

Seminar - Environment, occupation and cancer: etiology and prevention

Auditorium L8, Norwegian Institute of Public Health, Oslo

September 25th 2025

Welcome

08.00 - 09.00 Registration and coffee/tea

09.00 - 09.05 **Mieke C Louwe** - Senior Advisor, Cancer Registry of Norway-NIPH

09.05 - 09.15 **Giske Ursin** – Director, Cancer Registry of Norway-NIPH

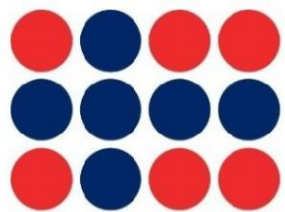
Welcome

- Purpose: to share latest findings, foster dialogue across epidemiology, toxicology, policy, exposure science etc.
- Goal: identify knowledge gaps, explore prevention strategies, and shape future collaborations
- Opportunity: connect researchers, early career scholars, policymakers, practitioners

Thematic research area on occupational and environmental exposures and cancer risk



This event has been supported by



NOFE

Norwegian Epidemiological Association
Norsk forening for epidemiologi



UNIVERSITY OF OSLO



The Research Council of Norway

Practicalities

Giske Ursin

Director, Cancer Registry of Norway - Norwegian Institute of Public Health

Session 1

Occupational cancer: Historical experiences and current challenges

Session 1 - Occupational cancer: Historical experiences and current challenges

09.15 – 10.35 **Keynote: Kurt Straif** - Research Professor, Morrissey College of Arts and Sciences, US
Lessons learned from identifying environmental and occupational carcinogens for evidence informed policy

Jo S Stenehjem - Senior Researcher, Cancer Registry of Norway-NIPH, University of Oslo

Benzen exposure from non-solid to solid tumors among offshore workers

Hilde Langseth - Senior Researcher, Cancer Registry of Norway-NIPH

Exploring Norwegian cohorts and biobanks in agricultural exposure and cancer

Break

10.35 – 11.00

Keynote Kurt

Environment, occupation and cancer: etiology and prevention

*Benzene exposure: from non-solid to solid tumors
in offshore petroleum workers*

Jo S. Stenehjem, PhD

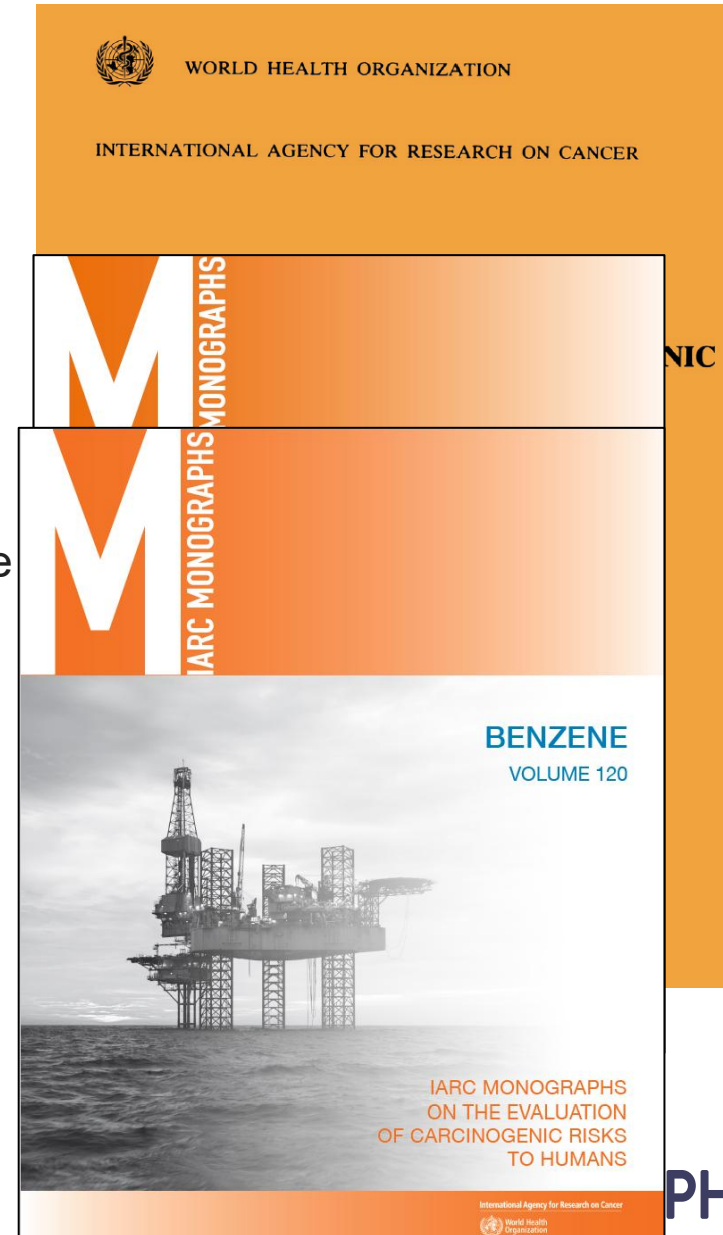
Senior Researcher

Cancer Registry of Norway, NIPH

Dept of Biostatistics, UiO

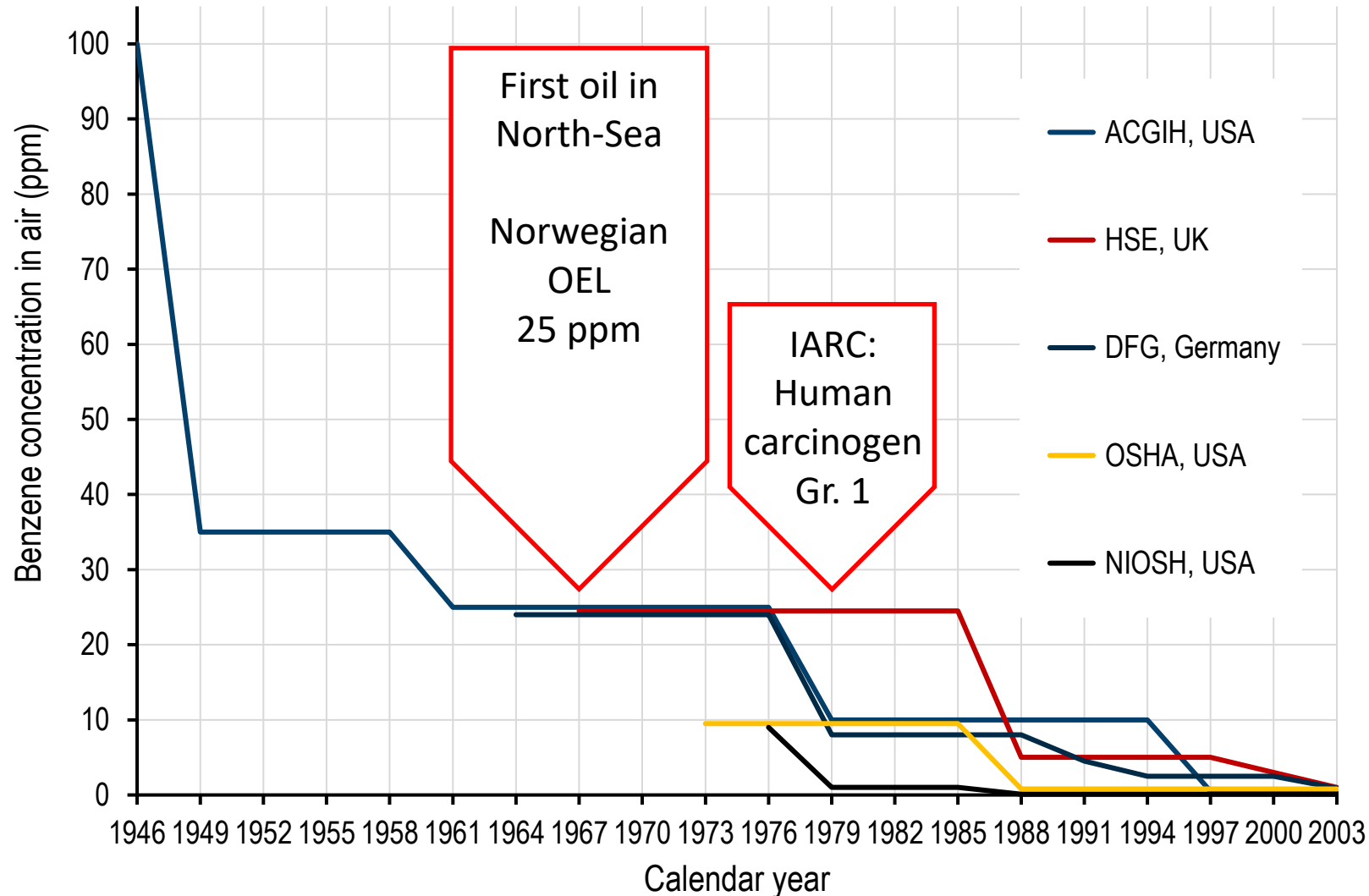
Background

- 1890s: Benzene linked to aplastic anemia in tire-manufacturing workers (Santeson, 1897)
- 1900-1950: Risks associated with benzene in printing, petro-refining, plastic manufacturing and leatherworking industries- → Suspected link between benzene and leukemia.
- 1960-1970: Studies by Dr. Muzaffer Aksoy linked benzene to leukemia in shoemakers and leather manufacturers.
- 1979: Benzene deemed carcinogenic to humans (IARC group 1) based on acute myeloid leukemia
- 1990-2010: Positive associations seen for major histological subtypes of lymphoma (IARC vol. 100F)
- 2010-2020: IARC vol 120: Confirming positive associations for lymphoma subtypes and lung cancer
- 2020-2025: New studies showing associations with bladder, lung and CRC



Occupational exposure limits for benzene over time

Kilde: Capleton AC, Levy LS. Chemico-Biological Interactions 153–154 (2005), p43–53



