NEW DEVELOPMENTS IN GENETIC KNOWLEDGE FOR DEFENDANTS IN ASBESTOS CANCER CASES

ToxicoGenomica

October 30, 2018

ToxicoGenomica

A multidisciplinary group of geneticists, scientific consultants, and lawyers offering ala cart and turn-key services in genomics & systems biology for toxic tort litigation

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Presenters

David H. Schwartz, Ph.D.
Moderator

Kirk T Hartley, J.D.
Legal Updates

Len van Zyl, Ph.D.
Scientific Updates

Webinar Overview

- Overview and Current Legal Developments
- Genomics Overview
- Recent, Key Mesothelioma Cancer Research
- Recent, Key Ovarian Cancer Research
- ToxicoGenomica Custom Genome Panels
- How to Pick Your Cases for Genomics Application
- Conclusions / Q&A

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Overview & Current Legal Developments

Genomics and Asbestos Litigation

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- **Genetic and molecular knowledge is exploding**
  - Genetic knowledge revolutionized criminal law
  - Exponentially more knowledge now exists – revolutionizing tort litigation
- **Genetic arguments already are increasingly common**
- **Plaintiffs are embracing individual susceptibility as part of "reptile" tactics**
  - Mesothelioma cases
  - Talc cases ($4+ billion Ingham verdict in 22 ovarian cancer cases)
- **Some asbestos defendants are successfully using genetic and molecular knowledge**
  - more should be using the knowledge
  - short term uses in particular cases
  - strategic long term use in subsets of cases

Examples of Genomic Defense Successes

Plaintiff	Age at diagnosis	Cancer	Toxicant	Principle
Mr. Cacoilo v. Sherwin-Williams *et al*.	24	AML	benzene	Person destined for cancer due to multiple inherited variants
Ms. Blackford-Cleeton v. Marathon Oil *et al.*	32	Peritoneal meso + melanoma	asbestos	Mesothelioma causation, including other toxins are relevant to some mesotheliomas due to *CDKN2A* mutation (sun tanning and smoking)
Mrs. Guzman v. Exxon Mobile *et al.*	28	papillary thyroid cancer	α-radium ($^{226}Ra/^{228}Ra$)	Gene expression tests sometimes can “fingerprint” a cause of cancer

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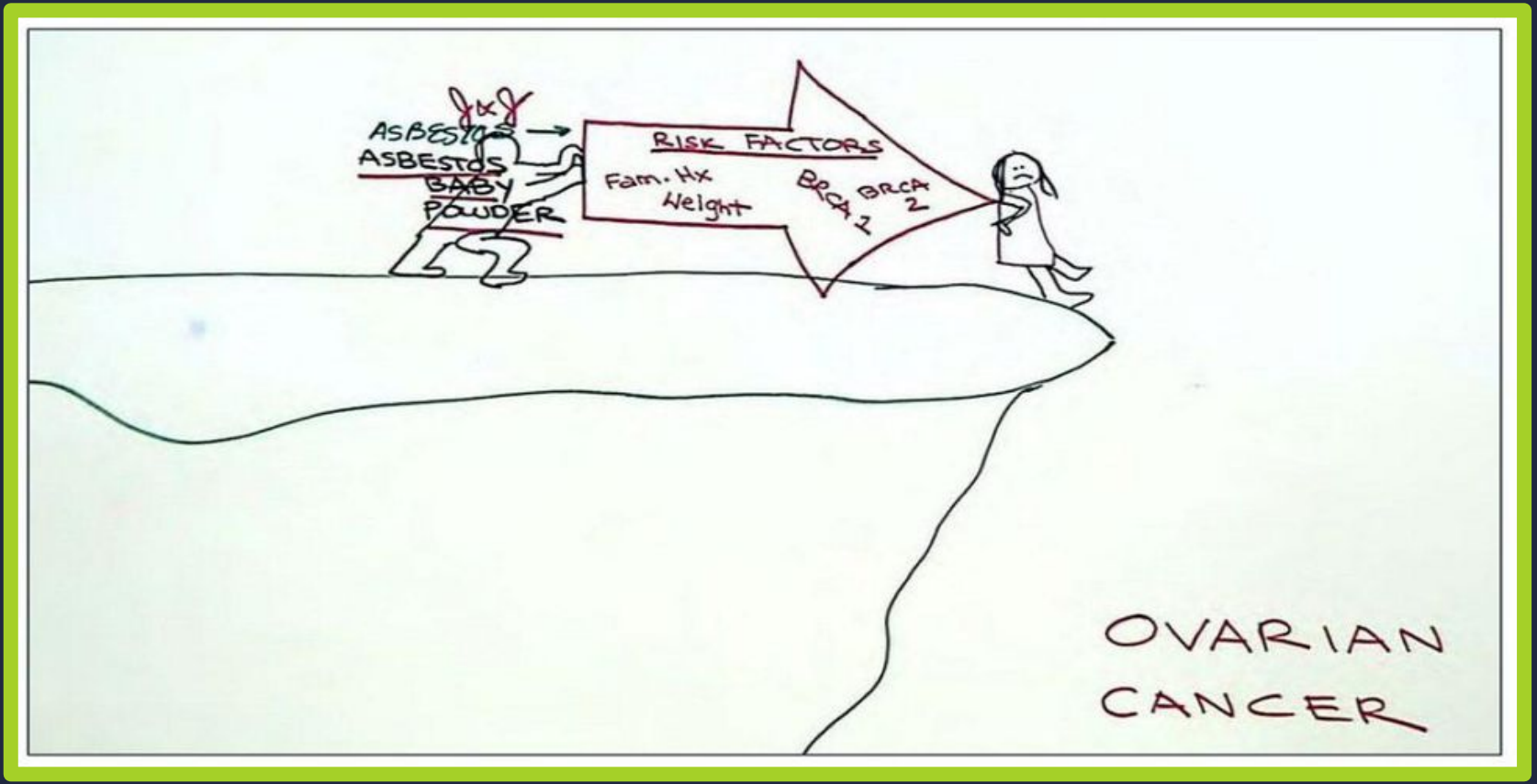
Plaintiffs Have Embraced Genetics



Mark Lanier

- Ingham produced $4+ billion verdict in 22 ovarian cancer cases
- Well credentialed plaintiff expert

New Developments in Genetics - Asbestos Cases

- Courts are allowing genetic testing
  - But Marshall in CA went the wrong way without genetic expert for defense

