So, good afternoon everyone. At the outset, I take this opportunity to thank Dr. Kumar Prabhash, Dr. Anuradhahan, Dr. Priting for inviting me here today to share the experience of a path kind, where sequencing of lung cancer patients is concerned. So, path kind is kind of a new entrant into this field. So, I have limited data that I would be sharing. I think it would be fair to say that cancer is actually a disease encoded in the genes, because now there is ample data to support the fact that whether we talk about the uncontrolled growth or the drug resistance or metastasis, but thankfully also the fact of targeted therapy, it's all because of the war genetic variants which are encoded in the genes themselves.

As a libertarian, the aim of genomic testing in oncology is of course to identify actionable targets as well as resistance markers, so as to enable the medical oncologist for better patient management and that is really what we focus on. For lung cancer at a minimum, as per NCCN, whatever panel, whatever stand alone testing is used, should ideally cover all these important biomarkers and mutations. So, in line with that, in our laboratory, primarily most of the testing that we do are NGS panels, having said that we are limited as a stand alone laboratory not only on NGS, but depending upon the patient requirement, we also do specific stand alone biomarker testing either using DDPCR, speech TR, FISH or IHC. An important case in point is PDL1. As of right now, at least in our laboratory, we are offering PDL1 testing only on the tissue sample, so hence just the IHC based. Which brings me to the fact that really whatever technology we use for oncology, for somatic mutation testing, tissue really is the issue, whatever recommendations, guidelines we look at, they very, very clearly underscore that this is the gold standard, the testing ought to be performed on tissue until and unless you have a scenario where the biopsy is either not possible, it has been exhausted and in only in that case, we resort to using the cell-free DNA or the liquid biopsy. The smallest lung panel that we use in R lab is the 12-gene panel and we use the platform the S5+, so the entorant platform is what we use and we perform a combined DNA RNA testing for this panel. The largest and this is actually the panel which gets referred more often, so the bulk of the data that we have from R lab is from the 52-gene panel which covers the relevant hotspots, the copy number variations as well as the gene fusions. The earlier panel that I had shown by and large in terms of the copy number variation, it only focuses on the met, so we know now that the herb B2 copy number

variations are
also important which is not covered in this panel but in the 52-gene panel, the herb B2 copy
number variations are also covered. For patients in which the cell block is cell or the cytology
block is either not available and we are going ahead with testing using the cell-free DNA,
the on-commine cancer panel is what we use and in terms of the genes the coverage, pretty much
it is like a sort of copy of the 52-gene panel that we do for somatic mutation testing.
So this is, this particular year we processed 564 samples for tissue biopsy for lung cancer
and of these 81.6 were adenocarcinomas and only about 11% were squamous. There were 7% which were
categorized as poorly differentiated carcinomas and 2% were small cell lung carcinomas, so this was the
histological distribution of the samples that came to us. Demographically there were 61% which
were male and 39% female and age wise we got most of our samples which were in the fifth and sixth
decade and overall we were able to identify variants in about 88% cases. There were 2.6% in which
the results were either unsatisfactory or we could not perform testing and about 9.9% in which
no variants were identified. Of these 88% there were 71% which were informative variants in terms of
being able to help the clinician for targeted treatment and between the various histologies
I have provided what is the percentage in which we were able to identify any variants at all
and versus not. So if we look at, though it on the face of it it looks as if like you know
in the squamous cell carcinomas we are able to identify less number of variants but then there
might be a skew there and because if we look at the total number of cases which were squamous
in the population that we had tested these are only about 50-60 cases. So there could be an
inherent skew because of the small number of sample size that we had for squamous.
This is the distribution and frequency of the variants that we saw in among the xenocarcinomas and in this we were able to identify variants in 91.2% cases
as like you know has been shown by other speakers also and in concordance with the already published
literature the largest mutation positivity was seen in EJFR which was 41.3 followed by TP53
RAS, ALK and so on. There were 9% cases in which we saw double mutations and most of these were
combination of two different kinds of EJFR mutations or EJFR with MET, EJFR with CT and BB
PIC3, TP53 and also combinations of PIC3 with KRAS and combinations of B2 again with
B2. So two different kind of mutations, point mutations as well as amplifications in this.
There were other cases to 0.4% in which there were more than so there were triple mutations
and beyond that. This is the scenario that we had for adenocarcinomas. Amongst these squamous
cell carcinomas 20% cases we could not identify any variants and the largest.

So this again is just to show that amongst the EJFR mutations the highest percentage
of the subtype that we saw was exon 19 mutations. For squamous cell carcinoma the highest percentage
of variants were seen in the TP53 gene followed by PIC3 and FGF2 along with TP53.
In the TP53 which was the highest one here we saw that the maximum number of mutations which
primarily were single nucleotide variants these were seen in exon 5 and exon 7.
So by and large this is like you know the data that we have we did process about 50 odd samples
for liquid biopsies as well but the data was kind of all over the place so I'm not showing that here.
So limited data but it's a start that we have made that path kind. So thank you.
Any questions?
If I don't hear the question with the questions.
So correct. What were they and did you take a follow up with the clinicians how did they take
the code? So the quad one is actually rather recent ones you know. So in fact like so but
for the double mutations yes we have a follow up of that as we do for the triple not for the
quad as yet. We have reached out but we don't as yet have information on that. But it was from
what I recall it was a combination of EJFR, TP53 along with CTNNB1 and I'm forgetting the fourth one.
Your total proportion of double mutant is also very high. So is this only EJFR double mutation or
EJFR with all genes? No so EJFR with so they're like you know three main ones EJFR with some other
genes which I've listed out here TP53 with a combination and PIC3 with a combination and a B2.
So four of these with combinations of other genes. Thank you. Thank you ma'am.