ACGIH, USA; American Conference of Governmental Industrial Hygienists

HSE, UK; Health and Safety Executive

DFG, Germany; Deutsche Forschungsgemeinschaft, Germany

OSHA, USA; Occupational Safety and Health Administration

NIOSH, USA; National Institute for Occupational Safety and Health

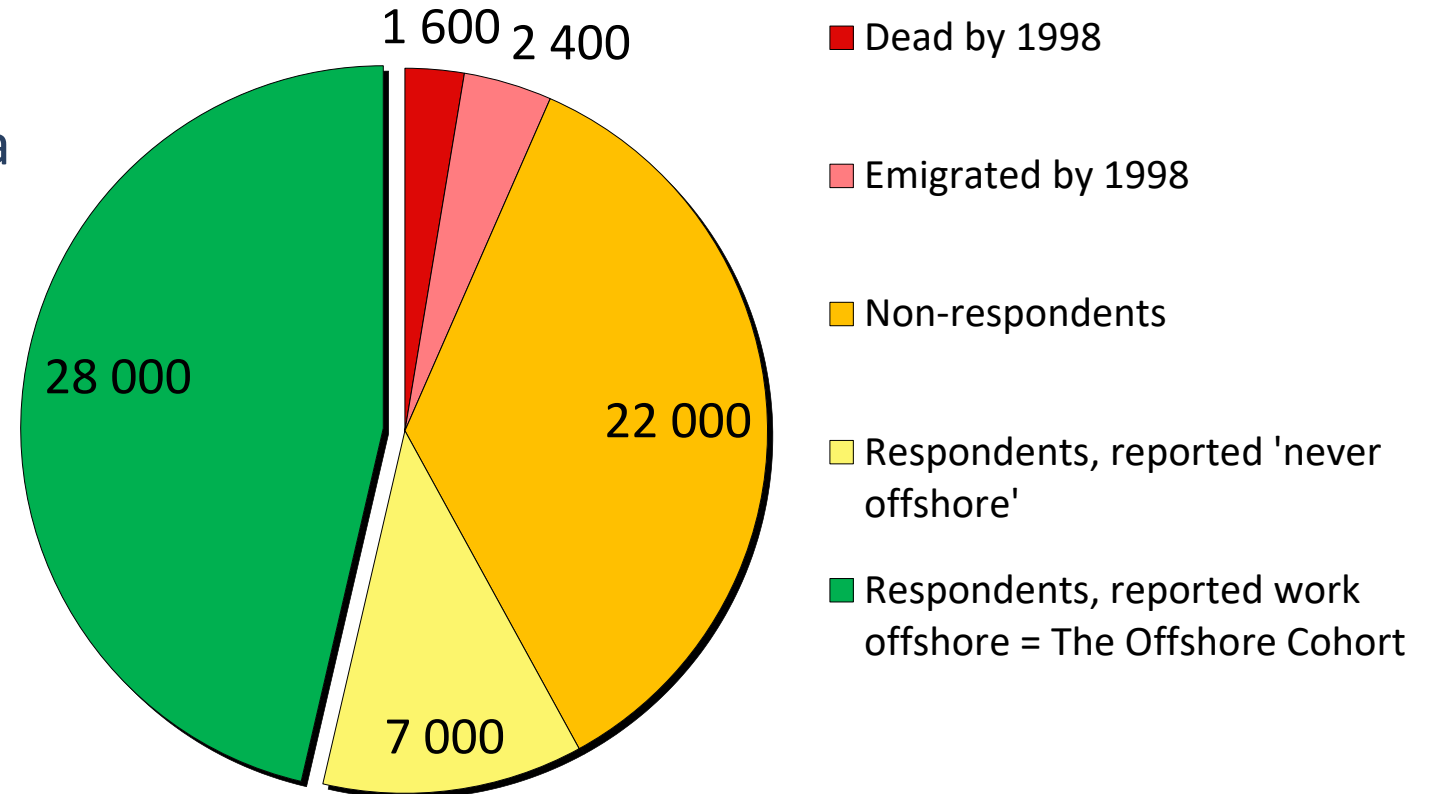
IARC; International Agency for Research on Cancer (WHO)

I dag:

- Norway 2021: OEL (8-h TWA): 0.2 ppm
- Netherland: OEL (8-h TWA): 0.2 ppm
- U.S. NIOSH recommend: 0.1 ppm
- EU (ECHA) suggests: 0.05 ppm

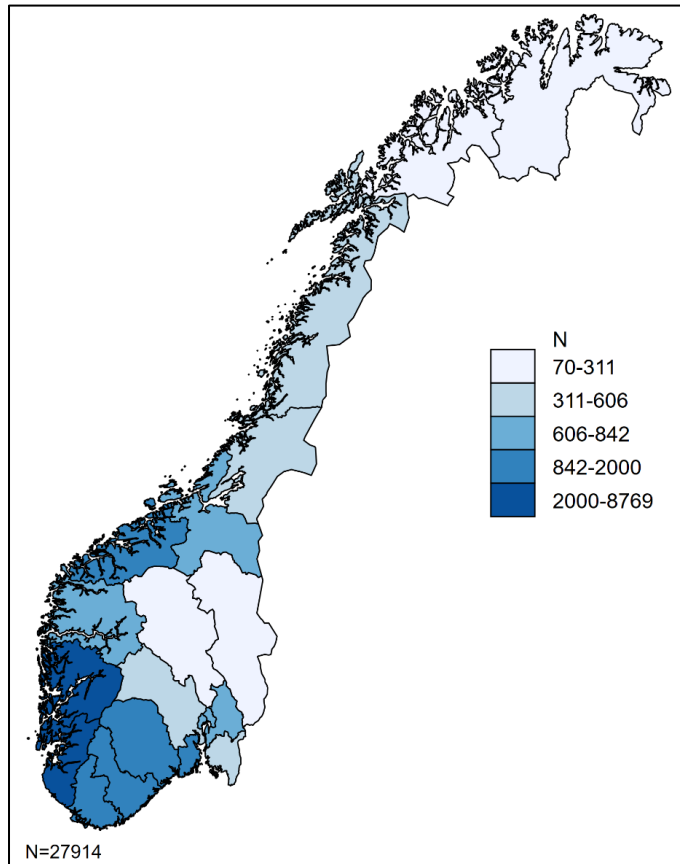
Norwegian offshore petroleum workers (NOPW) cohort

- 25 500 males employed between 1965 and 1998
- Baseline (1998) questionnaire data
 - Work history (offshore and onshore)
 - Education
 - Body mass index
 - Smoking
 - Alcohol intake
 - Red/processed meat intake
 - Physical activity

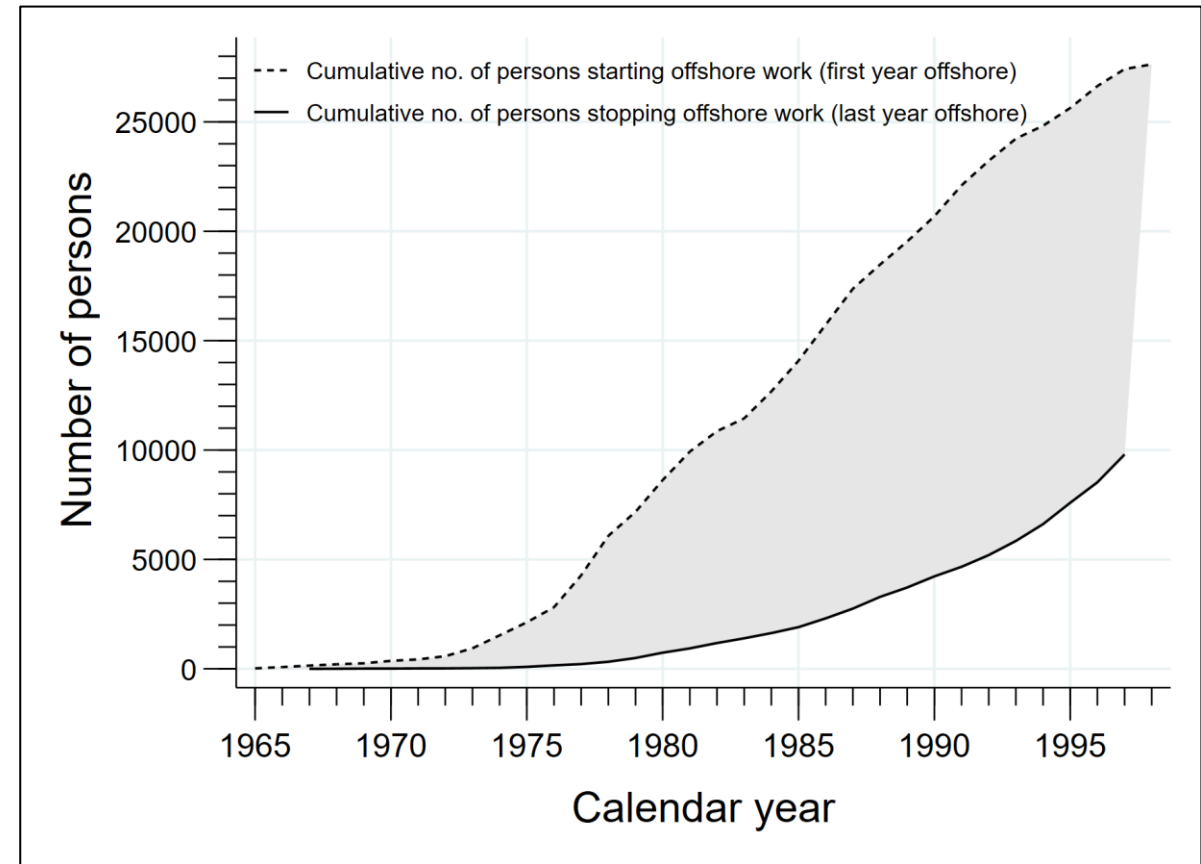


Recruitment

- Mostly recruited from the western part of Norway, but all counties represented
- Steep increase in number of new employees in 1970s and 1980s



