I think we are done. We'll move on to the panel discussion on EJFR- mutated lung cancers for the past two sessions. I would like to invite our moderator, Dr. Hulaz Batra, who's a senior medical oncologist from Rajiv Gandhi Cancer Centre Delhi. And our panelists, Dr. Praladheela Mardi, Dr. Manoj Mahajan, Dr. Roshni Pandey is I think not here. Dr. Pradi Pandapati, Dr. Pridesh Munod and Dr. Deepi Zao. I think somebody's earpond is... So I think Hulaz has not been introduced properly. He's one of the finest lung cancer oncologists in the country. I am going to get... Anyway sir, 11 abstracts in 30 minutes is a kind of a too much even for Dr. Sahu and us all combined together. But I thought I'll do initial analysis and you know we will try to do everything in one. But I think I have just divided my moderation into two lines. The first line, the second line, and then some real world practical scenarios. So when we talk about EJFR mutant non-smaller lung cancer trust me that last two years back, I mean the discussion would have been boring.
I mean all that you discuss is the NK regimen and you discuss OC-Mirt rhythm and which one will you actually give. But from the last 20, 23, 24 especially since the last SMO, I think the ECFC, EJFR is absolutely fantastic and you know now we have so many presentations which are there and it's going to be very nice and fun to treat EJFR mutant non-smaller lung cancer. So I'll start with this side Dr. Manoj. Your choice of treatment of first time EJFR mutant non-smaller lung cancer and Vanita is in the house.
So you are advising me one by answer. No I'm just telling you that. I still don't give Jeff to me. I still don't give that to Jeff. I'm telling you one on record here.
Yes sir, if the financial toxicity is not concerned then we start, go with OC. But most of the time it is a concern. So standard is either we combine Jeff plus PEM or if patients desire to go for a single legate. Dr. Sahu is going to go to Goa next one. Dr. Sahu, it is not yet time for Goa. You cannot, Dr. Sahu's eyes lit up like this. I wanted to, you know, that is why I purposely look at this. I hope there's a video of here which I can show you to your wife Dr. Sahu. It is just because I just got late and this was the only one that was available. So just fast smiling now Dr. Sahu. Okay.
Dr. Simeh Nipse.
OC Martin, yes. OC Martin, if not affordable, PEM car was F.
OC Martin, otherwise I am K. Rajaman.
So I think it cannot be a straight answer. Dr. Sahu, don't look at the picture over there.
Just look up and then talk.
I think it's not going to be a very straight answer. The type of mutation, the bulk of disease and the financial all put together will be...

Around 100 patients of lung cancer each year from...
So if I don't have a brain meds, it will change. So let's say I don't have a brain meds,
I probably will go for a third-gen TKI finances, not issue. But if I have a brain meds,
it will change totally. If finances is the issue reality, the Jeff Taney permit,
except protocol does come in. The take home should be that if you use a first-generation,
it should not be alone, it should be with something else. That's what it should be.
TKI plus something, fantastic.
If affordable third-generation TKI OC Martin, if not in carriage.
Okay. Nandini?
So like Sahu said, I've taken to multiple factors, whether there are any co-mutations,
volume of disease brain meds. So if in an ideal scenario, if the patient is very fit
and some adverse co-mutations are there and a large volume disease may be osmotenip chemo,
if the patient is lower volume disease, maybe not as fit osmotenip, and a lot of patients
who have uncommon mutation cannot afford osmotenip still have large volume do well
with Jeff Taneyp chemo therapies, who we commonly use Jeff Taneyp chemo therapy.
The NK regimen.
NK regimen, but we also use now osmotenip chemo in certain...
Not as a part of the co-mutations, even 1921 differentiation.
Okay, we will come to that. We will come to that.
So we will start with the first one. The OS update of the NK regimen in for people
who are just trying to embarrass Vanita more. The NK regimen has just been coined by
M.H.E. that term. That is the Narunoha Kumar kind of a regimen. It's an opposite
of KN regimen. And this was a research letter which was published in JAMA, I think.