- Mark Lanier and other plaintiff lawyers are expanding embrace of "susceptibility"
  - Ovarian cancer cases
  - Some defendants are not ready
    - Newly published scientific research defines multiple inherited genes now associated with development of mesothelioma
      - Panou paper (August 2018) from University of Chicago – Dr. Hedy Kindler
      - Repeated use of term susceptibility instead of predisposition

- Multi-gene testing is needed; testing only *BAP1* gene misses a wide range of information

New Developments in Genetics - Asbestos Cases (Cont'd)

- More attention to inflammation and other mechanistic explanations for asbestos and cancer
- New, ongoing studies with genetically altered mice exposed to chrysotile
  - Dr. Joseph Testa has testified as an expert for plaintiffs
  - Excess deaths reported in interim data (May 2018)
- Some defendants and defense experts are using epidemiology to urge that most/many peritoneal mesos in women are not caused by asbestos
  - No clear acceptance by juries (at least so far)
- Smoking and other factors are relevant in some small numbers of mesotheliomas
- Groups continue to work on developing "fingerprints" for asbestos-induced cancers

California and Genetic Testing After Ortwein

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- Nolan Lamb – Contra Costa County
  - Brayton firm for 33 year old male with peritoneal mesothelioma
  - CertainTeed sought genetic testing, and plaintiff stipulated to allow
  - Testing revealed *BAP1* variant (mutation)
  - Two pathology experts:
    - Plaintiff: Dr. Sobonya
    - Defense: Dr. Feingold
  - Defense verdict (but wisdom is verdict was based on other factors)

- Cynthia Marshall – Alameda County
  - Kazan firm opposed *BAP1* testing
    - Argued "no evidence" *BAP1* variant - by itself - will lead to mesothelioma
  - Plaintiff Verdict
    https://www.youtube.com/watch?v=3iGWLA93n3s

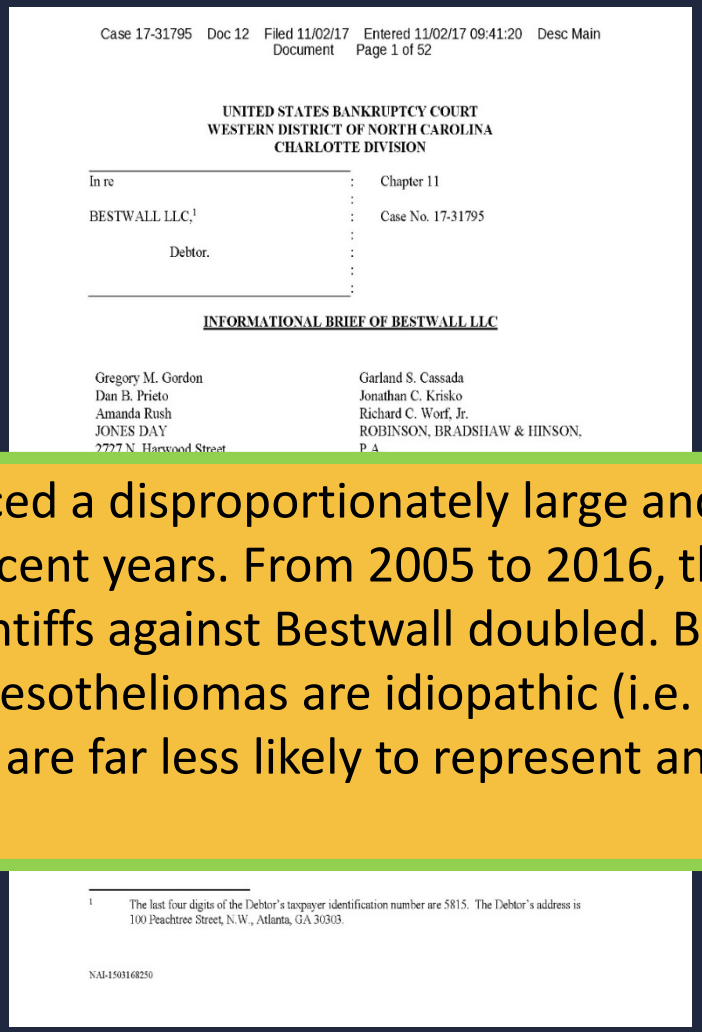
Genetic Testing – Outside of California

- Lanzo – Middlesex County, NJ (Judge Viscomi)
  - *TP53* genetic testing allowed
  - CMO motion for *BAP1* testing is still awaiting action

- Also in Middlesex County, NJ, one defendant sought genetic testing in two mesothelioma cases (Johnson and Lashley)
  - some other defendants joined formally or less formally, but motions mooted by dismissals

- Bailey – Lawrence County, TN
  - genetic testing allowed despite plaintiff's objections

Bestwall – Challenging Mesothelioma Causation



Case 17-31795 Doc 12 Filed 11/02/17 Entered 11/02/17 09:41:20 Desc Main Document Page 1 of 52

UNITED STATES BANKRUPTCY COURT
WESTERN DISTRICT OF NORTH CAROLINA
CHARLOTTE DIVISION

In re	:	Chapter 11
BESTWALL LLC,[1]	:	Case No. 17-31795
Debtor.	:	

INFORMATIONAL BRIEF OF BESTWALL LLC

Gregory M. Gordon
Dan B. Prieto
Amanda Rush
JONES DAY
2727 N. Harwood Street

Garland S. Cassada
Jonathan C. Krisko
Richard C. Worf, Jr.
ROBINSON, BRADSHAW & HINSON, P.A.

[1] The last four digits of the Debtor's taxpayer identification number are 5815. The Debtor's address is 100 Peachtree Street, N.W., Atlanta, GA 30303.

NAI-1503168250

"Notably, Bestwall has faced a disproportionately large and growing number of female mesothelioma cases in recent years. From 2005 to 2016, the annual number of mesothelioma cases filed by female plaintiffs against Bestwall doubled. Because recent studies show that the vast majority of female mesotheliomas are idiopathic (i.e. not connected to any particular cause or exposure), these cases are far less likely to represent any valid claims that can be attributed to Bestwall."

