CHARITÉ
UNIVERSITÄTSMEDIZIN BERLIN

Clinical interpretation of molecular data for personalized cancer therapy

- from best guesses to databases to AI?

Damian Rieke, Hasso-Plattner-Institut 2023

COI

Bayer

Lilly

Bristol Myers-Squibb

Roche

Agenda

1. Background
2. Standards
3. Challenges
4. Summary

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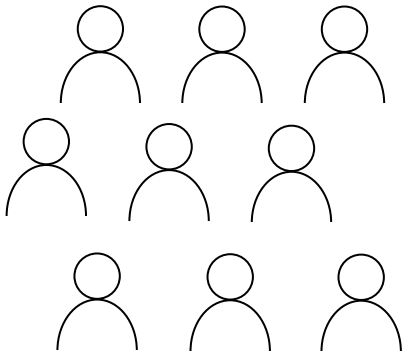
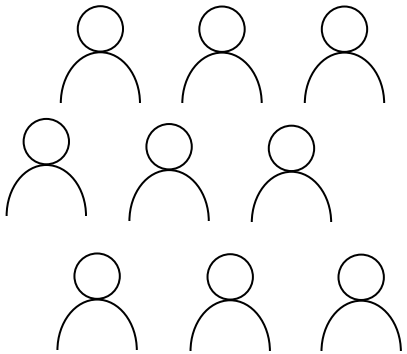
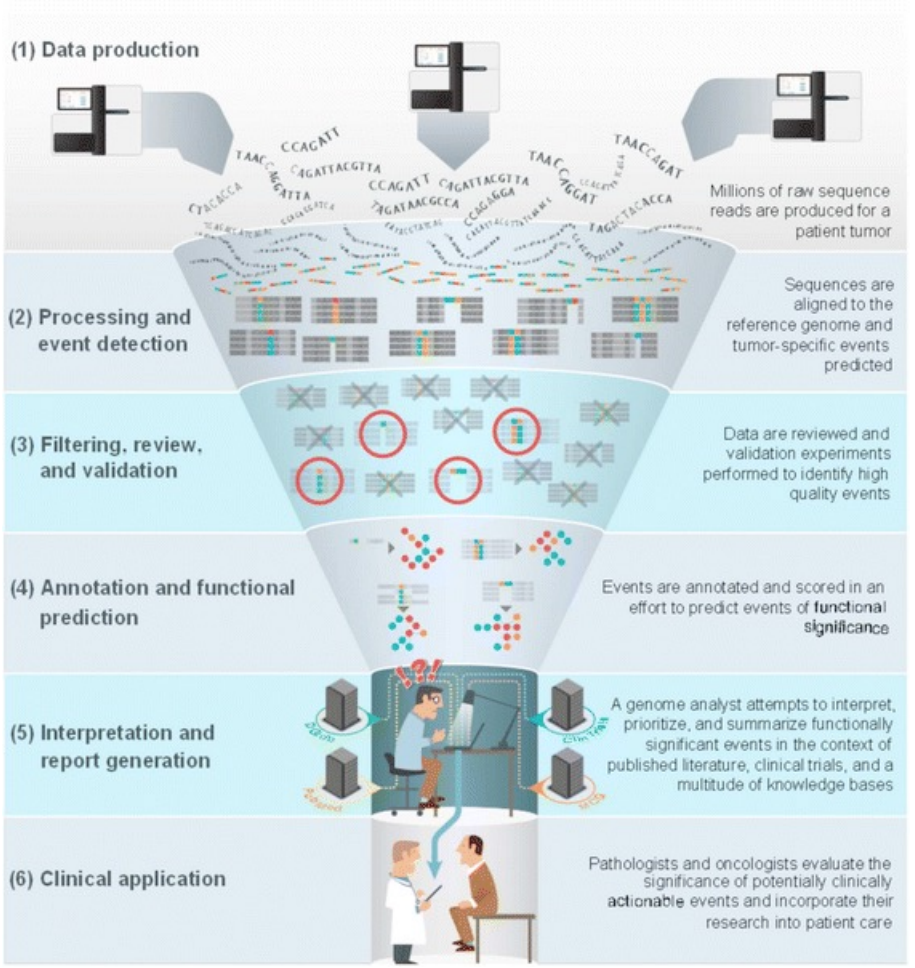
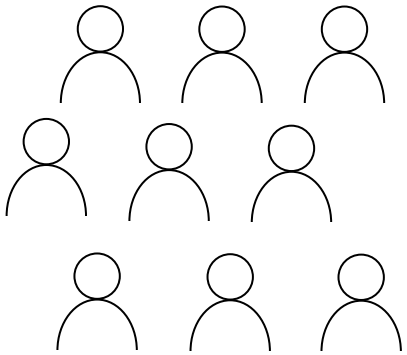
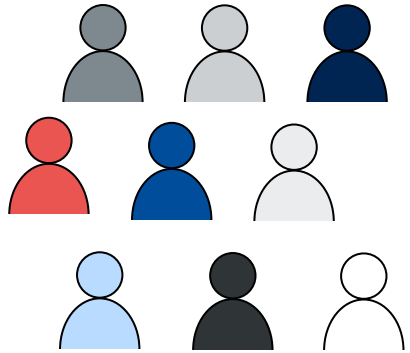






Good et al. Genome Biol . 2014





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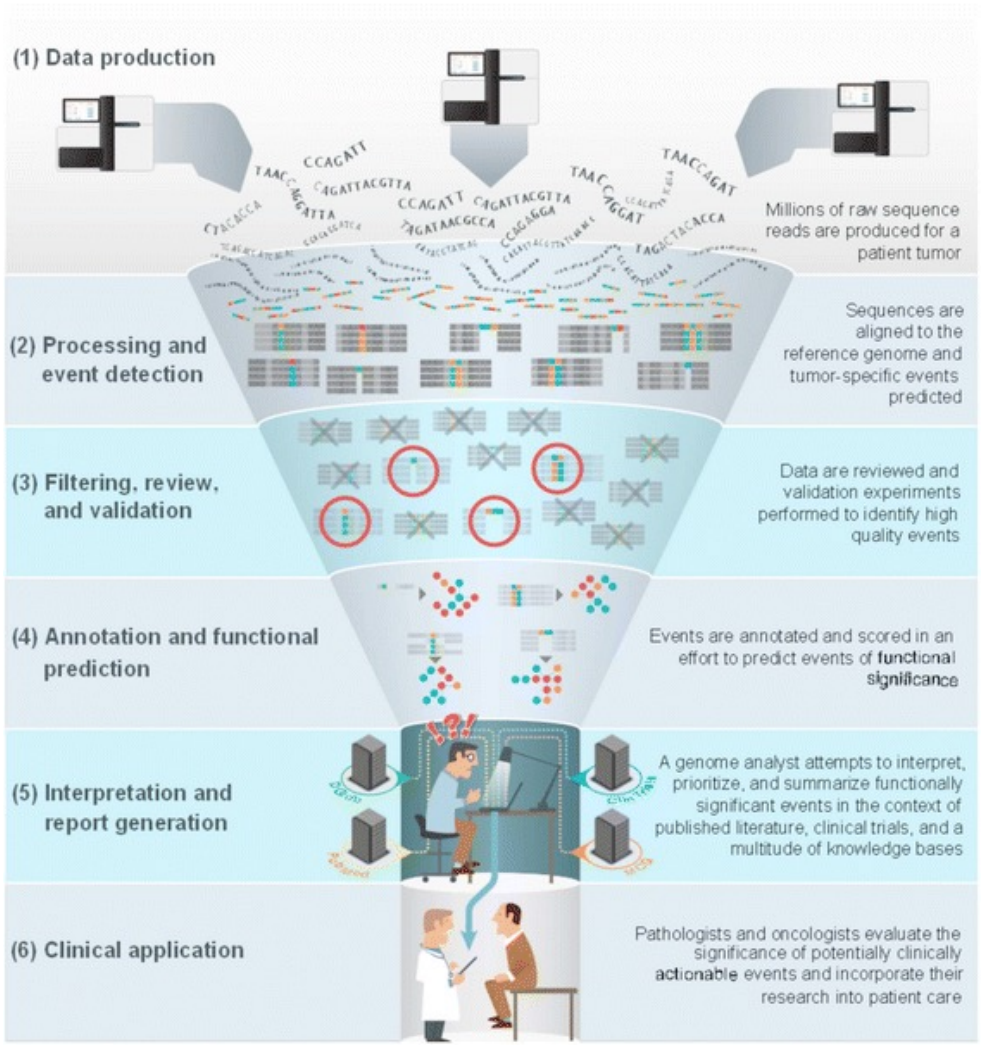
CHARITÉ
UNIVERSITÄTSMEDIZIN BERLIN
Charité Comprehensive Cancer Center
Leistungen
zurück
Präzisionsonkologische Sprechstunde
Für Patientinnen, Patienten & Interessierte
Für Ärztinnen, Ärzte & medizinisches Personal
Für Wissenschaftlerinnen & Wissenschaftler
Forschung
Karriere
Präzisionsonkologische Sprechstunde
Die präzisionsonkologische Sprechstunde am Charité Comprehensive Cancer Center bietet die Möglichkeit einer additiven Diagnostik für Patient:innen der Charité, für Betroffene aus externen Kliniken sowie onkologischen Praxen.
Durchsuchen Sie diese Website
Startseite › Leistungen › Plattform für personalisierte Krebsmedizin der Charité (PPK-C) ›
Präzisionsonkologische Sprechstunde
Präzisionsonkologische Sprechstunde - Reevaluation und unabhängige Beratung bei soliden Tumoren

