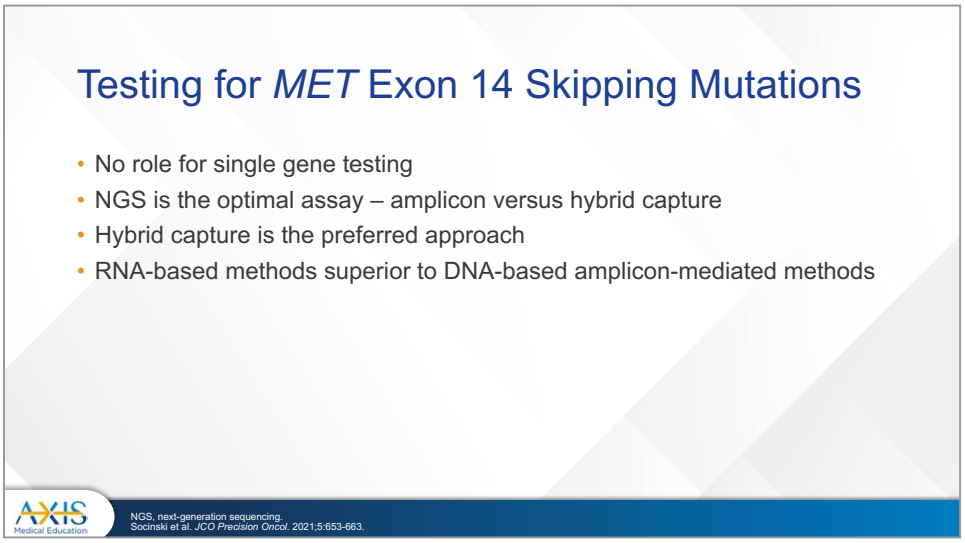


Mark Socinski, MD: Welcome. I'm Dr. Mark Socinski, Executive Medical Director at AdventHealth Cancer Institute. I'm joined today by Dr. Karen Reckamp from Cedars Sinai, and Dr. Patrick Forde from Johns Hopkins University.



Today we will be discussing and answering questions that were asked by clinicians during a recent educational series on advances with selective MET inhibitors in *MET* exon 14 altered non–small cell lung cancer. Our questions today will focus on three main topics. Number one: identifying *MET* exon 14 skipping mutations; two, patient and treatment selection; and three, safety and adverse events.





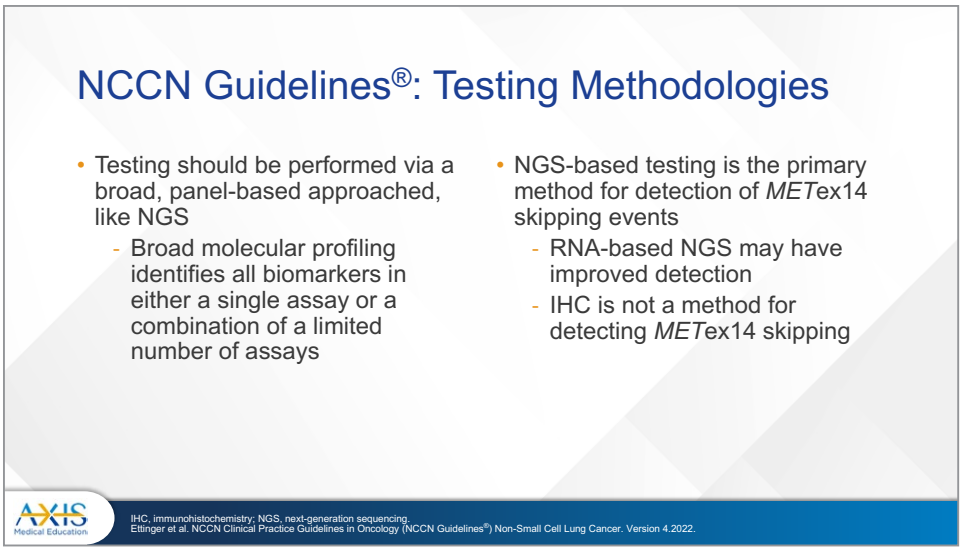
First, a very brief review of current guideline recommendations for molecular testing. Obviously in advanced stage disease, we now have 9 or 10 biomarkers, which the FDA has approved therapies for, so it's important to employ a comprehensive, broad-based platform to make sure that you cover all potential targets at the time of initial diagnosis.