And congrats, Vanita. I mean, this was 2023, but this was one of the more defining
moments of my journey as a medical professional to seeing you up there in the podium at 20, 18,
2019 was fantastic. Yeah, but I didn't get any Google's
NAP of that. So that is why I had to put 2023. But that's it.
So this was the five years of I will update where we basically got 27.6 versus 17,
17 months median OS. So we will start with Dr. Sahu.
Does it really work as well in the... I mean, you know, now that I have said congratulations
and put us NAP, let's get to real stuff. Does it work as well? And what are the toxicity that you have seen?
It works. That's for sure. But because as a comparator, see, if you're comparing with
the Oseumatinib, then it's a different ballgame. You're comparing with the Jeffatinib
addition of it works. That's for sure. I don't have an experience of 50.
I may be at 15, 20 patients. So on that experience, I can tell you, yes,
it is toxic more than what you would expect from a single agent. You would not be using it for 75 years old,
and it needs to be toxic. And the first two, three cycles is something,
first two cycles, something which I'm very careful. And then so, but it works.
And the good thing about it is, as I told you, even if your brain mates and their data showed,
and even if you look at Chinese data and the Japanese, also with that regimen,
it works better than Jeffatinib. In the... I think, but in the patient who had brain mates,
a lot of people had taken a whole... They had taken a whole brain. They had taken a whole brain.

They had taken a whole brain. They had taken a whole brain. Fantastic. I agree.
So it does work, but my concern, even with Florida too, when the NK regimen has been the kidney toxicity,
I mean, somewhere down the 8 and 10 cycles, I mean, initially we didn't know.
I mean, now that even with you go for the keynote 189 also, you find out about 13 cycles,
you actually have to stop by matrix set for some part or another. So that is...
That is obviously there. A low incidence of T790M
Manoj... I mean, they just showed 17%
this... I read here. I read here. I read here. 17%
rate of T790M. Did you read?
You were an author. You were an author of that. Okay. 17%
rate of that. Is it because... Because Florida too, we saw there was not much of change in the resistance mechanism. So what could be the explanation for that?
Really not sure. We are not really sure. So let's ask Dr.
Vannita, only Dr. Vannita. I mean, do you think we didn't test much?
Do you think what happened actually over there?
It's okay. You're loud enough. So 17% was from the original
paper with that and that at that time that was a very early
analysis. So a lot of patients are not progressed, etc.
So we are... Dr. Shiva, my student is actually
going to be publishing. No, I already analyzed. Going to be publishing the resistance mechanisms. There is a definite difference
in T790M emergence between the two arms. Far more in
Jefitenib than Jefitenib plus chemotherapy. Okay.
Exact number, I don't remember. I think it's like some maybe two. It's not what is described in the
chart. It's not as... It's like maybe in the 30th or 25th or
38th. It also depends upon the kind of testing that was used.
Yes. Actually, if you use a DDPCR, you will get much more if you use a normal capacity, you will not get that right. Absolutely. So the majority had tested with RTPCR. So not NGS testing, I have progressed. So I mean, again for the residency, you know, it is very easy to say T790M. But see whether it was not tissue
by the... see to the... they're not liquid by the upsea. If you do one DDPCR which is not yet available because the reagents during COVID actually got finished and the companies are not really promoting that, it actually...
it's a very sensitive technique. So it will become actually more. So just saying liquid by upsea or NGS or which technique is very, very important on that.
Any data about... about Nandini, about L85 at our
DEL19 commutations or there was no data, I guess. I mean, at least not what is reported in it. But still... so, Dr. Prateesh, what if
patient is affordable and OSIMART NIM is available?
Will you go for this or you will go for OSIMART NIM?
Go for OSIMART NIM. Fantastic. So, now the next question over here is
to you Prateep is, do you think in 2024 a treatment with a single generation, third gen... even third generation DGI? Is it good enough? Or is it not good enough on some or most of the EGFI mutant patients? I mean, do you think
OSIMART NIM was a gold standard before 2023, you know? I mean before the FLORA2 and MARIPOS everything came. But do you think it is good enough or you would want to add
OSI plus something in some of the patients? I still feel it is good enough, sir.
Not only in terms of efficacy, more in terms of the quality of life of the patient.
Quality of the patient, you are a strong believer of the Singaporean study which will come to later.
