As a panelist, I invite Dr. Manikandan, who is from nuclear medicine from Mumbai, Dr.
Shikhar Kumar from Hyderabad, Dr. Manavi Shuman from Rachi, Dr. Manshi Sharma from Delhi,
Dr. Omshri Shati from Mumbai.
Dr. Anil Manikandan, Mumbai.
All right.
So, good evening all of you.
I think we have very few people to understand what is happening.
The number of mutations are more than the people who are listening.
So, let us go through each.
We will not just abstract what was presented.
Let us see how best we can treat each mutation in our practice in the present day.
So, we have nuclear medicine consultant and let us start with him.
So, Dr. Manikandan, nuclear medicine and target mutation.
So, what do you have to say?
You know, we have medical on call we speak of p-values, whatever would you.
So, basically we can predict or like with our imaging features, sometimes we can predict
some like which mutation the patient can have.
Any example you want to give, like EGFR, this is the feature.
EGFR, we can like mostly they have a associated ground glass of pastities and they are like
solid but small tumors.
In early stage, they will be very small and they have a less chance for like the intratratic
metastasis in the early stage but they have a higher propensity for brain metastasis.
But if we move to like ALK positive, they are also solid tumors but the EGFR can have
aeroboncograms but the ALK positive are mostly solid.
Sometimes they can have cavitations but they have a higher propensity for the intratratic
metastasis like mediational nodes, plural metastasis and they have a they can have sclerotic
skeletal metastasis.
This rare mutations, most of the imaging features actually overlap but there are few
possible points to consider like in KRS, they are also solid aggressive tumors.
They have a high scheduled early on pet CT which represents the aggressiveness of the
killer.
Thank you, thank you.
So, there is some direction based on the images, maybe you can have some suggestions
about this.
Thank you.
So, let us start with JIT.
These are the two abstracts that was presented.
JIT will see, you know, it is not non-JIT will see.
It should be JIT will see with brain mates, you know, if you have second line post chemo
in you know, ad aggressive was better than dose it axis.
This is one abstract which we just, this is in which Dr. Darshit presented.
And the other one is Pembro chemo, Ollomer de Raseb, Ollomer de Raseb, second generation
KRS inhibitor where it can be used in first line.
So we saw data for ad aggressive in second line and we also saw data for Ollomer de Raseb

in first line along with chemo immunocompination.
So, this is how a KRS pathway looks like.
It is right in the top.
So it is mediating its signaling through both RAF pathway and also the PICK3 pathway and
it is important for, it is one of the first oncogene that was cloned.
It is seen mainly in smoke, smokers, males and mutated KRS means it is a switch, it is
a non-switch one and it keeps progressing.
And this is roughly what is the percentage of JIT will see and non-JIT will see.
JIT will see around 30% and rest all non-JIT will see around 70%.
And JIT will D has the best prognosis and JIT will V has the worst prognosis and JIT
will see somewhere in between and this is the only targetable one.
So why Dr. Nandini you want to share some experience and why this JIT will see is only
targetable.
So basically the structure of the KRS protein, the receptor has an on-state and an off-state
if the GTP bound is active and the GDB form is inactive for the longest time it was not
targetable but after discovering the structural pocket they saw there is a cysteine residue
so the recipe was actually designed to bind it and it is just behind that switch to which
is there and because it is able to bind it has become actionable.
Now the newer ones are binding more side so they are supposed to be more potent so that
is the reason why the structure of G2LC is important.
Thank you.
So one thing which we have to remember probably after this particular panel discussion is
G2LC is the only targetable as of now.
Why see?
Why not others?
Because C is for cysteine and it has a handle to which these drugs can bind.
If you look into the red one in that that is the only socket where these drugs can bind
whereas for non-G2LC as of now still drugs are maturing to bind but it is C, C for cysteine
therefore it is targetable.
So this curve just says that while type does good then mutant is a poor prognostic factor
and then this is one of the first KRS G2LC in a better it again had phase 2 and phase
3 the code break 100 trial with the good response rate and disease control rate with
an OS of around 12 months again this is second line trial but very importantly hepatotoxicity
40% if it was previous IO exposed and 18% without IO before and treatment disteroids.
So Dr. Shikha so we have so to rest in bed like you want to you know if at all if you
have access to these drugs any difference you want to state.
There does not seem to be much you know immediate look there is not much difference between
these drugs but I think adagrassip has been studied more in patients with brain metastasis
right.