Since we're focusing on the *MET* exon 14 skipping mutations, I want to ask Dr. Reckamp our first question: What's the rate or incidence of these mutations, and what's the optimal way to identify them?

Karen L. Reckamp MD, MS: Thank you, Mark. *MET* exon 14 skipping mutations occur in about 2% of patients with non–small cell lung cancer, and there's a multitude of mutations that can occur, and these can sometimes be larger mutations, and affects the membrane domain, so it's important to do a broad-based, next-generation sequencing (NGS) method to evaluate for these mutations. Preferably we're using hybrid capture to identify these, and even more so, RNA-based methods can often identify these alterations more frequently than DNA-based methods. This is not to be confused with *MET* amplification, which can occur in about 20%-30% of those patients who have tumors with *MET* exon 14 skipping mutation, so they can co-occur, but they are separate types of alterations.

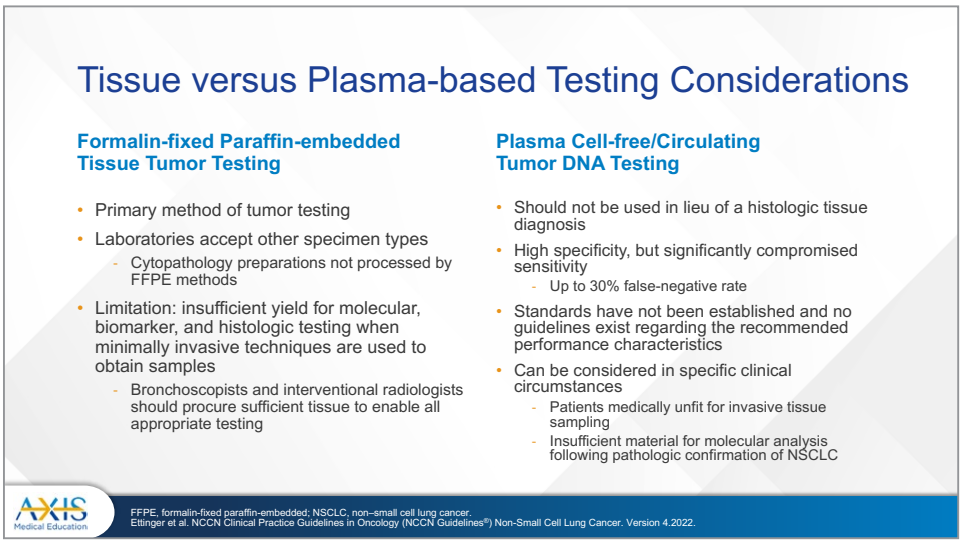
Dr. Socinski: Patrick, anything to add to Karen's comments?

Patrick Ford, MBBCh: I think with all the different modalities we have for testing having a clear idea of what we're looking for when we send the tests is going to be important—also for our colleagues who are providing the results to us, to make it clear to us as practicing physicians what a specific result may mean for that patient.



Dr. Socinski:
Karen, you mentioned the term both DNA and RNA NGS platform. Obviously, one should be familiar with what platform you're dealing with, and if it's just a DNA-based platform, how important is the RNA component, to cross-check this if you have an initial negative result?

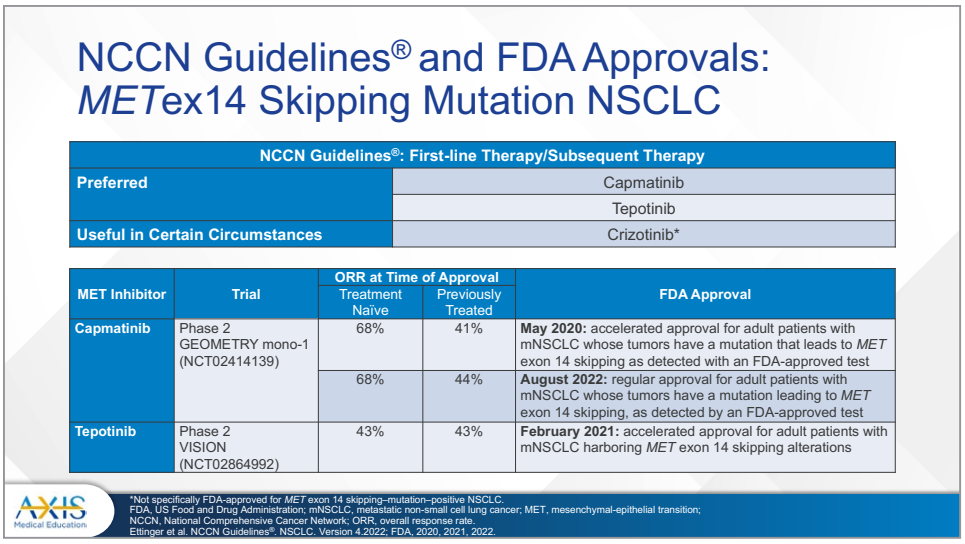
Dr. Reckamp:
There will be some false negatives if you use just a DNA-based platform, and so with a negative result moving on to an RNA-based platform would be ideal. Many of the platforms do incorporate RNA-based sequencing from the beginning, up front.