"Informational Brief" filed by Bestwall/Georgia Pacific on 11/2/17 (Footnote 35)
https://www.scribd.com/document/379701100/Bestwall-Georgia-Pacific-chapter-11-Doc-12c-Informational-Brief-as-Filed

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Experts Agree

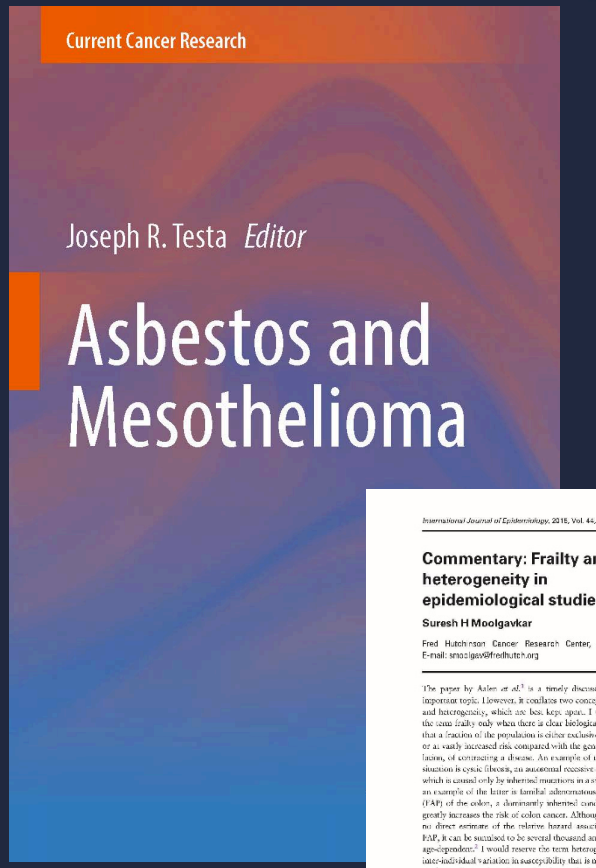
Individual Genetic Variables Do Matter

"…large differences in susceptibility are determined by major gene defects or by events occurring in embryonic life that alter populations of critical cells…."

"…existing models for incidence and development of cancer do not account for individual variability"

- Int. J. Epidemiol. 2015 Aug; 44(4):1425-6

Dr. Moolgavkar acknowledged epidemiology missed role of smoking in some small number of mesotheliomas (2017)



International Journal of Epidemiology, 2015, Vol. 44, No. 4 1425

Commentary: Frailty and heterogeneity in epidemiological studies

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International Journal of Epidemiology 2015; 1425–1426
doi: 10.1093/ije/dyv044
Advance Access Publication Date: 16 April 2015

The paper by Aalen *et al.*[1] is a timely discussion of an important topic. However, it conflates two concepts, frailty and heterogeneity, which are best kept apart. I would use the term frailty only when there is clear biological evidence that a fraction of the population is either exclusively at risk, or at vastly increased risk compared with the general population, of contracting a disease. An example of the former situation is cystic fibrosis, an autosomal recessive condition, which is caused only by inherited mutations in a single gene; an example of the latter is familial adenomatous polyposis (FAP) of the colon, a dominantly inherited condition that greatly increases the risk of colon cancer. Although there is no direct estimate of the relative hazard associated with FAP, it can be simulated to be several thousand and strongly age-dependent.[2] I would reserve the term heterogeneity for inter-individual variation in susceptibility that is much more modest and arises from factors that do not clearly single out a sub-population at greatly increased risk of the disease. Heterogeneity of susceptibility may arise from biological factors, such as polymorphisms in metabolizing enzymes or inter-individual variations in efficiency of DNA repair or cell proliferation, or from purely stochastic considerations.

The role of stochasticity as a factor in heterogeneity has been under-appreciated. To their credit, Aalen *et al.*[1] clearly recognize the importance of stochastic factors, and they correctly note that it is not easy to distinguish between stochastic and chaotic behaviour arising from unstable dynamical systems. I prefer to work with stochastic models. Many toxicology experiments are conducted with highly inbred strains of animals kept under identical environmental conditions. Yet, despite identical 'nature and nurture', these animals do not succumb to disease at identical ages, and they show considerable heterogeneity in the number and size distribution of lesions. The same would presumably be true of humans subjected to similar experimental conditions. To summarize, there are three sources of inter-individual variation in susceptibility in human populations first, at the most fundamental level, we have stochastic variations that are present even in genetically uniform populations living under similar or identical environmental conditions; second, we have inter-individual variations in the efficiency with which fundamental biological processes are carried out; and finally, large differences in susceptibility are determined by major gene defects (e.g. FAP) or by events occurring in embryonic life that alter populations of critical cells (perhaps exemplified by testicular carcinoma, as Aalen *et al.* note[1]).

Methods of analysis based on stochastic models of carcinogenesis automatically address stochastic heterogeneity if (and only if) the exact (stochastic) solutions to the models are used for data analyses. In this regard, I find the discussion of carcinogenesis models in this paper unclear. The models described in the papers by Heidenreich, Meza *et al.*, Moolgavkar *et al.* and Armitage and Doll cited by Aalen *et al.*[1] were not developed to describe hazard functions in individuals, as the authors state, but rather in populations of like individuals. If the exact (stochastic) solution of these models is used in homogeneous populations, stochastic heterogeneity is immediately introduced. Let $N_{t,1}$ and $N_{t,2}$ be random variables representing the number of pre-malignant and malignant cells, respectively, in a tissue at age t and let μ_{12} be the last mutation rate. Then, the exact stochastic solution to a one-step process model is equivalent to solving the following expression for the hazard function, $h(t) = P'(t)/[1-P(t)] = \mu_{12} E[N_{t,1} | N_{t,2} = 0]$, where $P(t)$ is the probability of a malignant cell at age t and E is the expectation. Quite often, approximate (deterministic) solutions, $h(t) = P(t) = \mu_{12} E[N_{t,1}]$, are used and these can yield misleading results and inferences. Thus, for example, the pioneering Armitage-Doll model is stochastically a pure birth process; the almost universally used power function approximation, which is the hazard function arising from a Weibull survival model, is only the first non-zero term in the Taylor series expansion of the exact (stochastic) solution.[3] In contrast to this approximation, which is monotonically increasing without bound, the exact hazard function approaches a finite asymptote, which would be expected with a heterogeneous population. Thus, starting with a homogeneous population, the (exact) Armitage-Doll and other stochastic models introduce stochastic heterogeneity.

