

DROPLET DIGITAL™ PCR (DDPCR™)

**BIO-RAD**



# What *do* *you* **Gene**

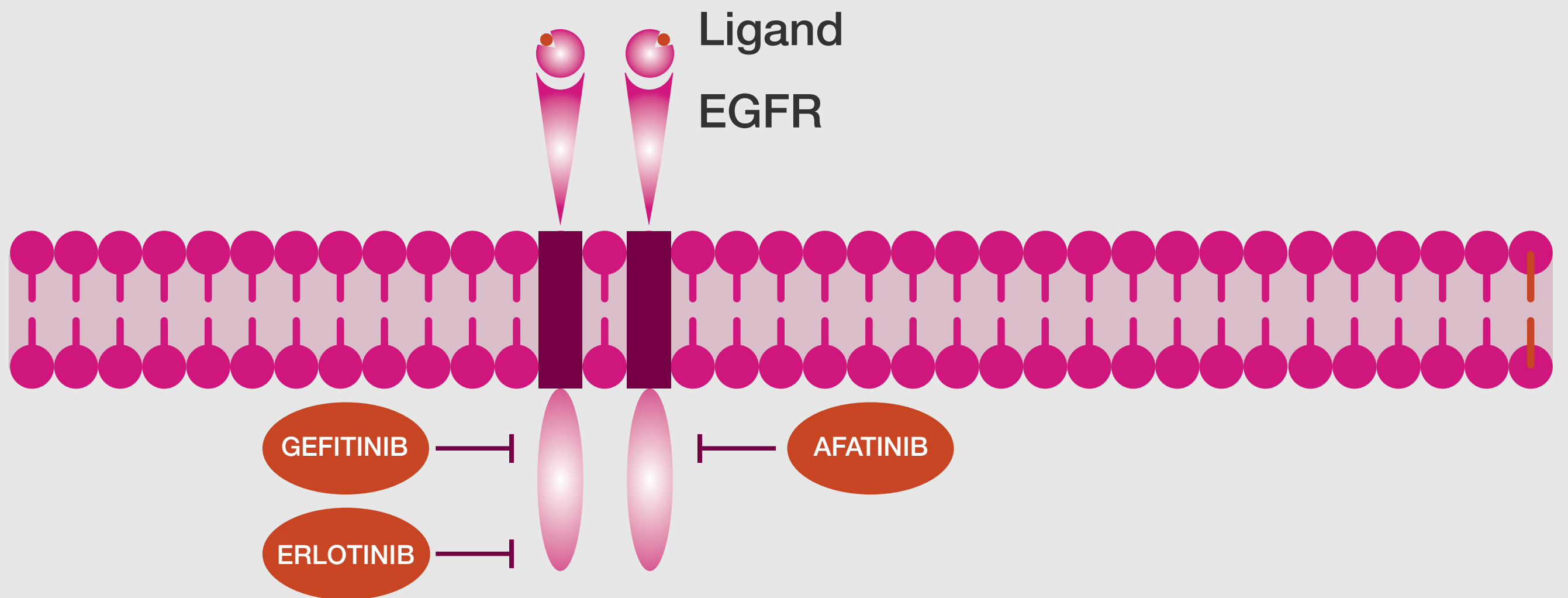
EGFR T790M

**A Gatekeeper Mutation**

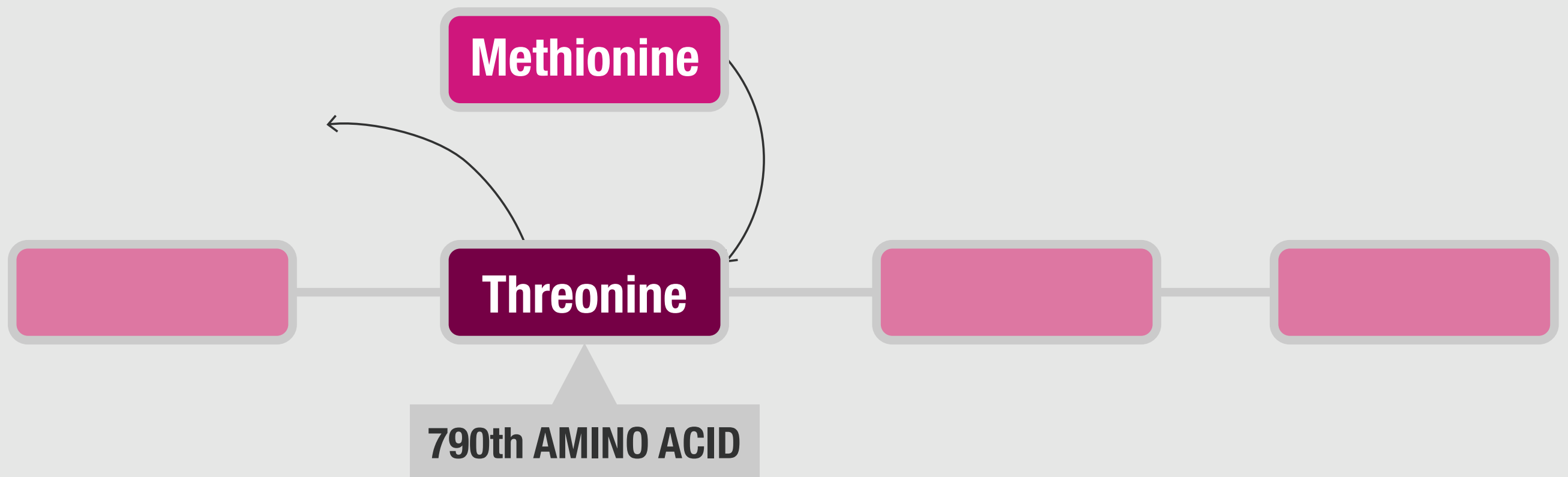
Activating EGFR mutations lead to metastatic non-small-cell lung cancer (NSCLC).



