

Médecine préventive et génétique: de la recherche à la pratique clinique Oncologie



Suzette Delaloge
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Liens d'intérêt

Aucun avec cette présentation

Intérêts financiers (tous à mon institution)

Elsan, Gilead, MSD, Novartis, BMS, Roche Genentech, Sanofi, Taiho :
investigation/comités scientifiques/conférencière invitée

DÉFIS

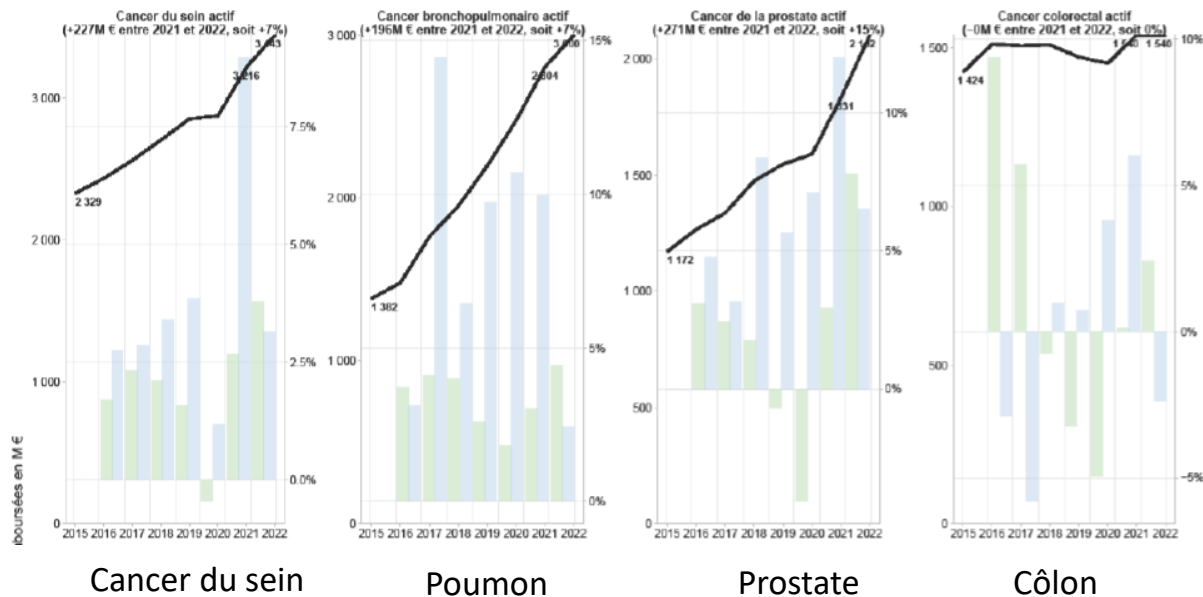


- Contre **les murs épidémiologiques** qui nous font face en oncologie
- Transformer nos **connaissances** sur la carcinogenèse en **stratégies** de prévention efficaces
- Élaborer des stratégies de prévention **efficaces acceptables abordables et équitables**
- **Remplacer les stratégies thérapeutiques** à long terme !

$$\frac{\lim_{n \rightarrow \infty} \frac{2^{2n} (n!)^2 \log 7}{(2n)! \sqrt{n}}}{\left[\int_0^\infty \frac{\sqrt{3} dt}{t^6 + 1} \right]^2 \left[\int_{-\infty}^\infty e^{-\pi t^2} dt \right] \left[\int_0^\infty e^{-t} t dt \right]} - e^i \sum_{k=0}^\infty \frac{8}{(4k+1)(4k+3)} \left| \int_0^\infty \frac{2t}{e^t - 1} dt \right| = 50$$

AUGMENTATION IMPORTANTE DES COÛTS LIÉS AU CANCER (FRANCE)

Données issues du système national d'assurance maladie 2015-2023

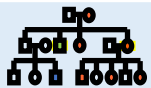


	2015	2023	AUGMENTATION
CANCER DU SEIN	2 833 783 414	4 683 899 421 €	+ 65%



LA PREVENTION PERSONNALISÉE : UN MÉLANGE DE DÉTECTION PRÉCOCE ET DE RÉDUCTION DES RISQUES

Déterminants sociaux et culturels



Âge, sexe, déterminants génétiques

Exposition

Tabac, alcool, agents infectieux, soleil, polluants, alimentation, excès de poids, rayonnement, sédentarité, hormones, etc.

Identification d'une situation présentant risque accru plusieurs années avant l'événement



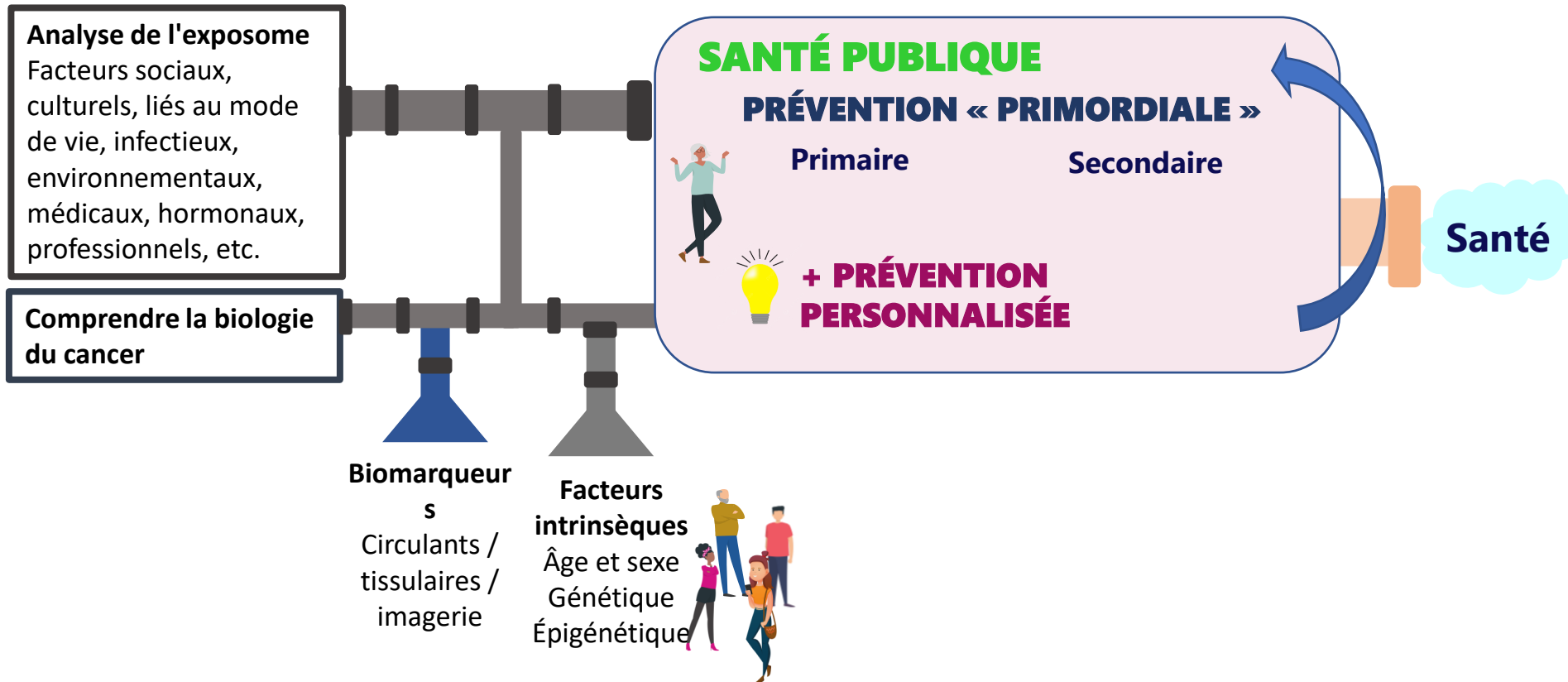
Intervention ciblée

Cancer

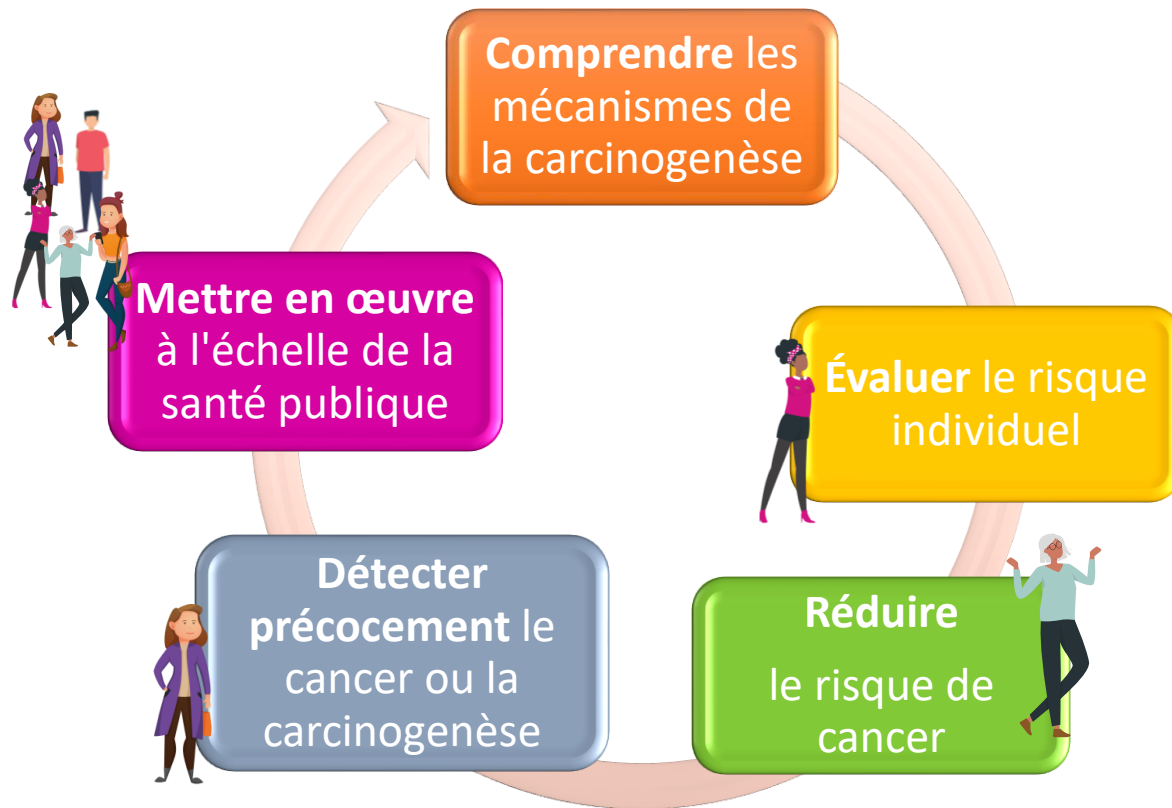


L'évaluation du risque individuel à un moment donné permet la mise en œuvre d'interventions appropriées.