Key Messages on Use of Genomics

- **Each person is unique** in genetic make up and experiences, therefore epidemiologic evidence has limits
- Genomic analysis is most obviously called for in cases of outlier plaintiffs:
  - early onset (before age 50)
  - multiple primary cancers
  - a “no dose” case that fits criteria merits a genomic defense evaluation
- Like any forensic investigation, results can cut both ways
  - risks can be mitigated using good initial review of facts
  - don’t apply genomics to all cases
- Genomic/systems biology defense strategies offer juries objective, **quantitative data specific to the individual**, thus avoiding the “it’s unknown” argument (idiopathic)

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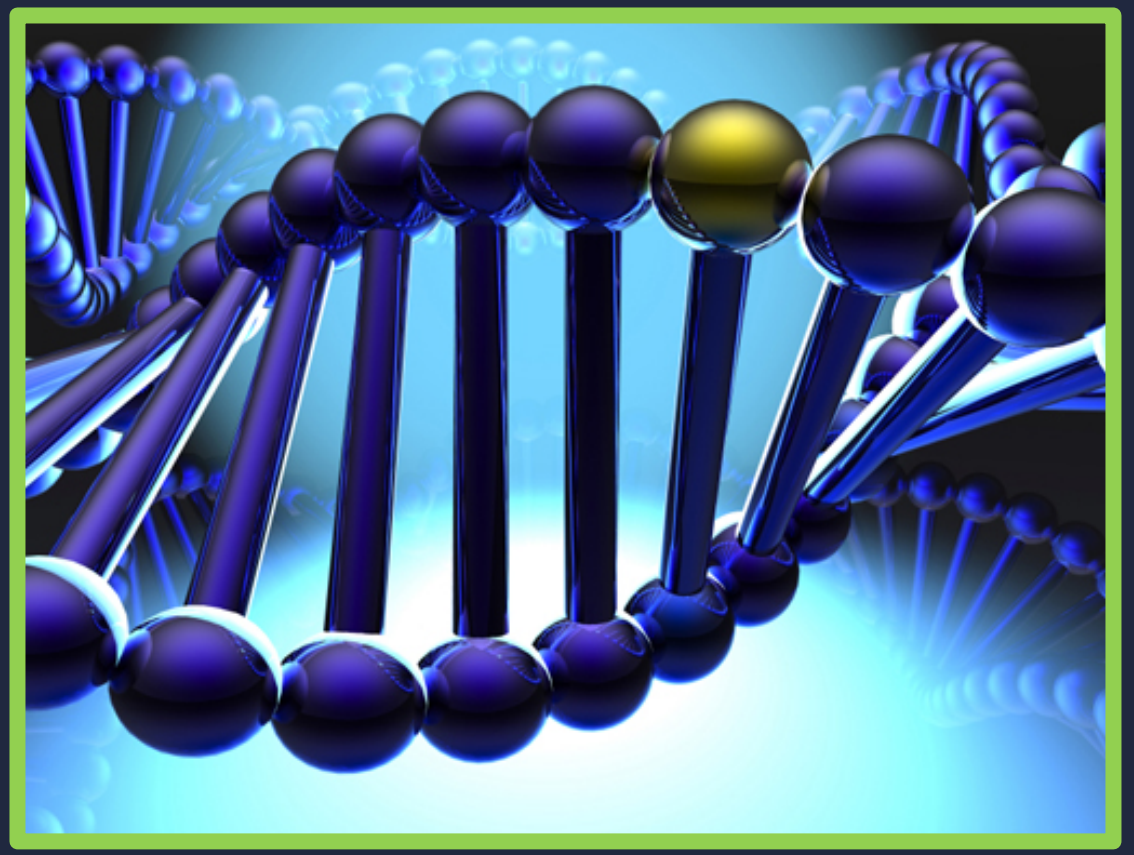
Genomics Overview