County of residency



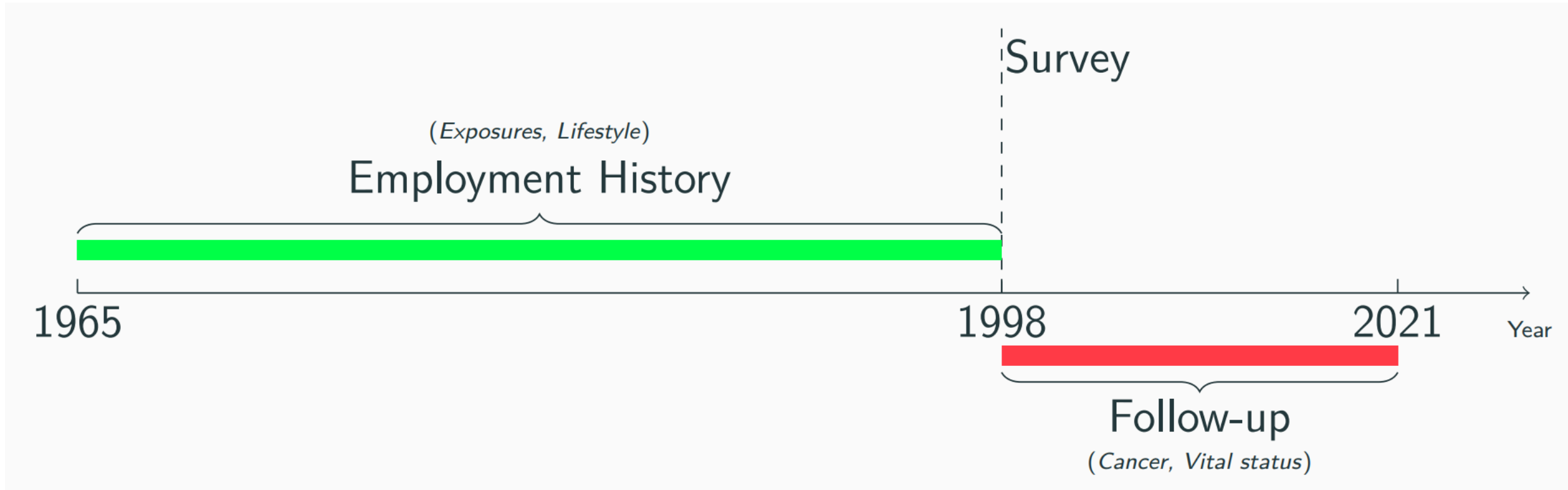
Employment flux

Benzene job-exposure matrix

- Independently developed job-exposure matrix
- Task oriented
- Semi-quantitative, using expert assessment and measurement data in combination
- Proven to detect *a priori* known associations with acute myeloid leukemia

Job category	Exposure burden (intensity x frequency x duration)			
	1970-79	1980-89	1990-99	2000 →
Process technicians ^a	2.4	2.4	2.1	1.8
Mechanics	1.9	1.9	1.6	1.4
Industrial cleaners	1.4	1.4	1.3	1.3
Process technicians ^b	1.4	1.4	1.1	0.9
Laboratory engineers	1.3	1.3	1.0	0.7
Deck crew	0.8	0.8	0.7	0.7
Plumbers and piping engineers	0.6	0.6	0.5	0.4
Non-destructive testing	0.5	0.5	0.4	0.4
Machinists	0.4	0.4	0.4	0.4
Electric instrument technicians	0.3	0.3	0.2	0.2
Scaffold crew	0.2	0.2	0.2	0.2
Sheet metal workers and welders	0.2	0.2	0.2	0.2
Insulators	0.2	0.2	0.1	0.1
Mud engineers and shale shaker operations*	*	*	-	-
Drill floor crew*	*	*	-	-
Surface treatment (painters)*	*	*	-	-
Drillers	-	-	-	-
MWD and mud loggers	-	-	-	-
Derrick employees	-	-	-	-
Well service crew	-	-	-	-
Control room operators	-	-	-	-
Electricians	-	-	-	-
Radio employees	-	-	-	-
Turbine operators	-	-	-	-
Hydraulics technicians	-	-	-	-
Chef and catering	-	-	-	-
Health, office and administration personnel	-	-	-	-

Timeline



Benzene and lymphohematopoietic cancers

Exposure metric*	AML (n=10)			CLL (n=12)			MM (n=17)		
	C	NC	HR† (95% CI)	C	NC	HR† (95% CI)	C	NC	HR† (95% CI)
Cumulative exposure (p.p.m.-years)									
Unexposed	2	547	1.0 (ref.)	1	547	1.0 (ref.)	4	547	1.0 (ref.)
T1 (<0.001–0.037)	2	372	1.4						
T2 (>0.037–0.123)	1	374	0.9						
T3 (0.124–0.948)	5	368	4.9						
p Trend									
Average intensity (p.p.m.)									
Unexposed	2	547	1.0 (ref.)	1	547	1.0 (ref.)	4	547	1.0 (ref.)
T1 (<0.001–0.007)	1	373	0.8 (0.1, 8.7)	2	373	2.9 (0.3, 31)	4	372	1.3 (0.3, 5.4)
T2 (>0.007–0.013)	3	371	2.5 (0.4, 15)	5	370	7.6 (0.9, 65)	3	373	1.2 (0.3, 5.2)
T3 (>0.013–0.040)	4	370	3.2 (0.6, 19)	4	371	5.9 (0.6, 56)	6	369	2.7 (0.8, 9.1)
p Trend			0.092			0.094			0.099
* Categories were generated according to tertiles (T1–3) among exposed workers.									
† Adjusted for age (as the time scale); benzene exposure from other work (yes, no); ever daily smoker (yes, no, unknown).									
Abbreviations: AML = Acute myeloid leukaemia; CLL = Chronic lymphocytic leukaemia; MM = Multiple myeloma; C = Cases (n); NC = Non-cases (n); HR = Hazard ratio; CI = Confidence interval; T = tertile; Bold results indicate statistical significance at a 0.05-level.									

FULL PAPER

BJC

British Journal of Cancer (2015) 112, 1603–1612 | doi: 10.1038/bjc.2015.108

Keywords: benzene; case-cohort; cancer incidence; lymphohaematopoietic; offshore workers; petroleum industry

ne exposure and risk of
haematopoietic cancers
000 offshore oil industry workers

¹, K Kjaerheim¹, M Bråtveit², S O Samuelsen³, F Barone-Adesi^{4,5}, N Rothman^{6,7}, Q Lan^{6,7}
and T K Ghislerud^{*,1,7}

Stenehjem et al., 2015

Benzene, crude oil and skin cancer

Agent	Arm (n=24)			RESEARCH ARTICLE	WILEY
	Overarm (n=10)	Forearm and hand (n=13)	Hand (n=8)		
	HR (95% CI)	HR (95% CI)	HR (95% CI)		
Crude oil				<div>Accepted: 31 May 2017 DOI: 10.1002/ajim.22741</div> <div>Aromatic hydrocarbons and risk of skin cancer by anatomical site in 25 000 male offshore petroleum workers</div> <div>Jo Steinson Stenehjem PhD¹ Trude Eid Robsahm PhD¹ Magne Bråtveit PhD² Sven Ove Samuelsen PhD³ Jorunn Kirkeleit PhD^{2,4,5} Tom Kristian Grimsrud MD, PhD¹</div>	
Unexposed	1.00 (reference)	1.00 (reference)	1.00 (reference)		
Tertile 1 (>0-6 yrs)	1.36 (0.30-6.19)	NA	NA		
Tertile 2 (>6-14 yrs)	1.53 (0.30-7.84)	5.02 (0.99-25)	5.26 (1.10-25)		
Tertile 3 (>14-34 yrs)	NA	11 (2.12-54)	18 (1.81-171)		
<i>P_{trend}</i>	0.188	<0.001	0.011		
Benzene					
Unexposed	1.00 (reference)	1.00 (reference)	1.00 (reference)		
Tertile 1 (>0-5 yrs)	1.36 (0.30-6.19)	NA	NA		
Tertile 2 (>5-13 yrs)	1.53 (0.30-7.84)	5.02 (0.99-25)	5.26 (1.10-25)		
Tertile 3 (>13-34 yrs)	NA	11 (2.12-54)	18 (1.81-171)		
<i>P_{trend}</i>	0.188	<0.001	0.011		

AMERICAN JOURNAL
OF
INDUSTRIAL MEDICINE

WILEY

Aromatic hydrocarbons and risk of skin cancer by anatomical site in 25 000 male offshore petroleum workers

Jo Steinson Stenehjem PhD¹ | Trude Eid Robsahm PhD¹ |
Magne Bråtveit PhD² | Sven Ove Samuelsen PhD³ | Jorunn Kirkeleit PhD^{2,4,5} |
Tom Kristian Grimsrud MD, PhD¹

Stenehjem et al., 2017

SUMMARY

Associations found, but only on the forearm! Probably due to local dermal absorption

Stenehjem et al., 2017

Benzene and bladder cancer


www.nature.com/bjc

British Journal of Cancer

ARTICLE OPEN

Check for updates

Exposure to benzene and other hydrocarbons and risk of bladder cancer among male offshore petroleum workers

Nita K. Shala^{1,2} , Jo S. Stenehjem¹, Ronnie Babigumira^{1,2}, Fei-Chih Liu¹, Leon A. M. Berge^{1,2}, Debra T. Silverman³, Melissa C. Friesen³, Nathaniel Rothman³, Qing Lan³, H. Dean Hosgood⁴, Sven O. Samuelsen⁵, Magne Bråtevit⁶, Jorunn Kirkeleit^{6,7}, Bettina K. Andreassen¹, Marit B. Veierød² and Tom K. Grimsrud¹

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BACKGROUND: Occupational exposures constitute the second leading cause of urinary bladder cancer after tobacco smoking. Increased risks have been found in the petroleum industry, but high-quality exposure data are needed to explain these observations.