And then the other option is liquid biopsy, if there is insufficient tissue, and we know that older patients tend to get *MET* exon 14 skipping mutations. The median age is in the 70s so sometimes doing an extra biopsy for extra tissue is not feasible, and so liquid biopsy is an option but you have to know that there is up to 30% false-negative rates for liquid biopsy.

Dr. Socinski:
Patrick, is this something you commonly do in your practice?

Dr. Forde:
I think our focus primarily has been on tumor testing. At our institution, we do a DNA-based test which is broad-based, as you mentioned, covering about 500 genes, but focusing on those important ones that have actionable implications. And allied to that, we have a more limited RNA-sequencing panel, which covers about 250 of the most well-described fusions, including *MET* exon 14 skipping mutations. Sometimes we will get a positive result on both. I think the main role, in terms of newly diagnosed patients where I use a liquid biopsy, is for those who do not have enough tissue for one or both of those methods. And I think that's not an insignificant number of our patients for the reasons Karen mentioned.



Dr. Socinski:
So again, the reason we're testing for these sorts of things is we have a number of FDA approvals, if you identify them, and none of these FDA-approved drugs, which are highly efficacious can be utilized by clinicians unless you make the diagnosis with comprehensive molecular testing.

In some of the data that we saw with the MET inhibitors, these drugs were evaluated in pretreated patients as well as the treatment-naïve patients. And, as we see in most cases, you don't see quite as good of a response rate in the pretreated patients. What are your thoughts about this? Is just related to tolerance, or resistance, or what are your thoughts?

GEOMETRY mono-1: Capmatinib Best Overall Response

	Pretreated, Cohort 4 (2/3L) N = 69		Expansion Cohort 6 (2/3L) N = 31	Treatment-naive, Cohort 5b (1L) N = 28		Expansion Cohort 7 (1L) N = 32
	BIRC	Investigator	BIRC	BIRC	Investigator	BIRC
Best overall response, %						
CR	0	1.4	-	4	0	-
PR	41	42.0	-	64	60.7	-
SD	36	30.4	-	25	35.7	-
Non-CR/non-PD	1	2.9	-	4	-	-
PD	9	10.1	-	4	3.6	-
Not evaluable	13	13.0	-	0	-	-
ORR, %	40.6	43.5	51.6	67.9	60.7	65.6
Median DOR, months	9.7	8.31	8.4	12.6	13.83	NE
DCR, %	78	76.8	-	96	96.4	-
Median PFS, months	5.4	4.8	6.9	12.4	12.0	10.8

All responses confirmed per RECIST 1.1. Response rates consistent between BICR and investigator assessment
BIRC, blinded independent review committee; CR, complete response; DCR, disease control rate; DOR, duration of response; NE, not evaluable; ORR, overall response rate; PD, progressive disease; PR, partial response. SD, stable disease.
Wolf et al. *N Engl J Med* 2020;383:944-957; Wolf et al. *J Clin Oncol* 2021;39:9020.

VISION: Tepotinib Best Overall Response

	Cohort C (confirmatory) N = 161	Treatment-naïve N = 69	Previously Treated N = 51
Best overall response, %			
PR	-	62.3	51.0
SD	-	24.6	31.4
PD	-	10.1	7.8
Not evaluable	-	2.9	9.8
ORR, %	54.7	62.3	51.0
Median DOR, mo	20.8	NE	12.6
DCR, %	80.1	87.0	82.4
Median PFS, mo	13.8	15.9	13.8

DCR, disease control rate; DOR, duration of response; NE, not evaluable; ORR, objective response rate; PD, progressive disease; PR, partial response; SD, stable disease.
Thomas et al. *J Thorac Oncol.* 2022;17:S9-S10.