One is yes very important one is both of them are in second line management of G2LC only
post chemo immuno but two important things one is the T half 24 hours whereas and the
end the intracurial responses are more impressive with adagrassip right and then comes response
to I mean if you look into the incidence of brain maths it is it is higher with those
with target mutations and KRS also has higher but they are not that radio sensitive therefore
you need to have CNS effective drugs and these are the resistance mutations and the
sensitivity so just like EGFR and all others that particular mutation is sensitive to that
particular drug.
So this is the crystal 12 trial which we discussed brain maths and this is the difference in
PFS and also in with and without brain maths and in impressive and this is one more devarassip
again showing impressive outcomes and non-G2LC the drugs there the mechanism is there but
it did not improve outcomes so non-G2LC is not a targetable.
Ollomerassip is a second generation G2LC inhibitor Dr. Manavi you want to share some
thoughts on Ollomerassip and using grass inhibitors in first line so this is how the mechanism
is like it locks in the inactive state covalent binding.
So if you look at this drug so it is having acceptable safety profile and it can be combined
with immunotherapy and chemotherapy and can be used as a first line as a monotherapy
capsule.
Right very important so Torelassip adagrassip are toxic enough to combine with chemo immuno
whereas Ollomerassip is safe enough to be combined plus it keeps the G2LC in a inactive
state with covalent binding.
So this is how the study looked like again just to show the differences this is the
response rates 50% objective response rates looks impressive and the phase 3 trial is
awaited.
Dr. Manavi you would take on these grass inhibitors which you just saw how do you know.
So I will summarize quickly first generation Keras inhibitors they have shown response
rates but they come with a toxicity profile and other response rates the PF is the OS
is not that mouthwatering right.
The other issue is because see Keras G2LC is also very immunotherapy sensitive and
first line we give immunotherapy or immuno-kimo combinations in these.
In fact there is some data to say that which says which has been seen that Keras actually
they respond better.
In fact we are going to publish our own chemo immuno data soon and that also we have seen
like 3 or 4 of our patients are extremely well on chemo immunotherapy.
So with the first generation TKIs the inhibitors you needed to have a wash out phase.
We were part of the trial one of the trials of the Keras inhibitors there has to be a

6 to 8 weeks wash out phase otherwise they land up with bad toxicities which were immune
mediated.
So that is one aspect coming to the first line setting we have seen with Flora too that
if you combine you know targeted therapy with the standard of care therapy you do get better
responses.
So working upon that and looking at pre-clinical activity of the combination of immunotherapy
and ulmarassif and talking about ulmarassif now like a second generation inhibitor there
seems to be some one positive sign that the combination is synergistic.
Second the toxicity is not that bad.
So this is a very I would say exciting field the phase 3 trial in fact we are going to
start enrolling patients on the phase 3 trial maybe next month at our site that is RGCI.
So we will also find out how it is going to be.
I would say that this is a very hopeful field and if the drugs do work together in combination
we may be looking at a new standard of care maybe a couple of years later.
Right.
I think very important points in the present day still we are using chemo immuno and this
is the results for chemo immuno ABCP 9LA 189 and also on they are shown efficacy even in
the RAS mutant subset as Dr. Manci was saying in the present day standard of care.
Though you want to say anything on the present day standard of care.
Right.
So this is the present day standard of care till now RAS inhibitors are there in the
second line post chemo immuno.
So in the present day first line standard of care is chemo immuno with or without VEGF
and second line you can use ad aggressive or sotoresib based on the approval we have.
So we are going on to RAS1.
So we do not have Dr. Rome tree Dr. Anthony you want to share some of the incidences
and experience of RAS1 in your practice IHC versus fish and so on.
So the incidence around similar less than 3% is what we see.
We are a center actually is started to do IHC but if it is positive then the confirm it
with an orthogonal testing so they do a fish or if there is an NGS report.
So we do not start treatment based on IHC alone based on the HCO they do a confirmatory
testing and ideally we would prefer to have the newer drugs for RAS but due to a lot of
times the resource limitations we commonly use chryzotenip for these patients and if
they progress and some of them do receive low latin.
Right.
So very important point IHC is screening but IHC is positive you need to do a fish and
confirm or an NGS and liquid biopsy has been some new development.
So liquid biopsy results are correlating with interacting responses.
So EGFR liquid biopsy and now RAS liquid biopsy is also concordant with the response