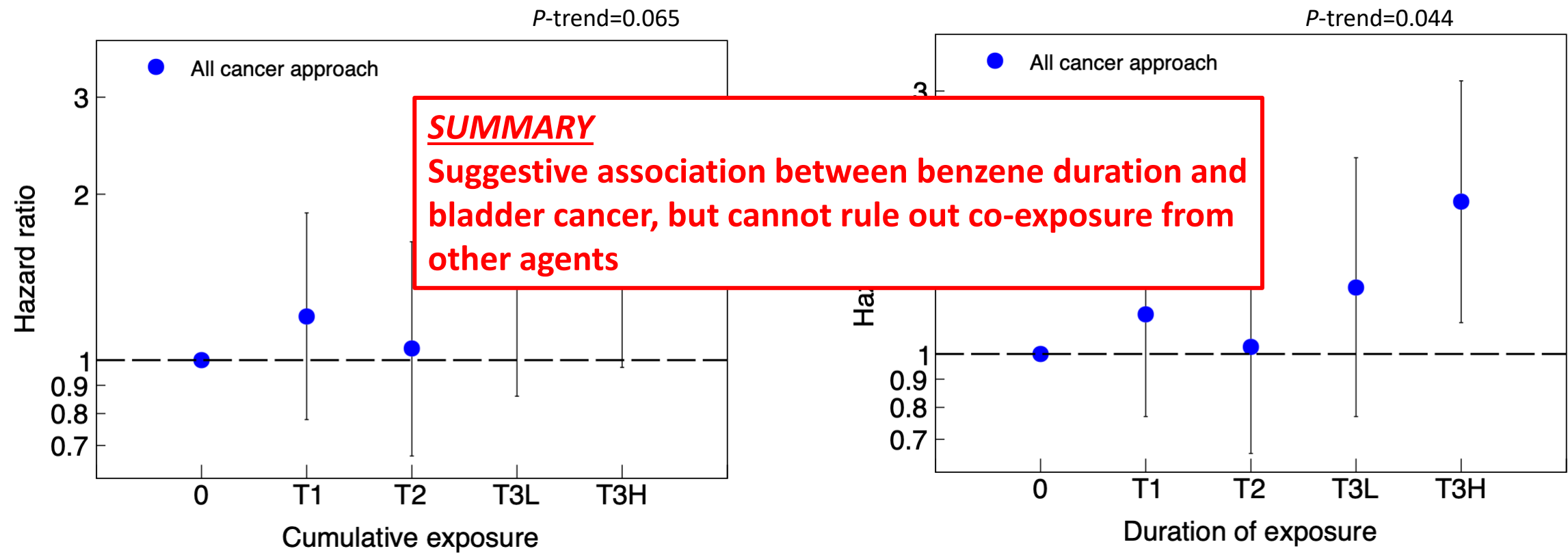
METHODS: Using a prospective case-cohort design, we analysed 189 bladder cancer cases (1999–2017) and 2065 randomly drawn non-cases from the Norwegian Offshore Petroleum Workers cohort. Cases were identified in the Cancer Registry of Norway, while work histories (1965–1998) and lifestyle factors were recorded by questionnaire at baseline (1998). Occupational petroleum-related hydrocarbon exposures were assessed by expert-developed job-exposure matrices. Hazard ratios were estimated by weighted Cox-regressions, adjusted for age, tobacco smoking, education, and year of first employment, and with lagged exposures.

RESULTS: Increased risks were found in benzene-exposed workers, either long-term exposure (≥ 18.8 years, HR = 1.89, 95% CI: 1.14–3.13; p -trend = 0.044) or high-level cumulative benzene exposure (HR = 1.60, 95% CI: 0.97–2.63; p -trend = 0.065), compared with the unexposed. Associations persisted with 20-year exposure lag. No associations were found with skin or inhalation exposure to crude oil, mineral oil (lubrication, hydraulics, turbines, drilling), or diesel exhaust.

CONCLUSIONS: The results suggest that exposures in the benzene fraction of the petroleum stream may be associated with increased bladder cancer risk.

British Journal of Cancer (2023) 129:838–851; <https://doi.org/10.1038/s41416-023-02357-0>

Benzene and bladder cancer




T=tertile; T3L=lower half, T3H=higher half.

Shala et al., 2023

Benzene and lung cancer

Table 4 HRs with 95% CIs of the major histological subtype: Adenocarcinoma (n=152)		
Adenocarcinoma (n=152)		
Benzene metric	C/NC	Model 3*
		HR† (95% CI)
Cumulative (ppm years)		
Unexposed	45/665	1.00 (reference)
Q1 (0.000–<0.019)	30/341	1.25 (0.60 to 2.61)
Q2 (0.019–<0.071)	20/355	1.01 (0.44 to 2.29)
Q3 (0.071–<0.175)	35/359	2.20 (0.98 to 4.90)
Q4 (0.176–0.879)	22/363	1.27 (0.50 to 3.28)
<i>P-trend</i>		0.986
Duration (years)		
Unexposed	45/665	1.00 (reference)
Q1 (1–4)	24/389	0.93 (0.43 to 2.03)
Q2 (5–10)	28/375	1.39 (0.62 to 3.11)
Q3 (11–16)	26/313	1.82 (0.80 to 4.17)
Q4 (17–34)	29/341	2.02 (0.80 to 5.11)
<i>P-trend</i>		0.044
Average intensity (ppm)		
Unexposed	45/665	1.00 (reference)
Q1 (0.000–<0.004)	28/337	1.15 (0.56 to 2.38)
Q2 (0.004–<0.007)	30/362	1.84 (0.81 to 4.17)
Q3 (0.007–<0.014)	27/352	1.65 (0.70 to 3.91)
Q4 (0.014–0.021)	22/363	1.27 (0.50 to 3.28)
<i>P-trend</i>		0.986






Workplace



OPEN ACCESS

Original research

Benzene exposure and risk of lung cancer in the Norwegian Offshore Petroleum Worker cohort: a prospective case-cohort study

Ronnie Babigumira ^{1,2} Marit B Veierød ² H Dean Hosgood,³ Sven Ove Samuelsen,⁴ Magne Bråtveit,⁵ Jorunn Kirkeleit,^{5,6} Nathaniel Rothman,⁷ Qing Lan,⁷ Debra T Silverman,⁷ Melissa C Friesen ⁷ Nita Kaupang Shala,^{1,2} Tom K Grimsrud ¹ Jo Steinson Stenehjem ¹

► Additional supplemental material is published online only. To view, please visit the journal online (<http://dx.doi.org/10.1136/oemed-2023-109139>).

For numbered affiliations see end of article.

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ABSTRACT

Objective The objective of our study was to examine whether occupational exposure to benzene is associated with lung cancer among males in the Norwegian Offshore Petroleum Workers cohort.

Methods Among 25 347 male offshore workers employed during 1965–1998, we conducted a case-cohort study with 399 lung cancer cases diagnosed between 1999 and 2021, and 2035 non-cases sampled

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Benzene is a known human carcinogen; however, the evidence for an association between benzene and lung cancer risk remained unclear in the latest evaluation of benzene by the International Agency for Research on Cancer.

Babigumira et al. 2023 OEM

DOI: <http://dx.doi.org/10.1136/oemed-2023-109139>

Benzene and lung cancer

New systematic review and meta-analysis including the NOPW cohort study

Review

A Section 508–conformant HTML version of this article
is available at <https://doi.org/10.1289/EHP15086>.

Occupational Benzene Exposure and Lung Cancer in Human Studies: A Systematic Review and Meta-Analysis

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⁴Department of Environmental and Occupational Health, University of California, Irvine, Irvine, California, USA

BACKGROUND: Benzene is classified as carcinogenic to humans based on evidence that benzene causes acute myeloid leukemia. However, there is limited evidence that benzene causes lung cancer.

OBJECTIVES: We performed a systematic review, quality assessment, and meta-analysis of published cohort and case–control studies on the association between occupational benzene exposure and lung cancer risk.

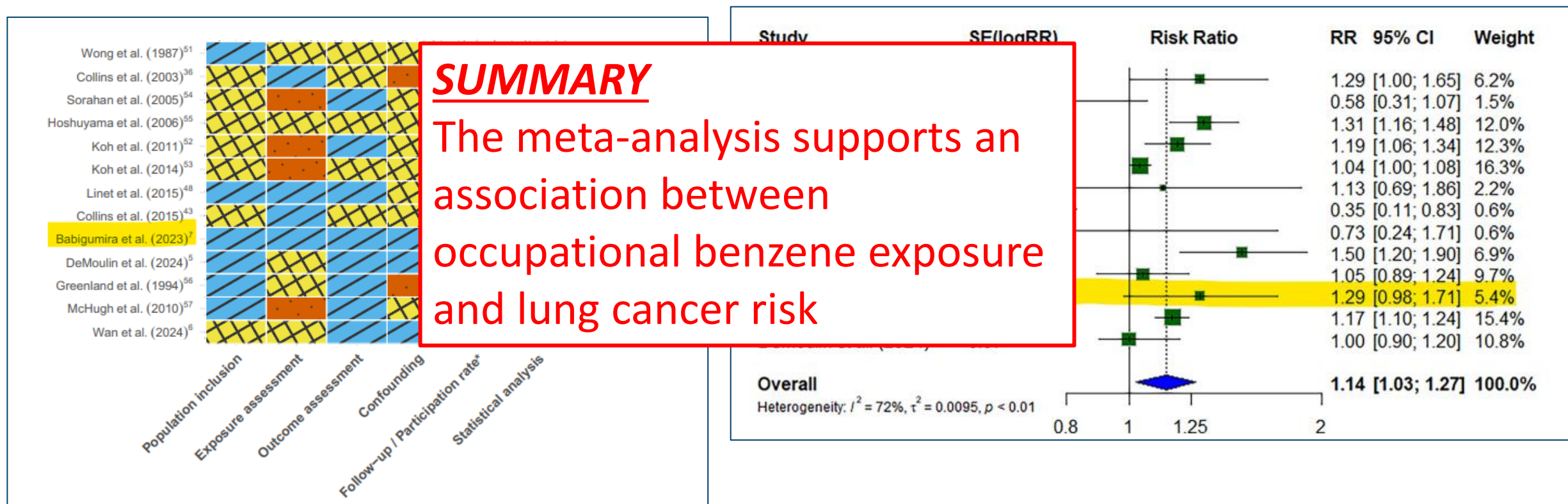
METHODS: We reviewed the relevant human epidemiological studies from PubMed and Embase databases to 19 August 2024. Data extraction included study characteristics, effect estimates, and exposure assessment details. Two investigators independently evaluated study quality using the Newcastle-Ottawa scale (NOS) framework and exposure assessment quality based on *a priori* criteria. Six risk of bias (ROB) domains were constructed from the NOS criteria to identify and quantify possible biases and their impacts on parameter estimates. Meta-analysis relative risk (pooled RR) and associated confidence intervals were calculated using random-effects models, and a flexible exposure–response meta-regression was fitted to assess the shape of the association. Subgroup analyses were conducted to explore the consistency of results.

RESULTS: Of 252 articles identified, 13 studies covering 366,975 participants (17,030 lung cancer cases) were included in our analysis. The meta-analysis of ever occupational benzene exposure showed an elevated risk of lung cancer (pooled RR = 1.14; 95% CI: 1.03, 1.27; $I^2 = 72$). Subgroup analyses revealed that larger pooled RRs in studies based on highly exposed groups had higher overall quality and better exposure assessments and included both males and females (as opposed to only males). A positive linear trend was observed in the exposure–response meta-analysis.