Sample selection
Technology selection



Follow-up/Trial infrastructure

Good et al. Genome Biol . 2014

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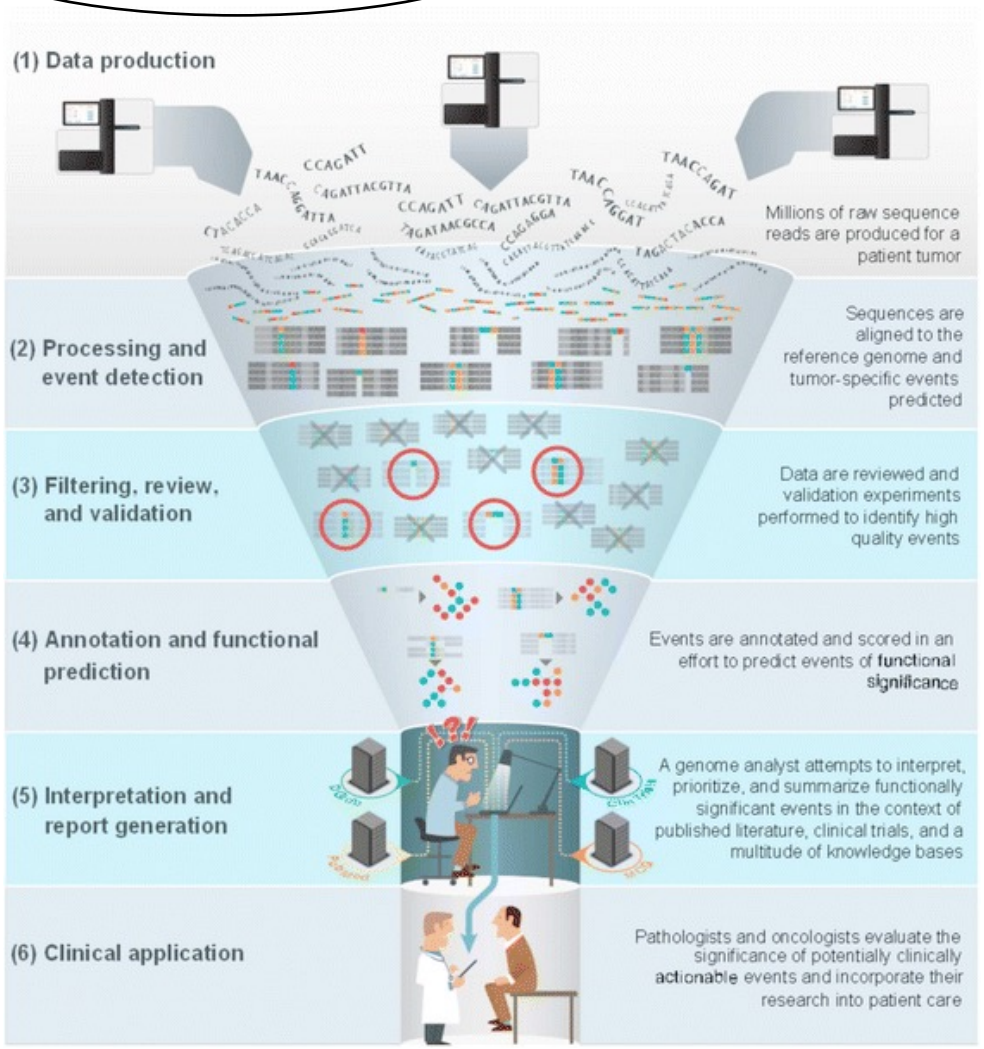


Follow-up/Trial infrastructure

Good et al. Genome Biol . 2014

NGS (accredited/validated)

- Oncomine Focus/Precision DNA Assay
- Oncomine Focus/Precision RNA Assay
- ColonLung Panel V2
- Cancer Hotspot Panel
- Myeloid Panel (Custom)
- (B-cell) Lymphoma Panel
- Oncomine cfDNA (Liquid Biopsies)
- Breast cfDNA Panel (Liquid Biopsies)
- BRCA1/2 Panel
- Tumor Mutational Burden (1.7 Mbases)
- Molecular Health 600+ Panel (3 Mbases); NextSeq
- Oncomine Comprehensive Assay V4 (500+) Panel
- TSO500 (DNA/RNA) Panel
- Ig/TCR Clonality Panel
- Archer RNA Panel

IHC/FISH

- nTRK screening
- TMB
- Other Targets (e.g. HER2, AR…)
- Fusion Gene validation

Other

- e.g. EPIC (Methylom)

DKTK MASTER

- WES/WGS
- RNASeq

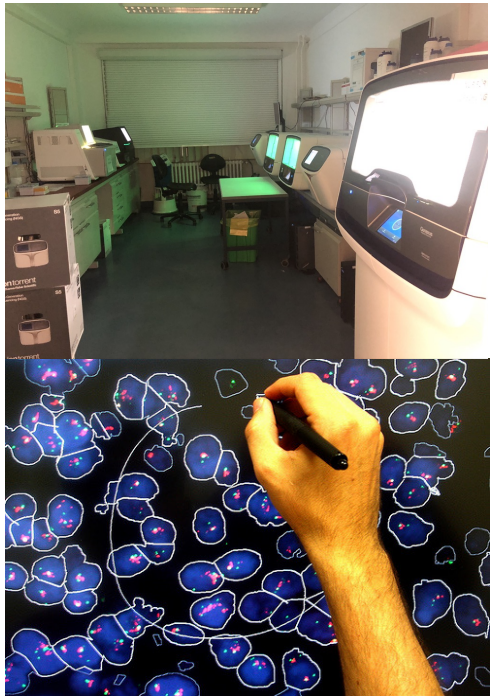
ExLiquid

- ctDNA

Functional Analyses

Single cell analyses

Portfolio
Molecular Diagnostics Pathology, Charité



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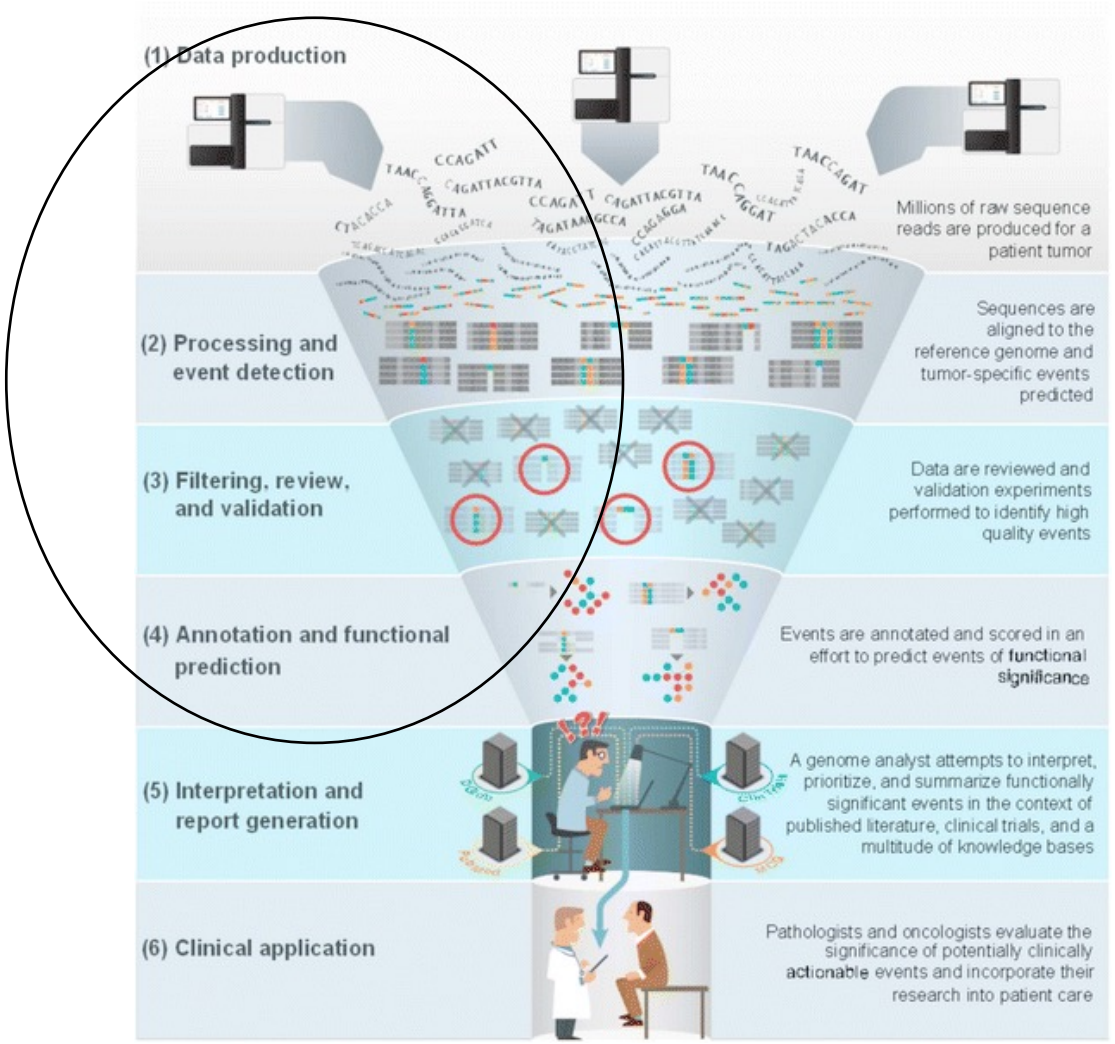

Follow-up/Trial infrastructure

Good et al. Genome Biol . 2014

Genetics in Medicine
Volume 24, Issue 5, May 2022, Pages 986-998

Special Article

Standards for the classification of pathogenicity of somatic variants in cancer (oncogenicity): Joint recommendations of Clinical Genome Resource (ClinGen), Cancer Genomics Consortium (CGC), and Variant Interpretation for Cancer Consortium (VICC)

Peter Horak [1], Malachi Griffith [2], Arpad M. Danos [2], Beth A. Pitel [3], Subha Madhavan [4], Xuelu Liu [5], Cynthia Chow [6], Heather Williams [7], Leigh Carmody [8], Lisa Barrow-Laing [9], Damian Rieke [10], Simon Kreutzfeldt [1], Albrecht Stenzinger [11], David Tamborero [12], Manuela Benary [10], Padma Sheila Rajagopal [13], Cristiane M. Ida [3], Harry Lesmana [14], Laveniya Satgunaseelan [15], Jason D. Merker [16] ...Dmitriy Sonkin [35]

Gen	Mutation [Exon: c.HGVS; p.HGVS]	AF [%] [1]	Potentielle Therapie-option [2]	Potentielle Kontra-indikation [2]	Potentiell anderweitig Relevant [2,3]
PIK3CA	Exon 21: c.3140A>G, p.H1047R	32	AMP IIc	-	-
HRAS	Exon 3: c.182A>G, p.Q61R	33	AMP IId	-	-

AR +

HER2 –

NTRK -

Diagnose
Unter Berücksichtigung der Vorbefunde ergibt sich ein vordiagnostiziertes Speichelgangskarzinom mit Nachweis von klinisch bzw. diagnostisch relevanten Mutationen und/oder Polymorphismen in den Genen PIK3CA, HRAS, NQO1, CYP2D6 TPMT sowie folgendem Profil:
- Tumor-Mutationslast: 10,9 Mut/Mb.
- Fusionsgen-Analyse: Keine RNA Untersuchung möglich, da Gewebe aufgebraucht..