Yes, your take. Especially if the patient doesn't have a
TP-50 mutation or some other extensive liver extensive metastatic disease.
So, what proportion of your patients you would be happy? I'm just a hypothetical situation.

Happy in giving OSI plus combination. Let's say money is not an issue right now.
Only fit young patients. Approximately.
Less than 10% percent. Yes.
There are multiple factors as Dr. Savu was pointing out. For the volume, higher volume of disease,
probably I will not say... I'll say it's not good enough. Fit patient, very minimal disease,
OC alone would be a good enough. Out of the last 100 patients that come up,
in the last one day, anybody, what does the thing of floratope, how many people have you given Dr. Savu?
Floratope, none. Why? I don't have patients who can afford OSU.
So, let's say... I'll give it.
If I have a patient, I'll...
Seriously now,
I'm taking it forward. Who would be looking... you would be looking beyond OSU, if I have to look into it.
So, how do you decide?
Yeah. So, I feel the brain meds is one point which I'll be very seriously looking into.
They are going to do poorer, but the hazards look much better for the brain meds when you add a chemo.
The TP53, I'm no more that convinced with the Exploratory Analysis data.
We may criticize Exploratory Analytial data, but the response date, if you look at the publication,
it's only 5% more. It's not a huge jump in the response.
Ocimatib, anyway, is doing so great. So, I think the brain meds, 40% is a patient or brain meds which you have involved.
Liver meds, Dr. Vannita?
No, I just wanted to ask about the brain meds. I totally agree with you and I have also tried it.
Whatever I've seen is the patients who have those brain meds, usually are the little bit poorer P.S. patients.
So, I'm not able to. So, I agree. I've been trying. I've maybe Ekadbar, I have given it, but otherwise they are not.
Actually, what we do, Dr. Vannita, is we do MRI brain for everyone.
And actually, the data which we have is 38% of people have got some brain meds at diagnosis
and not the big, big brain meds. 20% of them will have small, small, small asymptomatic
and we have been able to give that for everyone. So, just for the residents, how do you decide to
which give OCO receipt plus something? Look at the clinical factors, presence of liver, brain,
young age, high volume of disease or you look at the genomic factors that is the LH5,
Dhar, co-mutation and CT DNA is something which I'm going to come. We'll talk about in the afternoon session also.
So, the options that you have in the interest of time, I only tell you four options. You can add
chemotherapy, you can add ami-ventumab, you can add the favorite Ramisuramab also and you can
add North-Sar, you know, radiation oncology also, you can add something over there.
And let's do the cardinal sin of medical oncology. Let us just compare Mariposa versus
kind of a floratou. And what it basically does is that there is a slight increase,
HR is 0.62 for floratou, it is 0.70 for ami-lays plus chemo, so that is there.
Nandini,
do you think there is too much to differentiate between this or how you decide between, so one is,

let's say your FED patient has asymptomatic brain mate, has a Tp53 mutation. Now, how do you
decide Mariposa or floratou and money is not an issue? It's a difficult decision to make because
no one, I mean, they are not being head to head compared, compared. But the floratou has-
You're supposed to tell me to learn-
Sir, sir, sir, sir, floratou.
Come to the floor.
Come to the floor.
I'm sorry, I'm sorry.
I'm sorry.
And there you people say,
I'm sorry, I'm sorry, I'm sorry.
I send somebody for a second opinion, there's only the bushloaf.
I said, I don't know how that-
I didn't even know anything.
Just say yes to whatever we say.
So, don't need anything.
Please, please go in.
Although, I mean, sir, I would prefer to use floratou.
Floratou.
The only concern with Mariposa, though the data is good, there's so much of family ventilma related toxicity in patients.
So much of accessibility.
Uh, chemo-
maybe it's probably individual experience, but the chemo therapy, TKI, toxicity is better managed because-
Because we have been experienced that from the last-
Probably so many years.
I mean, so that is probably the reason.
Is it-
Yes, sir.
Yes, sir. You had a point and then-
Yeah, yeah, yeah.
Okay.
So, actually, the point was brought out.
The toxicity of my want-to-man management requires another understanding, another art.
So it's a different art which we will learn over time.