DISCUSSION: Our meta-analysis supports an association between occupational benzene exposure and an increased risk of lung cancer. <https://doi.org/10.1289/EHP15086>

Wan et al. 2024 Environ Health Persp.
DOI: <https://doi.org/10.1289/EHP15086>

Benzene and lung cancer: New systematic review and meta-analysis including the NOPW cohort study





Benzene and colorectal cancer


Environmental Research 276 (2025) 121407

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Benzene exposure and risk of colorectal cancer by anatomical subsite in the Norwegian offshore petroleum workers cohort

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Results – benzene and risk of colorectal cancer

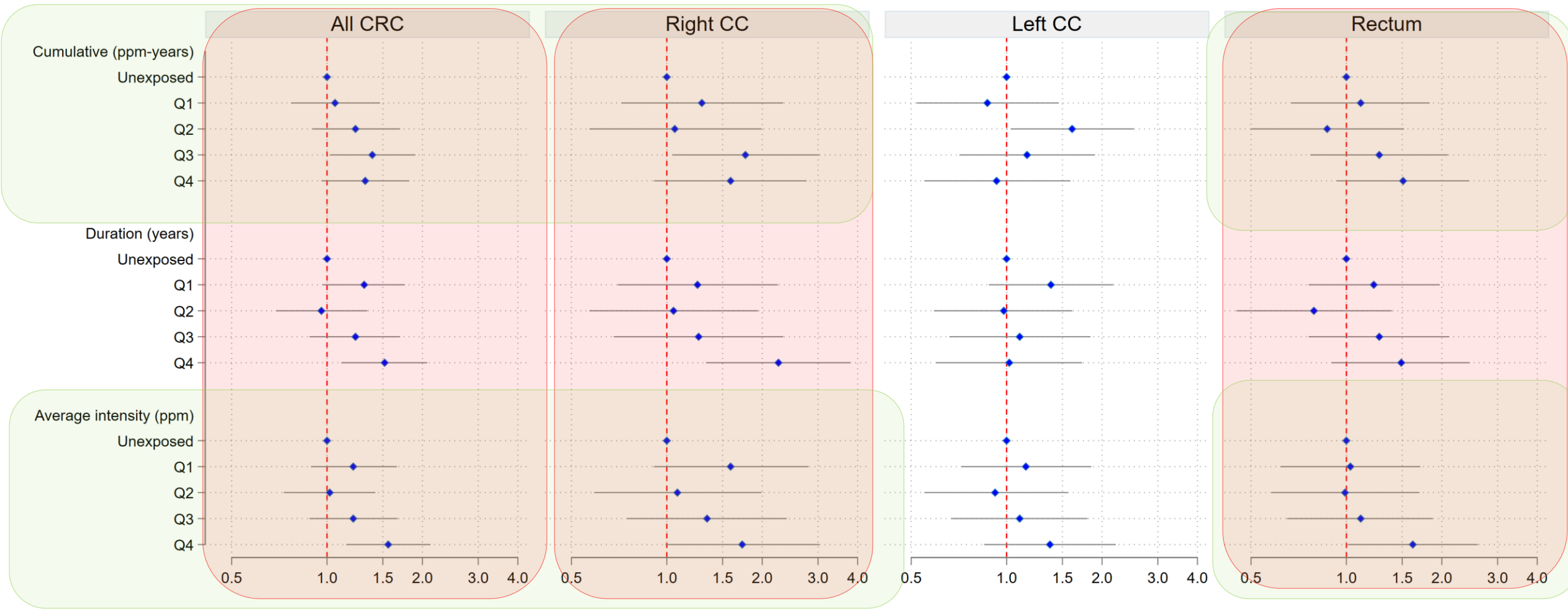
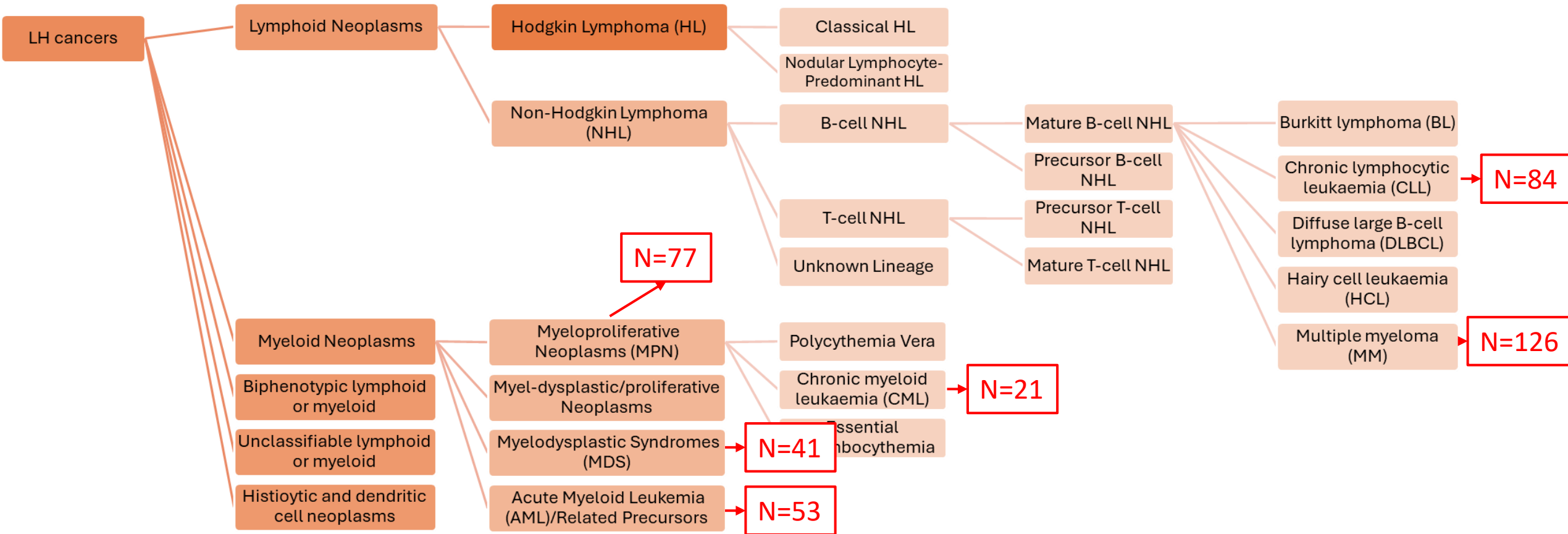


Figure: Hazard ratios (HRs) with 95% confidence intervals (logarithmic scale)

Major histological subtypes of LH cancer

For new studies on benzene and LH cancer – case number for selected subtypes in the offshore cohort



Summary

- More studies on rare LH cancer subtypes (e.g. ALL, CML) are needed
- More studies needed on bladder and CRC
- What about other solid tumours such as head-neck and upper aerodigestive cancer tract?
- What about co-exposures from other hydrocarbons than benzene?

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Summary of findings

Exposure	Cancer type	Association
Benzene	Lymph/leuk	Yes
Benzene, crude/mineral oil, sun	Skin	Yes
Benzene, hydrokarboner	Bladder	Suggestively
Benzene	Lung	Yes
Benzene	Colorectal	Yes

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www.nature.com/bjc

Review
A Section 508-conformant HTML version of this article is available at <https://doi.org/10.1289/EHP15086>

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Benzene exposure and risk of colorectal cancer by anatomical subsite in the Norwegian offshore petroleum workers cohort

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ABSTRACT
Objective: To investigate the association between low levels of benzene exposure (≤0.879 parts per million [ppm]-years) and risk of colorectal cancer (CRC) including its anatomical subsites.
Methods: Among 25,347 male workers in the Norwegian Offshore Petroleum Workers (NOPW) cohort with offshore work history (1965–1998), 455 CRC cases were diagnosed 1999–2021. We compared these with a subcohort (n = 2031) drawn from the full cohort. Work histories were linked to a previously developed industry-specific benzene job-exposure matrix (JEM). Cox regression for case-cohort analyses was used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for CRC, adjusted for age, body mass index, smoking, alcohol intake, red/processed meat intake, and physical activity.
Results: Risks of CRC increased with increasing benzene exposure. For all CRC, the HRs (95% CI) for the most exposed [quartile 4] vs. the unexposed were 1.32 (0.96 to 1.81, [0.177–0.879 ppm-years]; p-trend = 0.085) for cumulative, 1.52 (1.11 to 2.07, [17–34 years]; p-trend = 0.032) for duration, and 1.56 (1.15 to 2.12, [0.015–0.046 ppm]; p-trend = 0.005) for average intensity of benzene exposure. For right-sided colon cancer, the association was most evident for exposure duration (HR = 2.25 (1.33 to 3.80), quartile 4 [17–34 years] vs. unexposed; p-trend = 0.007). Sensitivity analyses showed consistent associations.
Conclusion: This study found positive exposure-response associations between low-level benzene exposure and CRC risk in offshore petroleum workers. These findings add to emerging evidence that benzene can be associated with solid tumours including lung and bladder, which potentially has important occupational and public health implications.

* Corresponding author. Cancer Registry of Norway, Norwegian Institute of Public Health, P.O. Box 5313 Majorstuen, NO-0304, Oslo, Norway.
E-mail address: roba@kreftregisteret.no (R. Babigumira).

<https://doi.org/10.1016/j.envres.2025.121407>
Received 29 August 2024; Received in revised form 13 March 2025; Accepted 14 March 2025
Available online 19 March 2025
0013-9351/© 2025 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>).

31

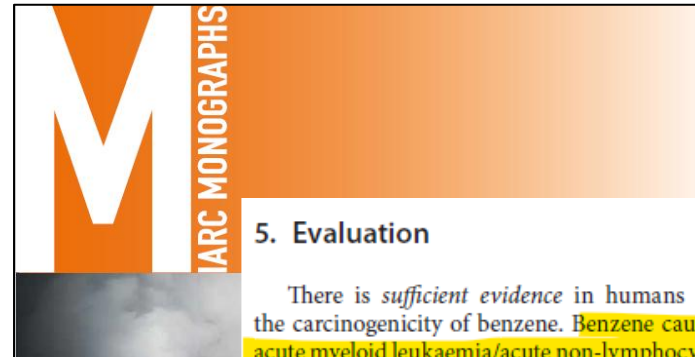
Background

9. BENZENE (Group 1)

Benzene has shown no evidence of carcinogenicity when mice by skin application. Other animal experiments were to be inadequate to evaluate the carcinogenicity of benzene.

Several case reports as well as an epidemiological case study suggest a relationship between benzene exposure and acute myeloid leukaemia. Two cohort studies^{5,6} showed an increased incidence of acute myeloid leukaemia in workers exposed to benzene. There has been an additional report of a large number of leukaemia cases (mostly acute non-lymphocytic) among a group of workers exposed to benzene^{7,8}.