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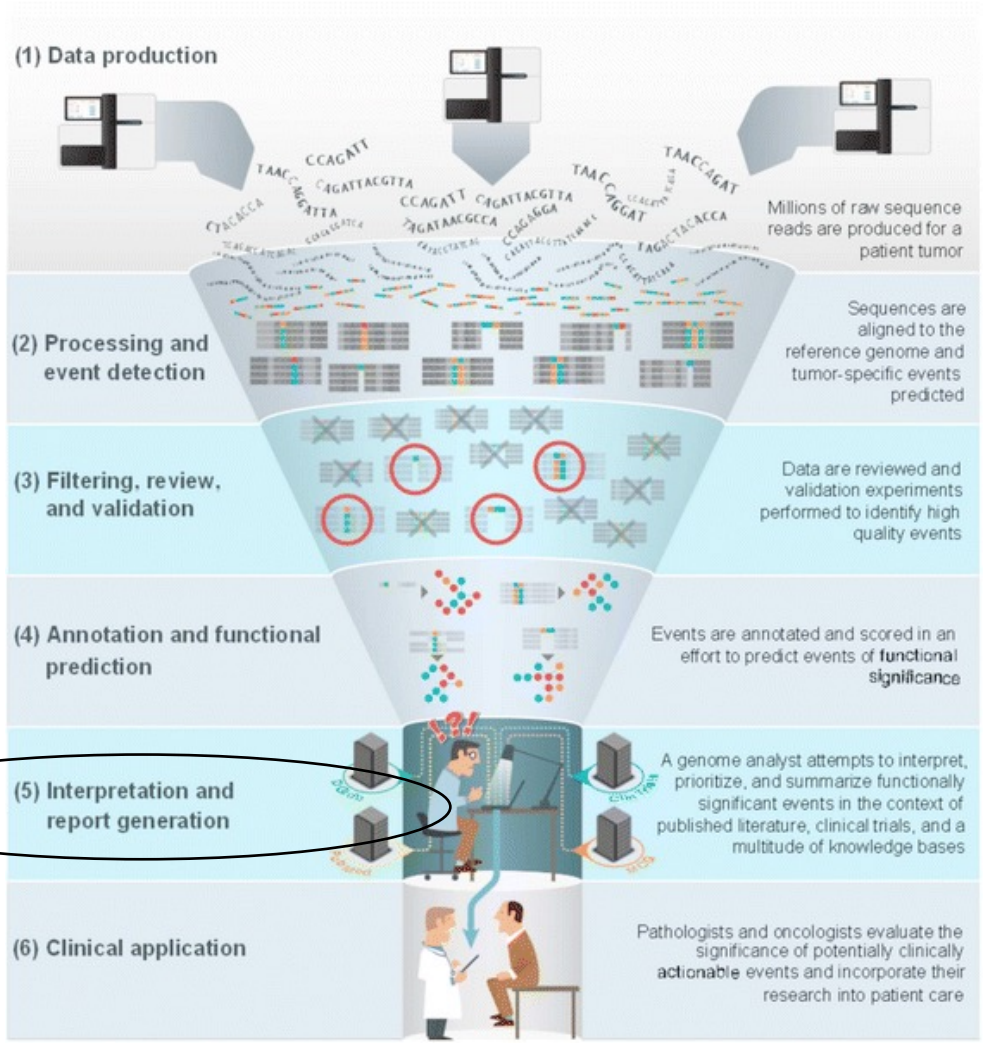

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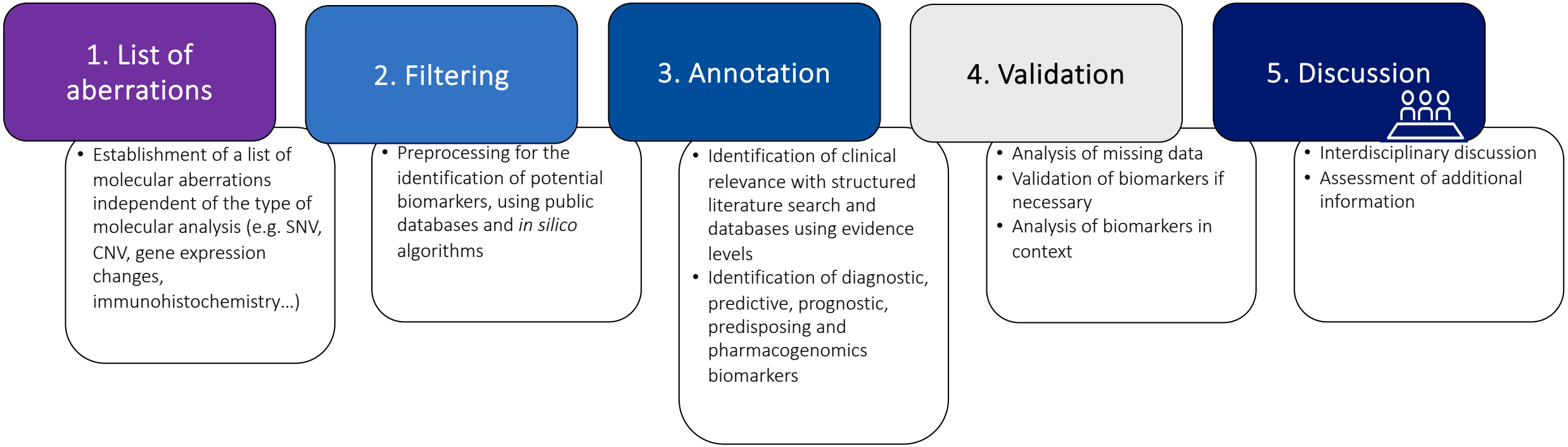
Good et al. Genome Biol . 2014


1. List of aberrations
• Establishment of a list of molecular aberrations independent of the type of molecular analysis (e.g. SNV, CNV, gene expression changes, immunohistochemistry…)
2. Filtering
• Preprocessing for the identification of potential biomarkers, using public databases and *in silico* algorithms
3. Annotation
• Identification of clinical relevance with structured literature search and databases using evidence levels
• Identification of diagnostic, predictive, prognostic, predisposing and pharmacogenomics biomarkers
4. Validation
• Analysis of missing data
• Validation of biomarkers if necessary
• Analysis of biomarkers in context
5. Discussion
• Interdisciplinary discussion
• Assessment of additional information

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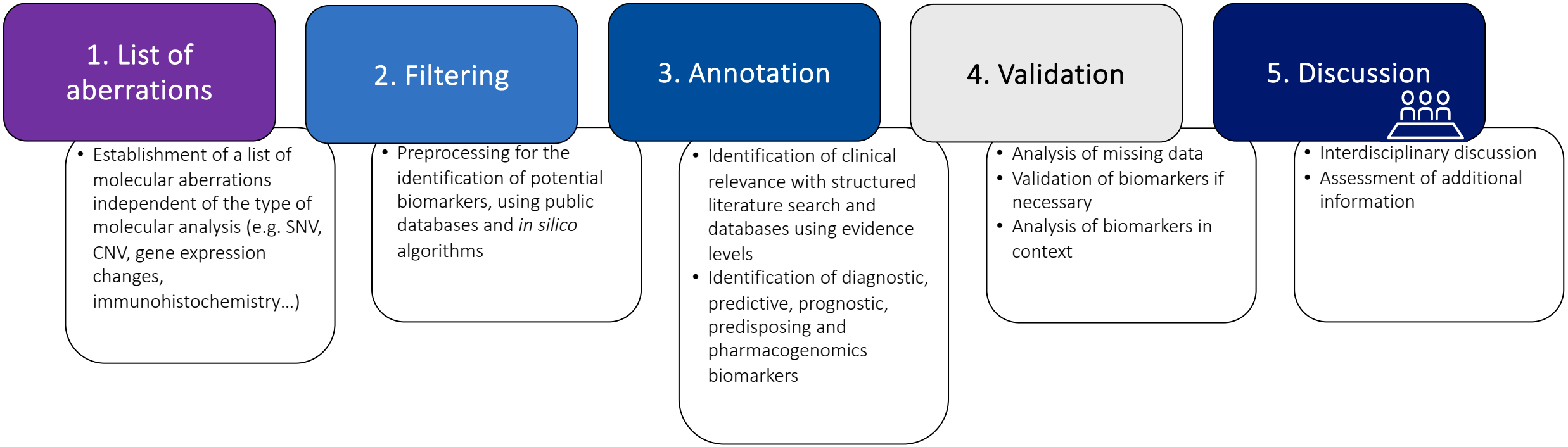
Medical History: 07/22 ED met. Salivary duct carcinoma
Manifestations: local, pulmonary
Sampling: 07/22 CT guided biopsy pulmonary metastasis
IHC/MolPath: TMB10,9 Mut/Mb, Her2 negativ (pulm, 1+ lokal), TRK negativ, Androgen receptor (strong positive)

	Aberration evidence	Drug/s	Mechanism	EvL	PMIDs/NCT	VAF	Histology
AR Expression							
PIK3CA p.H1047R							
HRAS p.Q61R							
TMB 10.9 Mut/Mb							