so this is some new development in RAS.
Dr. Manci you want to share any of your experiences of these RAS inhibitors in practice.
Okay so we have used chryzotenip in patients in fact we did analyze our data.
The median PFS in the studies were around 19 months.
RAS came out to be around 14 months.
Entrectinib we have not yet given in RAS positive patients but my trial has started.
I mean the trial for entrectinib is also starting soon and the trial for reputrectinib in India
is also starting soon both in the first line setting.
One allows one previous line of chemotherapy also.
So the overall response rates in these drugs and the studies that have already gone on
are good.
There is excellent entrectinib activity also for both entrectinib as well as reputrectinib
between chryzotenib, entrectinib and reputrectinib.
I would say reputrectinib has the best data in terms of 80s you know response rates being
in 80s including entrectinib response rates but the phase 3 trial we are waiting for that
data.
Secondly yeah one more thing if residents are here there is some data that even they respond
nicely to pemeteric-side based chemotherapy also.
So that should also be something that you should be aware of in regular clinical practice
as Dr. Nandini said we do not really not everybody can afford these drugs and they are not available in India as well.
Right so thank you Dr. Manci.
So this is how the Lola trial looked at.
So for post-chryzotenib progression or second line we did not have data and here is the
data of Lola dinner 70 patients impressive you can see the responses like 43% responses
in the second line post-chryzoten-chemo progression and even the duration of response
PFS looks impressive even in the second line setting.
So Lola tini can be safely used in the second line.
So Dr. Nandini your thoughts on Lola what about Lola line.
So we have been using in ALK and now Ross.
So this study also shows that this objective is sponsored and most important is the site
of progression which is very often intracranial has a very remarkable control with the Lola
tini black we have seen in ALK.
So I think this is a good strategy and I think Lola tini was also one of the terms which
we have in India.
So I think failure on chryzotenib this would be a good option if we can.
Right.
Dr. Shikha we know for EGFR it is T790M and for ALK it is 1202R any thoughts and required
resistance for Ross.
So like we have the solvent front mutation for ALK, G2-0-3-2-R which is the most commonly
characterized mutation on chryzotenib that is this mutation.
Now when I read up about Ross I looked at the data set of Lola tini post-

chryzotenib
disease progression and it was very clear that Lola tini does not have any good efficacy
against the G2-0-3-2-R mutation because out of six patients there were zero responses.
But when it comes to repo tectinib we got that Trident1 published in the New England
Journal earlier this year in that almost 55% patients responded in those who had the G2-0-3-2-R
mutation.
So I think that is where I think we are headed now that ideally those who progress on chryzot
or entrectinib you end up looking for the G2-0-3-2-R if they are positive for that then
probably by that time if repo tectinib is available you put them on that and if that
is negative then probably they can also give them Lola also.
Right very important points G2-3-0-2-R in Ross and G1-2-0-2-R in ALK these are the
resistance mechanisms these numbers you need to make note of and we have the first in the
present day if you have Ross positive you can use TKI in the first line setting chryzot
in a bar entrectinib or the treatment of choice and once they progress you have some data
for Lola tundib and as Dr. Shikhar was saying repo tectinib and Dr. Manci also delve into
it the response rates are best with repo tectinib and there is one drug NVL 520 that I have
shown very promising once it is mature we can use this just to pressure.
And not just the responses because anyhow in the high 70s and 80s but it is the duration
of response which was very striking with the repo tectinib it was around 35 months
but with all the other chryzot-n-trick Lola it is around 20 months.
The repo tectinib looks to be I think it is going to be launched in a couple of years
or so but it is going to be important repo tectinib is the game changer but Ross positive
in the present day TKI is the treatment of choice.
Then coming on to this drug T.V. Telly saw Tuzumab video tundib.
So we all know met is a game changer met it binds to hyperto side growth factor and mediates
the signals causes proliferation and so on and we just saw the presentation.
So met amplification is different met exon 14 skipping is different and met expression
is different this met expression is just I H is just like hard to.
Hard to mutation is different you know hard to is in around 30% of breast something like
that whereas hard to expression 1 plus or 2 plus in enough to 60 to 70% this is something
like that this is met expression but it is not met mediated.
Met mediated is met amplification and met skipping.
So we should not confuse met I H C expression versus amplification and skipping met expression
may be there but there may not be met driven cancer.
So we should know how these are the criteria for reading of I H C from met 2 plus 3 plus
just like her 2 and if there is met positive it has some implication on survival.
So Dr. Manavi you want to share something about this new I H C biomarker and survival.