¹ IARC Monographs, 1: 69-73, 1972.



5. Evaluation

There is sufficient evidence in humans for the carcinogenicity of benzene. Benzene causes acute myeloid leukaemia/acute non-lymphocytic leukaemia.

Also, a positive association has been observed between exposure to benzene and acute lymphocytic leukaemia, chronic lymphocytic leukaemia, multiple myeloma, and non-Hodgkin lymphoma.

There is sufficient evidence for the carcinogenicity of benzene in experimental animals.

There is strong evidence that benzene metabolites, acting alone or in concert, produce multiple genotoxic effects at the level of the pluripotent haematopoietic stem cell resulting in chromosomal changes in humans consistent with those seen in haematopoietic cancer. In multiple studies in different occupational populations in many countries over more than three decades a variety of genotoxic changes, including chromosomal abnormalities, have been found in the lymphocytes of workers exposed to benzene.

Benzene is carcinogenic to humans (Group 1).



6. EVALUATION AND RATIONALE

6.1 Evidence in humans

There is sufficient evidence in humans for the carcinogenicity of benzene. Benzene causes acute myeloid leukaemia/acute non-lymphocytic leukaemia in adults.

Also, a positive association has been observed between exposure to benzene and acute lymphocytic leukaemia, chronic lymphocytic leukaemia, multiple myeloma, chronic myeloid leukaemia in children, and non-Hodgkin lymphoma.

However, if the Working Group considered that benzene also causes non-Hodgkin lymphoma, a separate small minority considered that a positive association was not observed for cancer of the lung.

6.4 Rationale

Support for Group 1 from mechanistic data

A Group 1 evaluation was supported by mechanistic data demonstrating that benzene exhibits many of the key characteristics of carcinogens. In particular, there is strong evidence, including in exposed humans, that benzene: is metabolically activated to electrophilic metabolites; induces oxidative stress and associated oxidative DNA damage; is genotoxic, inducing DNA damage and chromosomal changes; is immunosuppressive; and causes haematotoxicity.

Exploring Norwegian cohorts and biobanks in agricultural exposure and cancer

Hilde Langseth, PhD

Senior Researcher and Section Chief Molecular Epidemiology and Infections

Research Department

Cancer Registry of Norway, NIPH

Seminar – Environment, Occupation and cancer: etiology and prevention

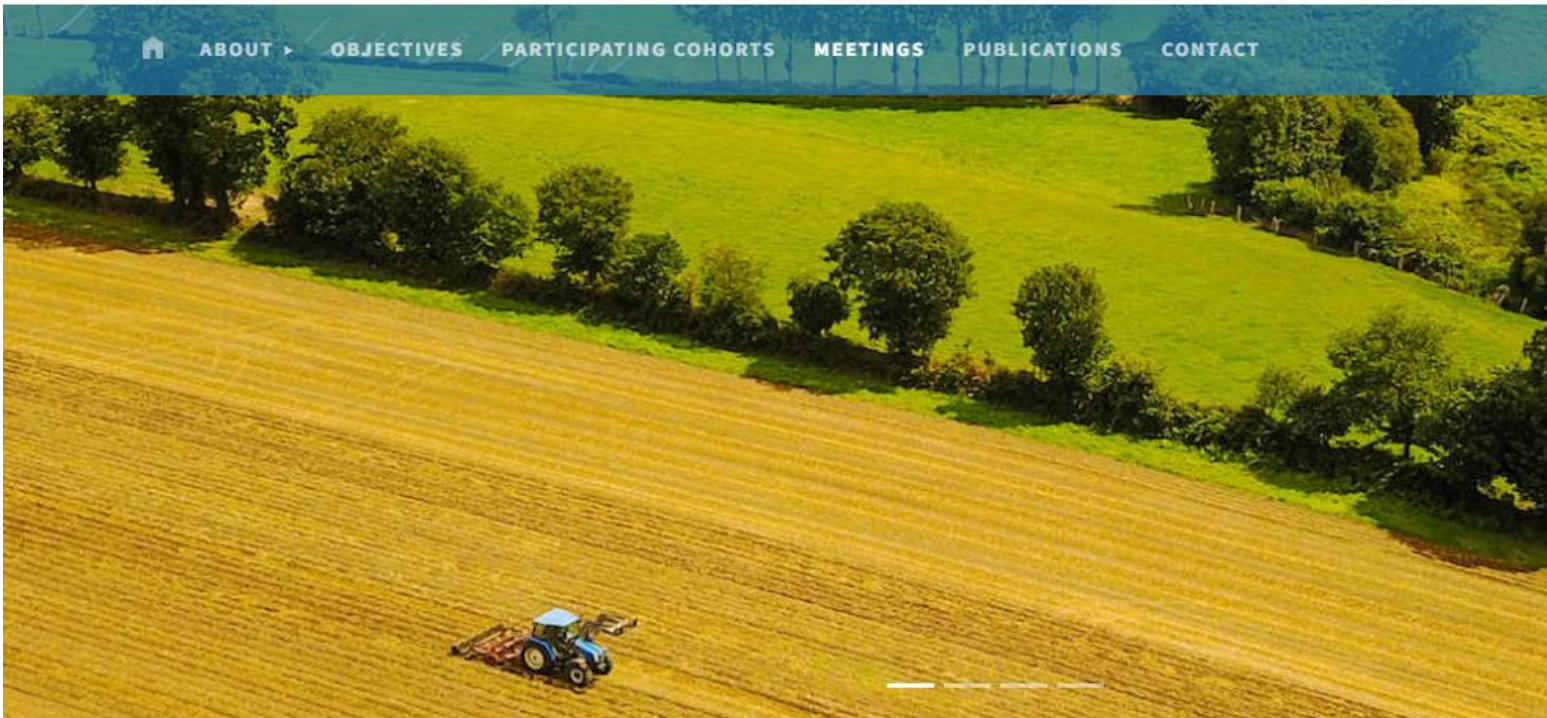
25 september 2025

Background

- Agricultural populations consistently shows distinctive patterns of health and disease than other populations
- Workers may be exposed to potential carcinogens including pesticides and solar radiation
- Physically active work routines and low prevalence of smoking partially explain a reduced incidence of lifestyle related cancers like lung and colorectal cancer
- Higher risk of hematological and lymphatic cancers associated to pesticide exposures
- Regular exposure to ultraviolet rays has been linked to increased risk in lip cancer
- Cancer Registry of Norway in international consortium (AGRICOH)
- Three Norwegian Cohort with data from Cancer Registry, Census data, health survey data and/or biological material

AGRICOH: A Consortium of Agricultural Cohort Studies

- Established 2010
- 29 cohorts from 5 continents
- CRN with Janus since 2012



The consortium is interested in identifying environmental and occupational exposures in agricultural settings associated with excess risk of chronic illnesses, including cancer, respiratory, neurologic and auto-immune diseases, reproductive and allergic disorders as well as in factors associated with a decreased morbidity risk.

[AGRICOH: Home](#)

Int. J. Environ. Res. Public Health **2011**, *8*, 1341–1357; doi:10.3390/ijerph8051341

OPEN ACCESS

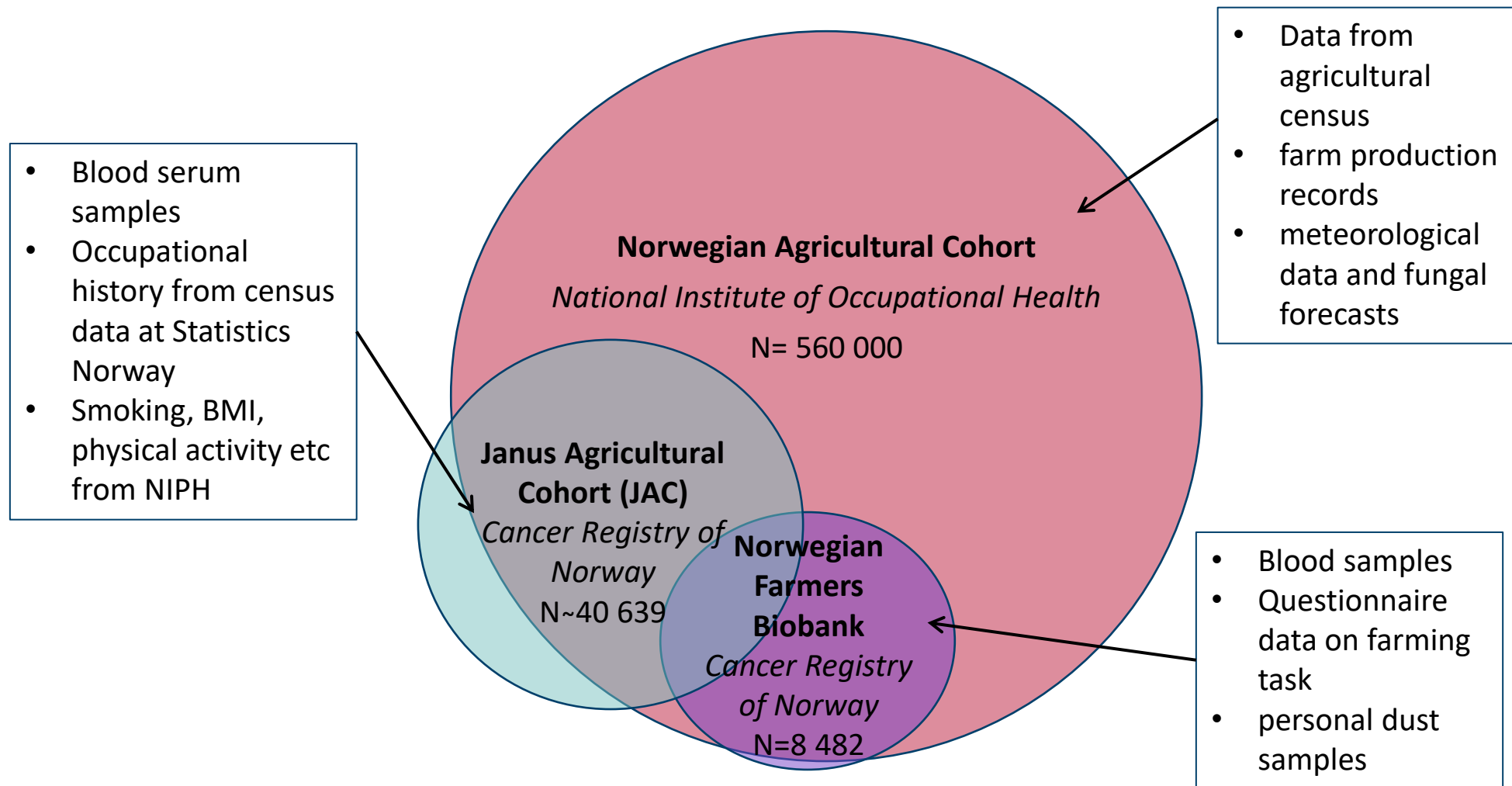
International Journal of
Environmental Research and
Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Communication