And the problem-
The problem will be solved to some extent when the substituting-
The sub-ute, the sub-ute one comes.
Here, stop the management.
So I feel the combination like TKI plus chemo combination needs some drug dosage management
to optimize the- maintain our optimism- efficacy.
Because we are dealing with the toxicity in both the syncy-
situation- Oc plus chemo or the Gf plus chemo.
So we need some dose-fine adjustment- probably generating data from the real world-set.
Okay.
Although with the- there's no comparison at all told,
but still, if I have a co-mutation I met, I will probably-
I know which agent I'll choose for.
That's one point.
You will choose?
I'll go for the ami-vant-a-man.
Waste poster call.

So second, if I have a TP53 with that Explode analysis,
I will choose a Mariposa rather than the floor-
I will like to take Dr. Sahu who has said, you know, what exactly was there.
So I just told you about the clinical factors that I will show you some graphs.
If you have brain meds, you know, let's do another grave cardinal-syn.
Now let us compare subset analysis of a cross-style comparison.
You know, when first we did a cardinal-synth, then we'll- let's do a more-
so for Dr. Sahu is smiling over here after this we'll go up.
That's a more cardinal-synth on his reunion party.
So, you know, I mean, have a video of that and see what is happening around.
Okay.
So let's compare a subset analysis of these two and I mean something which we should not do.
I mean, let us be very honest about it.
In which florid-to is slightly better than Mariposa.
Both in L-8 5-1 and TP53 mutation, Mariposa and florid-to-hids,
although there was no formal comparison of- in the florid-to about the TP-2,
numerically it was better.
In the liver meds also and baseline CTDNA also,
Mariposa was slightly better.
So my question over here to start with to you, Dr. Prithesh would be,
do you think, you know, we are eating too much into this data, you know, I mean, are we
actually torturing the data or you will decide it on the basis of a- let's say,
if you have a brain meds, you will give florid-to.
If you have a CTDNA positivity, you will give amylaz and chemo,
are we eating too much into this data?
You will just decide I'll give OC plus something, by not their same.
So there will be two things.
Number one, I'll give both options to patients.
Correct.
Of course.
When there are multiple-
When there are multiple-
Opel is a good one.
Multiple, say for example, as already mentioned, multiple high-risk features,
whether to use OC plus chemo versus Mariposa,
I'll just explain these are the side effects at the end of the day,
you have to choose, but I'll be more comfortable with OC plus chemo,
personally, because we are more used to-
Nice, good structure.
Fantastic.
Toxicity.
And Nandini?
Sir, there- I mean, we may be touching the data, but right now,
this is all we have to make decisions on till there is more randomized data,
probably we'll have to make it-
So you will say if somebody's CTDNA doesn't clear off in two months,
or it is positive, you will want to go for amylaz.
Somebody's a liver med because the data is more,
you will want to go for amylaz, somebody's brain, you will go for this.
Is this the way or you will say something and then we'll decide upon our own toxicity?
Sir, I think consider the data and then ultimately,
dissertation, fitness and toxicity.
Okay, I think this is all exploratory.
I think we should be taking the med is one which I think will be one of the guiding,
otherwise for brain meds, even the amylventa, Lazartini population-

Not a bad name.
Good.
No, Lazartini person is a very strong-
Who also with the amylventa med, chemo, they showed in good-
It's a good-
Good CNS, man.
It is some-
I think we're in too much end to this, you know,
I mean, we go to all these adaboards and company people come and tell us,
there isn't too much end to this.
I think the basic thing would be to say whether we want to give OC or OC plus something.
After OC plus something, it is your decision whether you want to go for the chemo toxicity
or you want to go for the skin and the anticoagulant shock.
So if we see wherever the data is coming positive,
it is because of the number of patients in that subgroup.
Yes.
If it is smaller in OC plus chemo 65, that is why it is not-
So I don't think that makes much sense to come.
I think we are torturing the data a bit too much with this.
So I don't think we should decide about whether you want to give single agent or combo
and then combo we decide on later over there.
Safety, very, very important.
We all know it's only the hematological and the nephrotoxicity which will be there in the floratou.