Sir out of the 3 met over expression is the most common compared to met exon skipping
mutation and exon amplification and does the poorest market for the survival negative
promotion factor it has if they are met over expressed on the cell surface.
Right so again so met expression is I H C met amplification and exon 14 skipping is different to the mansey you want to share any thoughts on this exon 14 skipping amplification
and all this.
Okay so as I was speaking with you earlier so met is very variable right we have multiple
biomarkers we have multiple drugs right.
So now we are trying to fit them all together that is exactly what is happening in the met
area right now.
So as we all discussed before met I H C as of now was not a biomarker on this trial it
may be a biomarker but I think more data is needed I think we need more trials we need
bigger studies to actually be sure about this as a biomarker.
Second thing is with regard to amplification so amplification you can do check either by
copy gene number or you can see by met sep ratio sep 7 ratio again lot of variability amongst
all the drug trials nobody has come to a consensus yet it is like TMB right nobody is really
come to a consensus yet.
Thirdly now we have three drugs in the pipeline for met exons skipping mutation we have targeted
treatment we know that works well we have a sub wallet and a ventapotanib which work
very very well we also have to result in it right.
So that is where more work is going on to obviously get better responses better outcomes
but as of now that seems to be settled with regard to amplification I think we need more
data especially with the two newer kids on the block one is slightly old now which is
your ami-vanta map where we do have some data that it works again no consensus on the amplification
part yes consensus on the exons skipping mutation part still not FDA approved or come
into guidelines right.
The new kid on the block the newest kid on the block is Tevolo I am sorry.
Teleso to Azuma Wado to me.
Yes sorry so this is a bi specific and you know this is not a bi-space it is an antibody
drug conjugate sorry so this is an antibody drug conjugate might take on this trial do
you want that also no yeah go ahead just talk okay.
This is how it is.
So my take on this trial is that yes with IHC 3 plus we did see some responses I think
around 30% 20 22% overall and around 30% right but there were toxicities if you look
at the toxicity data there were two treatment related deaths because of ILD and almost 10%
of patients had ILD so again and another thing was I think almost 90% patients or 80% patients

received immunotherapy earlier before they started it so these are all concerning factors
right I think if we do take this further we'll have to design a very good trial to take
into account all these factors right so there are a lot of red flags in this trial seems
to be something that we can you know might be able to target in the future as of today
I am not very sure.
Just a point that you know we like to add met inhibitors when there is met amplification
but the trial of T portinip actually it had two cohorts one cohort with met exone 14
skipping and one cohort with the application and it failed to meet the criteria for efficacy
pre-defined efficacy in the amplification cohort.
There is a vision study.
There is a vision study so that's what I mean you know we like to find these biomarkers
but the drugs might not always work in them you know.
So what you guys need to understand is met amplification not much data and met takes on
14 skipping kept metinip, T portinip fantastic and this is something new for today met IHC
expression something like her to IHC expression one plus two plus three plus is there if it
is two plus three plus there we go we have an antibody drug conjugate telesortosomeb
veroutin showing something so you may need along with alkyl C, ros IHC, met IHC also
may come in future if this drug comes into practice.
So we finished and now coming on to NTRK I think this is how NTRK it's common in rare
cancers and rare in common cancers so.
Romchi no problem mom you please come here so there were lot of slides you know where
you had to say I was struggling to explain no problem.
You could have come directly you know right.
Right so we were speaking of NTRK so NTRK this is the ones you know the rare cancers
you see high expression whereas in common cancers you do not see them it's rare in common cancers.
So let's come to very important is NTRK postivity we do see in the reports of NGS but we need
to know what is important what is pathogenic not all NTRK mutations are pathogenic for
NTRK to be positive you need it should not be SNV it should not be a CNV and this is
how so mom you just want to add on this was a slide for you NTRK postivity we are very
excited but not all NTRK postivities are actionable so which NTRK you would say that for a clinician
for us that it here is the mutation that we can act.
It's the NTRK fusions that we are looking at specifically NTRK 1 NTRK 2 NTRK 3 again
in that there are those in frame fusions are the one which would be the one which to look
for because the outside the like frame mutations would not lead to any truncating effects so