AGRICOH: A Consortium of Agricultural Cohorts

Maria E. Leon ^{1,2,*}, Laura E. Beane Freeman ^{3,4}, Jeroen Douwes ^{4,5}, Jane A. Hoppin ^{5,6}, Hans Kromhout ^{6,7}, Pierre Lebaillly ^{7,8}, Karl-Christian Nordby ^{8,9}, Marc Schenker ^{9,10}, Joachim Schüz ^{1,2,4}, Stephen C. Waring ^{10,11}, Michael C.R. Alavanja ³, Isabella Annesi-Maesano ¹¹, Isabelle Baldi ¹², Mohamed Aqiel Dalvie ¹³, Giles Ferro ^{1,2}, B éatrice Fervers ¹⁴, Hilde Langseth ¹⁵, Leslie London ¹³, Charles F. Lynch ¹⁶, John McLaughlin ¹⁷, James A. Merchant ¹⁸, Punam Pahwa ¹⁹, Torben Sigsgaard ²⁰, Leslie Stayner ²¹, Catharina Wesseling ²², Keun-Young Yoo ²³, Shelia H. Zahm ³, Kurt Straif ^{1,2} and Aaron Blair ³

Norwegian Agricultural Cohorts participating in the AGRICOH consortium



Results from the AGRICOH Consortium



Cancer incidence in agricultural workers: Findings from an international consortium of agricultural cohort studies (AGRICOH)

Kayo Togawa^{a,*}, Maria E. Leon^a, Pierre Lebaillly^b, Laura E Beane Freeman^c, Karl-Christian Nordby^d, Isabelle Baldi^e, Ewan MacFarlane^f, Aesun Shin^g, Sue Park^h, Robert T Greenlee^b, Torben Sigsgaardⁱ, Ioannis Basinas^j, Jonathan N. Hofmann^c, Kristina Kjaerheim^k, Jeroen Douwes^l, Rachel Denholm^{a,1}, Gilles Ferro^a, Malcolm R. Sim^f, Hans Kromhout^m, Joachim Schüz^a

Lower risk in many cancers. Increased risk of Prostate cancer, multiple myeloma and malignant melanoma in women

Cancer Causes & Control (2023) 34:995–1003
https://doi.org/10.1007/s10552-023-01748-1

ORIGINAL PAPER

No association

Exposure to pesticides and risk of Hodgkin lymphoma in an international consortium of agricultural cohorts (AGRICOH)

Joanne Kim¹, Maria E. Leon¹, Leah H. Schinasi², Isabelle Baldi³, Pierre Lebaillly⁴, Laura E. Beane Freeman⁵, Karl-Christian Nordby⁶, Gilles Ferro¹, Alain Monnereau^{7,8}, Maartje Brouwer⁹, Kristina Kjaerheim¹⁰, Jonathan N. Hofmann⁵, Kurt Straif^{11,12}, Hans Kromhout¹³, Joachim Schüz¹, Kayo Togawa¹



International Journal of Epidemiology, 2019, 1519–1535
doi: 10.1093/ije/dyz017
Advance Access Publication Date: 18 March 2019
Original article



Occupational Health

Pesticide use and risk of non-Hodgkin lymphoid malignancies in agricultural cohorts from France, Norway and the USA: a pooled analysis from the AGRICOH consortium

Maria E Leon,^{1*} Leah H Schinasi,^{1,2} Pierre Lebaillly,³ Laura E Beane Freeman,⁴ Karl-Christian Nordby,⁵ Gilles Ferro,¹ Alain Monnereau,^{6,7} Maartje Brouwer,⁸ Séverine Tual,³ Isabelle Baldi,⁹ Kristina Kjaerheim,¹⁰ Jonathan N Hofmann,⁴ Petter Kristensen,⁵ Stella Koutros,⁴ Kurt Straif,¹¹ Hans Kromhout⁸ and Joachim Schüz¹

Associations of pesticides with NHL appear to be subtype- and chemical-specific

BMJ Journals

Occupational & Environmental Medicine

ORIGINAL RESEARCH

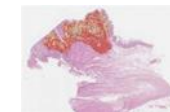
No association

Animal farming and the risk of lymphohaematopoietic cancers: a meta-analysis of three cohort studies within the AGRICOH consortium

Sonia El-Zaemey^{1,2}, Leah H Schinasi,^{2,3} Gilles Ferro,² Séverine Tual,⁴ Pierre Lebaillly,⁴ Isabelle Baldi,⁵ Karl-Christian Nordby,⁶ Kristina Kjaerheim,⁷ Joachim Schüz,² Alain Monnereau,^{8,9} Maartje Brouwer,¹⁰ Stella Koutros¹¹, Jonathan Hofmann,¹¹ Petter Kristensen,⁶ Hans Kromhout,¹⁰ Maria E Leon,² Laura E Beane Freeman¹¹



Janus Serum Bank Cohort



123 719
cancer cases

1972 1973 1976 2004 2005 2015 2023

● Janus Serum Bank established

89% Norwegian Regional Health Studies

Cancer follow-up

10%

Red Cross donors

Cancer follow-up

1%

Post-diagnostic samples from Radium Hospital Cancer follow-up

Baseline
information on
smoking, BMI,
physical activity

Total: 318 628



Males: 166 137

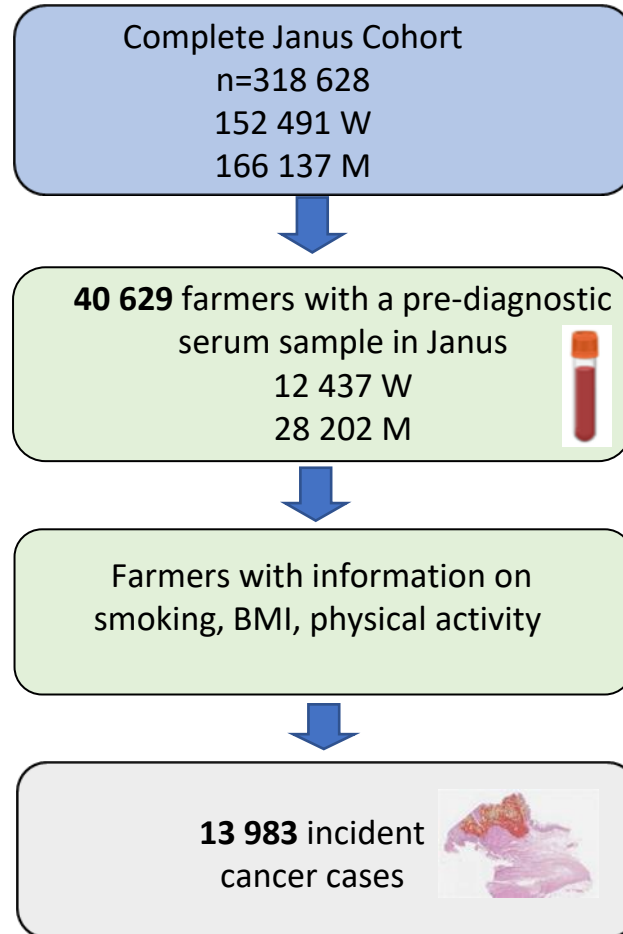


Females: 152 491

Langseth et al, Janus Cohort profile, *Int J Epidemiol*, 2016
Hjerkind et al, Janus Cohort Profile update, *Int J Epidemiol*, 2017



Janus Agricultural Cohort (JAC)



Linkage to Census data and agricultural census at Statistics Norway

Crop: 4 065 Animal: 13 031 Mixed: 8 570

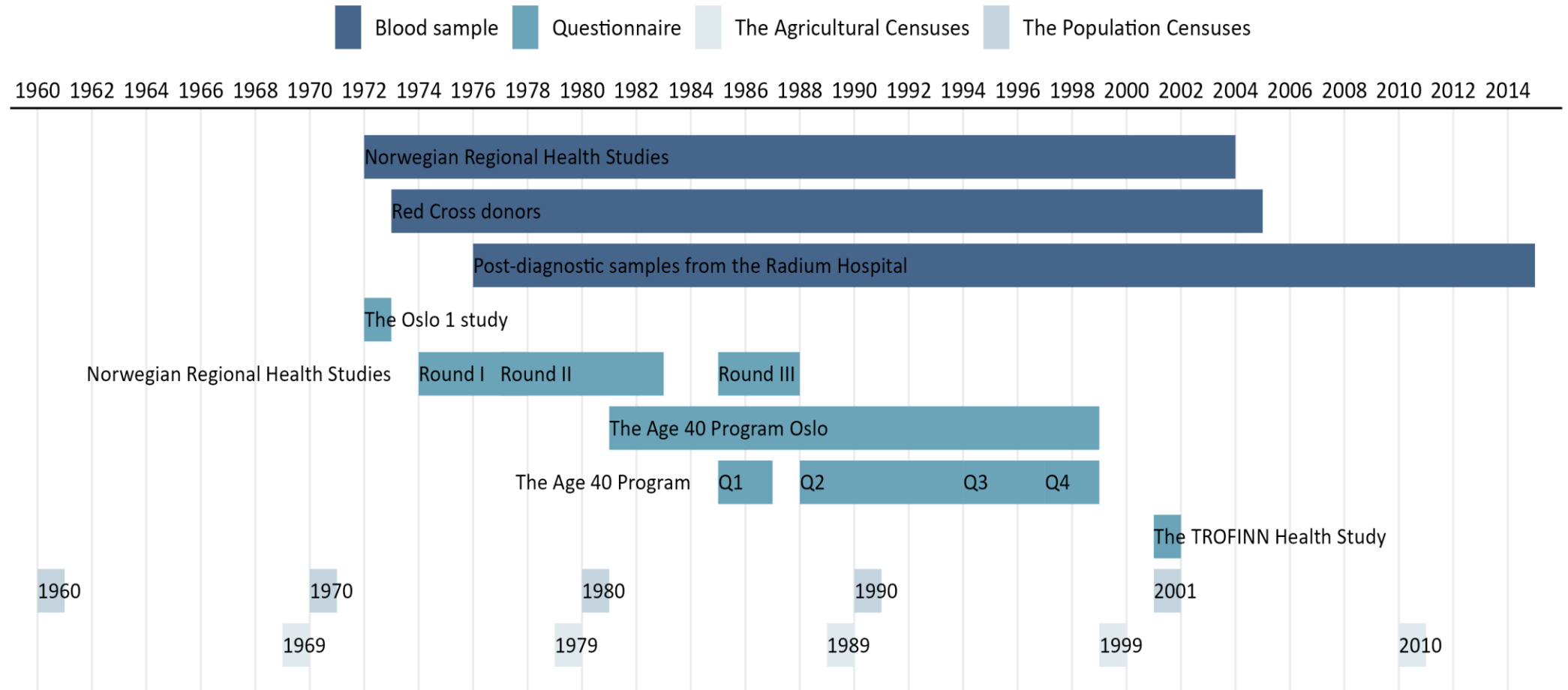
Linkage to questionnaire data at National Inst of Public Health