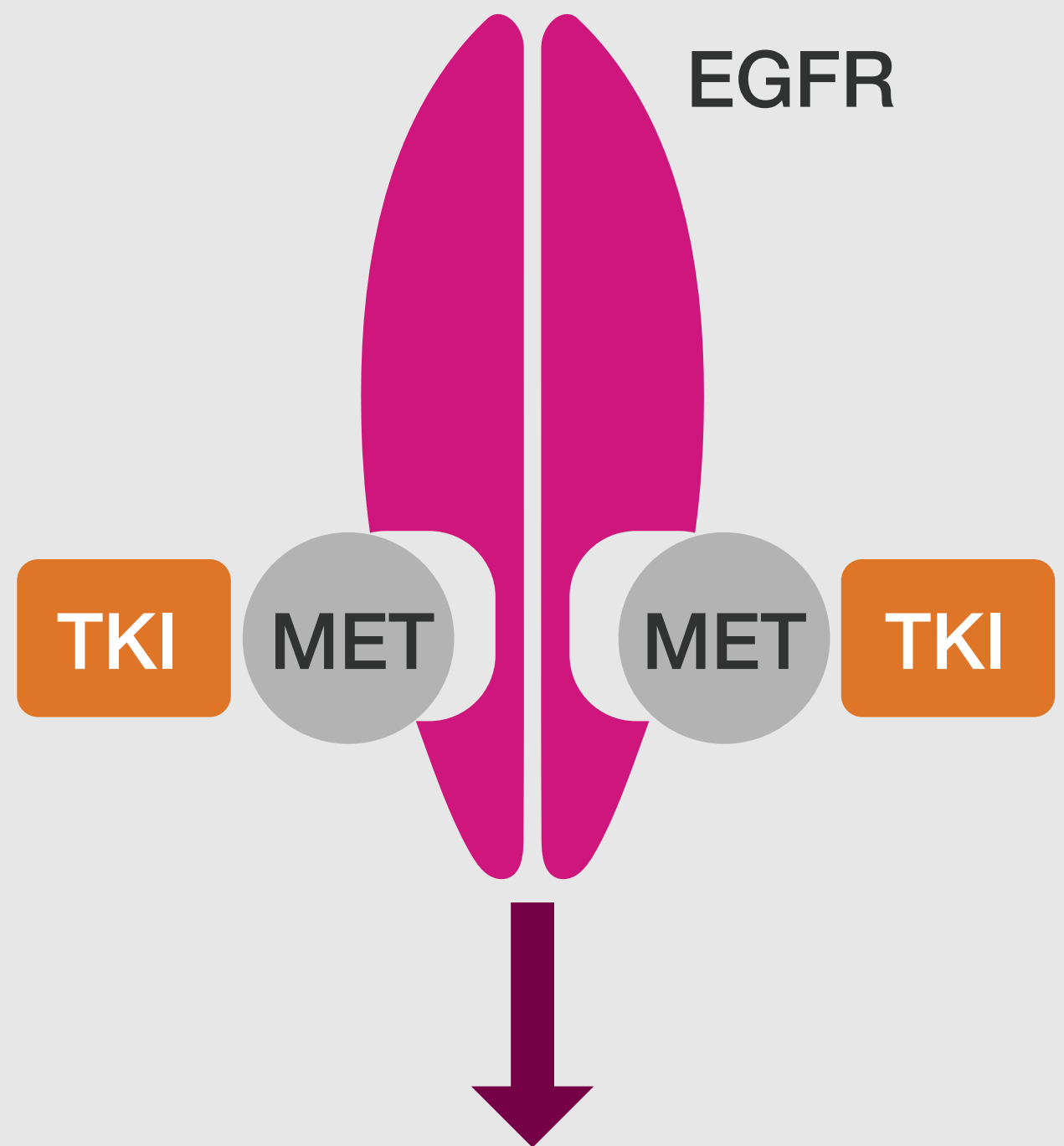
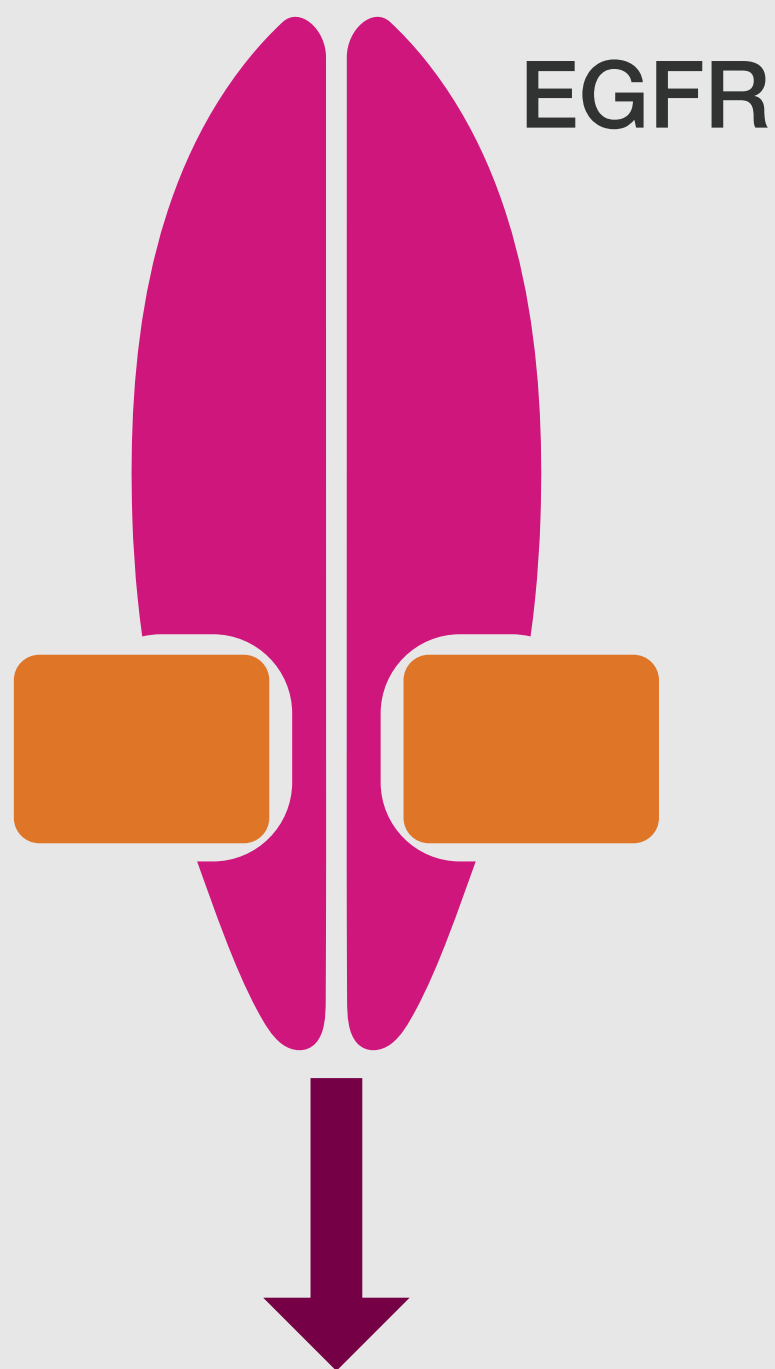
First- and second-generation tyrosine-kinase inhibitors (TKI), including gefitinib, erlotinib, and afatinib, are initially effective against NSCLC.