Dr. Reckamp:
I think that for some of these patients, it may be about the tolerance again because we are looking at older adults. Some of the data, it's smaller numbers and so the response rates also may even out a little bit as numbers get higher and they're treated for longer periods of time. But there is potentially a slight benefit to treating in the frontline setting, and so that just highlights the need to do full, next-generation sequencing up front for patients, so that we can treat them with the appropriate most beneficial therapy as the first-line setting. But again, I'm not certain what causes that difference. Generally, when these targeted agents work, they work. But *MET* also has a number of co-mutations that may cause differences in efficacy. So, I don't think we quite understand some of those differences that we see.

Faculty Discussion

Dr. Socinski:
Very good. We saw the response rates broken out by several different categories—smokers versus never-smokers—and identifying this particular abnormality, either in tissue versus plasma. Patrick, what are your thoughts about the response rates in these different subgroups, and particularly, not necessarily response, but some of the progression-free survival (PFS)-overall survival (OS) data with regard to identifying tissue versus plasma?

Dr. Forde:
In terms of significance, patients who do have a tissue-confirmed alteration tend to have a longer PFS, and do somewhat better with these agents overall, and I guess there's a few things we could postulate about that. One is, perhaps, there is some subclonal element in plasma which we're picking up which may be subclonal and not necessarily in a single biopsy taken, and perhaps in a tissue biopsy, you're looking more at a clonal mutation which is driving the tumor to grow across multiple metastatic sites. That was an interesting finding, and, more broadly in terms of *MET*-altered tumors they're probably a more heterogeneous group than we see with other driver mutations, for example, with *ALK* or with *ROS1* we rarely see those occurring in smokers whereas with *MET* we can, and there can also be implications for other forms of therapy for MET. For example, patients with PD-L1 high, *MET*-altered tumors who are smokers can actually have benefit from immunotherapy as well. It's a complicated group of patients and needs a lot of nuance in terms of how we manage them.

Dr. Socinski:
Just to build on that, Karen mentioned the false-negative rate with plasma. One of the other hypotheses would be related to the bulk of disease, and if you find it in plasma, of course we don't understand much about shedding of tumor DNA and these sorts of things, but is if you find it in plasma, do you think that that bulk of disease may be one of the other variables why you might see a difference?

Dr. Forde:
That's quite possible, so it's one metric. Those patients who are detectable in plasma, all must certainly have more advanced disease on average than those patients who are undetectable. And perhaps have progressing disease at the time of the sample taken. So that could influence PFS as well.

Dr. Socinski:
Karen, we're going to give you the toughest question here. How do you choose whether to give capmatinib or tepotinib as first-line treatment?

Dr. Reckamp:
That is a tough question, and fortunately for our patients, these are excellent medications that are very efficacious, have CNS penetration, and are tolerable. And so, for our patients they're both very good options. There are subtle differences. They both have the toxicity of peripheral edema, which we'll talk a little bit more about in a bit. But capmatinib is given twice a day, and so sometimes with compliance that may be a little more difficult, but sometimes with dose reductions, that may be a little easier. And tepotinib is a once-a-day medication, so obviously a little easier to remember once a day. But overall, they're both very, very good drugs for patients with *MET* exon 14 mutation.

Dr. Socinski:
Patrick, do you treat these patients until progression? Do we know much about optimal duration?

Dr. Forde:
In the VISION trial, patients were treated until progression or unacceptable toxicity. What I do tend to utilize with these patients is dose reductions, bearing in mind that these are older patients on average, for example, compared to our other targeted therapy patients. And they often have comorbidities. The toxicity I've seen which has been most noticeable is peripheral edema. But it is relatively responsive to dose reductions, and that tends to be my first approach rather than stopping the drug or treatment breaks. But occasionally treatment breaks are needed, you know? And I would not hesitate, perhaps at a short treatment break, and then trying to restart at a lower dose. What are your thoughts, Mark?

Dr. Socinski:
No, that's similar. I think sometimes a short break can help with some of the toxicities that we see. As Karen alluded to, we still don't have a good sense of why the edema occurs or the optimal management strategy for it, but I do think—as you point out, Patrick—the combination of dose reduction; sometimes if disease is controlled, giving them a little break from treatment can be very helpful in terms of tolerating these sorts of things.

Moving on, let's get back to Karen. You know, it's quite rare—we've all probably in our practices had a few patients in which you encounter multiple oncogenic drivers on these sorts of situations. Have you seen that? And if you did see it, how would you think about the management of those patients?