!!! LA PRÉVENTION PERSONNALISÉE DU CANCER DOIT ÊTRE MISE EN PLACE AU NIVEAU DE LA SANTÉ PUBLIQUE



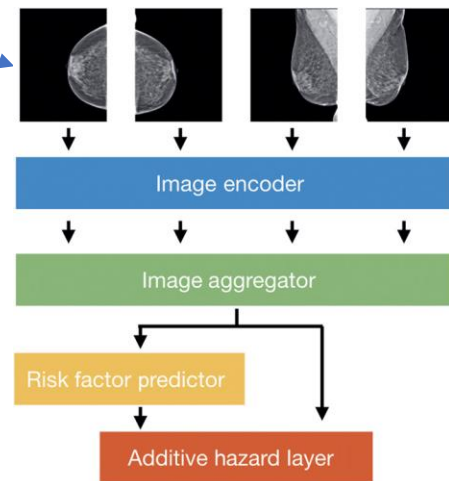
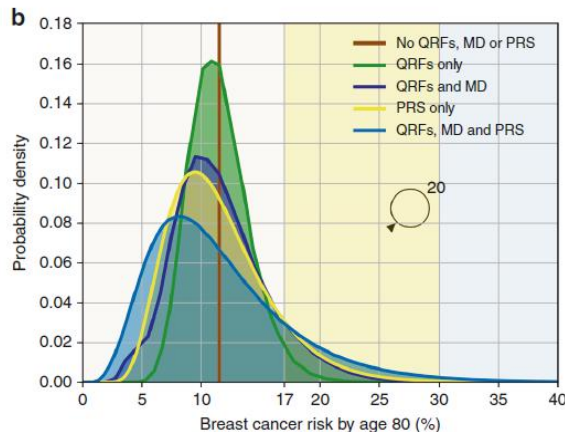
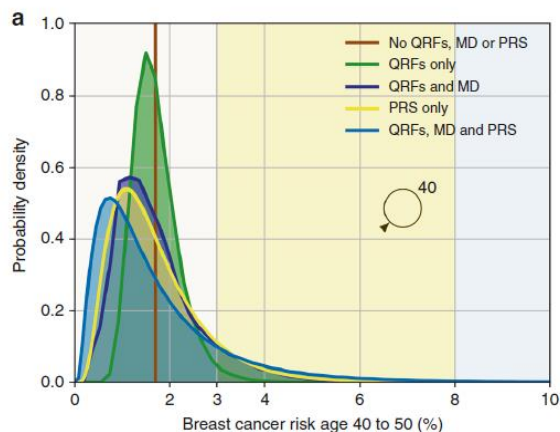
ÉLABORER LA PRÉVENTION PERSONNALISÉE NECESSITE UNE APPROCHE MULTIDISCIPLINAIRE



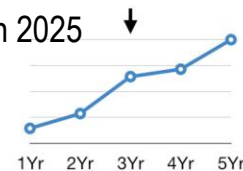
EVALUER LE RISQUE: SCORES COMPLEXES + GÉNÉTIQUE vs RADIOMIQUE?



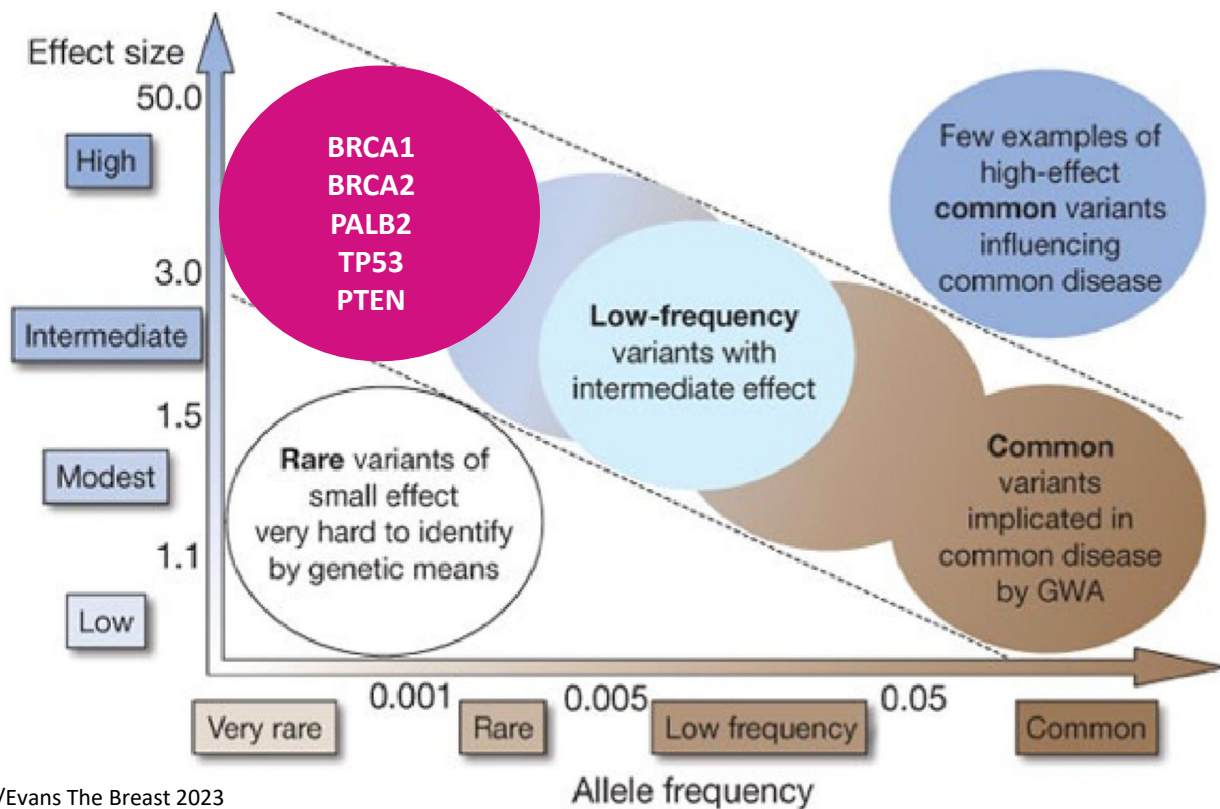
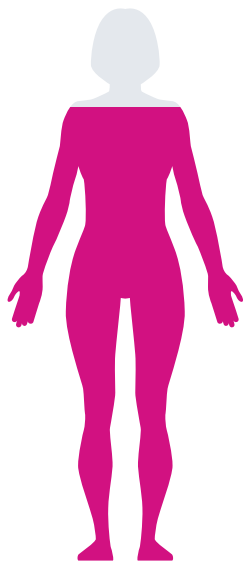
Socle + génétique (scores SNP) + expositions + biomarqueurs



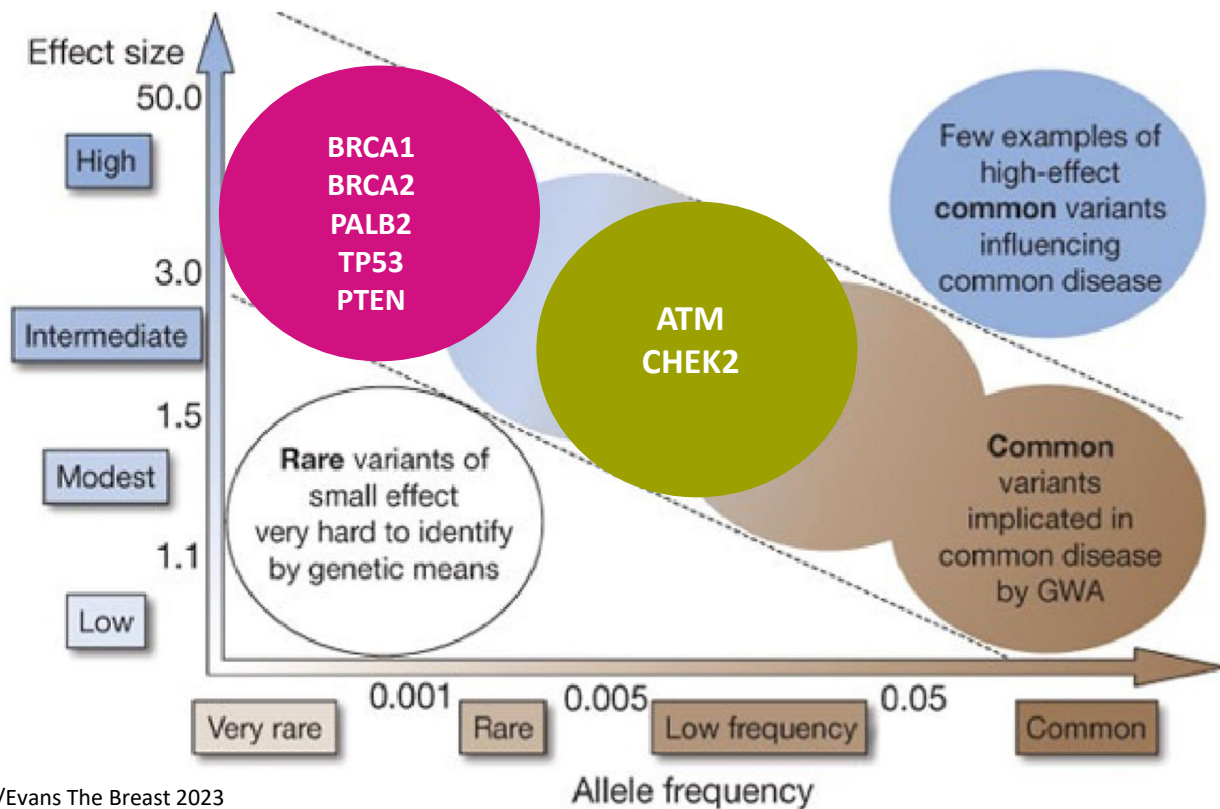
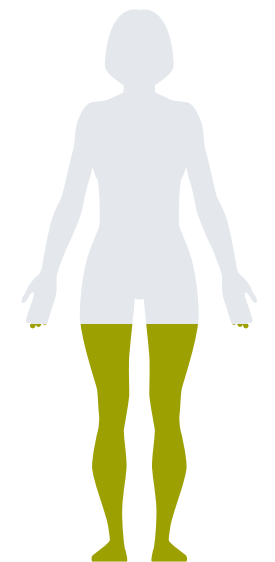
Clarity™ Autorisation de la FDA en juin 2025



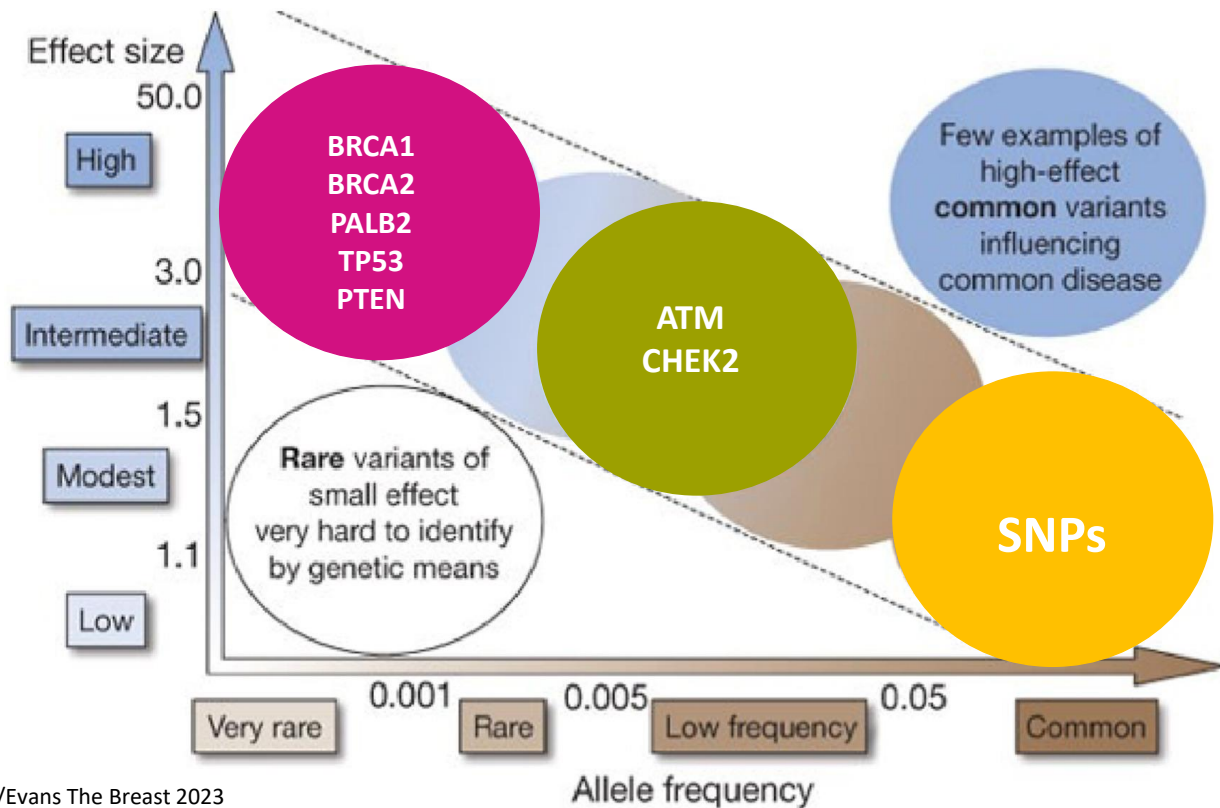
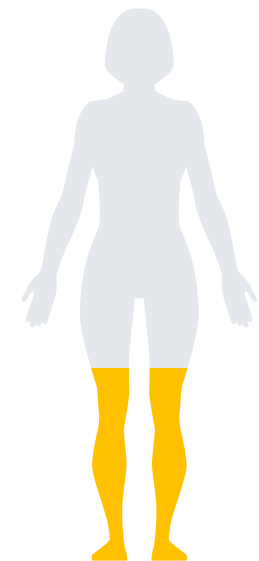
Génétique constitutionnelle et cancer du sein