Dermatological infusion-related and VTE, very, very important is whenever you're giving me last,
please start them with Duax to start with over there.
So pros and cons I think we know of both the regiments.
The toxicity is basically a concern.
My take on whether it decide between floratou and maripar size,
apna apna d'eclo.
Jojis ko thik lege bodego.
As long as you decide about which one to give first, there is no one choice.
I will go to both.
I will promote both this thing.
But I think there is nothing, nothing will be to choose between the two.
So on Monday morning, Dr. Pradi, will you ask a broad-based NGS panel,
including met amplification, co-mutation for your patients at diagnosis?
Yes, sir.
You will ask, yes.
Okay.
Dr Manoj, will you ask for MRI brain and consider giving PKIs plus something for majority of your patients?
MRI brain, we like locally advanced or we do commonly.
You look.
I'll put it in front.
Okay.
In all these, you will do commonly.
Fantastic.
And will you want to do gift DKI plus something with the proportion increase?
If A is symptomatic, then we can manage it.
Okay.
Or Dr. Sahu, what says and happens, what happens in Vegas, stays in Vegas.
What happens in ViAR, stays in ViAR.
And what happens in Goa, stays in Goa.
Will you change your practice or you will not change your practice?

So which practice?
Your practice.
How will it go out?
No.
My point is, we already, see, we have to do MR and flora and flora.
The metamplification?
The metamplification, I think it makes sense with the drug which is already there as a
bi-specific.
The MR brain should be now done.
The flora, flora to just data set to look into.
Okay.
So, so if you look at it, you see that 20% in flora comes to 40% flora.
Are catching nearly double the brain nets, although most of it is symptomatic.
Yes.
Dr. Bhanita.
I think yes, up TK, I really want to know what is happening in Goa and can we also come along?
Kia hai.
So what is happening in Goa?
You, we will not never know and you can never come along over there please because what happens in Goa,
let's say is in Goa.
So for me, definitely a NGS panel, we will ask for, we will definitely ask for metamplification by fish also, which a normal panel doesn't really cover.
Considering MRI brain definitely, I will not, I mean, you know, we have been trying to get a
florid to regimen for a lot of people, but over a period of time, I think not more than 10% of
people or 15% of people actually are willing for a chemo plus OC combination because you know,
first you do an NGS test, you tell them what is the advantage of NGS test that, you know,
you can avoid chemo.
Then when the NGS report comes, you said you want to give chemo.
So it becomes difficult.
I think the way we have to counsel them.
Just keep this light out of the context, huh?
Just feel that a person is read two nights telling the same answer, I am not read anything
in a Goa mode.
So randomized trial, so don't read much.
Okay, so that this one was basically, this is very, very important for me.
This was a trial which was a DS-Kelishabee, did for intensification of therapy.
Now let's do what the de-intensification of therapy.
With this, Dr. I think I'll go to you, Petesh.
Can you cure bio-addicted non-small addicted non-small cell lung cancer stage four?
Because this is what they're trying to do, they're trying to shock therapy over there.
Are you a believer of concentration, RT?
This is something which is practically very, very important.
Are the current molecules that assess sensitive enough and guiding treatment decision and does
re-challenge work in biomarker, it's been long small.
So this is, for me, this paper and the PEM, car wipper was very nice.
So you are taking on this.
So cure, no.
Believer of consolidation, RT.
Believer of consolidation, RT in metastatic, no sir.

Sensitive enough in guiding treatment decisions, yes.
Re-challenge work, I've not had the experience.
Nandini, you are taking on this.
Cure, I wouldn't say no, because CML also was like, no, no in the past and now we are going for TFR.
So we don't know.
So we don't know.
No, we don't know.
So probable cure may happen if techniques of, I'm sure the SAs are sensitive enough now,
but we may have more sensitive assays in the future and the concept of MRT may also come
into play, which is so much of data, which we discussed yesterday also.
Consolidation RT in our center, we have trial ongoing to look at it.
So in certain patients who do well, they are looking at it and now there is upcoming data for
even an EGFR whether a concentration RT may or may not help, but right now there are mixed results
with the energy at U002 showing no benefit versus benefit.