Dr. Reckamp:
I would say de novo, I have not seen multiple driver mutations that include *MET* exon 14. I have had a patient with an *EGFR*-mutated tumor develop a *MET* exon 14 as a resistance mechanism and have successfully given both MET inhibition and osimertinib together, and it's been tolerable. And we have some examples of that for patients who develop *MET* amplification. But generally *MET* exon 14 skipping mutations, as a de novo alteration, is an oncogenic driver, and very uncommonly presents with other co-mutations that are oncogenic drivers. There are co-mutations that may affect efficacy, and again, the presence of amplification or even protein efficacy at the time of diagnosis may have some indication of whether response will be deeper, or prolonged. But generally it is an oncogenic driver on its own.

Dr. Socinski:
Yeah, I agree with you. I've not seen it in the *MET* exon 14 space, but certainly in other spaces we sometimes see these at the time of initial diagnosis.

Karen, you made the point that these are both great drugs for this sort of thing. Any evidence that one may work after the other?

GEOMETRY mono-1: Capmatinib Safety Summary

	Cohort 4 (2/3L), N = 69		Cohort 5b (1L), N = 28	
	All Grades	Grade 3/4	All Grades	Grade 3/4
Any adverse event, n (%)	68 (99)	52 (75)	28 (100)	21 (75)
Most common adverse events, n (%)				
Peripheral edema	37 (54)	10 (14)	21 (75)	3 (11)
Nausea	32 (46)	0	13 (46)	0
Vomiting	18 (26)	0	7 (25)	0
Blood creatinine increased	23 (33)	0	10 (36)	0
Dyspnea	19 (28)	7 (10)	6 (21)	2 (7)
Fatigue	18 (26)	6 (9)	4 (14)	1 (4)
Decreased appetite	15 (22)	1 (1)	8 (29)	0
Constipation	10 (14)	2 (3)	4 (14)	0
Diarrhea	12 (17)	0	5 (18)	0
Cough	10 (14)	1 (1)	7 (25)	0
Back pain	11 (16)	2 (3)	4 (14)	0
Pyrexia	9 (13)	1 (1)	2 (7)	0
ALT increased	8 (12)	6 (9)	4 (14)	2 (7)
Asthenia	6 (9)	3 (4)	4 (14)	2 (7)
Pneumonia	7 (10)	4 (6)	2 (7)	0
Weight loss	9 (13)	0	3 (11)	0
Noncardiac chest pain	5 (7)	1 (1)	1 (4)	0
Event leading to discontinuation	14 (20)	8 (12)	6 (21)	5 (18)

ALT, alanine aminotransferase.
Wolf et al. *J N Engl J Med.* 2020;383:944-957.

VISION: Tepotinib Safety Summary

TRAE, n (%)	Overall (N = 291)	Age subgroup, y			
		<65 (n = 64)	≥65-75 (n = 107)	≥75-85 (n = 96)	≥85 (n = 24)
Any grade Grade ≥3	264 (90.7) 86 (29.6)	52 (81.3) 9 (14.1)	105 (98.1) 28 (26.2)	84 (87.5) 39 (40.6)	23 (95.8) 10 (41.7)
Leading to dose reduction	90 (30.9)	10 (15.6)	36 (33.6)	36 (37.5)	8 (33.3)
Leading to temporary interruption	114 (39.2)	14 (21.9)	39 (36.4)	46 (47.9)	15 (62.5)
Leading to permanent discontinuation	41 (14.1)	4 (6.3)	14 (13.1)	17 (17.7)	6 (25.0)
Most common all-cause AEs, n(%)	**Overall (N = 291)**	**Age subgroup, ys**			
		<65 (n = 64)	≥65-75 (n = 107)	≥75-85 (n = 96)	≥85 (n = 24)
Peripheral edema	191 (65.6)	35 (54.7)	75 (70.1)	61 (63.5)	20 (83.3)
Nausea	87 (29.9)	16 (25.0)	35 (32.7)	32 (33.3)	5 (20.8)
Diarrhea	81 (27.8)	17 (26.6)	27 (25.2)	30 (31.3)	7 (29.2)
Hypoalbuminemia	81 (27.8)	15 (23.4)	27 (25.2)	31 (32.3)	8 (33.3)
Blood creatinine increase	76 (26.1)	13 (20.3)	30 (28.0)	29 (30.2)	4 (16.7)
Dyspnea	60 (20.6)	9 (14.1)	21 (19.6)	22 (22.9)	8 (33.3)
Decreased appetite	48 (16.5)	3 (4.7)	21 (19.6)	22 (22.9)	2 (8.3)
Constipation	46 (15.8)	9 (14.1)	17 (15.9)	19 (19.8)	1 (4.2)
Fatigue	45 (15.5)	8 (12.5)	16 (15.0)	20 (20.8)	1 (4.2)

AE, adverse event; TRAE, treatment-related adverse events.
Garassino et al. *Ann Oncol.* 2021;32(suppl 5):S949-S1039.