Génétique constitutionnelle et cancer du sein



Génétique constitutionnelle et cancer du sein

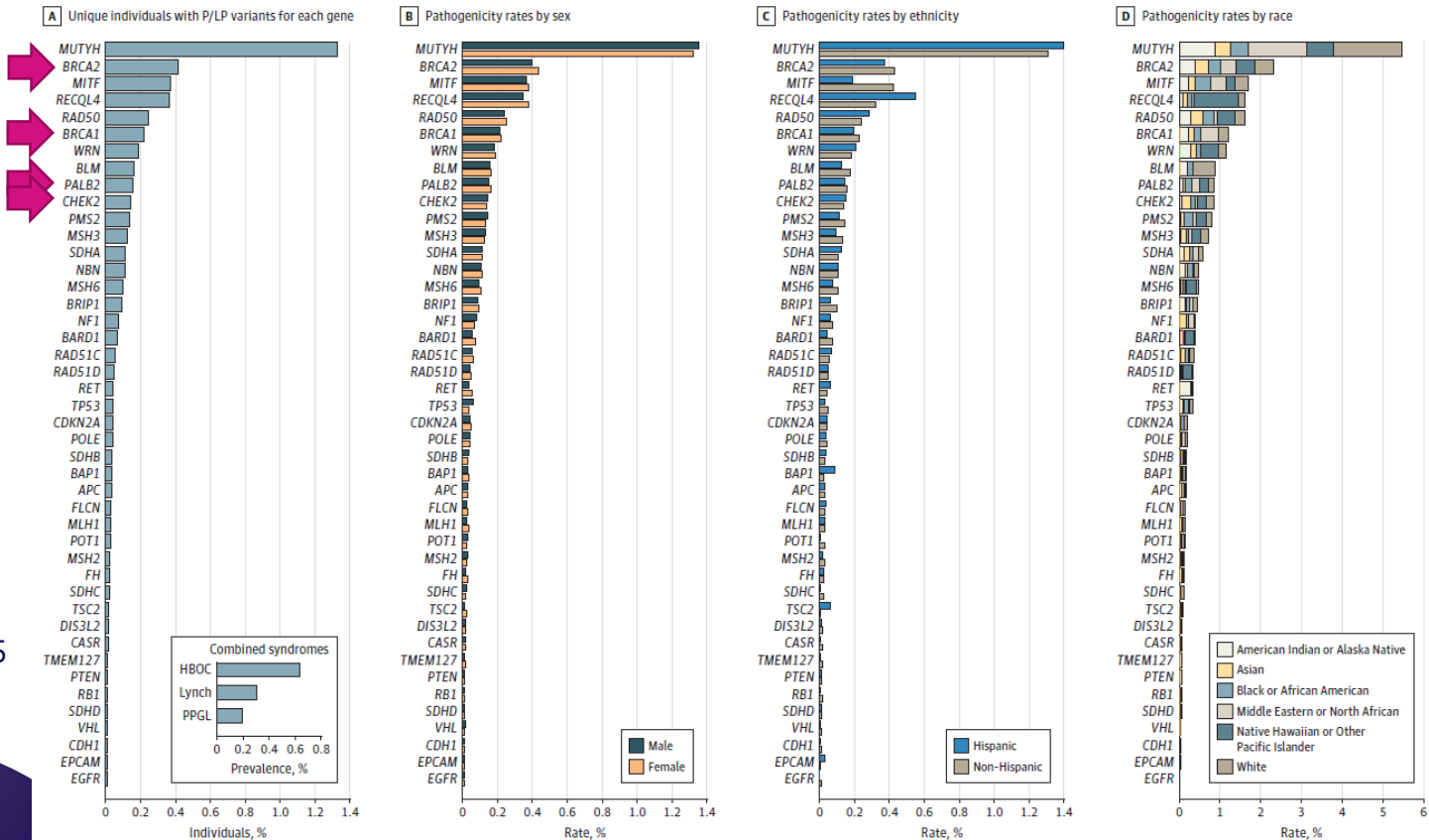


PRÉVENTION DE PRÉCISION : LA GÉNÉTIQUE GERMINALE A OUVERT LA VOIE

Altérations germinales - cancer	Moyens de détection précoce (LoE)	Mesures de réduction des risques (LoE)
BRCA1/2 – sein et ovaire	IRM mammaire (+ mammographie) (IIA)	Mastectomie prophylactique (IIA) et annexectomie (IIA), traitements endocriniens (IIIB), interventions sur le mode de vie (IV)
Autres gènes BC – sein et ovaire	Preuves de faible niveau, attitude dérivée des données BRCA1/2	
TP53 - large spectre	IRM mammaire (III), IRM du corps entier (IIB), autres	Mastectomie prophylactique (IV) Mode de vie (IV)

Prévalence de variants de prédisposition tous cancers en **population générale** (All of US, N=414830)

Figure. Gene-Level Distribution of Individuals With P/LP Variants



Idumah et al JAMA 2025

Prévalence de variants de prédisposition au cancer du sein en population générale: étude prospective WISDOM (USA)

Table 2. Overall Frequency of Germline Pathogenic or Likely Pathogenic Variants (PVs) in the WISDOM Trial

PV	Frequency	WISDOM trial participants with a PV	Comparison rates from other studies ^a
High-penetrance breast cancer genes			
<i>BRCA1</i>	0.14% (1 in 697)	0.09% (1 in 1134)	
<i>BRCA2</i>	0.69%	0.36% (1 in 280)	0.25% (1 in 400)
<i>PALB2</i>	0.19% (1 in 522)	0.14% (1 in 726)	
Lower-penetrance breast cancer genes			
<i>ATM</i>	0.44% (1 in 228)	0.35% (1 in 287)	
<i>CHEK2^b</i>	1.08%	0.64% (1 in 156)	0.58% (1 in 172)
<i>CHEK2</i> low penetrance ^{c,d}	0.83% (1 in 121)	NA	
Syndromic breast cancer genes			
<i>CDH1</i>	0.01% (1 in 7663)	0.02% (1 in 6658)	
<i>PTEN</i>	0	0.01% (1 in 14 902)	
<i>STK11</i>	0	<0.01% (1 in 46 733)	
<i>TP53</i>	0.02% (1 in 4597)	0.01% (1 in 13 606)	
Total	2.63%	2.63% (1 in 38)	0.92% (1 in 109)

29.8% of 605 women with PVs (29.8%) did not report any family history nor Jewish ancestry

N=23098

Fergus et al JAMA Internal Med 2025

Table 3. Prevalence of Germline Pathogenic or Likely Pathogenic Variants (PVs) According to Family History of Breast or Ovarian Cancer

Family history	Total, No.	Women, No. (%)			
		Any (n = 605)	High-penetrance and syndromic PV (n = 167) ^a	Moderate-penetrance PV (n = 248) ^b	<i>CHEK2</i> low-penetrance PV (n = 190) ^c
No family history of breast or ovarian cancer	10 144	229 (2.3)	53 (0.5)	101 (1.0)	75 (0.7)
1 Female relative with breast cancer ≥50 y	5208	133 (2.6)	39 (0.7)	54 (1.0)	40 (0.8)
1 Female relative with breast cancer <50 y	1437	43 (3.0)	11 (0.8)	15 (1.0)	17 (1.2)
≥2 Female relatives with breast cancer any age	3555	117 (3.3)	32 (0.9)	53 (1.5)	32 (0.9)
≥1 Female relative with ovarian cancer	1052	29 (2.8)	7 (0.7)	11 (1.0)	11 (1.0)
≥1 Female relative with ovarian cancer and ≥1 female relative with breast cancer	1445	48 (3.3)	22 (1.5)	12 (0.8)	14 (1.0)
≥1 Male relative with breast cancer	148	6 (4.1)	3 (2.0)	2 (1.4)	1 (0.7)

Risque de cancer du sein par type de variant (population)

Table 2. Associations between Pathogenic Variants in Established Breast Cancer–Predisposition Genes and Risk of Breast Cancer.*

Breast Cancer– Predisposition Gene ^{1,2,7}	Case Patients (N = 32,247) no. with pathogenic variant (%)	Controls (N = 32,544) no. with pathogenic variant (%)	Odds Ratio (95% CI) [†]	P Value
ATM	253 (0.78)	134 (0.41)	1.82 (1.46–2.27)	<0.001
BARD1	49 (0.15)	35 (0.11)	1.37 (0.87–2.16)	0.18
BRCA1	275 (0.85)	37 (0.11)	7.62 (5.33–11.27)	<0.001
BRCA2	417 (1.29)	78 (0.24)	5.23 (4.09–6.77)	<0.001
CDH1	17 (0.05)	6 (0.02)	2.50 (1.01–7.07)	0.06
CHEK2	349 (1.08)	138 (0.42)	2.47 (2.02–3.05)	<0.001
NF1‡	19 (0.06)	11 (0.03)	1.93 (0.91–4.31)	0.09
PALB2	148 (0.46)	38 (0.12)	3.83 (2.68–5.63)	<0.001
PTEN	8 (0.02)	3 (0.01)	NA	NA
RAD51C	41 (0.13)	35 (0.11)	1.20 (0.75–1.93)	0.44
RAD51D	26 (0.08)	14 (0.04)	1.72 (0.88–3.51)	0.12
TP53‡	19 (0.06)	2 (0.01)	NA	NA
Total	1621 (5.03)	531 (1.63)	—	—