the best thing to go for is to in frame fusions but any which ways in NTRK fusion is always
a good news but any other like in an NGS report when you are doing SNVs you see many of those
mutations but mostly that need to be traded with a caution that is not something that
you can jump up and say you know I got something so it's only the fusions and there are IHC
like you know PANTI RK IHCs but that is still not a foolproof method so the best thing to
go for is to either do fish or NGS and just confirm it.
Thank you. So very important is NTRK IHCs was cleaning NTRK IHC positive or some SNV
positive does not mean it is actionable as Madam said it should be a proper fusion it
should be an in frame means it should be the coding region and the kinase domain should
be intact and it should manufacture a functional protein only then it is actionable just some
SNV some rearrangement or loss of kinase domain these are not actionable so we should
be making sure whether this is a actionable NTRK or not we should not be excited just
I see an NTRK and that is where to go right so I think this is some data of LEROTRAK TANIP
is not accessible to us impressive very impressive CNS fantastic duration of action so anyone
anyone has any NUTAN you have any experiences of and interact in practice giving some drug
and doing well not LEROTRAK TANIP but on compassionate access there are one or two
patients who are getting interact in it before the compassionate access program close but
they are still I mean disease control for about more than two years so there was one boy
oh some 60 16 year old boy with but Jari's syndrome sarcoma something filled with liver
NTRK IHC positive and the tissue was not available and he didn't if he was in
the cabo and with cabo he did extremely well so cabo is something like a pan TKI maybe
that is also used when you don't have access to these drugs now coming on to BRAF okay
quickly in three minutes BRAF is important you see in multiple heresilukemia melanoma
and so on and we have three types of BRAF and what matters is 600E right class 1 2 3
and it is drugable is only BRAF V600E just like EGFR it is heterogeneous and the best
prognosis is V600E and it has map kinase activity and V600 non-V600 this differentiation has
to be made it is V600 that is targetable so Dr. Ampray again if you want to test for BRAF
in the present day you know what is the way to go so of course I mean if NGS is not accessible
earlier Sanger sequencing was the only thing and it is still the gold standard but the issue
with Sanger is that the limit of detection I mean you would go as low as maybe 10% or
in an extremely good scenario maybe max 5% but not necessary that every time you would

get that and now BRAF IHC is also there which is very specific to V600E then comes to NGS
is something which would be able to pick up everything like you know an exon 15 of BRAF
you will be able to pick up Sanger D non-V600E class 1 class 2 class 3 any of it having said
that the targetable one is the BRAF V600E but even real time PCR assays are also there
but sometimes those assays would just give you like you know V600X they will not tell
you the exact genotype so that is the caveat when you are sending anything for real time
PCR ensure that those laboratories are giving you because real time PCR is also a sensitive
technique but most of the commercial kits usually do not say whether it is E or whatever
other part is non-V600E that you are not able to pick it up another excellent tool would
be DDPCR if it is accessible and if it is available number one it is cheaper number two
it is extremely sensitive it is even doable in liquid biopsies and even for follow ups
if BRAF V600E is initially detected so these are the various testing methods which are
available thank you so NGS DDPCR this is the two things that we need to do so we need
to remember this is a Faro study I think we already saw the Faro study results and
Encoref and Binamatine versus Deb I mean it was not versus it was just seen whether tolerable
or not but Encoref and Binamatine the sequential inhibition of RAF and MEC this is what is important
and there is some comparison saying that Encoref and Binamatine probably does better and W
is not possible so in the present day yeah Dr. Shikha how do you treat BRAF inhibition
in the present day so to be honest I am not completely sold on BRAF MEC as first line
treatment for these mutations I presented some data on BRAF incidentally in the same
venue last year and when I looked up the literature there was plenty of retrospective data that
the tumor micro environment in BRAF mutant disease is actually not that dissimilar from
a oncogene negative disease and the PFS with IO chemo combination is actually quite good
so and we know how difficult it is to manage the toxicities of these drug with frequent
interruptions you know high levels of pyrexia and etc so first line would be in my opinion
IO chemo and then reserve BRAF making a bit as per second line.
Any experiences of BRAF?
Yes sir but like just like Shikha said giving BRAF and making sure the patient is able to
take it properly is a challenge and also since there is no real randomized data to say what
sequencing strategy is better these patients also do reasonably well with chemo and IO
and I think that is also a reasonable strategy use it first and use BRAF as second line no