Dr. Reckamp:
Not to my knowledge or in my experience. These are both very similar drugs, and the toxicity is very similar, so when we see edema developing from one drug to stop and switch to the other, I haven't seen improvements or differences in that toxicity. Basically, all MET inhibitors cause some development of edema, and the edema occurs over time, so the longer you're on it, the more prevalent it becomes, and fortunately for some patients, they can be on it even for years but that edema you're continuing to manage. I haven't seen any evidence that you can utilize one or the other to overcome resistance at this time. And looking at new generations that may overcome some mechanisms of resistance, or potentially combinations as we see a multitude of resistance mechanisms starting to emerge as we utilize these medications.

MET Inhibitor Summary: Warning and Precautions

Capmatinib

- **ILD/pneumonitis**
  - Monitor for new or worsening pulmonary symptoms indicative of ILD/pneumonitis (dyspnea, cough, fever)
  - Immediately withhold in patients with suspected ILD/pneumonitis
  - Permanently discontinue if no other potential causes of ILD/pneumonitis are identified
- **Hepatotoxicity**
  - Monitor LFTs (ALT, AST, and total bilirubin) prior to the start of treatment, every 2 weeks during the first 3 months of treatment, then once a month or as clinically indicated
  - Withhold, dose reduce, or permanently discontinue based on severity
- **Pancreatic toxicity**
  - Monitor amylase and lipase levels at baseline and regularly during treatment
  - Withhold, dose reduce, or permanently discontinue based on severity
- **Risk of photosensitivity**
  - Advise patients to limit direct ultraviolet exposure
- **Embryo-fetal toxicity**
  - Advise patients of potential risk to a fetus and to use effective contraception

Tepotinib

- **ILD/Pneumonitis**
  - Monitor patients for new or worsening pulmonary symptoms indicative of ILD/pneumonitis (eg, dyspnea, cough, fever)
  - Immediately withhold in patients with suspected ILD/pneumonitis
  - Permanently discontinue if no other potential causes of ILD/pneumonitis are identified
- **Hepatotoxicity**
  - Monitor liver function tests (including ALT, AST, and total bilirubin) prior to the start of treatment, every 2 weeks during the first 3 months of treatment, then once a month or as clinically indicated
  - Withhold, dose reduce, or permanently discontinue based on severity
- **Embryo-fetal toxicity**
  - Advise of potential risk to a fetus and use of effective contraception