	Aberration evidence	Drug/s	Mechanism	EvL	PMIDs/NCT	VAF	Histol.
CYP2D6 splice-site, p.P34S							
TPMT p.A154T							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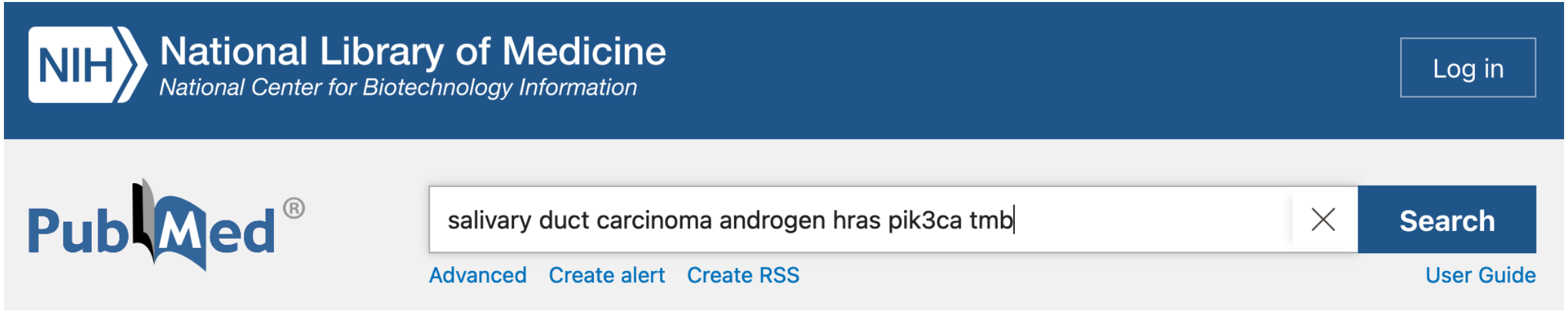
NIH
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National Center for Biotechnology Information
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salivary duct carcinoma androgen hras pik3ca tmb
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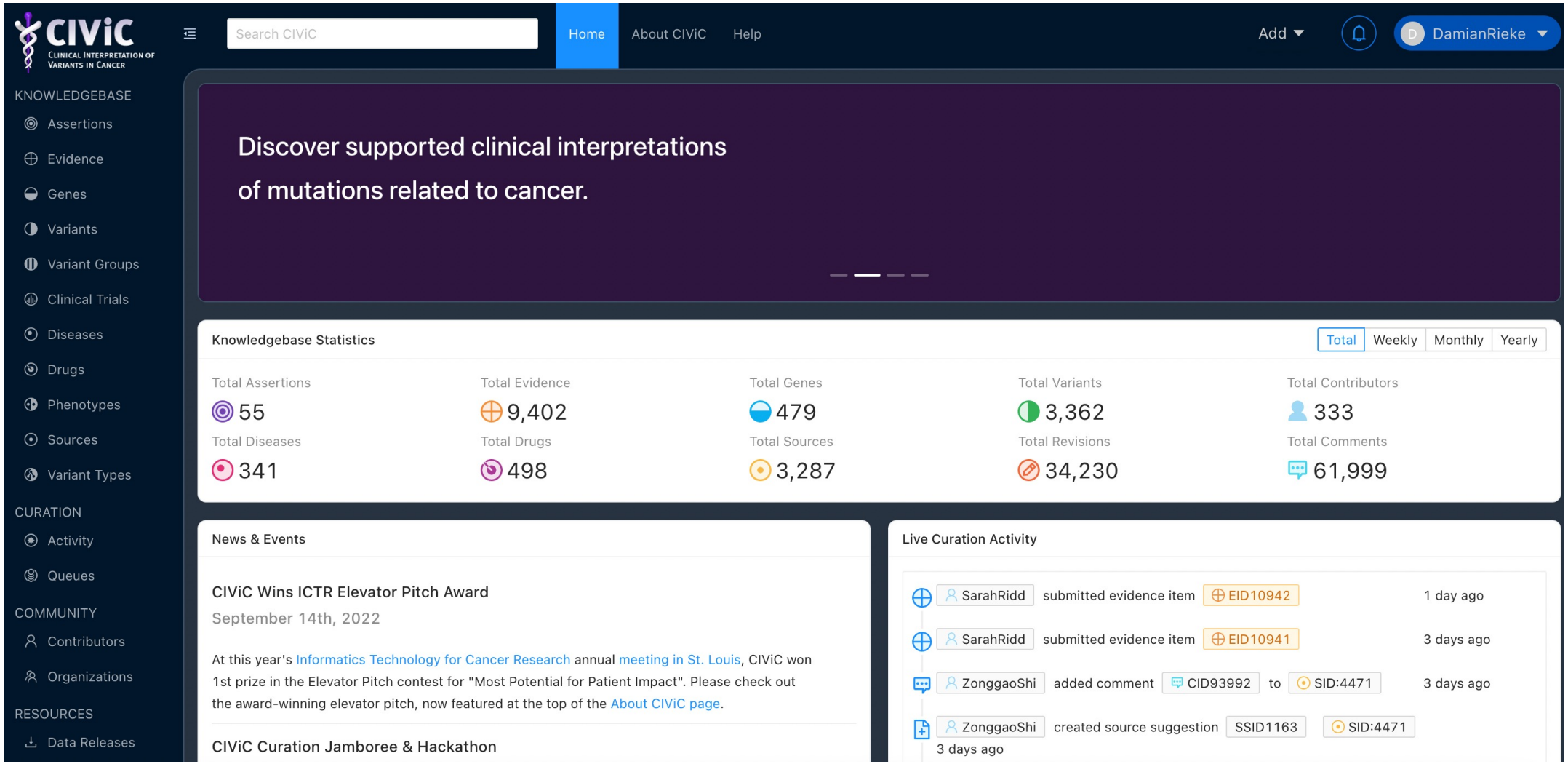
Advanced
Create alert
Create RSS
User Guide



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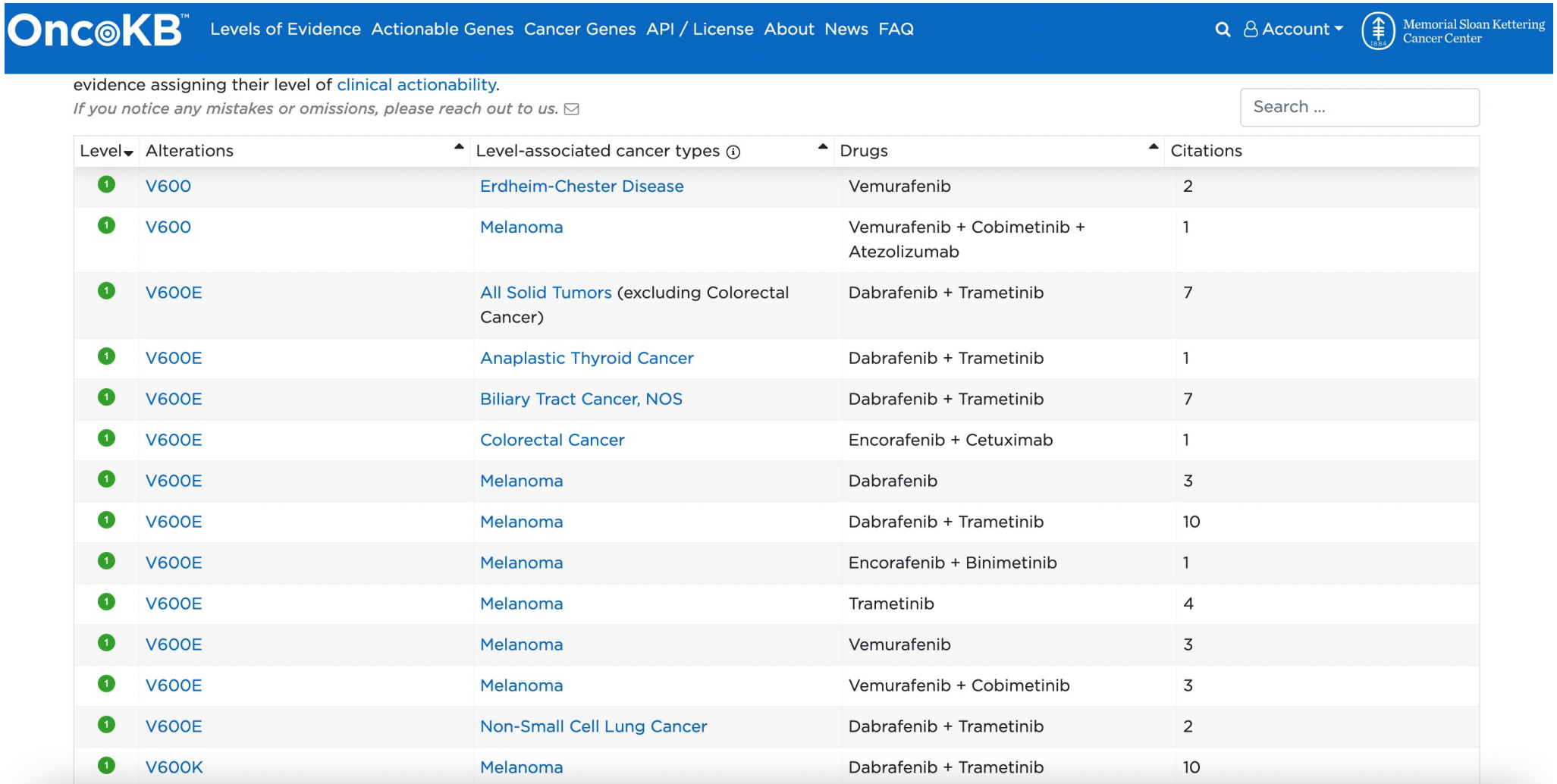
Griffith et al., Nat Genet. 2017
Krysiak et al., Nat Cancer. 2022
Krysiak et al., Nucleic Acids Res. 2022



Level	Alterations	Level-associated cancer types	Drugs	Citations
1	V600	Erdheim-Chester Disease	Vemurafenib	2
1	V600	Melanoma	Vemurafenib + Cobimetinib + Atezolizumab	1
1	V600E	All Solid Tumors (excluding Colorectal Cancer)	Dabrafenib + Trametinib	7
1	V600E	Anaplastic Thyroid Cancer	Dabrafenib + Trametinib	1
1	V600E	Biliary Tract Cancer, NOS	Dabrafenib + Trametinib	7
1	V600E	Colorectal Cancer	Encorafenib + Cetuximab	1
1	V600E	Melanoma	Dabrafenib	3
1	V600E	Melanoma	Dabrafenib + Trametinib	10
1	V600E	Melanoma	Encorafenib + Binimetinib	1
1	V600E	Melanoma	Trametinib	4
1	V600E	Melanoma	Vemurafenib	3
1	V600E	Melanoma	Vemurafenib + Cobimetinib	3
1	V600E	Non-Small Cell Lung Cancer	Dabrafenib + Trametinib	2
1	V600K	Melanoma	Dabrafenib + Trametinib	10

Gleiche Tumorentität	**m1A**	In der **gleichen Tumorentität** wurde der prädiktive Wert des Biomarkers oder die klinischeWirksamkeit in einer **Biomarker-stratifizierten Kohorte** einer adäquat gepowerten **prospektiven Studie** oder **Metaanalyse** gezeigt.
	m1B	In der **gleichen Tumorentität** wurde der prädiktive Wert des Biomarkers oder die klinische Wirksamkeit in einer **retrospektiven Kohorte** oder **Fall-Kontroll-Studie** gezeigt.
	m1C	Ein oder mehrere **Fallberichte** in der **gleichen Tumorentität**.
Andere Tumorentität	**m2A**	In einer **anderen Tumorentität** wurde der prädiktive Wert des Biomarkers oder die klinische Wirksamkeit in einer **Biomarker-stratifizierten Kohorte** einer adäquat gepowerten **prospektiven Studie** oder **Metaanalyse** gezeigt.
	m2B	In einer **anderen Tumorentität** wurde der prädiktive Wert des Biomarkers oder die Klinische Wirksamkeit in einer **retrospektiven Kohorte** oder **Fall-Kontroll-Studie** gezeigt.
	m2C	Unabhängig von der Tumorentität wurde beim Vorliegen des Biomarkers eine **klinische Wirksamkeit** in einem oder mehreren **Fallberichten** gezeigt.
In vitro oder Tiermodell	**m3**	**Präklinische Daten** (*in vitro*-/*in vivo*-Modelle, funktionelle Untersuchungen) zeigen eine Assoziation des Biomarkers mit der Wirksamkeit der Medikation, welche durch eine wissenschaftliche Rationale gestützt wird.
Biologische Rationale	**m4**	Eine **wissenschaftliche, biologische Rationale** legt eine Assoziation des Biomarkers mit der Wirksamkeit der Medikation nahe, welche bisher **nicht durch (prä)klinische Daten** gestützt wird.