one really knows so many of our patients do receive chemo and IO and then eventually
go on to receive BRAF in the second line probably we need to have any driver mutation
where you want to use IO is one is met and another one is BRAF I think HER2 we already
saw my my one minute comment of HER2 IHC you know they used IHC in lung for HER2 2 plus
3 plus so what is your thought of HER2 IHC in lung?
I don't think so it is recommended and I guess HER2 what we see in breast is completely
different than HER2 what we see in lung when we are talking about HER2 in breast we are
talking about amplification of the gene which is also corroborated with phase or IHC and
there is a recommended guidelines even for gastric HER2 there is a recommendation like
what need to be done but the same doesn't translate to I mean lung in lung what we are
seeing is basically the alterations I mean like HER2 exone 20 duplications or insertions
are the most common one which are not detected by IHC so I mean like if it is like during
the screening if HER2 sometimes it is directly because still the unified guidelines are not
there that you know you need to see for expression and one plus two plus would have some implications
only thing is that presence of HER2 mutations would indicate that you know other targeted
therapy can be given or response to immunotherapy and that is what it indicates so these two
are completely two different things when it comes to lung and when it comes to breast
right so to detect that again the best method is NGS because that would pick up any of
these exone 20 alterations like duplications insertions whereas maybe if there are CNVs
are there even that can be picked up which IHC will not be able to tell you so in lung
HER2 IHC would not give you much of information as much as NGS would give and most of the
panels do include HER2 as one of the genes in the panel.
So Raudhana I think this is the last trial which was read out you know like in this destiny
lung 0 3 they used IHC 3 plus and 2 plus for lung and they are showing some responses
to it what do you think I mean as of now as Madam said we are not using IHC so this is
some trial they are saying that you know IHC can also be used 2 plus 3 plus can be used
as a target what do you think.
So I think it is something similar to the CMA expression that we saw whether that is
a separate subgroup altogether who do not have the mutation but have a good expression
and how they would do because we know in second line TDXT has very good data and even
the ongoing destiny lung 4 we have some patients and they are doing remarkably well so maybe

HER2 mutations might be a different subgroup and maybe like the CMA kind of thing hurt
2 expression might be a different subgroup and maybe that might be effective in the process.
You made lot of sense if you are having a target mutation there you need something in the gene
but if you are adding an antibody drug conjugate like a TDXT or the TV which you saw the cell
has to have a surface expression but did not be driven by that particular gene just an
expression the cell surface is sufficient it may not be hurt to driven but maybe just
a surface expression that is what we are saying maybe in future we will see some HER2 IHC also
being a marker for these drugs because then they may not be driven but those are targets
where the drug will attach and get the chemotherapy moiety inside the cell to mancy.
Just one little thought actually we do not know the biomarker data for these population
you know somewhere it was mentioned that they gave EGFR TKAs also so we need to be very clear
about what biomarkers do these patients have before we go ahead and jump upon IHC 3 plus
you know GIFTEDXT and lastly I think one point for all the residents here that I would
want to make is on NGS all these fusions that we have talked about we look at them
better in RNA NGS as compared to the DNA which is like BRAF and all that or HER2 that we
look better at the DNA NGS right okay so that is a take home message for the students here
as well today.
So man made a very good point about RNA NGS I will share one experience of mine which I
know sharing probably all forums it was the only and first case of NTRCA1 case which
I actually picked up this was actually Mistona NGS because it was an amplicon based NGS in
our one of our local Indian labs and then the patient progressed and then I sent it to
foundation so they do a hybrid capture based NGS we all know that hybrid capture takes
up more fusions like met and NTRCA because of the way it is done and it came out an
NTRACMON positive so the patient did remarkably well on a LAROTRCA NIP.
Eight nine months of disease control in a heavily heavily treated radiative refractory
C.A. thyroid came off from almost oxygen support and gained 30 kilos.
Eight nine months of disease control now again of course he is having some progressions so
again we have again sent foundation to look at what kind of NTRAC mutation is happening
because we need to know that so that is what I want to say that RNA based NGS and also
look at whether your lab is doing regular amplicon based sequencing or are they doing
hybrid capture based sequencing.
All right okay I think we learned little about all the small small mutations and I would
thank all the panelists for their active participation and thanks again all of you.

Thank you.