Cases - large
population
control

CARRIERS
study

Hu et al NEJM 2021

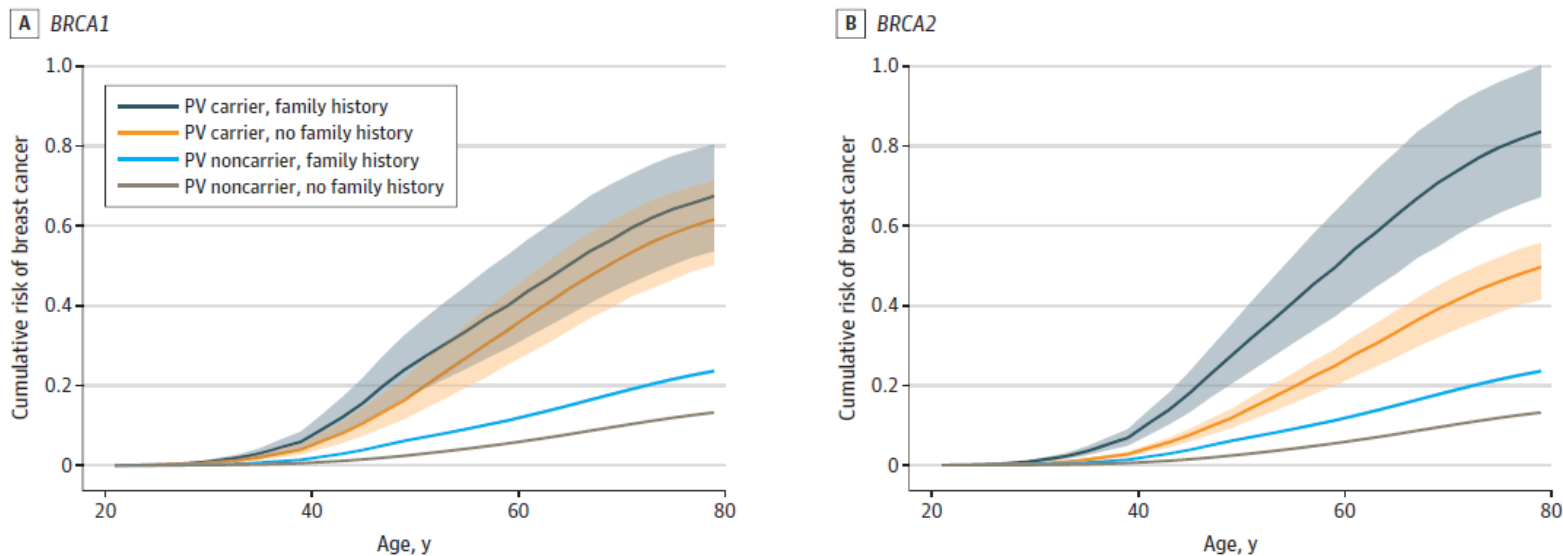
* Germline pathogen variant



Risque de cancer du sein par type de variant et histoire familiale

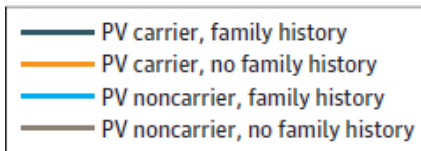
CARRIERS study

Figure 2. Cumulative Risk of Breast Cancer (Invasive or In Situ) by Age and First-Degree Family History of Breast Cancer Based on Hybrid Models

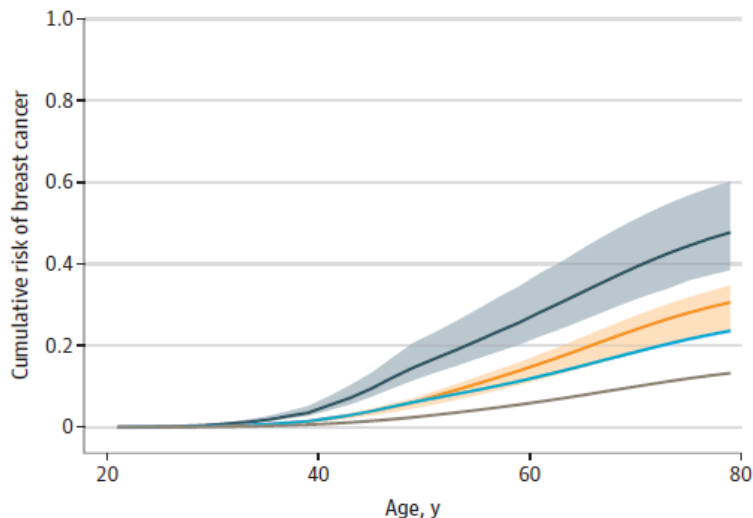


Risque de cancer du sein par type de variant et histoire familiale

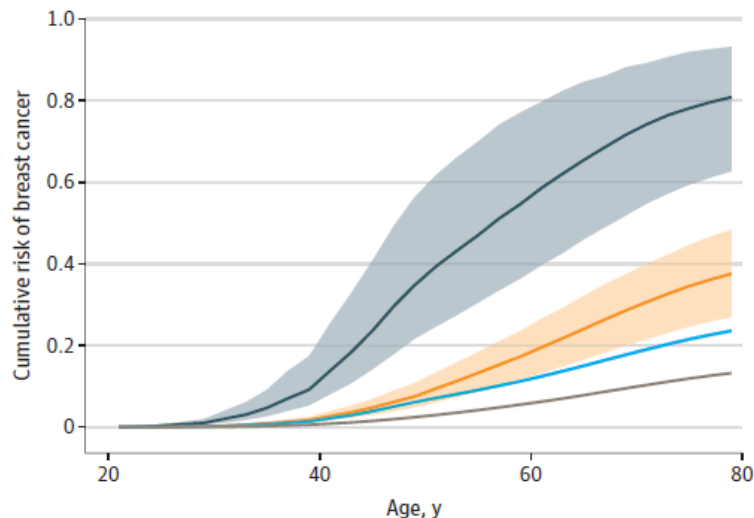
CARRIERS study



C CHEK2



D PALB2



Impact of MRI screening on mortality – gBRCA1/2

MRI use versus not in a prospective cohort

Table 2. Breast Cancer-Specific Mortality and All-Cause Mortality Stratified by Magnetic Resonance Imaging (MRI) Surveillance Status^a

Events	No./total No. of deaths (%)	HR (95% CI)	P value
All deaths			
Breast cancer death ^b			
No MRI surveillance	21/732 (2.9)	1 [Reference]	NA
MRI surveillance	14/1756 (0.8)	0.23 (0.11-0.48)	<.001
BRCA1 sequence variation			
Breast cancer death			
No MRI surveillance	18/562 (3.2)	1 [Reference]	NA
MRI surveillance	12/1442 (0.8)	0.20 (0.10-0.43)	<.001
BRCA2 sequence variation			
Breast cancer death			
No MRI surveillance	3/170 (1.8)	1 [Reference]	NA
MRI surveillance	2/317 (0.6)	0.87 (0.10-17.25)	.93
All-cause mortality			
No MRI surveillance	47/732 (6.4)	1 [Reference]	NA
MRI surveillance	45/1756 (2.6)	0.42 (0.26-0.66)	.001

Figure 1. Incidence of Breast Cancer Over 20 Years of Follow-Up in Women with *BRCA1* or *BRCA2* Sequence Variations Stratified by Magnetic Resonance Imaging (MRI) Surveillance Status

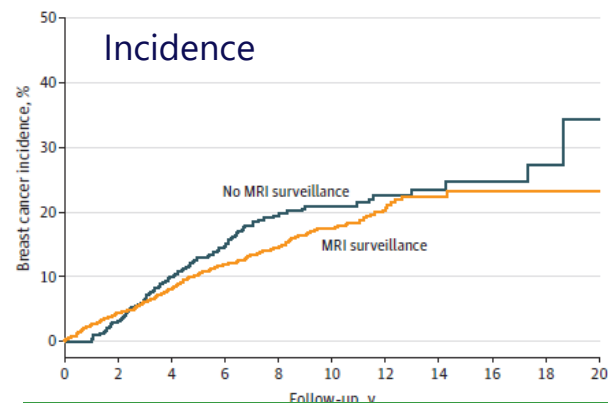
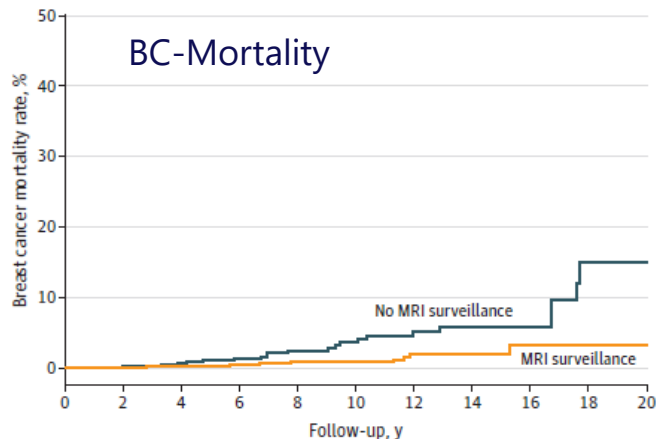


Figure 2. Breast Cancer Mortality Over 20 Years in Women with *BRCA1* or *BRCA2* Sequence Variations, By Magnetic Resonance Imaging (MRI) Surveillance Status



Impact of prophylactic mastectomy

Breast cancer incidence and death after bilateral prophylactic mastectomy, *BRCA1/2*

Prospective pseudo-randomisation

- 827 RRM 827 controls
- mean follow-up of 6.3 years
- 20 versus 100 breast cancers
- **2 versus 100 BC death**

RRM: probability of dying of breast cancer at 15 years = 0,95%.

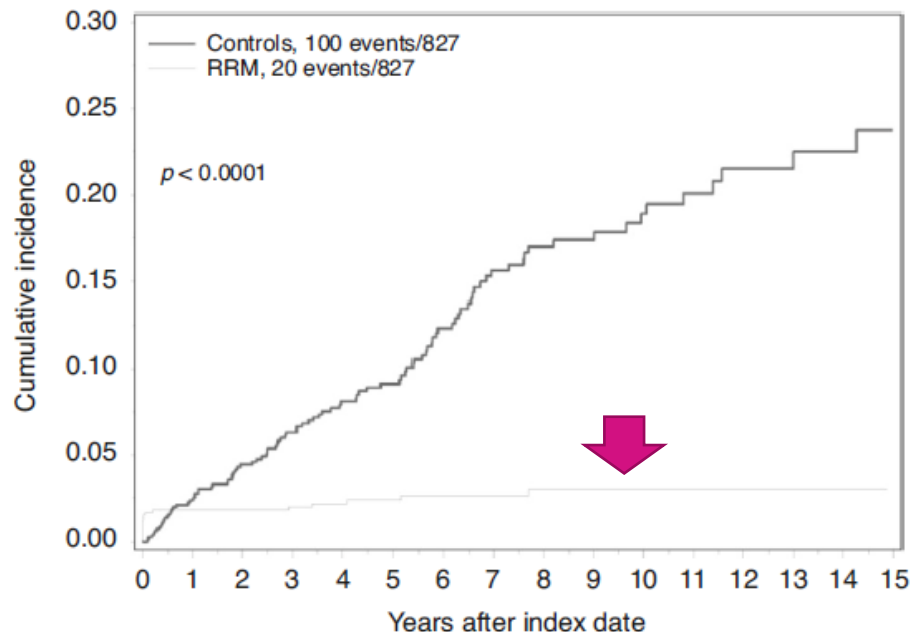


Fig. 1 Breast Cancer Incidence. Cumulative incidence of breast cancer from index date for RRM versus Control.