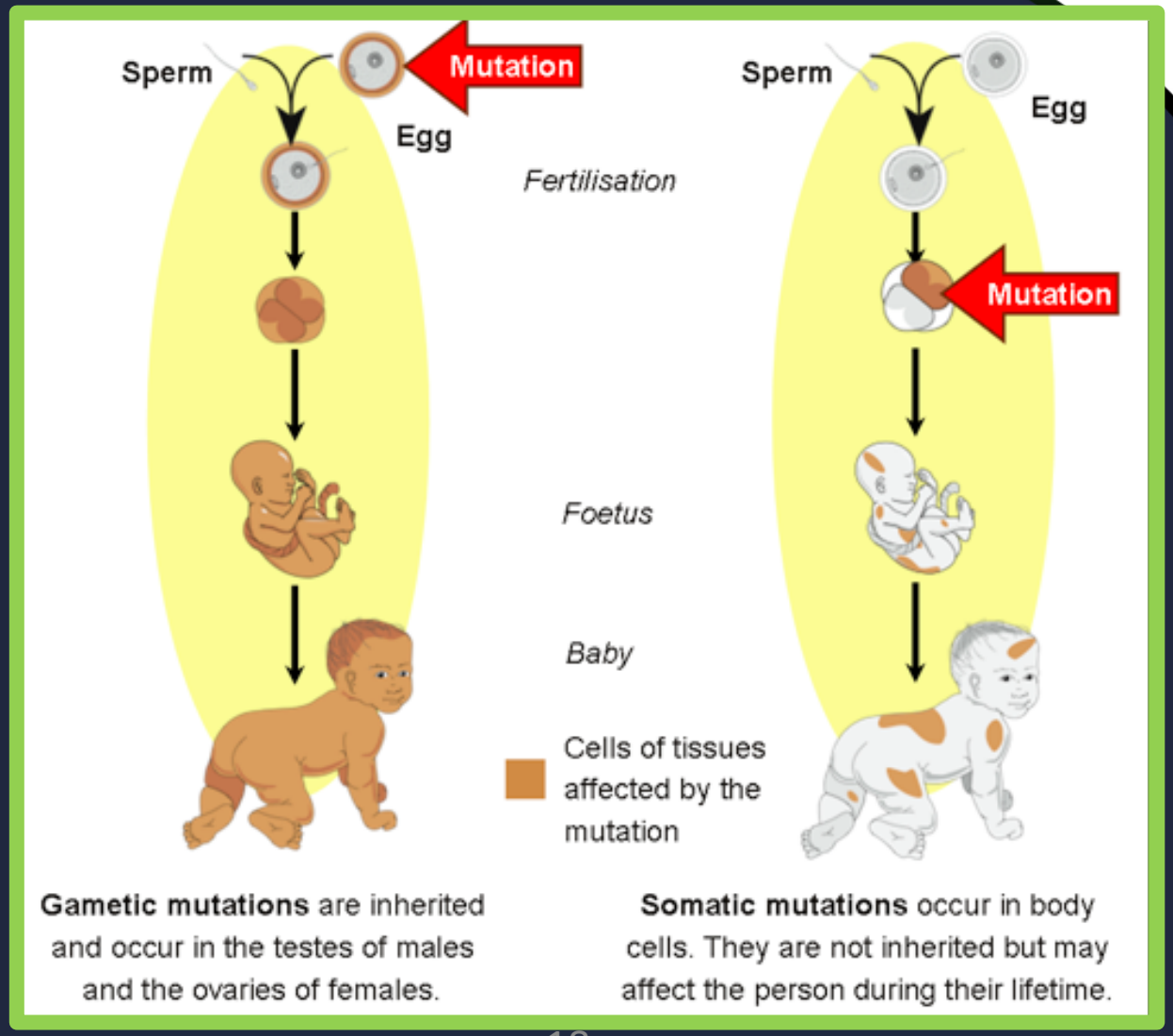
Cancer is a Disease of the Genome

At the molecular level, all cancers are caused by the accumulation of genetic lesions that disrupt normal cellular processes.



Cancer is from Gene Mutations

- **Germline Mutations**
  - Present in egg or sperm
  - Are heritable
  - Cause hereditary cancer syndromes
- **Somatic Mutations**
  - Occur in non-germline tissues
  - Are non-heritable
  - Later onset



Genetic Predisposition

- Many diseases and conditions arise from inherited genetic mutations
  - e.g., cystic fibrosis, sickle cell anemia, Huntington's disease, etc.
- Inherited disease syndromes have given rise to the **genetic counseling** profession
- There are over 200 known **Hereditary/Familial Cancer Predisposition Syndromes**
  - Genetic mutations **CAUSE** these cancer syndromes
  - No toxin required
- Highly **penetrant** mutations result in **early onset cancers**
- Defense case should emphasize genetic predisposition (when possible)

Genetic Resistance

- **Vast majority of people (> 98%) in any given year do not have cancer** (https://ourworldindata.org/cancer)
- Most people are genetically resistant due to protective variants (known as “alleles”)
- Defense strategy should be to emphasize that “resistance” is normal
- Genetic testing can objectively assess presence of genetic resistance

Confronting Genetic "Susceptibility"

- Susceptibility is a complex concept
- Plaintiff counsel and experts increasingly use and oversimplify
- **There are inherited genetic variants that influence the response to a toxicant**
  - **Some genetic variants protect from toxicant-induced cancer**
  - **Some genetic variants will increase "susceptibility" (but concept can be easily misused)**
- Objectively measurable
  - For some toxins (e.g., benzene), genetic variants are well defined
  - For asbestos and others, knowledge is still developing
- If "susceptibility" is asserted by plaintiff's expert, the validity of that assertion can be objectively tested

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Recent
Key Mesothelioma Research

Genomics in Mesothelioma:
4 Key Take-home Messages

1. **Mesothelioma has a significant CAUSAL Hereditary Genetic Predisposition Component**
2. "Genetic Resistance/Susceptibility" can be tested
3. Immune system response to inflammation is intensely individual
4. Research groups continue seeking fingerprints for asbestos-induced disease

Hereditary Predisposition to Mesothelioma

- *BAP1* was the first gene known to predispose to mesothelioma
- Current data suggests that 5-20% of all mesotheliomas are initiated by germline mutations in genes known to cause onset of clinically, well defined Hereditary/Familial Cancer Predisposition Syndromes, in the absence of asbestos exposure (Kraynie *et al*., 2016)
- There are currently ~60 germline genes known to initiate mesothelioma in a hereditary context (and more to come!)

Hereditary Predisposition to Mesothelioma (cont'd)

- Yet to be discovered (non-asbestos) genetic factors may be responsible for mesothelioma in *BAP1*-unmutated families (Ascoli *et al.,* 2016)
- *CDKN2A* predispose to melanoma and mesothelioma *(*in addition to *BAP1* germline mutations) [Betti *et al*., 2016]
- Numerous pathogenic germline mutations predispose to malignant mesothelioma: *PALB2*, *BRCA1*, *FANCI*, *ATM*, *SLX4*, *BRCA2*, *FANCC*, *FANCF*, *PMS1*, *XPC* (Betti *et al*., 2017)

Hereditary Predisposition to Mesothelioma (cont'd)

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In 2018 Alone, Many Published Articles Have Demonstrated Hereditary Predisposition

- Inherited germline genetic mutations have a significant role in the development of mesothelioma (Kharazmi *et al*., 2018)
- 198 patients with mesothelioma showed mutations in 13 known cancer predisposition genes, including *BAP1 (*younger, minimal asbestos exposure, a second cancer, and peritoneal malignant mesothelioma (Panou *et al*., 2018)
- 3/88 patients with peritoneal mesothelioma had *ALK* gene fusions (rearrangements) – ALL lacked asbestos exposure
- Numerous mutations identified in **11 (non-BAP1)** "Cancer Census Genes" in a Belgian family with history of mesothelioma
- Infant with mesothelioma belonged to a family with known pathogenic mutations in *ATM* (gene known to increase risk of various malignancies
- Germline *PTEN* mutation in a patient with mesothelioma