ZPM Standard, according to
https://pct.mdanderson.org/pctService/resources/imageManager/image/loe

Medical History: 07/22 ED met. Salivary duct carcinoma
Manifestations: local, pulmonary
Sampling: 07/22 CT guided biopsy pulmonary metastasis
IHC/MolPath: TMB10,9 Mut/Mb, Her2 negativ (pulm, 1+ lokal), TRK negativ, Androgen receptor (strong positive)

	Aberration evidence	Drug/s	Mechanism	EvL	PMIDs/NCT	VAF	Histology
AR Expression	AR+ HER2+, AR+, PIK3CA p.E545K, HRAS p.Q61R	Leuprorelin/Bicalutamid ADT Alpelisib/Bicalutamid	Antiandrogen Antiandrogen + PI3K	M1a m1b m1c	29211833 (1) 29272069 34036229 (2)		SDC
PIK3CA p.H1047R	p.E545K	Alpelisib/Bicalutamid	Antiandrogen + PI3K	m1c	34036229 (2)	32%	
HRAS p.Q61R	HRAS mut	Tipifarnib	HRAS	m1a	32557577 (3)	33%	
TMB 10,9 Mut/Mb	TMB high AR+, TMB unknown	Immune Checkpoint Inhibitor Pembrolizumab	ICI PD-1	M1a m1c	34083238 32352883		

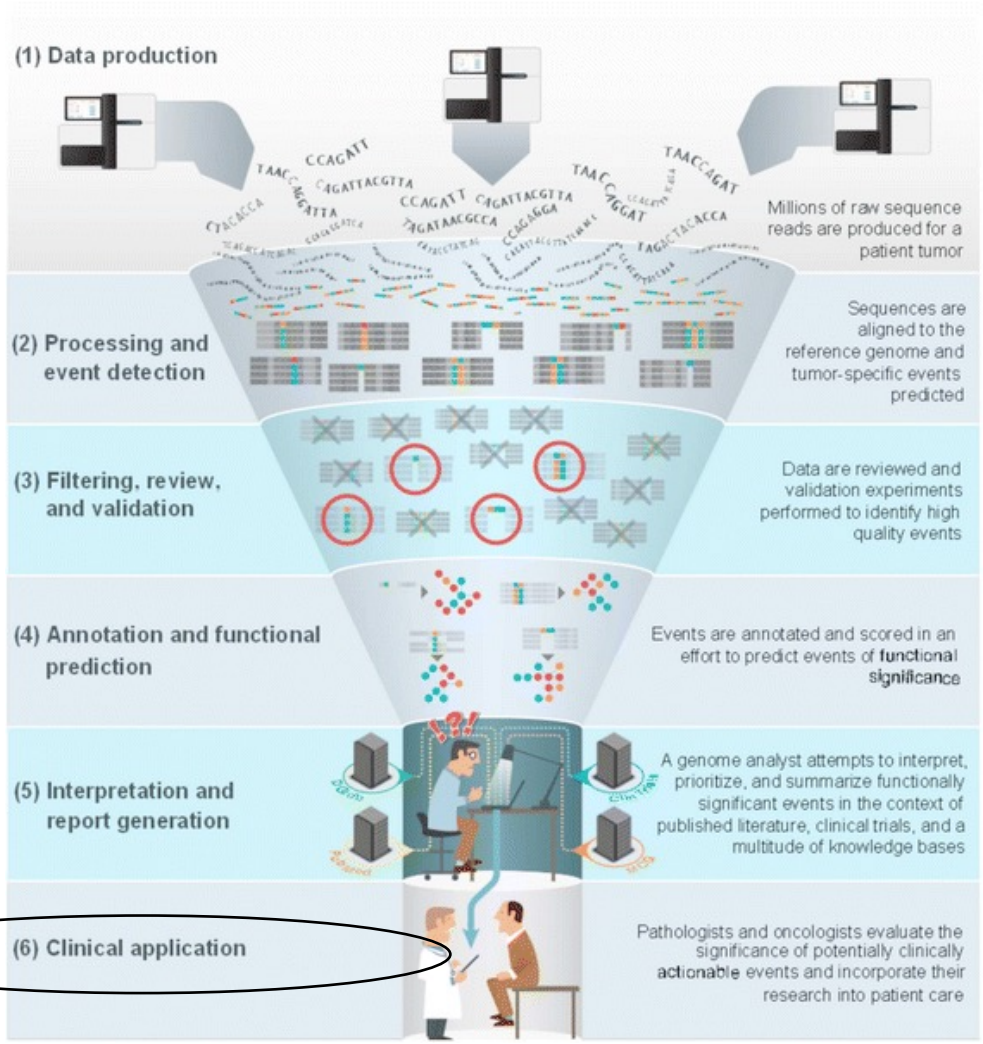
	Aberration evidence	Drug/s	Mechanism	EvL	PMIDs/NCT	VAF	Histol.
CYP2D6 splice-site, p.P34S		Codein, Tamoxifen, Gefitinib, Tramadol	toxicity			33, 47%	
TPMT p.A154T		Mercaptopurin, Thioguanin	toxicity			35%	

(1) Phase 2 study. 36 patients. ORR 41.7%. mPFS 8.8m

(2) Case report of metabolic response > 12months after prior progression on HER2-directed therapy

(3) Prospective trial, 13 R/M HRASmut (mostly Q61R) SGC. 1 Response, 7 SD.

Sample selection
Technology selection



Follow-up/Trial infrastructure

Good et al. Genome Biol . 2014

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Charité Comprehensive Cancer Center

Leistungen

zurück

Molekulare Tumorkonferenz

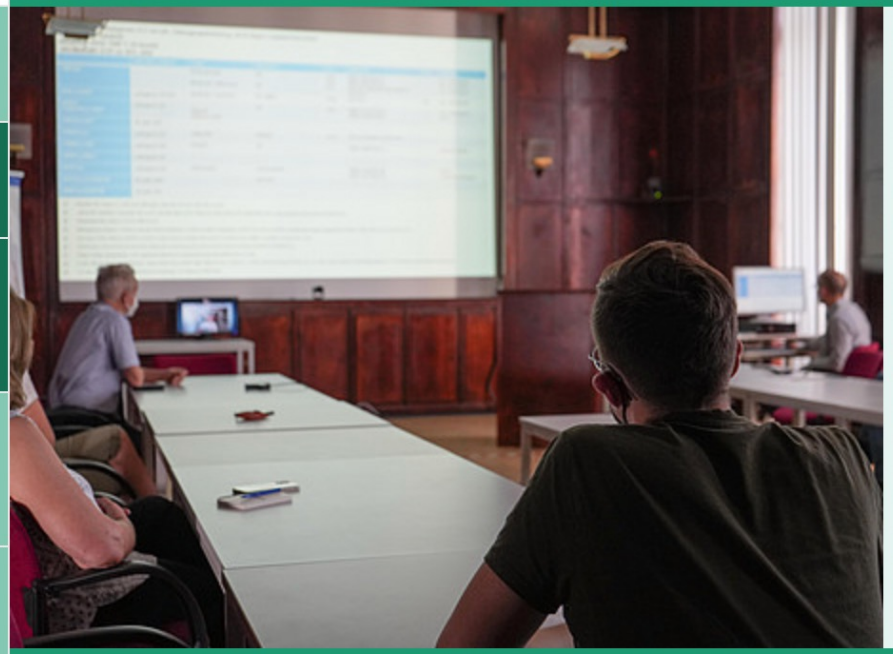
Für Patientinnen, Patienten & Interessierte

Für Ärztinnen, Ärzte & medizinisches Personal

Für Wissenschaftlerinnen & Wissenschaftler

Forschung

Karriere



Molekulare Tumorkonferenz: Präzisionsonkologie in der klinischen Routine

In der molekularen Tumorkonferenz werden gemeinsam mit Forschern Gensequenzierungen in die Therapieentscheidung mit einbezogen.

Im Charité Comprehensive Cancer Center finden regelmäßig wöchentlich molekulare Tumorkonferenzen statt.