Impact of prophylactic adnexectomy

Impact on general mortality: benefit for *BRCA1* + + +

Table 5. Annual Mortality Rates of Women With *BRCA1* and *BRCA2* Sequence Variations (All-Cause) by Oophorectomy Status

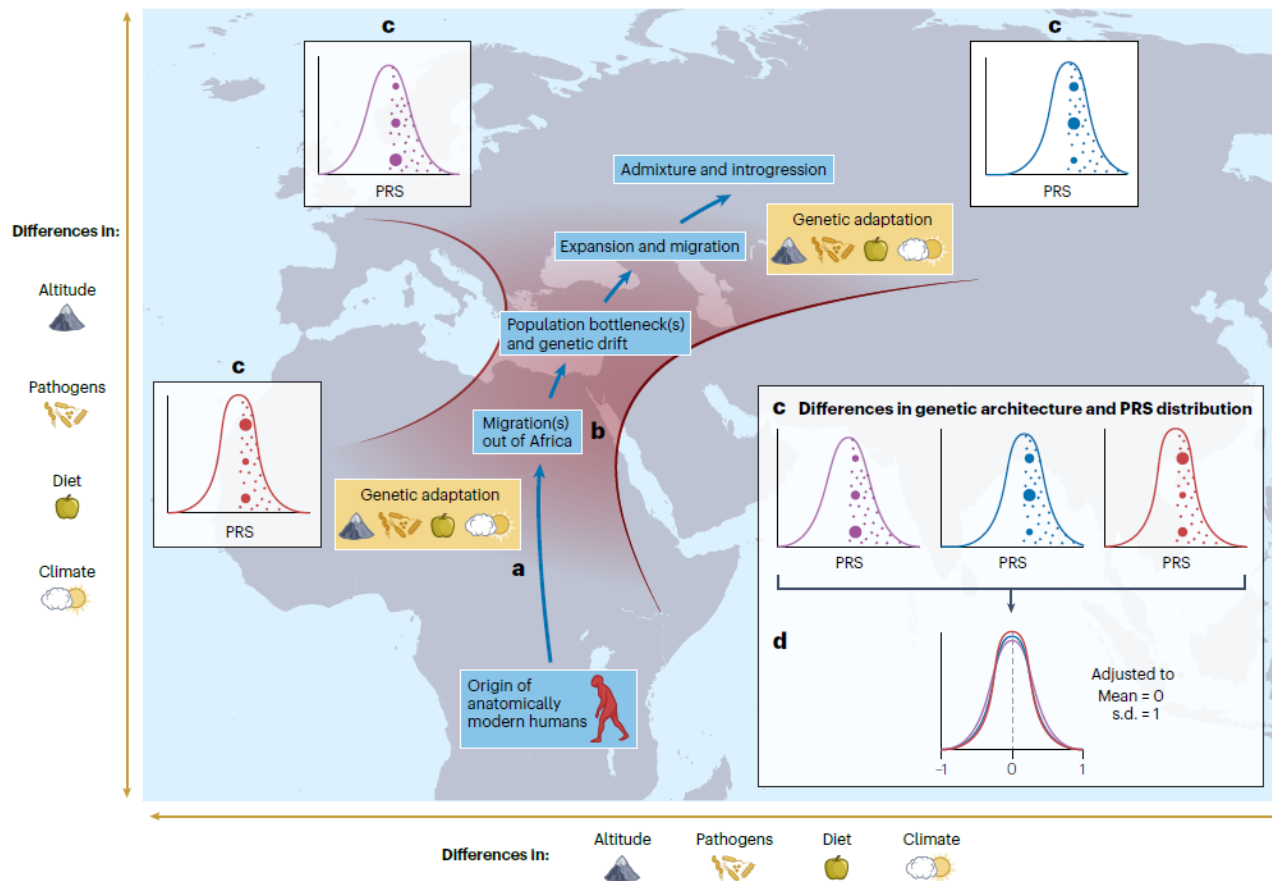
	No oophorectomy (<i>BRCA1</i> , n = 1071; <i>BRCA2</i> , n = 329)			Oophorectomy (<i>BRCA1</i> , n = 2106; <i>BRCA2</i> , n = 826)			
Age group, y	Person-years	Deaths, No.	Annual rate, % ^b	Person-years	Deaths, No.	Annual rate, % ^b	<i>P</i> value ^a
<i>BRCA1</i>							
35-44.9	6370.3	25	0.39	3768.5	7	0.19	.07
45-54.9	2248.8	40	1.78	6694.3	34	0.51	<.001
55-64.9	791.0	28	3.54	4045.1	35	0.87	<.001
65-74.9	276.0	10	3.62	1249.0	16	1.28	.07
All	9686.2	103	1.06	15 756.9	92	0.58	NA
<i>BRCA2</i>							
35-44.9	1669.4	3	0.18	887.7	0	0.00	.56
45-54.9	906.2	1	0.11	2253.6	7	0.31	.45
55-64.9	441.5	6	1.36	1746.3	8	0.46	.05
65-74.9	203.7	3	1.47	723.9	5	0.69	.38
All	3220.8	13	0.40	5610.6	20	0.36	NA

Genes with management guidelines, breast cancer, US

ACS, ACOG, ASCO, ClinGen, and/or NCCN Recommendations	Genes (n=48)
Annual screening breast magnetic resonance imaging	<i>ATM, BARD1, BRCA1, BRCA2, CDH1, CHEK2, NBN, NF1, PALB2, PTEN, STK11, TP53</i>
Earlier and more frequent colonoscopy/endoscopy	<i>APC, AXIN2, BMPR1A, CHEK2, EPCAM, GREM1, MLH1, MSH2, MSH6, PMS2, MSH3 (homozygous, h.); MUTYH (h.), NTLH1 (h.), POLD1, POLE, PTEN, SMAD4, STK11, TP53</i>
Risk-reducing mastectomy	<i>BRCA1, BRCA2, PALB2, PTEN, STK11, TP53</i>
Risk-reducing salpingo-oophorectomy, +/- hysterectomy	<i>BRCA1, BRCA2, BRIP1, EPCAM, MLH1, MSH2, MSH6, PMS2, RAD51C, RAD51D</i>
Risk-reducing colectomy	<i>APC</i>
Risk-reducing gastrectomy	<i>CDH1</i>
Other targeted screening (e.g., RCC, pheochromocytoma)	<i>MEN1, NF2, RB1, RET, SDHAF2, SDHB, SDHC, SDHD, TSC1/2, VHL, TP53, WT1</i>

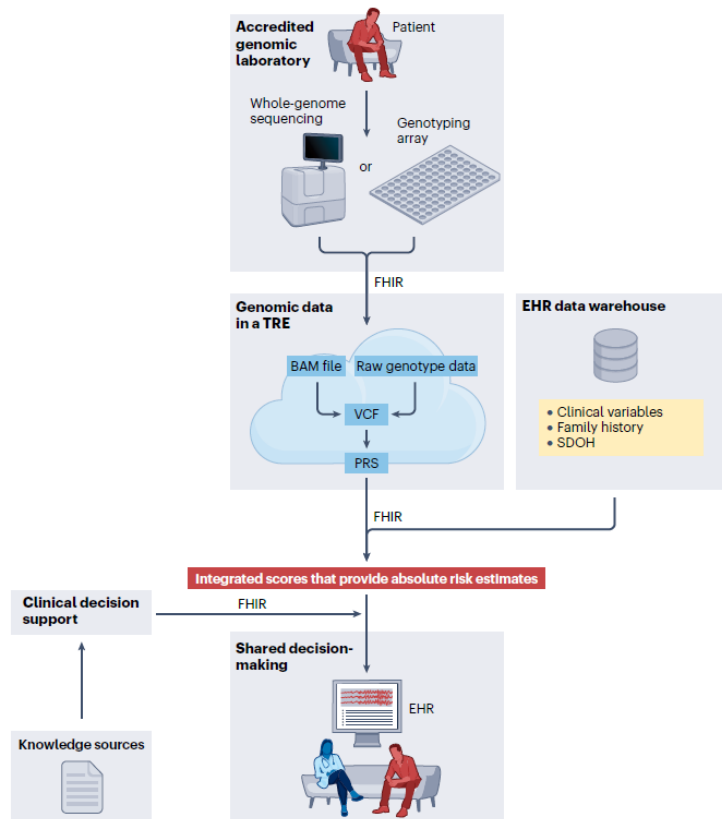
ET LES SCORES DE POLYMORPHISMES?

Kullo et al Nature Rev Genet 2025



UTILISATION CLINIQUE DES SCORES DE POLYMORPHISMES

Enjeux éthiques, circuits d'organisation, représentativité, communication....

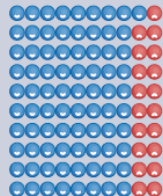


Kullo et al Nature Rev Genet 2025

a Icon array to convey absolute risk

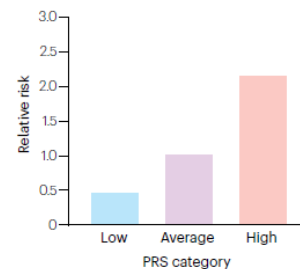
Risk for 100 people like you, over 10 years (based on PCE and PRS)

19 people will have a heart attack
81 people will not have a heart attack



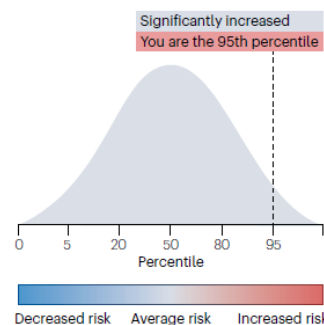
Risk over 10 years (based on several factors including your PRS) is shown. Out of 100 people like you, 19 will have a heart attack in the next 10 years, 81 will not have a heart attack.

b Bar graph to convey relative risk



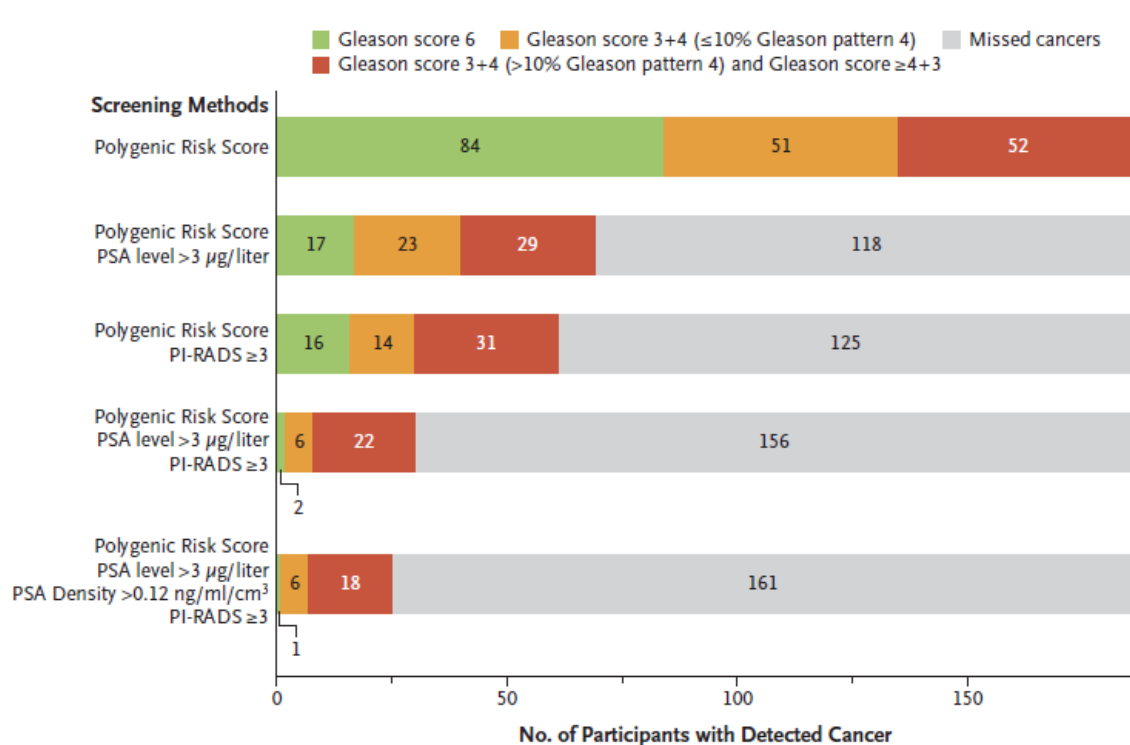
Your PRS is in a higher category, indicating that your risk of disease is twice that of a person with a PRS in the average category.

c Risk percentile rank



Your score is higher than average, meaning that you have increased genetic risk of disease compared with most people. If your polygenic score is in the 95th percentile, you do not have a 95% chance of developing the disease. Rather, it means that — out of 100 people — your polygenic score is higher than 95 people and the same or lower than 5.