So I think still evolving data and re-challenge in biomarker, there is a mechanistic resistance
mechanism between not work, but if we stop because of some other reason that toxicity then may be
reached out.
That's our hope.
Mostly agree with all the things except for the yes,
consolidation RT in oligometastatic setting.
It's all you already said it.
Or a no, or a induced oligometastatic, it really especially in the EGFR setting really
the data is encouraging.
So my take on this is can you cure a biomarker rated long term control?
Yes, cure I am not so sure you will have to give something over there.
Believer of concentration RT we do.
I mean, but please remember, a consolidation or oligometristic protocol is going to turn a
good disease into a better disease.
It is not going to run up poor disease into a good disease.
That's the difference.
Somebody is doing well.
You absolutely make it better, but not a poor disease into a better disease.
Are the current molecular assays good enough?
I don't think so.
I think we need much more assays over there.
Does the re-challenge work in biomarker rated?
It is reasonably well used for sometime if there's no resistance,
but we need more studies on that and that is very, very important.
So now plan B and we have five more minutes.
So let's say somebody has got an OC mortality.
How do you manage a patient on, let's say we have given OC what is the first time?
So I'll go to you Pradeep.
How do you manage a patient?
Somebody has got a good systemic progression.
Then what happens?
Do you order biopsy?
No, sir.
We are not doing biopsy.
So anybody who does a routine biopsy?
Yes.

So Nandini, what have you got till now?
How many times?
Maybe 15% times.
And do you act on that met?
You do ask for that.
We have treated patients with tipper and everything.
Do you biopsy routinely?
Rotally.
Rotally.
You get something out of it?
Yeah, I think.
Sure.
Not sure.
Because again, very important.
I have occasionally seen the transformation.
Yes.
And that is something that makes the decision.
So we need to look at the plan B, Ashwin is going and we hope
and Rohit and Coley go soon.
So we need to look at the transition from plan B.
The resistance pattern, I think I'm just going to change.
The plan B options are here,
but just again for the residents over here.
Once you have our resistance, look at the three things.
One, you look at the on target.
One, you look at the off target.
Third, as Dr. Sahu says, you look at the phenotyping.
If you have not yet understood, just remember this.
This is Shah Rukhant since Dr. Sahu is going to go to Bua
for the reunion.
So you know, you can have an off target mechanism.
You already have somebody in Bhopal
and you look at the reunion.
That's an off target mutation.
You have somebody in Bhopal, the girl and the guy becomes better
after the union.
That's an on target mutation.
The guy changes into somebody else.
That's an absolutely phenotyping transformation.
So you can have an off target mechanism.
You can have a resistance mechanism.
That's not me, sir.
This is a small cell transformation.
That's the way it goes.
So how do you, this is chemo immunotherapy.
So somebody with a PDL1 more than 80%
EGFR DEL19 mutant will you give a Pembro plus Pemcabo?
So post progression, only IEO plus chemo.
No.
No.
So that is absolutely there.
I mean, even the little girl knows over here.
So no irrespective of a PDL1, please do not go ahead
and give them any immunotherapy.
Now important thing is Mariposa 2.
Prithesh, your take on Mariposa 2.
Impress with the results.
Yes, but I've not given it.
You have not yet given because of the cost

and because that is also not available.
Again, very, very important is it's a good regimen.
But A, three things you have to look for is
one is the infusion-related reaction.
Please give the first drug in the ICU,
at least week in the ICU setting.
Make very good friends with the dermatologist.
Third is give the DVT profile excess and doe-act over there.
So ROI on this, I don't know how much will it get.
Our favorite.
Can I ask something?
How many of you have tried the 350 on day one and the rest?
We had to give in the trial, sir.
So how did you find the reactions, Alissa?
Nothing.
Same.
If it happens with the U.D., it happens with the U.D.
So it is...
Because the first dose usually is the highest and then...
So we do 350 and the other one and the first one goes to ICU
and then we give because in the trial we had a bad...
But again, it's a grade one, grade two.
But somebody is spending five lakh rubies
and having a grade one also is a problem.
I would like two minutes.
I'll just finish it off.
I would like Manos, you are...