Durchsuchen Sie diese Website

Startseite › Leistungen › Plattform für personalisierte Krebsmedizin der Charité (PPK-C) › Molekulare Tumorkonferenz

Molekulare Tumorkonferenz: Präzisionsonkologie in der klinischen Routine

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Molecular rationale for treatment with

Priority 1: antiandrogen therapy (m1a, off-label)

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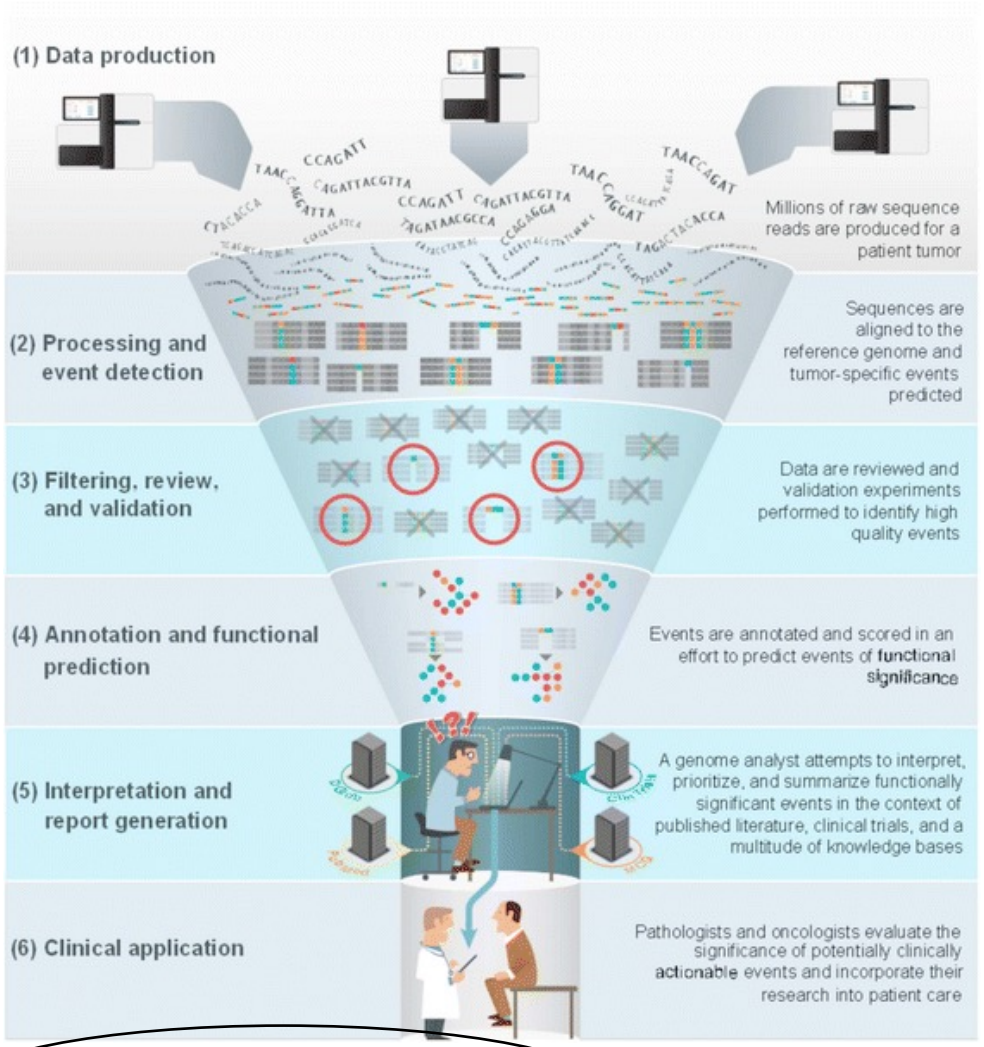

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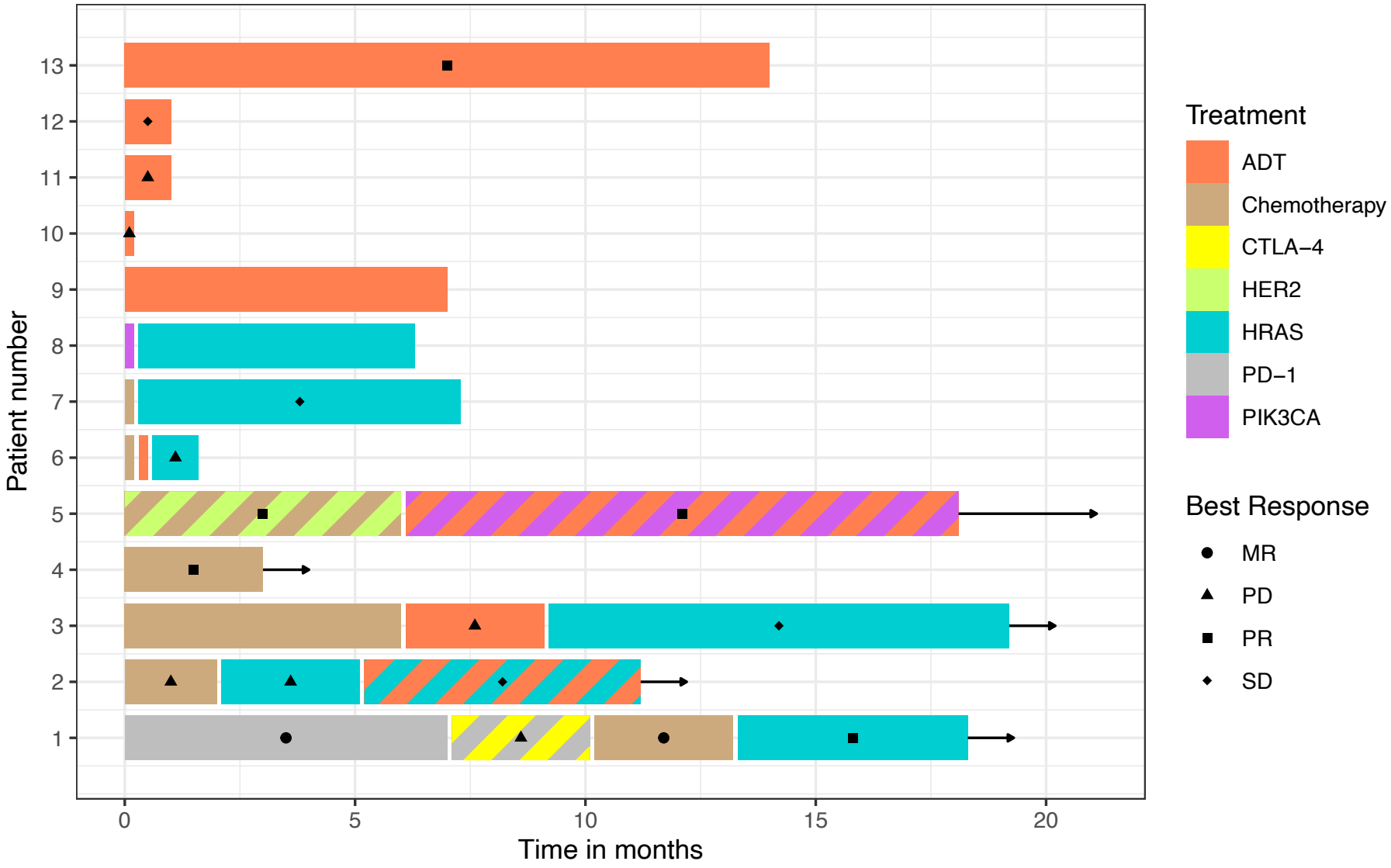

Rieke et al., Front. Oncol. 2023

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Publication [citation]	Matched treatment [8]	Off-label	Treated in trials	ORR [9]	SD [9]	PFS/TTF [9]	OS [9]	Other data
Zehir et al. 2017 [2]	24% (n=537)	n.r.	11% (n=527)	n.r.	n.r.	n.r.	n.r.	
Tsimberidou et al. 2014 [3]	27% (n=143)	none	100% (n=379)	12% vs. 5%	16% vs. 12%	3.9 m vs. 2.2 m	11.4 m vs. 8.6 m	
Massard et al. 2017 [4]	48% (n=199)	25% (n=50)	75% (n=149)	11%	52%	2.3 m	11.9 m	PFS2/PFS1 ≥1.3 : 33%
Burkard et al. 2017 [5]	28% (n=9)	89% (n=8)	11% (n=1)	17%	n.r.	n.r.	n.r.	
Le Tourneau et al. 2015 [6]	34% (n=99)	none	100% (n=195)	4% vs. 3%	n.r.	2.3 m vs. 2.0 m	n.r.	
Sicklick et al. 2019 [7]	49% (n=73)	none	100% (n=73)	23%	5%	3.67 m	11.8 m	PFS2/PFS1 ≥1.3: 75% vs. 36.6% in low matching score group
Rodon et al. 2019 [8]	42% (n=107)	none	100% (n=107)	11.2%	15%	2.01 m	5.9 m	PFS2/PFS >1.5: 22.4%
Tsimberidou et al. 2012 [19]	46% (n=211)	none	100% (n=352)	25% vs. 4%	23% vs. 10%	4.4 m vs. 2.3 m	11.4 m vs. 10.2 m	
Jameson et al. 2014 [20]	89% (n=29)	none	100 % (n=25)	20%	32%	n.r.	7.8 m	PFS2/PFS1 ≥1.3 : 44%
Wiesweg et al. 2013 [21]	45% (n=62)	69% (n=43)	31% (n=19)	n.r.	n.r.	n.r.	n.r.	
Jones et al. 2015 [22]	n.r.	n.r.	n.r.	n.r.	n.r.	n.r.	n.r.	
Dalton et al. 2017 [23]	21% (n=28)	46% (n=11)	54% (n=13)	n.r.	n.r.	5.0 m	n.r.	
Sohal et al. 2015 [24]	22% (n=24)	38% (n=9)	50% (n=12)	n.r.	n.r.	n.r.	n.r.	
Johnson et al. 2014 [25]	21% (n=18)	39% (n=7)	61% (n=7)	22%	28%	n.r.	n.r.	
Radovich et al. 2016 [26]	100% (n=44)	none	100% (n=101)	n.r.	n.r.	2.8 m vs. 1.6 m	n.r.	PFS2/PFS1 ≥1.3 : 43.2% vs. 5.3%
Stockley et al. 2016 [27]	n.a.	none	100% (n=245)	19% vs. 9%	n.r.	n.r.	16 m vs. 13 m	any tumor shrinkage: 62% vs. 32%
Schwaederle et al. 2016 [28]	48% (n=87)	n.r.	n.r.	n.r.	n.r.	4.0 m vs. 3.0 m	12.7 m vs. 12.4 m	PFS2/PFS1 ≥1.3 : 45.3% vs. 19.3%
Von Hoff et al. 2010 [29]	79% (n=66)	n.r.	n.r.	10%	n.r.	n.r.	5 m	PFS2/PFS1 ≥1.3 : 27%
Tredan et al. 2017 [30]	11% (n=101)	n.r.	n.r.	17%	34%	2.8 m	n.r.	
Cobain et al. 2017 [31]	n.a.	n.r.	n.r.	n.r.	n.r.	n.r.	n.r.	
Hoefflin et al. 2018 [32]	32% (n=33)	67% (n=22)	6% (n=2)	33%	24%	n.r.	not reached	PFS2/PFS1 ≥1.3 (off label): 57.1%
Basse et al. 2018 [33]	10% (n=45)	n.a.	100% (n=45)	11%	n.a.	n.a.	n.a.	
median	**34%**	**46%**	**100%**	**17% [10]**	**24% [10]**	**3.2 m [10]**	**11.4 m [10]**	