CANCER DE PROSTATE: UTILITE CLINIQUE D UN SCORE DE POLYMORPHISMES POUR GUIDER LE DEPISTAGE



SCORES DE RISQUE AVEC POLYMORPHISMES POUR STRATIFIER LE DÉPISTAGE DU CANCER DU SEIN EN POPULATION GÉNÉRALE



**PERSPECTIVE
PROCAS
autres**

**Essais contrôlés
randomisés**

**Cohortes
prospectives**

Développement



Validation



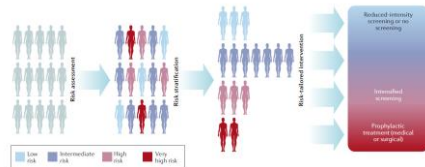
Validité clinique



Utilité clinique



Mise en œuvre

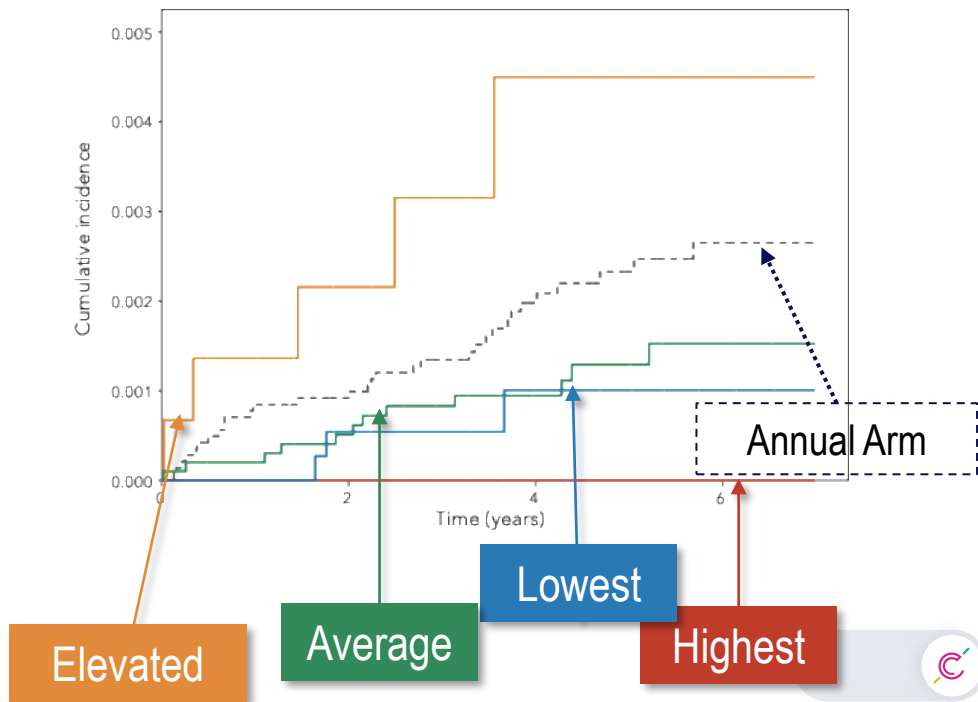
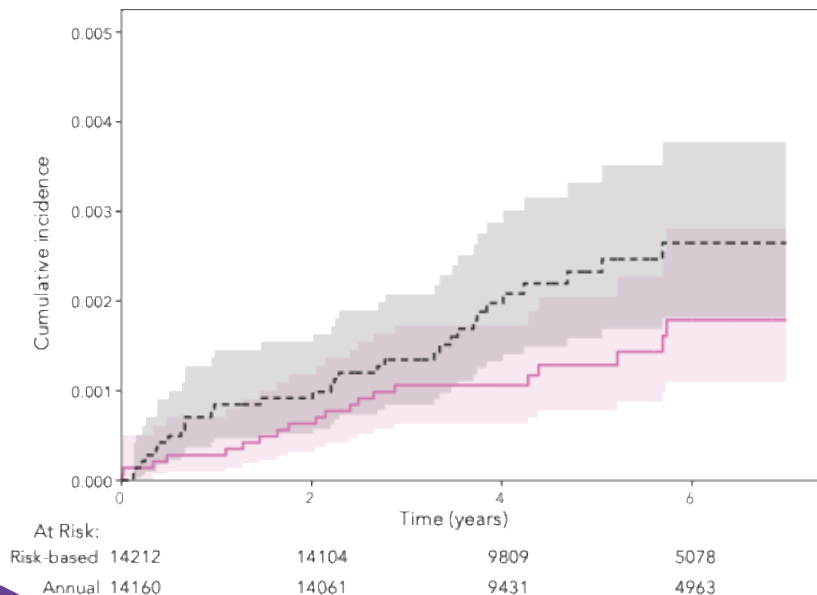


PREMIERS RESULTATS PROSPECTIFS DE DEPISTAGE STRATIFIE DU CANCER DU SEIN: ETUDE WISDOM

Esserman et al JAMA 2025

Non infériorité de la stratégie stratifiée sur le endpoint principal: incidence de cancers du sein de stade IIB+

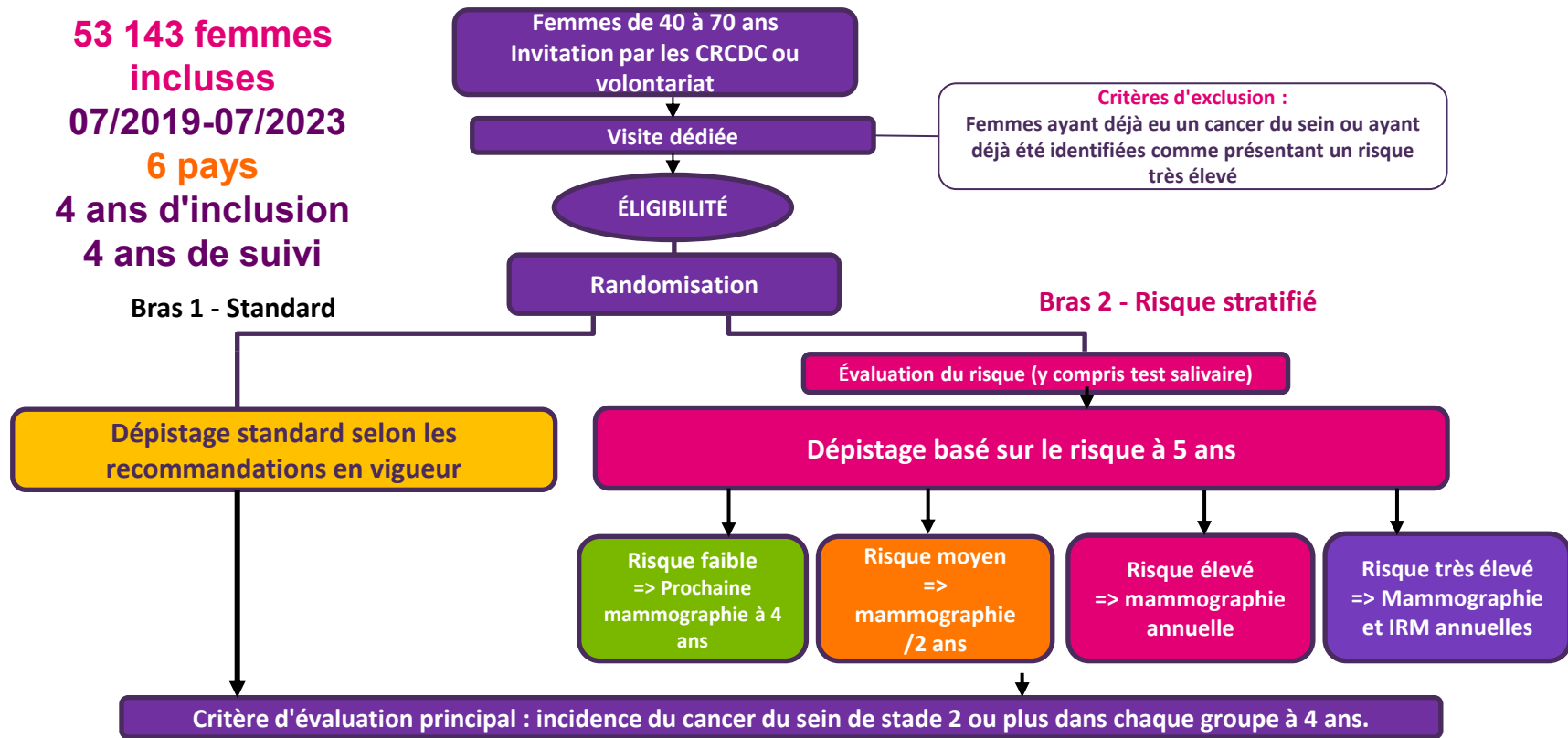
Rate of stage IIB cancers (risk-based: 30.0 [95%CI,16.3-43.8] vs annual: 48.0 [95%CI, 30.1-65.5] per 100 000 person-years; rate difference, -18.0 per 100 000 person-years [95%CI, -40.2 to 4.1]).





Etude MyPeBS en Europe: résultats fin 2027

**53 143 femmes
incluses
07/2019-07/2023
6 pays
4 ans d'inclusion
4 ans de suivi**



A 10 ET 15 ANS :

SUIVI À LONG TERME, Y COMPRIS LA MORTALITÉ DUE AU CANCER DU SEIN

STRATIFICATION : UK BIOBANK VS MYPEBS

Elie Rassy



Risk categories	Low risk < 1%	Average risk $1 \leq \text{Risk} < 1.67\%$	High risk $1.67\% \leq \text{Risk} < 6\%$	Very high risk $6\% \leq \text{Risk}$
UK Biobank Weighted mean density imputation**				
Participants from European ancestry				
MR + PRS313	67372 (34.3)	67221 (34.2)	61239 (31.2)	485 (0.2)
Participants from non-European ancestry				
MR + PRS313	13209 (33.8)	12958 (33.2)	12781 (32.7)	136 (0.3)
MyPeBS				
	9155 (36.58)	7260 (29.01)	8266 (33.03)	344 (1.37%)

** Imputation according to the breast density reported by Holley et al. [22]: 9.0% for density A, 45.1% for density B, 38.1% for density C and 7.7% for density D



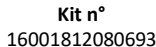
Scores de compréhension au départ par pays

	PAYS							
	BEL	FR	SP	IT	UK	ISR	Total	Kruskall-Wallis H
N	2 191	10 433	2 994	14 693	6 265	8 822	45 398	P<0,001
Médiane (min-max)	11,00 (6,00-12,00)	12,00 (4,00-12,00)	11,00 (4 h 00-12 h 00)	12h00 (0,00-12,00)	11h00 (4,00-12,00)	12,00 (4,00-12,00)	12,00 (0,00-12,00)	
Moyenn e	11,12	11,20	10,99	11,38	10,81	11,30	11,21	
SD	1,05	11,20	1,10	0,94	1,37	0,97	1,07	

Niveau de compréhension très élevé avec un score médian de 12 sur 12 questions : bonne compréhension du cancer du sein, du dépistage du cancer du sein en général, des avantages et des risques.