I mean, you know, our favorite whipping boy of medical oncology,
artisanalismab and buvusosimab.
Do you think there is a chance for an IEO plus anti-angiogenic pathway in this?
Are you impressed with this result?
No, not yet, really.
Okay.
Dr. Pradi?
Yes, sir.
There is conflicting data.
So I think we should avoid giving in this situation.
We should avoid giving in this situation.
Fantastic.
Dr. Pratish?
ABCP regime and that's what Adiso Baye-Patli-Carbo is what I'm talking about right
now.
Wow.
Generally don't give.
Generally don't give.
Dr. Sahu?
It's alternative.
You have a choice in the guidelines.
You do it if you have...
You have finished one or two lines.
That's all of us go to that.
Okay.
But it's not a...
It's out of choice.
They got this.
It's a lottery for them.
Okay.
I think the only...
I mean, I will show you this data in the end over there.

But I think if there is a place of...
For immunotherapy in EGFR, we do non-small cell lung cancer.
That is this.
I mean, our TZO, Baye-Plusser combination.
And I think all these drugs have shown a good HR.
If you look at this 0.6, 0.51, 0.62, the HR is good.
And as usual, it's bad you will never get an OS actually in that.
So that's the issue.
This one.
This is something which is very, very important for me.
I'll go to you.
Yeah.
So you have given OC, OC Martinip.
If you get 100 WhatsApp questions, 90% you will last.
So should we continue OC Martinip with Bemit extra carb,
that is two more minutes.
So do you continue in the real blood practice?
No, sir.
You don't continue.
Fantastic.
Nandini?
So somebody has got OC.
And then you give on PD OC Pemcabo.
Sir, we don't routinely do it.
But I think, I mean, it could be considered in certain...
It's the volume of progression is important.
And that volume systematic progression.
New liver mess...
There are multiple new lesions.
I don't think that makes sense to continue.
To continue over there.
Actually, this is to me the most practical and doable things,
which can actually give you a 4.35 months of PFS.
However, there have been retrospectives studies
which have already said this concept doesn't work.
So please don't go ahead and look just because of Singapore
and Tony of the hospital.
The point here is that if you only keep on,
you would have got that 4.5.
Yes, 4.5 months over there.
So we don't know.
But the point is the only place where I use it,
if I really want to have a brain protection.
So somebody has got some brain meds,
asymptomatic, you don't want to really...
So OC plus femme is a good regimen for the brain.
That's it. But it needs further tiles.
You just look at this.
Actually, the maximum advantage that you are seeing is with
a 4- drug combination of OC and...
OC...
Sorry.
Atizo, Bev and the Singh, which is real.
So it is very, very confusing now.
And trials are very, very important.
So I will ask Nandri now.
Nandri.
On Monday morning, will you get a repiopsy
for most of the patients progressing on 30th year?

Yes.
Okay.
Will you consider giving IO chemo in patients?
Only IO chemo.
Pembroke, Pemka.
No.
IO chemo, Bev.
In the past few patients, but after in Power 151,
I'm not so confused that we should offer all patients with me.
Fantastic.
Mariposa 2.
Yes.
Is this possible?
Consider for our clinical trial.
Definitely.
Yes, yes, yes.
So I think this is what I even said.
I think, yes, I'll help.
We will.
Mariposa 2, regimen versus...
Tropian trial, which is going to...
Which is going to come.
So I think what we will do is,
yes, for our biopsy, look for met amplification.
Again, very important is,
don't send the 12 gene coupon panel.
It does not look for met.
We just now had a patient who had a herd to new.
We had a patient who had a be-raff.
So look for a broad-based panel in this.
Please do not go ahead and give them single-legent Pembroke
or Pembroke M-Carbo,
even if their PDL 1 is 120%,
which is not possible.
Consider giving ABCP 4 regimen.
Yes, I will consider.
Some data is there and that,
also because I'm going to speak on this session in the evening.
So I can't be saying yes,
no right now in case in the evening.
Mariposa 2, yes,
but make good friends with your dermatologist.
Will you consider for our clinical trial?
Definitely.
We would like to consider for that.
And with this, thank you.
Thank you, organizers for giving this opportunity.
And thank you for all the panelists.