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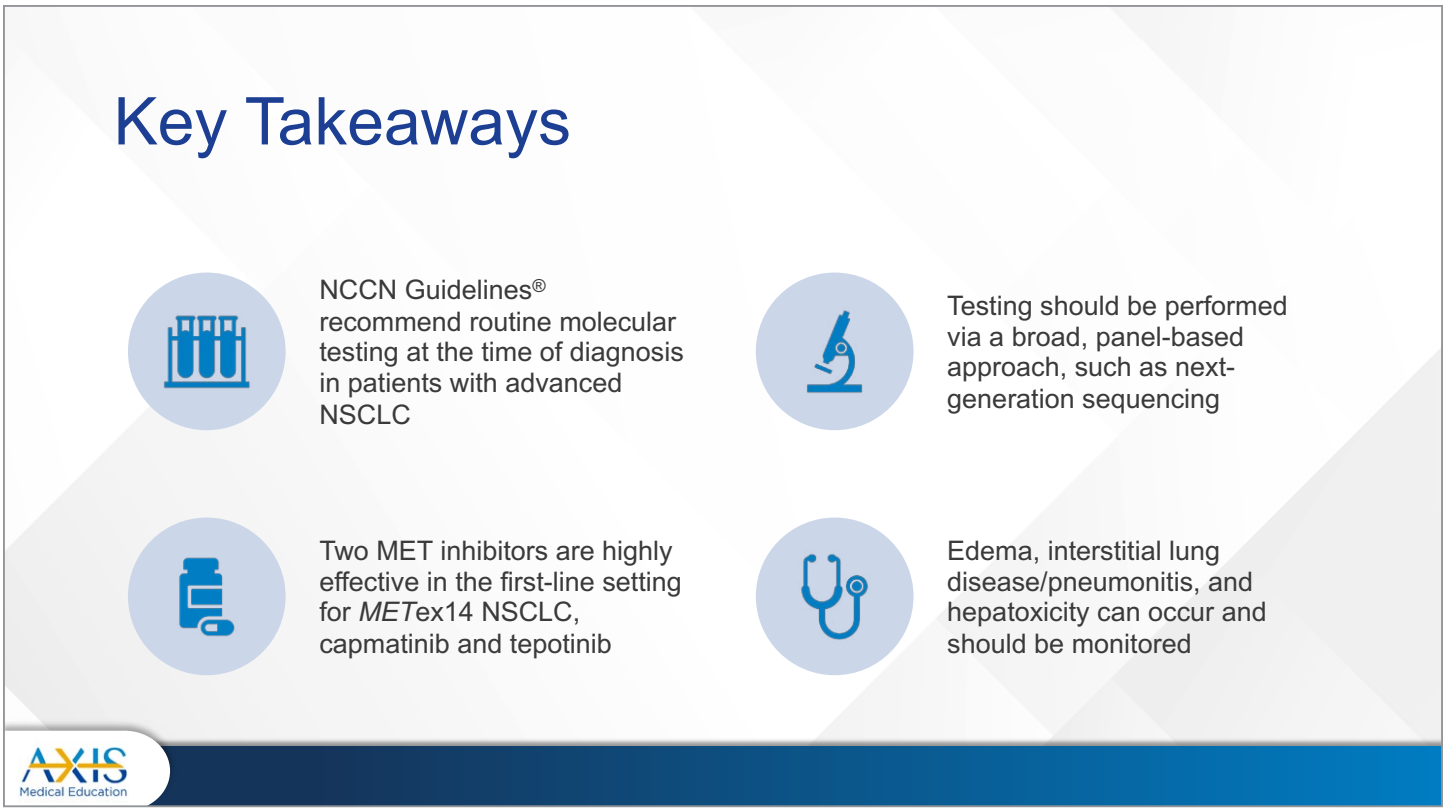
ALT, alanine aminotransferase; AST, aspartate aminotransferase; ILD, interstitial lung disease.
Capmatinib prescribing information, 2022; Tepotinib prescribing information, 2022.

Dr. Socinski:
This is the one thing that stands out about this class of drugs is the edema, and as I mentioned before, the pathophysiology of that in optimal management remains somewhat elusive other than the common sense sorts of things. We also see a little bit of GI toxicity.

But I wanted to get back to Patrick, and say, for those patients who you suspect interstitial lung disease (ILD), while on a MET inhibitor, is this a drug-specific side effect or class effect? Would you rechallenge patients if they've resolved? Or what are your thoughts about the ILD issue?

Dr. Forde:
This is something we've become more used to managing as oncologists over the past few years, with immunotherapy and more recently with targeted therapies, and so in particular the TKIs for example, for EGFR. In the MET space, both of these drugs can lead to interstitial lung disease, and it can be difficult, as we know, to differentiate this out from the underlying lung cancer, from infection, but once we do that, and we discover that we're attributing the changes in the symptoms to the drug, it's recommended that we discontinue that drug if a drug-related ILD occurs. In general, I would not recommend switching to, say, tepotinib if it happened on capmatinib, or vice versa, because it is more of a class effect and something which can recur if the patient is treated, particularly if it's a higher grade event.

Dr. Socinski:
I agree with that, and I would also make the point that the same thing would be true for the edema. If you, say, develop edema on capmatinib to the point where you feel like you have to discontinue the drug. I don't think switching to tepotinib would be a good strategy, because you would probably end up with the same degree of edema.



We're getting toward the end of our time. This has certainly been a great conversation and opportunity to answer the questions we got from clinicians about *MET* exon 14. I'd like to wrap up by providing a few take-home messages and those messages would be we can't emphasize enough the importance of comprehensive genomic testing at the time of diagnosis in advanced stage non–small cell lung cancer. Optimally, this should be by a DNA/RNA-based next-gen sequencing platform since there are 9 or 10 different alterations that we need to know at the time of diagnosis. If you identify a target, we have targeted therapies that are highly effective. We've discussed today two of them in the *MET* exon 14 space in terms of its activity, and that would be following the paradigm of getting the right treatment to the right patient at the right time.