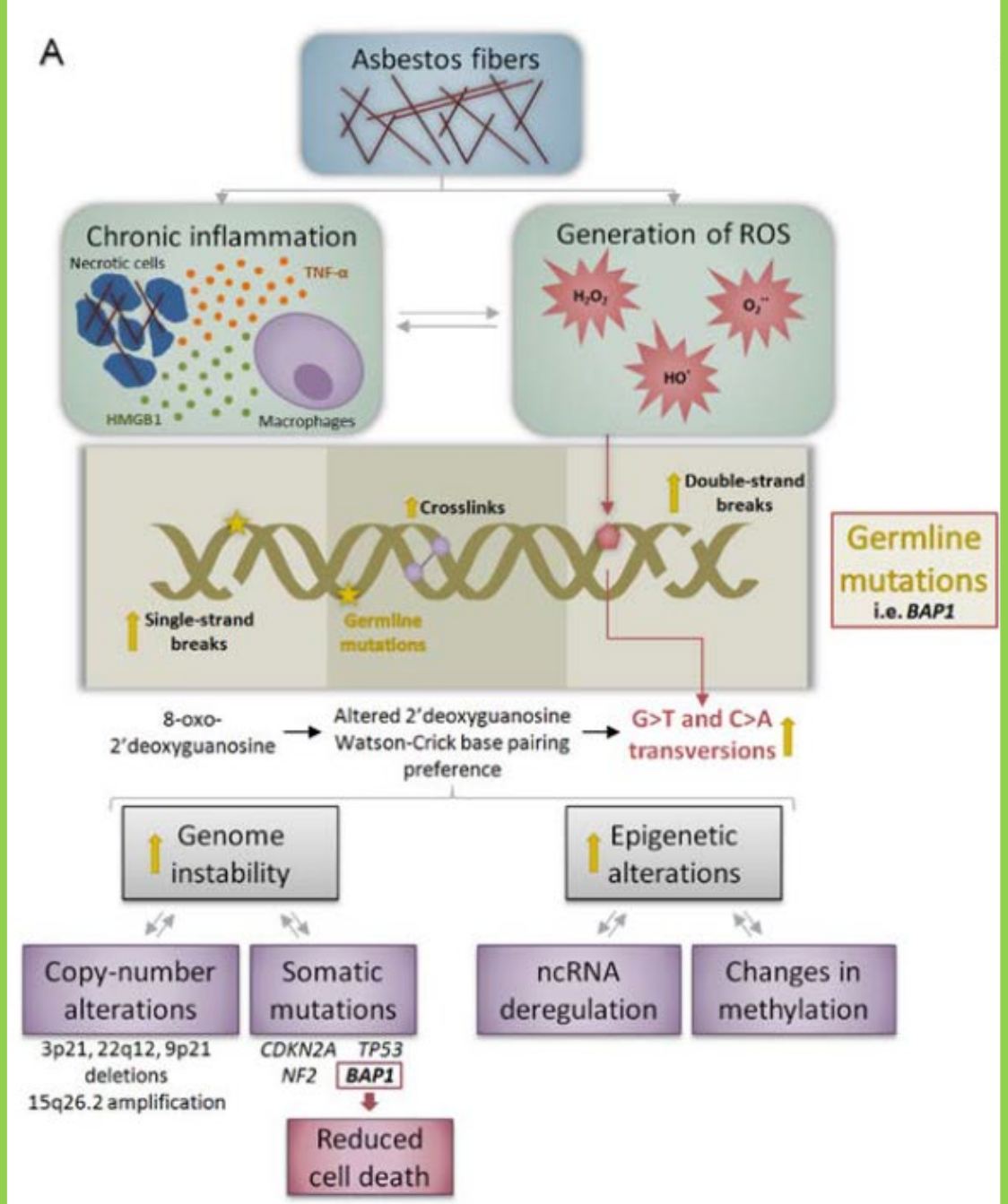
Defense Opportunity

- Currently, there are approximately ~60 hereditary cancer predisposition genes for mesothelioma (not just *BAP1*)
- Multi-gene testing is available and should be utilized

Genetic Resistance/Susceptibility Can be Tested

New and Progressing Research is Identifying Objective Genetic Markers Associated with Risk

- Presence or absence of these factors can be ruled in or out
- Review article summarizing numerous genetic variants for how a person responds to asbestos, including *CRTAM*, *SDK1*, and *RASGRF2* genes
- 10 genetic variants identified that influence how a person responds to asbestos (Matullo *et al., 2013)*
  - Further analysis of this Italian population validated existence of variants



Mechanism of Asbestos Carcinogenicity

"Asbestos-related carcinogenic effects mainly occur through two mechanisms: **activation of chronic inflammation** and generation of reactive oxygen species (ROS). Both mechanisms are known to promote DNA damage in the forms of single-strand breaks, crosslinks, and double-strand breaks."

Sage *et al.*, 2018

Immune System Response to Inflammation is Intensely Individual

- **Inflammation** is a complex biological response to harmful toxicants and/or pathogens
  - Inflammation is a protective response involving our **immune cells**, blood vessels, and molecular mediators
- **Each individual will respond differently to asbestos exposure based on inherited genetics regulating immune response**
- 25%-75% per cent of our immune defense is genetically inherited/determined
  - “The strength of someone's constitution is thus genetically determined for each stimulus.” University Medical Center Groningen (UMCG) and the Broad Institute of MIT and Harvard

Research Groups Continue Seeking Fingerprints for Asbestos-induced Disease

Fingerprints are Sought Using New Genetic Technologies and Biomarkers

- miRNA expression is altered soon after exposure to occupational and environmental carcinogens like asbestos
- Serum miRNAs are ideal biomarkers since they are non-invasive, stable, they vary little in the general population, and are not expensive to analyze (Micolucci *et al*., 2016)
- Prior studies seek to differentiate causes (e.g., smoking-induced lung cancer from asbestos-induced lung cancer)

Recent Efforts to Fingerprint Asbestos-Induced Mesothelioma

- Recent study identified four serum miRNAs (i.e. *miR-126*; *miR-205*, *miR-222* & *miR-520g*) that were shown to be directly implicated in asbestos-induced malignant diseases (i.e. mesothelioma and lung cancer) [Santarelli *et al*., 2018]
  - These miRNAs are well known to be involved in major pathways linked to cancer initiation and development/tumorigenesis
- Authors concluded that the discovery of a miRNA panel for asbestos-induced malignancies could have great medico-legal impact
- Limitations of the study: relatively small study population; difficulty to estimate asbestos exposure