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Publication [citation]	Matched treatment [8]	Off-label	Treated in trials	ORR [9]	SD [9]	PFS/TTF [9]	OS [9]	Other data
Zehir et al. 2017 [2]	24% (n=537)	n.r.	11% (n=527)	n.r.	n.r.	n.r.	n.r.	
Tsimberidou et al. 2014 [3]	27% (n=143)	none	100% (n=379)	12% vs. 5%	16% vs. 12%	3.9 m vs. 2.2 m	11.4 m vs. 8.6 m	
Massard et al. 2017 [4]	48% (n=199)	25% (n=50)	75% (n=149)	11%	52%	2.3 m	11.9 m	PFS2/PFS1 ≥1.3 : 33%
Burkard et al. 2017 [5]	28% (n=9)	89% (n=8)	11% (n=1)	17%	n.r.	n.r.	n.r.	
Le Tourneau et al. 2015 [6]	34% (n=99)	none	100% (n=195)	4% vs. 3%	n.r.	2.3 m vs. 2.0 m	n.r.	
Sicklick et al. 2019 [7]								
Rodon et al. 2019 [8]								
Tsimberidou et al. 2012 [19]								
Jameson et al. 2014 [20]								
Wiesweg et al. 2013 [21]								
Jones et al. 2015 [22]								
Dalton et al. 2017 [23]								
Sohal et al. 2015 [24]								
Johnson et al. 2014 [25]								
Radovich et al. 2016 [26]								
Stockley et al. 2016 [27]	n.a.	none	100% (n=245)	19% vs. 9%	n.r.	n.r.	16 m vs. 13 m	any tumor shrinkage: 6
Schwaederle et al. 2016 [28]	48% (n=87)	n.r.	n.r.	n.r.	n.r.	4.0 m vs. 3.0 m	12.7 m vs. 12.4 m	PFS2/PFS1 ≥1.3 : 45.3% vs. 19.3%
Von Hoff et al. 2010 [29]	79% (n=66)	n.r.	n.r.	10%	n.r.	n.r.	5 m	PFS2/PFS1 ≥1.3 : 27%
Tredan et al. 2017 [30]	11% (n=101)	n.r.	n.r.	17%	34%	2.8 m	n.r.	
Cobain et al. 2017 [31]	n.a.	n.r.	n.r.	n.r.	n.r.	n.r.	n.r.	
Hoefflin et al. 2018 [32]	32% (n=33)	67% (n=22)	6% (n=2)	33%	24%	n.r.	not reached	PFS2/PFS1 ≥1.3 (off label): 57.1%
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median	**34%**	**46%**	**100%**	**17% [10]**	**24% [10]**	**3.2 m [10]**	**11.4 m [10]**	

Publication [citation]	Matched treatment [8]	ORR [9]	SD [9]	PFS/TTF [9]	OS [9]
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CHARITÉ

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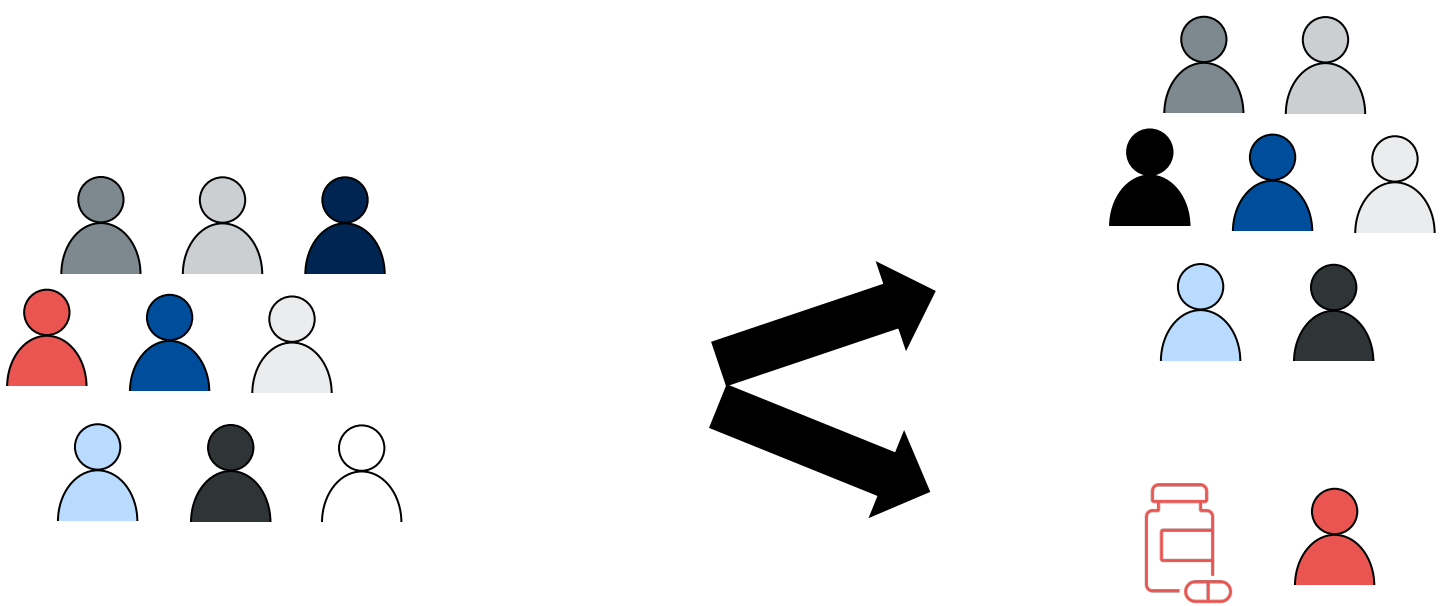

Table 1. Sample Patients as Provided to the Molecular Tumor Boards

Patient	Clinical Information	Additional Information	Sequencing	Fusion Genes	Copy No.	Gene Expression
1. 56-year-old patient	Initial diagnosis October 2014: TTF1-positive lung adenocarcinoma T3N3M0 (UICC IIIB) with negative *ALK/ROS* translocation and negative *EGFR* mutation status. Definite chemoradiotherapy. Widespread metastases to the peritoneum diagnosed in Nov 2015. Progressive after platinum-based chemotherapy and PD-1 inhibition (nivolumab). ECOG 1.	Tumor specimen from diagnostic mediastinoscopy Oct 2014: 25% tumor content, 580× mean sequencing depth.	Panel sequencing: *KRAS* G13D, *TP53* A276G, *PTPRS* R238*, *ZFHX3* F2994L, *CDH1* D433N	N/A	N/A	N/A

Table 3. Treatment Recommendations as Provided by the Respective Molecular Tumor Boards for Patient 1

Tumor Board	Recommendation	Provided Rationale	Additional Recommendation
1	Pan-Raf inhibitor	Downstream effect *KRAS* mutation	
2	No targeted therapy		*MET* and *RET* testing
3	Clinical trial for *KRAS* mutation	*KRAS* mutation	Genetic counseling for *CDH1* and *TP53* mutations (potential germline mutation)
4	Sorafenib clinical trial	*KRAS* mutation	
5	Docetaxel and selumetinib	*KRAS* mutation (data from phase II clinical trial)	Genetic counseling (*CDH1* mutation; potential germline mutation)
6	N/A because of missing information		

NOTE. Rationales were provided for only some of the recommendations and asked for if missing initially.
Abbreviations: N/A, not available; RET, Ret proto-oncogene.

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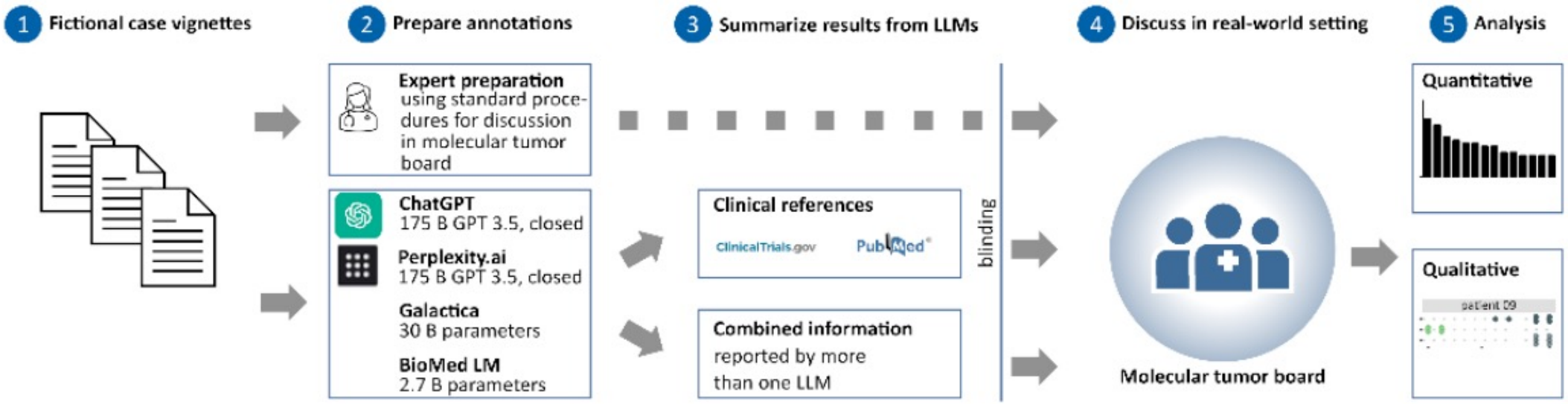