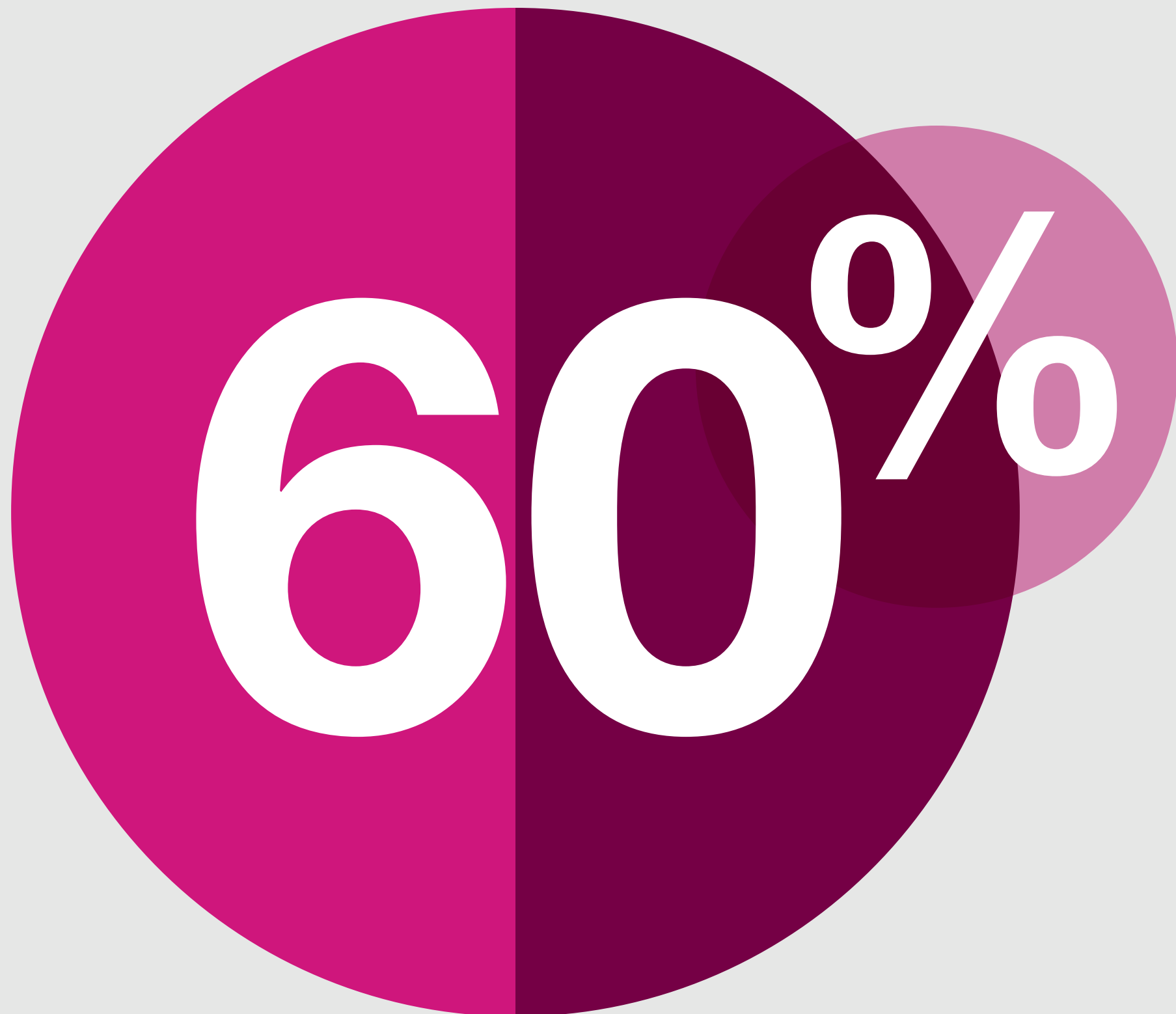
The switch from threonine to the larger methionine at position 790 sterically hinders interaction with the TKI inhibitor drug.