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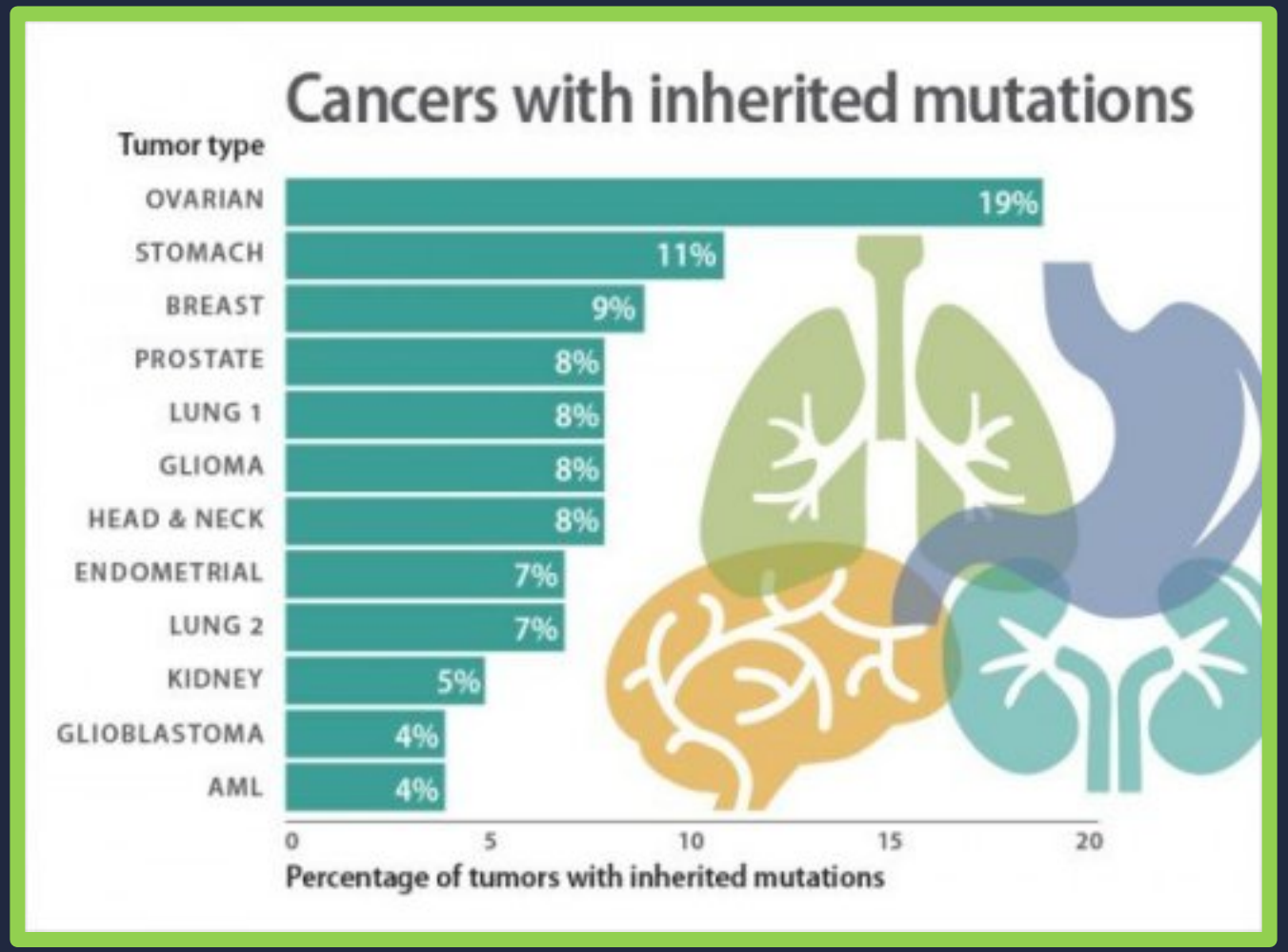
Recent Key Ovarian Cancer Research

Genomics in Ovarian Cancer
Key Take-home Messages

1. Ovarian cancer is one of the most genetically predisposed cancers
2. There are approximately 37 genetic drivers of ovarian cancer - it's not not just about *BRCA1* and *BRCA2* anymore
3. Other –omics factors (e.g., epigenetics) also drive ovarian carcinogenesis
4. Old genetic screening criteria are inadequate

Germline Genetic Mutations Drive Ovarian Cancer Predisposition



https://www.sciencedaily.com/releases/2015/12/151222084730.htm

Germline Genetic Mutations Drive Ovarian Cancer Predisposition

- Current data indicate 19% - 23% of ovarian cancers are due to a wide range of inherited driver mutations (Toss *et al*., 2015)
- **12 novel predisposition loci for OC identified** *(*Phelan *et al. 2017)*
- Ovarian cancer patients had genetic variants in genes associated with breast and ovarian cancer - most frequent were *BRIP1* and *MSH6 (*Minion *et al.* 2015)
- Pathogenic variants found in 4.5% of *BRCA*-negative patients (Lincoln *et al.,* 2015)
- 8.7% of women sequenced with breast cancer or OC harbored mutations in at least **1 of the 19 genes**: *ATM, CHEK2, PALB2, MSH6, NBN,* and *RAD51D* (Crawford *et al*., 2017)

Germline Genetic Mutations Drive Ovarian Cancer Predisposition (cont'd)

- *RAD51* paralogs (*RAD51B, RAD51C, RAD51D, XRCC2*, and *XRCC4*) [DNA repair mechanism] have been found to be involved in ovarian cancer (Golmard *et al.,* 2017)
- Eoh *et al.,* 2017 found variants in 6 no-*BRCA1/2* germline genes that were pathogenic or likely pathogenic in ovarian cancer patients. Those genes include *CHECK2, MSH2, POLE*, and *RAD51C.*
- Jessica Lang and William Hendricks in 2018 found that the tumor suppressor *SMARCA4* mutations can drive the development of ovarian cancer.
- Hirasawa *et al*., 2017 identified 11 genes, including *ATM, MRE11A, FANCC,* and *GABRA6*, harboring pathogenic variants frequently found in Japanese women diagnosed with ovarian cancer at a younger age.
- Earp and coworkers, 2018 found that germline variants in the GTPase superfamily of signal transducers are linked to familial ovarian cancer risk.

Old Genetic Screening Criteria Are Inadequate

Therefore defendants should not depend only on information previously generated

Rowely *et al*. (2018) concluded that HBOC [Hereditary Breast and Ovarian Cancer] genetic testing was well accepted, and the majority of high-risk gene carriers identified would not meet eligibility criteria for genetic testing based on their existing family history.