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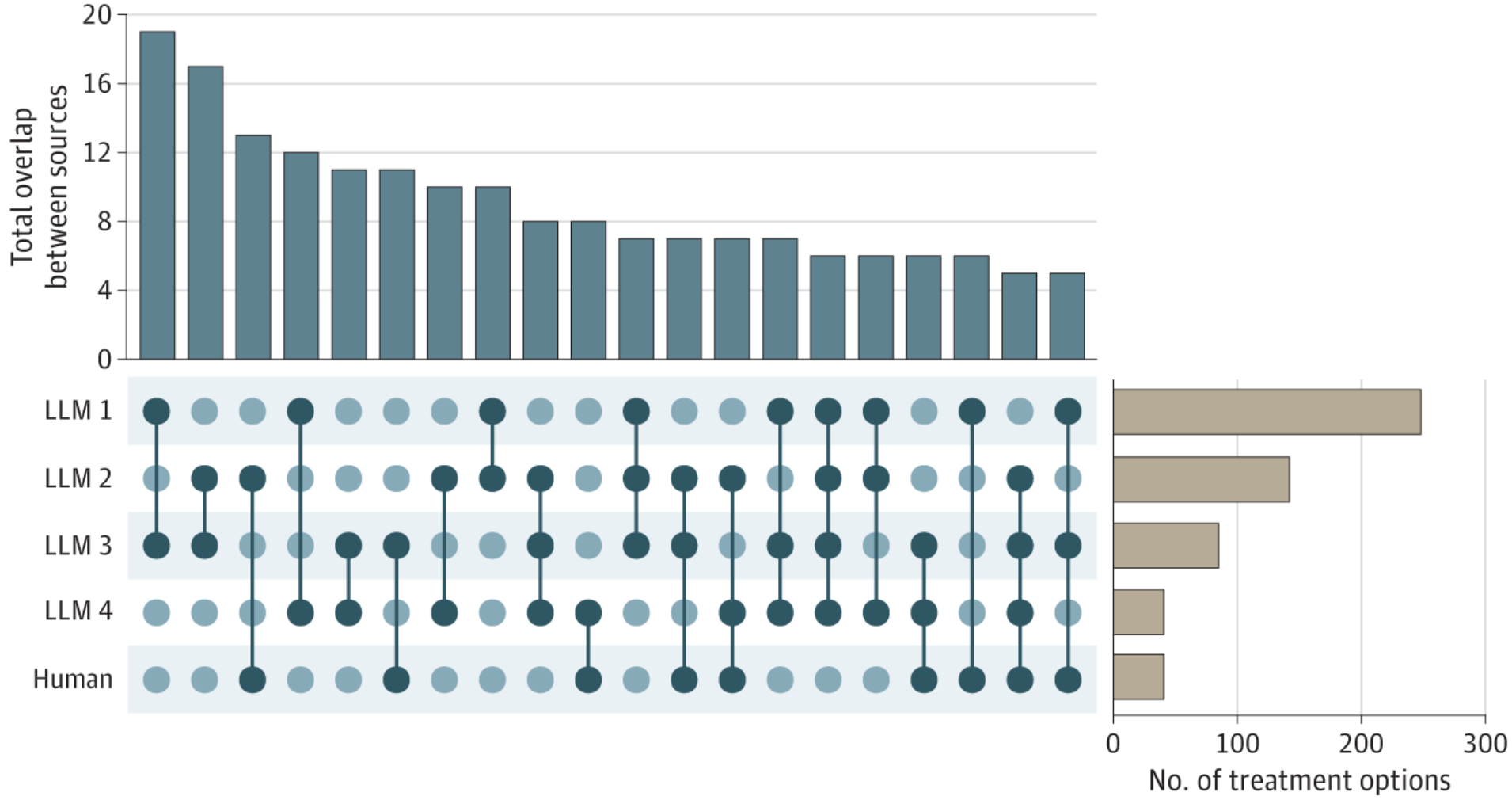

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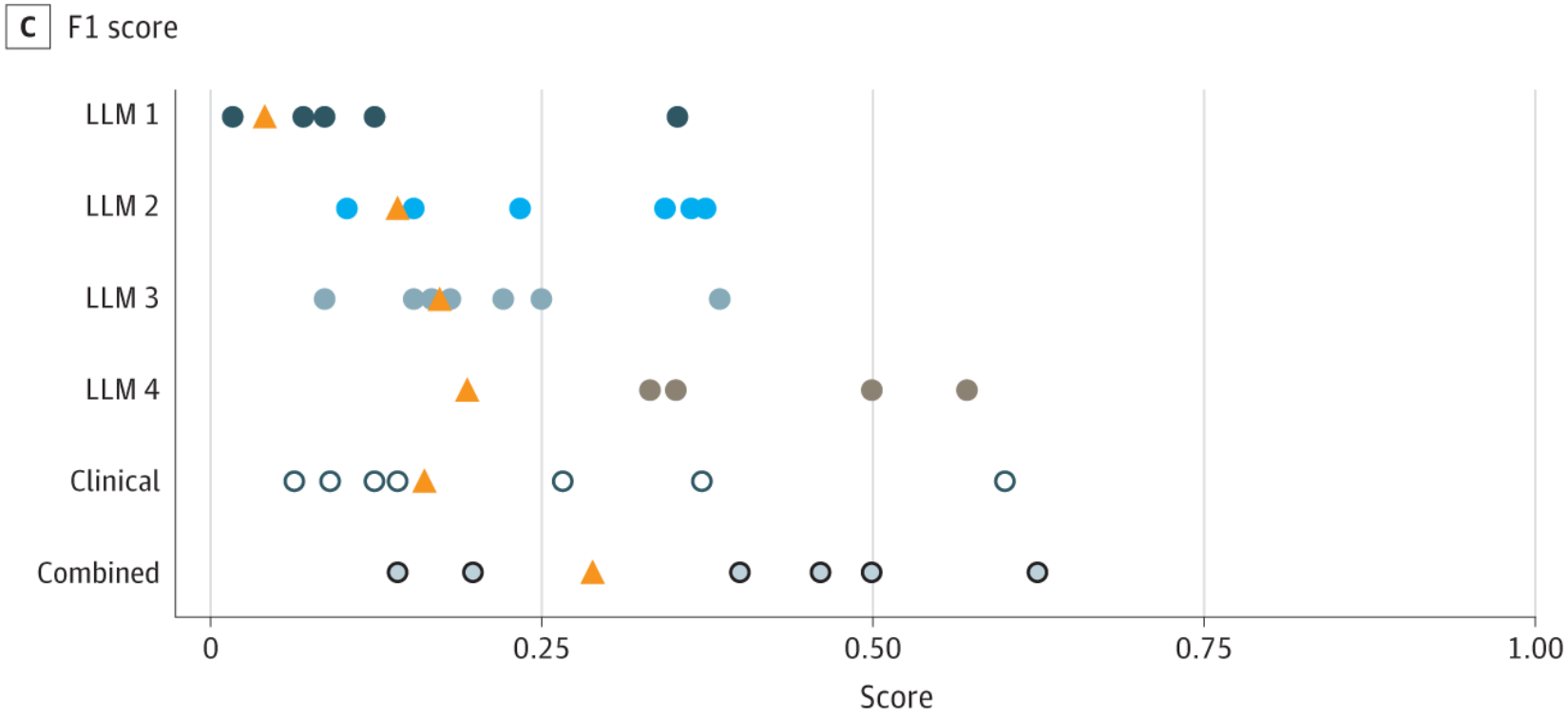

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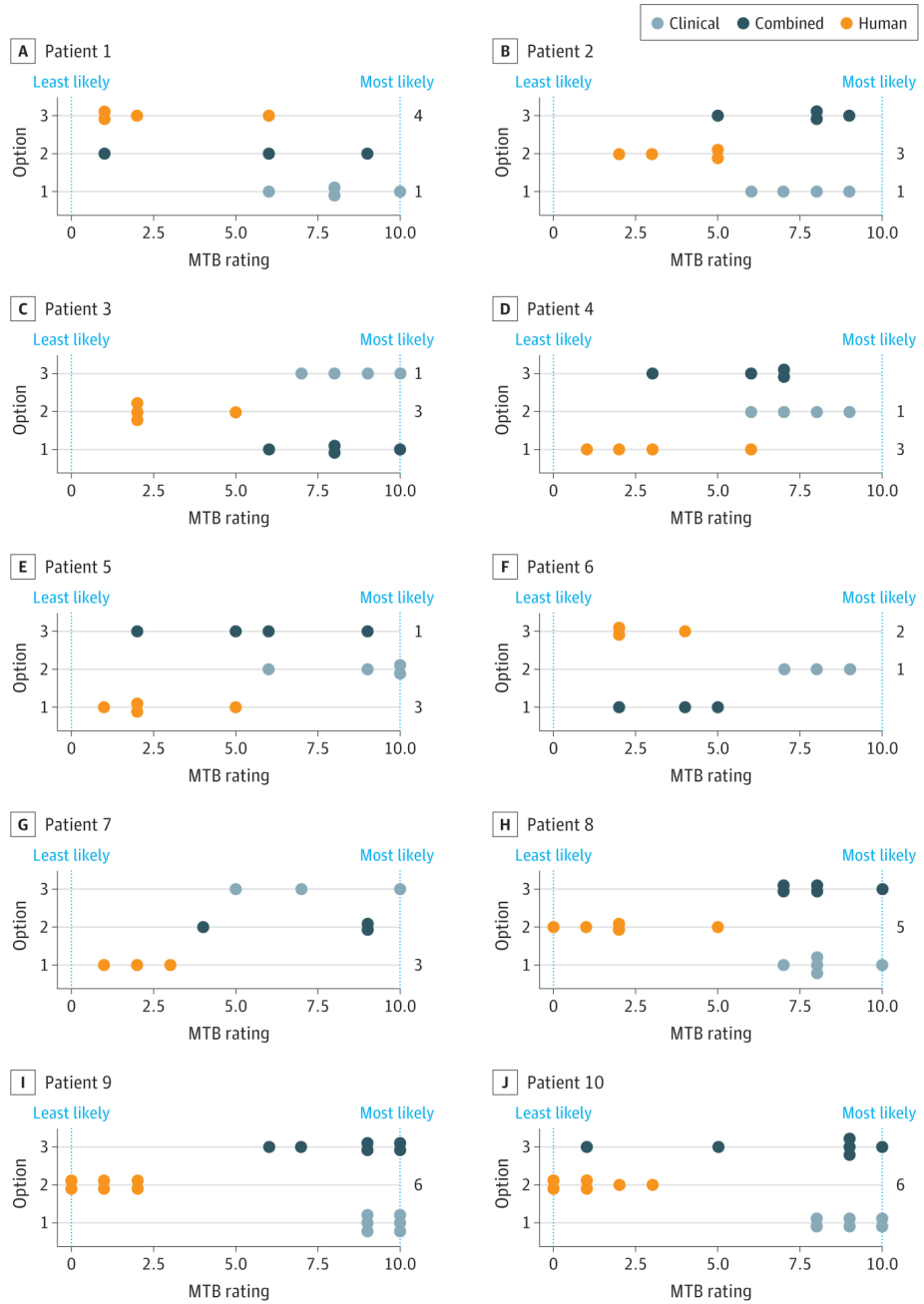

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The unique treatment strategy was antiandrogen therapy in a patient with salivary duct carcinoma with *HRAS* and *PIK3CA* variation. *HRAS* and *PIK3CA* comutated salivary duct carcinoma usually stain positive for the androgen receptor in immunohistochemistry. Antiandrogen therapy was not suggested by the human expert because no immunohistochemistry results were provided.

Agenda

1. Background
2. Standards
3. Challenges
4. Summary

- Multi-Step Process
- Integration of novel analyses, treatments, combinations
- Integration of complex data and multiple data layers
- Clinico-genomic database for research use
- Future application of AI?

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thank you!

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