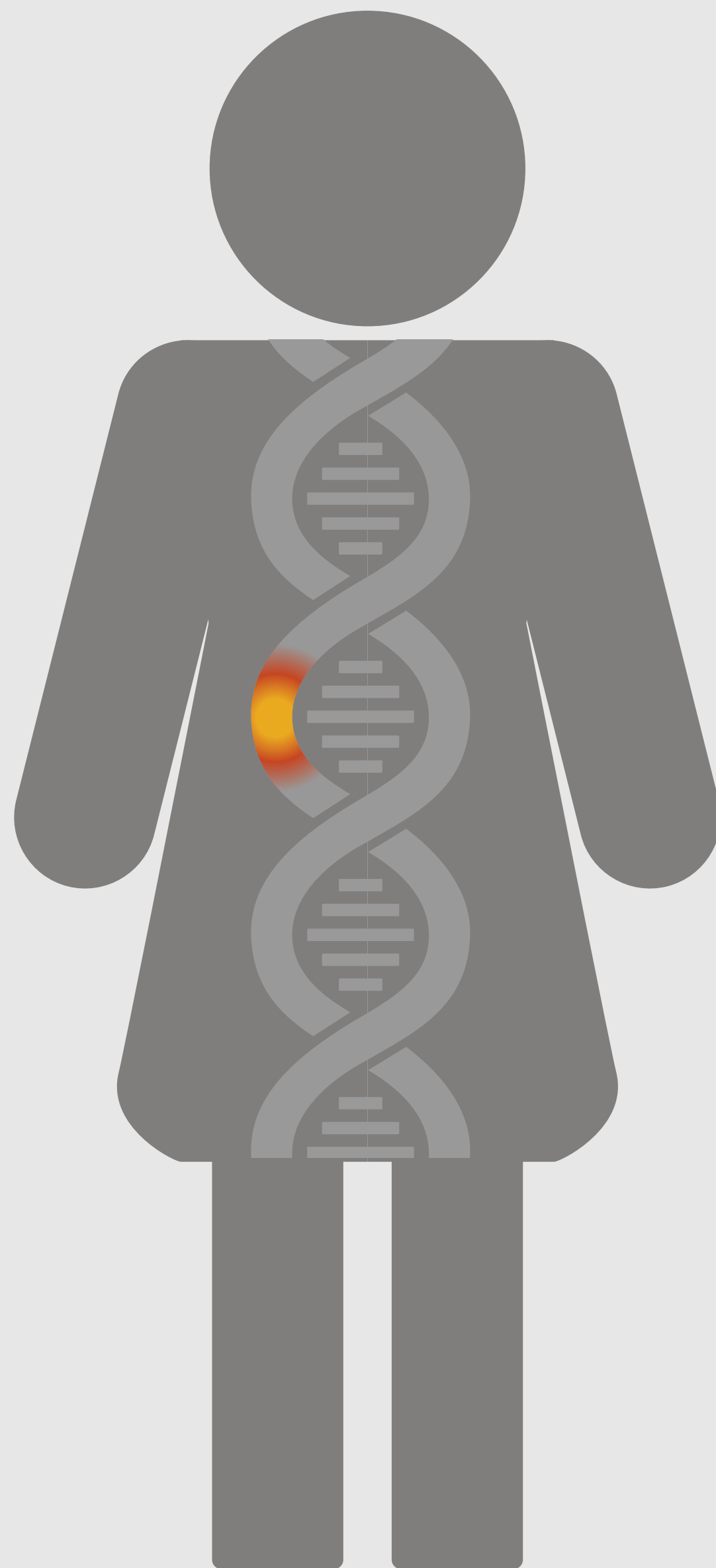
T790M is called a “gatekeeper” mutation because it prevents TKIs from binding the EGFR kinase domain.



Drug resistance can eventually develop in ~60% of NSCLC cases occurring after acquired T790M somatic mutation.



If the T790M mutation is detected in NSCLC patients, therapy can be switched to treatment that is active against the T790M alteration.



Cell-free tumor DNA (ctDNA) testing of a blood sample can be used to detect T790M mutations.

CtDNA or liquid biopsy also allows for sampling of the entire tumor burden and potentially circumvents the problem of tumor heterogeneity.







# EGFR

## T790M

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### DROPLET DIGITAL PCR (ddPCR) AND T790M DETECTION

- ddPCR enables quantitative, highly sensitive monitoring of the T790M mutation in blood plasma or cerebrospinal fluid
- Targeted testing by ddPCR enables serial testing for early resistance and therapeutic intervention in lung cancer

Visit [bio-rad.com/digital-assays](https://www.bio-rad.com/digital-assays) for more information on ddPCR Assays.  
For research use only:

## Reference

Lovly C et al. (2016). EGFR c.2369C>T (T790M) Mutation in Non-Small Cell Lung Cancer. My Cancer Genome [www.mycancergenome.org/content/disease/lung-cancer/egfr/4/](http://www.mycancergenome.org/content/disease/lung-cancer/egfr/4/), accessed October 30, 2018.

Kim Y et al. (2021). Exosome-based detection of EGFR T790M in plasma and pleural fluid of prospectively enrolled non-small cell lung cancer patients after first-line tyrosine kinase inhibitor therapy. *Cancer Cell International* 21, 50.

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