Defense Opportunity

- Currently, there are approximately 37 hereditary cancer predisposition genes for ovarian cancer (not just *BRCA1/BRCA2*)
- Multi-gene testing is available and should be utilized

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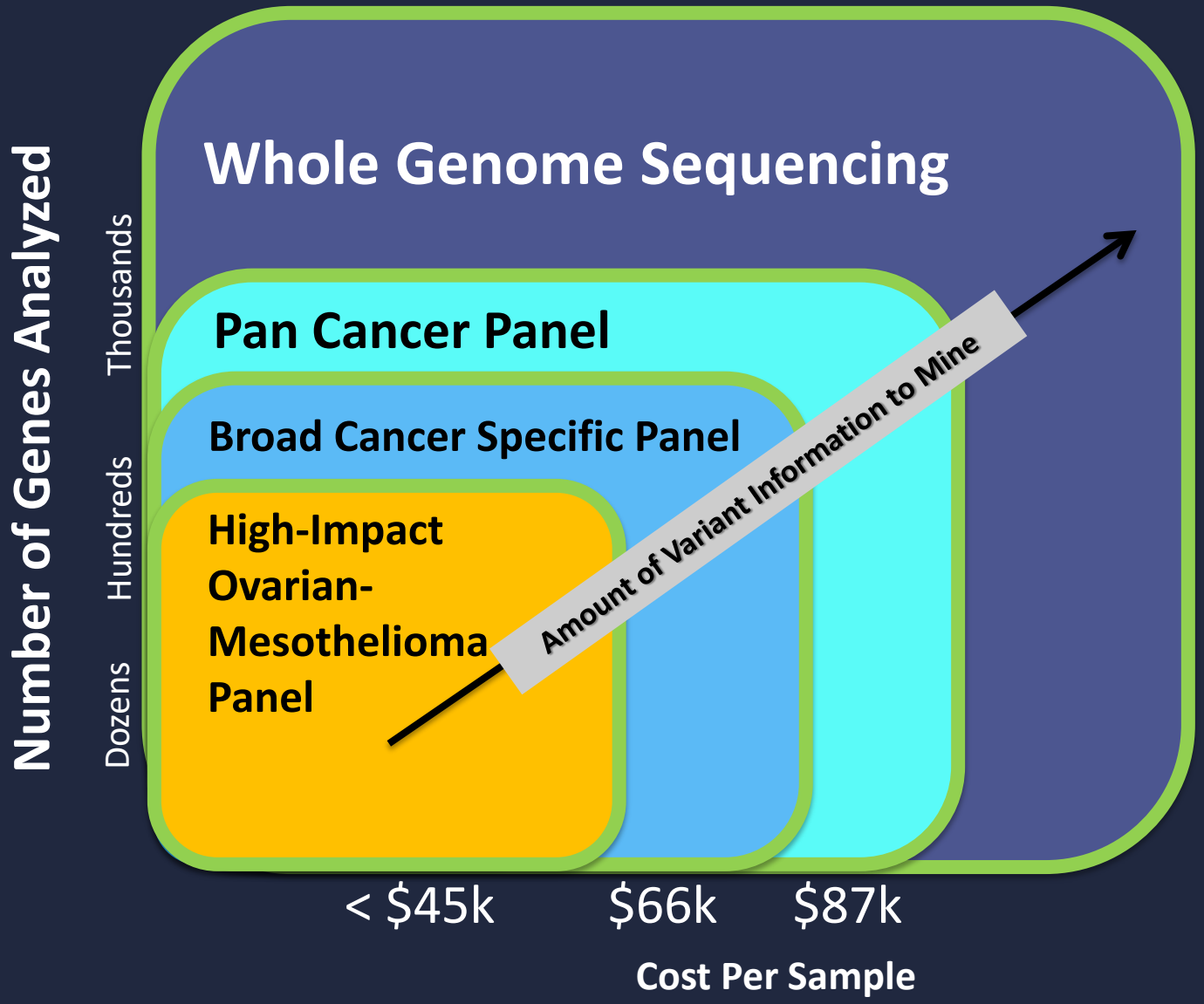
ToxicoGenomica Custom Genome Panels

Multiple Levels of Genetic Testing

- TG Custom Gene Panels for Germline (Healthy) Tissue
  - Lowest cost per case
- Whole Exome Testing
  - More comprehensive approach, but more costly
- Full Genome Testing
  - Most comprehensive approach, but most costly

Single Plaintiff Case - Data vs. Cost Trade Off



- Cost assumes single plaintiff
- Costs drop to < $15K for Tier 1 simultaneously running multiple DNA samples (multiplexing)

Types of Reporting

Basic Reports

(included with each tier)

- List of all relevant “high profile” variants
- Raw sequence of genes screened
- Easy to read format for disclosure to adverse parties and presentation to expert witnesses

Expert Reports

Curated Report

(more comprehensive)

Basic Report plus:

- Reports clinical relevance identified in public databases, as of date x
- Includes citations to relevant peer-reviewed scientific literature, as of date x

Comprehensive Report

(most comprehensive)

Basic & Curated Report plus:

- In-depth investigation of the individual’s genome
- Detailed analysis of molecular mechanisms
- Further development of more linkages and a direct connection to the family pedigree
- Fully up-to-date

Benefits of Custom Gene Panels

- Tiered custom gene panels increase flexibility
- Start with Tier 1 with high likelihood of finding inherited mutations
- A more concrete defense instead of arguing about unknown causes (i.e., idiopathic disease)
- Provides objective data that cancers arise from genetic causes
- Custom panels can be limited to fewer genes if so ordered by courts

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Case Selection Criteria

Family Pedigree

- 2+ relatives on same side of family diagnosed with related cancers (e.g. mesothelioma, melanoma, breast cancer, kidney cancer, pancreatic cancer and lung cancer)
- Evidence of autosomal dominant transmission (e.g. 1st degree relatives)
- Ancestry (e.g. Ashkenazi ancestry)

Personal Medical history

- Early age at diagnosis (<50 yrs)
- Multiple primary tumors
- Bilateral/multiple rare cancers
- Constellation of tumors consistent with known cancer syndrome (e.g. hematologic cancers, breast and ovary)

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Concluding Points

Concluding Points

Trials Increasingly Involve Low Dose Claims

- Plaintiff lawyers increasingly embrace genetic “susceptibility” arguments
- Defendants need to prepare for:
  - Plaintiff “susceptibility” arguments (including mouse data)
  - Expert depositions and cross examination for trial

Defendants Should Change the Paradigm

- Focus genetic investigation in selected cases with outlier facts
- Focus on genetic predisposition to Hereditary/Familial Predisposition Cancer Syndromes
- Introduce objective genomic data to support defense arguments
- Genetic data can provide powerful “alternative cause” arguments
- For appropriate situations, sequencing multiple plaintiffs is cost-effective

Q & A

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- The individual and collective slides are not intended to cover all factors that may be relevant to a particular situation, and should be considered in context.
-