With that, I thank our audience for listening in, and certainly thank you, Dr. Forde and Dr. Reckamp, for joining me and for sharing your incredibly valuable insights and expertise. It was great speaking with both of you today.

Dr. Reckamp:
Thank you so much.

Dr. Forde:
Thank you so much.

REFERENCES

Ettinger DS, Wood DE, Aisner DL, et al. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) Non-Small Cell Lung Cancer. Version 4.2022. © 2022 National Comprehensive Cancer Network, Inc. https://www.nccn.org/professionals/physician_gls/pdf/nscl.pdf

Garassino MCC, Felip E, Sakai H, et al. Efficacy and safety of tepotinib in patients (pts) with advanced age: VISION subgroup analysis of pts with MET exon 14 (METex14) skipping NSCLC. *Ann Oncol*. 2021;32(suppl 5):S949-S1039.

Socinski MA, Pennell NA, Davies KD, et al. MET exon 14 skipping mutations in non-small-cell lung cancer: an overview of biology, clinical outcomes, and testing considerations. *JCO Precision Oncol.* 2021;5:653-663.

Tabrecta (capmatinib) [prescribing information]. August 2022. Novartis Pharmaceuticals Corporation. https://www.novartis.com/us-en/sites/novartis_us/files/tabrecta.pdf

Tepmetko (tepotinib) [prescribing information]. February 2021. EMD Serono, Inc. https://www.emdserono.com/us-en/pi/tepmetko-pi.pdf

Thomas M, Garassino M, Felip E, et al. Tepotinib in patients with MET exon 14 skipping NSCLC: primary analysis of the confirmatory VISION cohort C. *J Thorac Oncol.* 2022;17(9):S9-S10.

US Food & Drug Administration. May 6, 2020. FDA grants accelerated approval to capmatinib for metastatic non-small cell lung cancer. https://www.fda.gov/drugs/resources-information-approved-drugs/fda-grants-accelerated-approval-capmatinib-metastatic-non-small-cell-lung-cancer?utm_campaign=PR-%20Lung%20Cancer%20x1_%20PEG-21-MS00775&utm_medium=email&utm_source=sfmc

US Food & Drug Administration. February 3, 2021. FDA grants accelerated approval to tepotinib for metastatic non-small cell lung cancer. https://www.fda.gov/drugs/resources-information-approved-drugs/fda-grants-accelerated-approval-tepotinib-metastatic-non-small-cell-lung-cancer.

US Food & Drug Administration. August 10, 2022. FDA approves capmatinib for metastatic non-small cell lung cancer. https://www.fda.gov/drugs/resources-information-approved-drugs/fda-approves-capmatinib-metastatic-non-small-cell-lung-cancer

Wolf J, Seto T, Han J-I, et al. Capmatinib in MET exon 14–mutated or MET-amplified non–small-cell lung cancer. *N Engl J Med.* 2020;383(10):944-957.

Wolf J, Garon EB, Groen HJM, et al. Capmatinib in MET exon 14-mutated, advanced NSCLC: Updated results from the GEOMETRY mono-1 study. *J Clin Oncol*. 2021;39:9020-9020.

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The executive leadership of AXIS combines 75 years of experience in adult learning theory, curriculum design/implementation/assessment, continuing education accreditation standards, and medical meeting planning and logistics. Our team has a deep understanding of the governing guidelines overseeing the medical education industry to ensure compliant delivery of all activities. AXIS employs an experienced team of medical and scientific experts, medical writers, project managers, meeting planners, and logistics professionals. This team is dedicated to meeting the unmet educational needs of healthcare professionals, with the goal of improving patient outcomes.

AXIS believes that partnerships are crucial in our mission to deliver timely, relevant, and high-quality medical education to healthcare professionals. To that end, AXIS partners with other organizations and accredited providers to offer added expertise and assist in expanding access to our educational interventions. AXIS also partners with numerous patient advocacy organizations to provide recommended patient education and caregiver resources in specific disease areas. AXIS finds value in these partnerships because they complement our core clinical curriculum with validated and relevant supplemental resources for busy clinicians and their patients.

The mission of AXIS is to enhance the knowledge, skills, competence, and performance of the interprofessional healthcare team to ensure patients receive quality care, resulting in improved patient outcomes. We engage healthcare professionals in fair-balanced, scientifically rigorous, expert-led certified educational activities designed to foster lifelong learning that is applicable to clinical practice and patient-centered care.

To learn more and to see our current educational offerings, visit us online at www.AXISMedEd.com.

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