Nora Moumjid





Acceptability of risk-based breast cancer screening among professionals and healthcare providers from 6 countries contributing to the MyPeBS study

Acceptabilité par les professionnels de santé

Alexandra Roux¹, Lucile Hervouet², Francesca Di Stefano³, David P. French⁴, Livia Giordano³, David Ritchie⁵, Marie-Eve Rougé Bugat⁶, Debbie Keatley⁷, Rachel Cholerton⁸, Lorna McWilliams⁴, Paolo Giorgi Rossi⁹, Corinne Balleyguier⁹, Michal Guindy¹⁰, Fiona J. Gilbert¹¹, Jean-Benoit Burri¹², Marta Roman¹³, Cécile Vissac-Sabatier¹⁴, Daniel Couch¹⁴, Suzette Delaloge⁹, Sandrine de Montgolfier^{1,15*} and On behalf of the MyPeBS Investigators and the MyPeBS Consortium¹



**Une majorité est plutôt à l'aise avec l'annonce des risques
Mais une minorité est très à l'aise**

Table 7 Difficulties with announcing risk, explaining high and low risk categories and opinions about training and training material to give back risk estimation

	1- Not comfortable at all	2	3	4	5-Very comfortable	Total
<i>Informing women of their risk category</i>	0% (0)	0.9% (1)	10.6% (12)	52.2% (59)	36.3% (41)	100% (113)
<i>Explaining high risk categories</i>	0.9% (1)	6.2% (7)	22.1% (25)	52.2% (59)	18.6% (21)	100% (113)
<i>Explaining low risk category and 4-years mammography intervals</i>	4.5% (5)	4.5% (5)	25.9% (29)	39.3% (44)	25.9% (29)	100% (112)
	Did not receive any training	Not sufficient at all	A little bit insufficient	Sufficient	Very sufficient	Total
<i>Opinion on your training to give back risk</i>	4.4% (5)	3.5% (4)	18.6% (21)	49.6% (56)	23.9% (27)	100% (113)
	Never	Rarely	Sometimes	Often	Total	
<i>Frequency of need to refresh training for the trial</i>	13.1% (26)	26.3% (52)	50% (99)	10.6% (21)	100% (198)	

Formation et éducation nécessaires

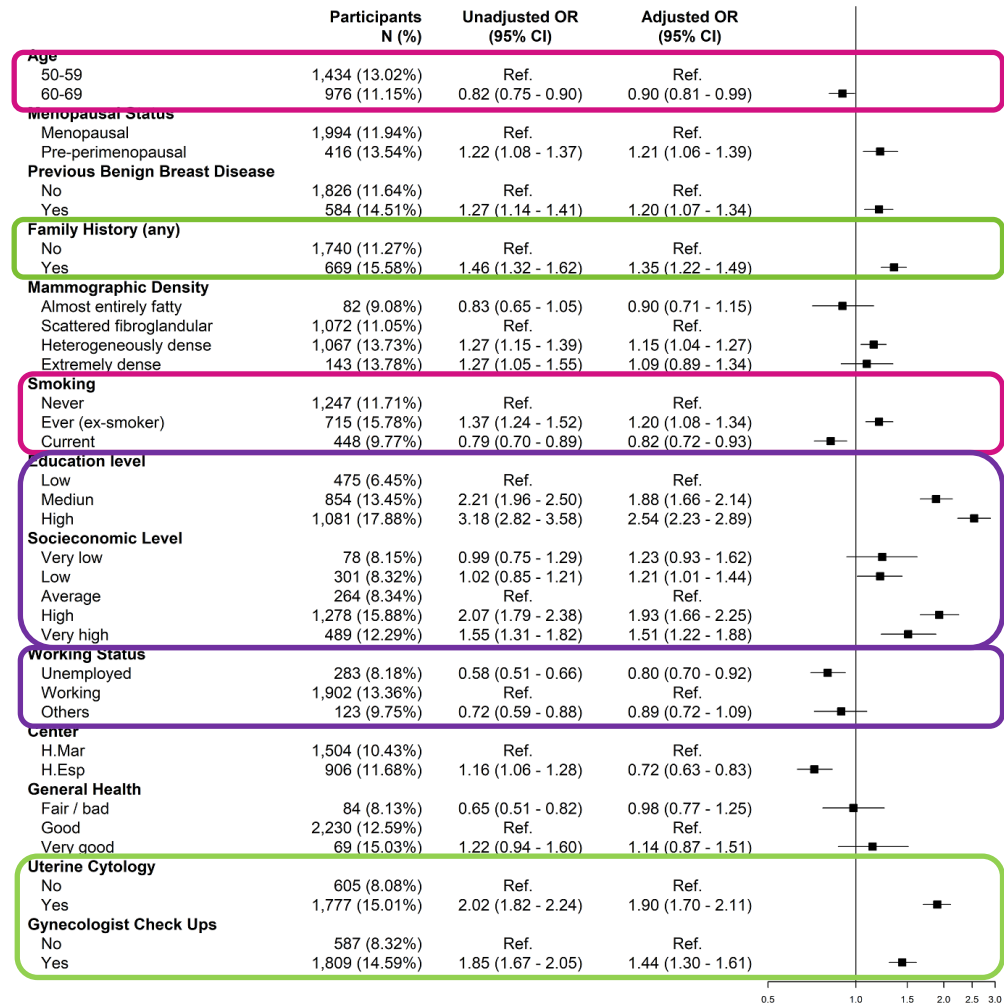
ÉTUDE DE L'IMPACT DES DISPARITÉS SOCIO DÉMOGRAPHIQUES SUR L'ACCEPTATION DE PARTICIPER À MYPEBS



Marta Roman
Hôpital Del Mar
Barcelone

Espagne : 19 768 femmes invitées
à participer à MyPeBS, 2 410
(12,2 %) se sont inscrites

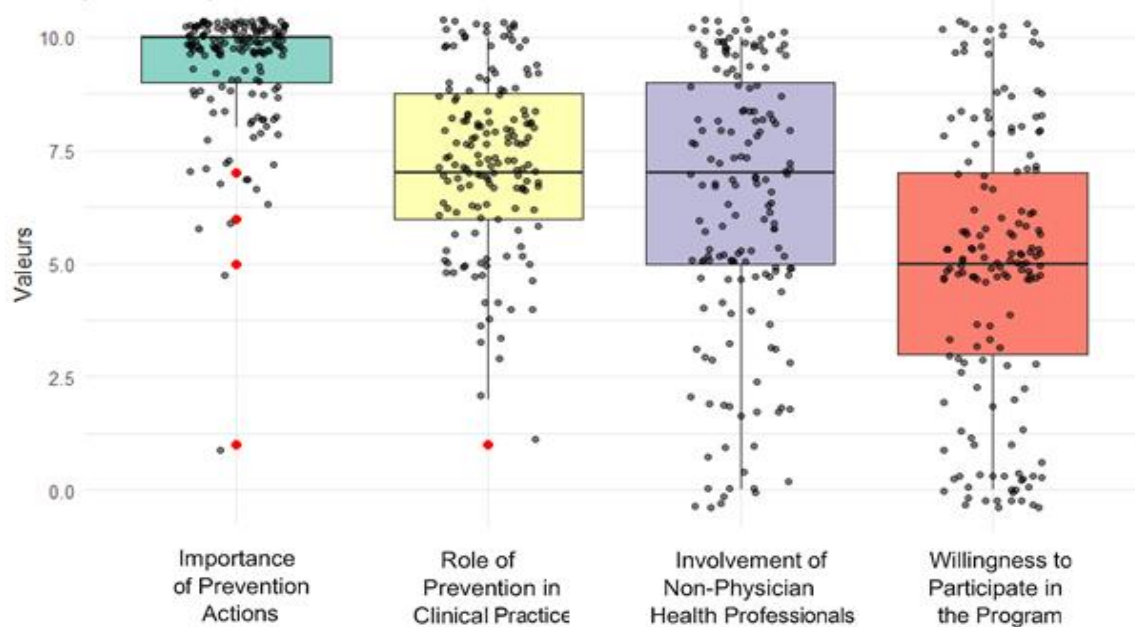
Soumis, Br J Cancer



Qui est responsable de la prévention (personnalisée) du cancer ? Une étude utilisant des méthodes mixtes menée auprès de médecins généralistes en France

- Les médecins généralistes considèrent qu'il est de leur devoir de prévenir le cancer, mais ils disposent de **très peu de leviers** (principalement cognitifs) et sont confrontés à **de nombreux obstacles** (manque de temps, absence de récompense, activités concurrentielles, etc.).
- Peu de connaissances sur les approches de prévention personnalisées

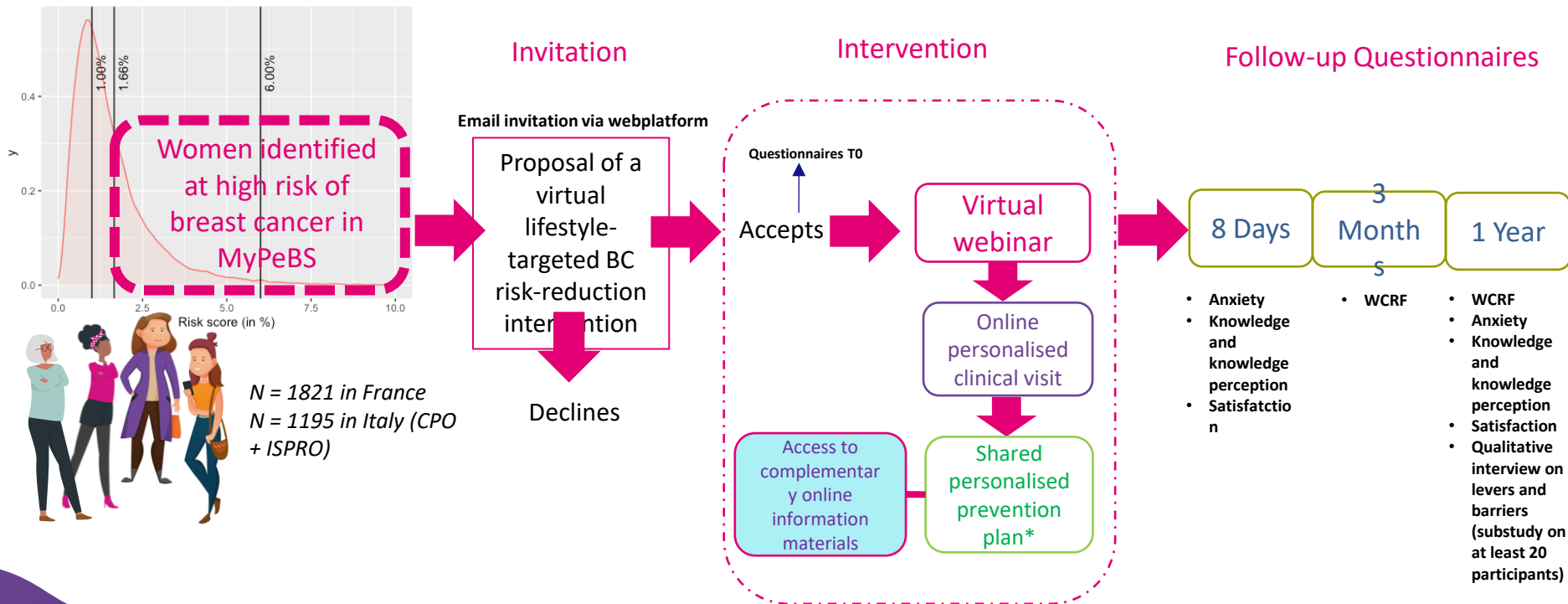
Boxplot of Opinions on Cancer Prevention



Tarek Ben Ahmed et al ESMO 2025

GUIDER LE DEPISTAGE MAIS AUSSI LA PREVENTION!

MyPREV (JA-PREVENT NCD) NCT07120087



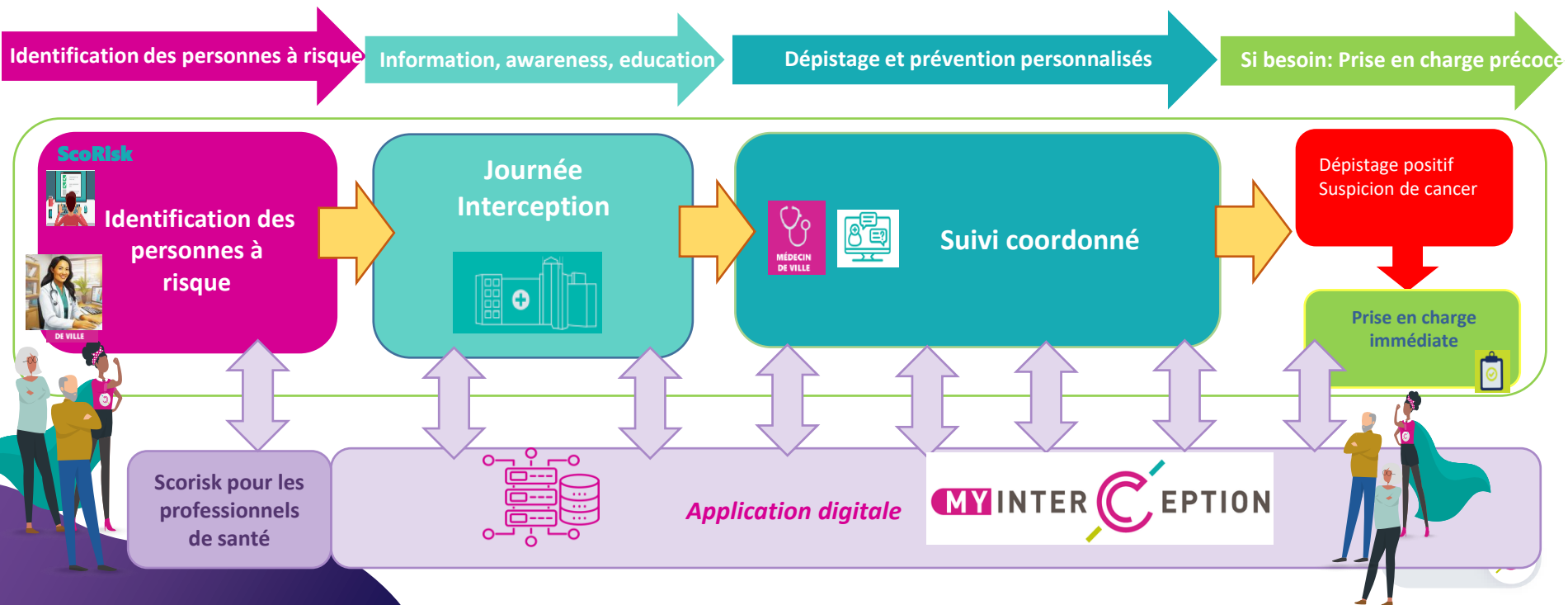
*Personalised (stratified and predefined) measures regarding weight, nutrition, physical activity, hormone exposures, other exogenous exposures to be discussed and decided in a shared decision process



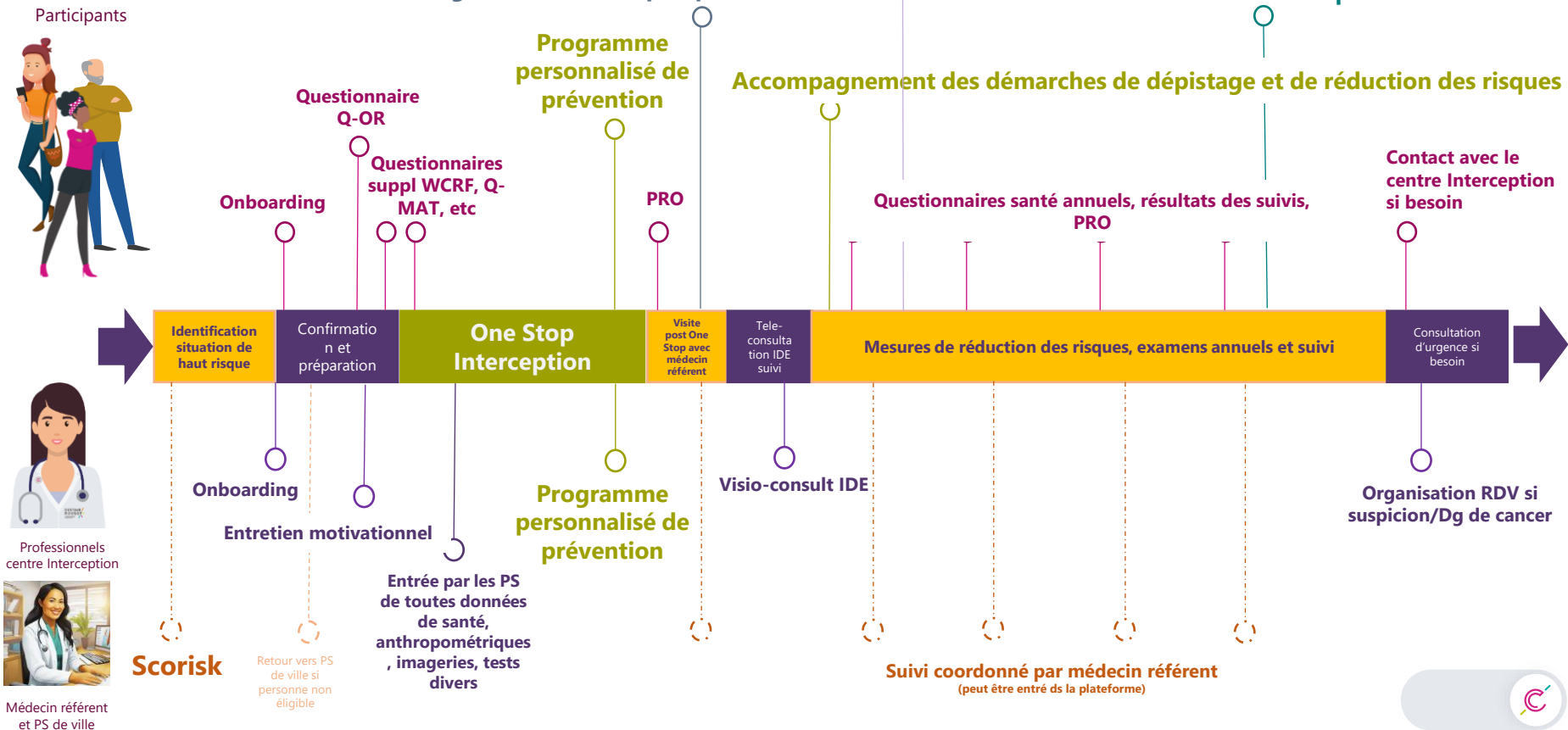
SYSTEMATISER LA CONSTRUCTION D UNE PREVENTION PERSONNALISEE DES CANCERS



Un parcours mixte ville-hôpital phygital de prévention personnalisée chez les patients à risque élevé de cancer, déployé de façon nationale



E-parcours Interception

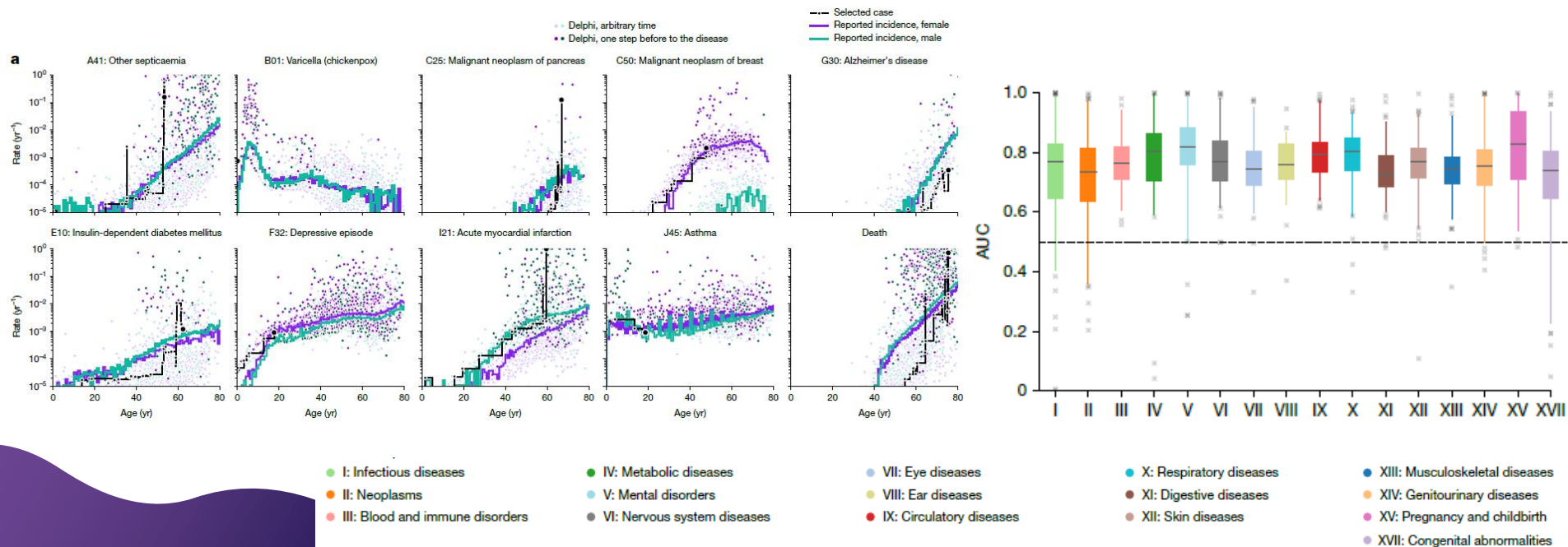


Les activités en pointillés sont en dehors de la plateforme MyInterception

PREDIRE LE RISQUE DUNE MALADIE OU DE (TOUTES LES) MALADIES?

Learning the natural history of human disease with generative transformers

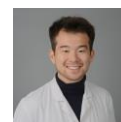
<https://doi.org/10.1038/s41586-025-09529-3>



CONCLUSIONS ET PERSPECTIVES

- La génétique constitutionnelle est le fondement du développement de la prévention personnalisée des cancers mais
 - Elle doit s'associer à l'analyse de l'exposome individuel!
 - Cette nouvelle prévention nécessite une démonstration prospective à grande échelle avant sa mise en œuvre
 - Doit être développée au niveau de la santé publique
- Grandes questions aujourd'hui/demain: doit on proposer un génotypage en population générale? L'analyse de variants constitutionnels?
- L'évaluation des risques, la détection précoce et les stratégies de réduction des risques doivent être combinées pour avoir plus d'impact
- Une collaboration étroite entre les domaines de recherche permet d'accélérer les développements et anticiper l'implémentation

MERCI!



À tous les participants MyPeBS !

MyPeBS ExCo et CTSC : Paolo Giorgi Rossi, Harry de Koning, Livia Giordano, Michal Guindy, Jean-Benoit Burrion, Marta Roman, Stefan Michiels, Sandrine de Montgolfier, Jean-François Deleuze, Fiona Gilbert, Corinne Balleyguier, Debbie Keatkey, Gareth Evans, Paul Pharoah, David French, Hélène Blanché, Anne Boland, Aloys Dubois d'Aische, Emilien Gauthier, Efrat Slonim, Francesca di Stefano, Xavier Castells, Damien Drunay, Eleni Michalopoulou, Yan Chen, Margarita Posso, Stéphane Ragusa, Nora Ferdjaoui, Ludwig Lebrun, Jaimie Taylor

Membres **du comité exécutif et du comité consultatif scientifique de MyPeBS**

Tous les chercheurs et équipes MyPeBS

Équipes Unicancer : Daniel Couch, Cécile Vissac-Sabatier, Camille Baron, Sonia Mardinian, Lamia Ghezali, Matou Diop, Aude Sirven, Muriel Dahan, Beata Juzyna, Jérôme Lemonnier

- **La Commission européenne, l'Institut national du cancer français, les sponsors de MyPeBS**
- **Groupe de travail EDEPT de l'ESMO** : Bruno Achutti-Duso, Claire Turnbull, Brigette Ma, Neil Iyengar, Banu Arun, Daniel Munoz-Espin, Nora Pashayan, Rebecca Fitzgerald, George Kapenatakis, Judith Balmana, Andrea De Censi, Monica Cigignoni, Klizia Marinoni, Georges Pentheroudakis
- **Équipe Interception de Gustave Roussy** : Olivier Caron, Lucie Veron, Pamela Abdayem, Thomas Pudlarz, Claire Bladier, Tarek Ben Ahmed, Elie Rassy, Bruno Achutti-Duso, Diane Le Stradic, Hélène Natchez, Agustina Ipinia