Linkage to Cancer Registry of Norway for detailed information on cancer diagnosis

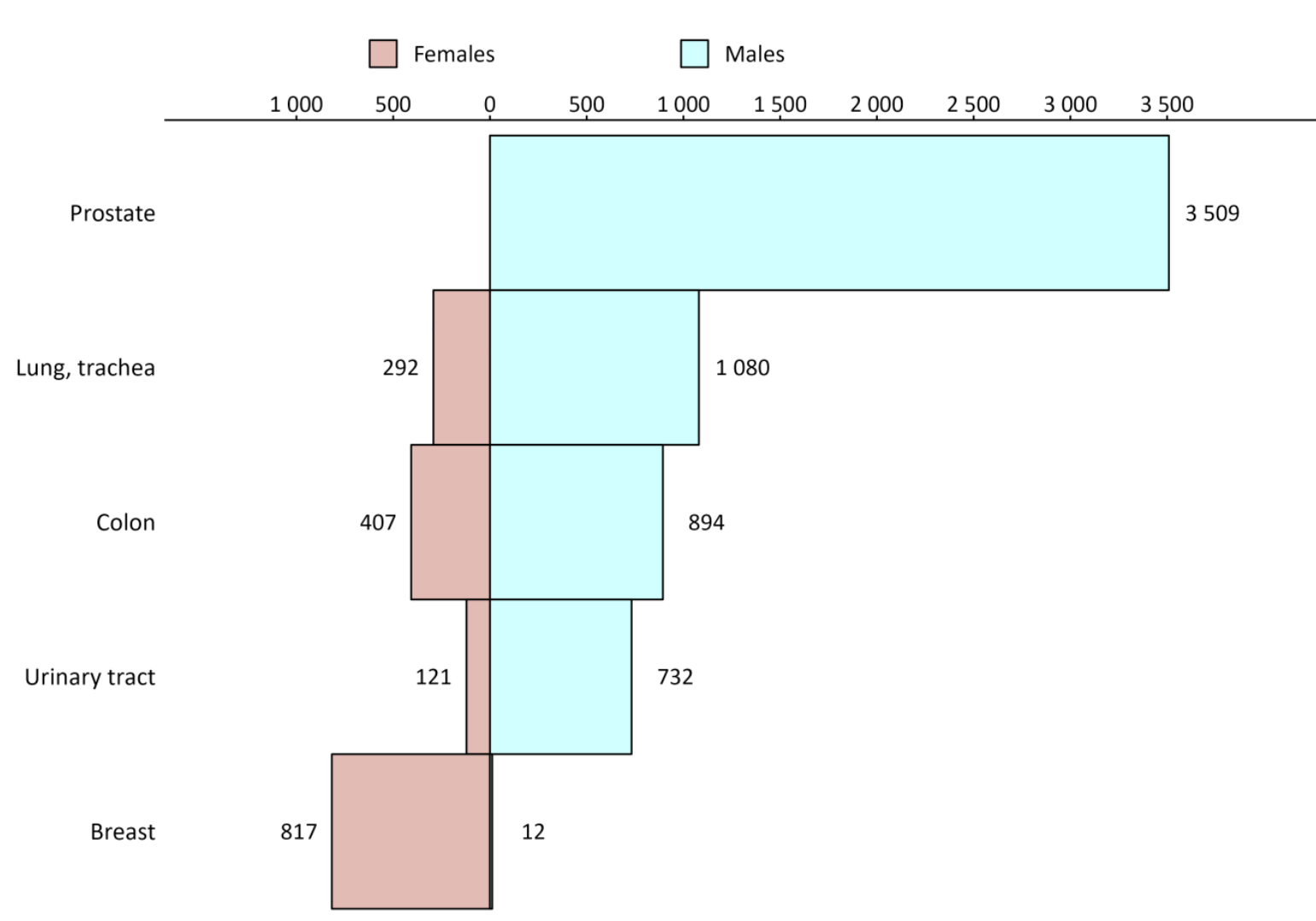


NORWEGIAN CANCER SOCIETY

Data material - timeline



The 5 most common cancer types in JAC



Standardized incidence ratios (SIR) analyses

- To compare the cancer incidence in the JAC with the general Norwegian population we calculated SIR by dividing the observed number of cancer cases in JAC with the expected numbers in the population
- The expected numbers of cancer cases were calculated using the five-year age-specific incidence rates for each year for the entire Norwegian population
- The 95% confidence intervals were estimated assuming a Poisson distribution of the cases
- There was an inverse association between cancer incidence and working as farmers. Except from male **lip cancer** (SIR=1.58) the observed numbers were significantly lower in farmers compared to the Norwegian population

Results: SIR analyses for selected cancer types

ICD10	Site	Men (N = 28 192)			Women (N = 12 437)			Total (N = 40 629)		
		Obs	SIR	95% CI	Obs	SIR	95% CI	Obs	SIR	95% CI
C00	Lip	82	1.58	1.26-1.97	16	1.51	0.86-2.45	98	1.57	1.28-1.92
C18	Colon	844	0.88	0.82-0.94	381	0.81	0.73-0.90	1 225	0.86	0.81-0.91
C19–20	Rectum, rectosigmoid	492	0.84	0.77-0.92	155	0.86	0.73-1.00	647	0.85	0.78-0.92
C22	Liver	42	0.44	0.31-0.59	15	0.58	0.33-0.96	57	0.47	0.35-0.61
C25	Pancreas	198	0.65	0.56-0.75	95	0.74	0.60-0.91	293	0.68	0.60-0.76
C33–34	Lung, trachea	916	0.66	0.62-0.70	261	0.57	0.50-0.64	1 177	0.64	0.60-0.67
C43	Melanoma of the skin	494	0.81	0.74-0.89	202	0.84	0.73-0.97	696	0.82	0.76-0.88
C50	Breast	12	0.69	0.36-1.21	780	0.80	0.74-0.86	792	0.80	0.74-0.86
C53	Cervix uteri	-	-	-	71	0.64	0.50-0.81	71	0.64	0.50-0.81
C56, C57.0–4, C48.2	Ovary etc.	-	-	-	176	0.85	0.73-0.98	176	0.85	0.73-0.98
C70–72	Central nervous system	264	0.87	0.77-0.98	141	0.88	0.74-1.04	405	0.88	0.79-0.97
C91–95	Leukaemia	305	0.75	0.67-0.84	111	0.81	0.67-0.98	416	0.77	0.70-0.85
C00–96	All sites	10 343	0.86	0.84-0.88	3 640	0.80	0.78-0.83	13 983	0.84	0.83-0.86

Corresponds to results from The Nordic Occupational Cancer Study (NOCCA) (SIR 1.60, 95% CI: 1.55–1.66)

Mroueh R et al, Acta Oncological 2023, vol 62, no 6, 541-549

Cancer incidence by farm type

Males

ICD10	Site	Crop (N = 3 556)			Animal (N = 11 473)			Mixed (N = 7 314)		
		Obs	SIR	95% CI	Obs	SIR	95% CI	Obs	SIR	95% CI
C00	Lip	10	1.94	0.93-3.57	33	1.41	0.97-1.98	22	1.66	1.04-2.51

Females

ICD10	Site	Crop (N = 509)			Animal (N = 1 558)			Mixed (N = 1 256)		
		Obs	SIR	95% CI	Obs	SIR	95% CI	Obs	SIR	95% CI
C00	Lip	2	5.47	0.66-19.76	5	4.14	1.34-9.66	0	0.00	0.00-3.75

Norwegian Farmers Biobank



Farmers biobank

Enrollment years and organization	1989-1991 established by The National Institute of Occupational Health through a research program looking at health risks from dust in agriculture. Transferred to Cancer Registry 2012
Inclusion criteria	Active farmers and those who had retired during the last 3 years
Number of participants <ul style="list-style-type: none"> • Males • Females 	8 572 5 622 2 950
Age at enrollment	20-67 years, mean age 49 years
Biological specimens and data	Serum samples 3 aliquotes à 150µl Questionnaire data and lung function measurements
Location	Stored at -25 at Janus storage



Project team and collaborators

Project team at CRN



Hilde Langseth
PI JAC and Farmers



Kristina Kjærheim
Senior Researcher



Jan Ivar Martinsen
Datamanager



Tove Slyngstad
Research assistance



Ronnie Babigumira
Datamanager



Marie Udnesseter Lie,
Datamanager

Collaborators and steering group members Farmers Biobank at STAMI



Karl-Christian Nordby



Wijnand Eduard

Break

10.35 – 11.00

Session 2 – Chemicals, environmental toxins and cancer: Etiology and prevention

11.00 – 12.45 **Keynote: Therese Haugdal Nøst** – Associate professor, University of Tromsø
Investigating environmental contaminants and health: Insights from population-based studies in Northern Norway

Marcin Wojewodzic - Senior Researcher, Cancer Registry of Norway-NIPH
PFAS in Occupational and Environmental Contexts

Tom K Grimsrud - Senior Consultant, Cancer Registry of Norway-NIPH
Nickel and cancer in the green shift area

Bente Oftedal – Senior Scientist, Department of Air Quality and Noise, NIPH
Air pollution and lung cancer in a national cohort

Lunch and poster display

12.45 – 13.45



UiT The Arctic University of Norway

Investigating environmental contaminants and health: Insights from population-based studies in Northern Norway

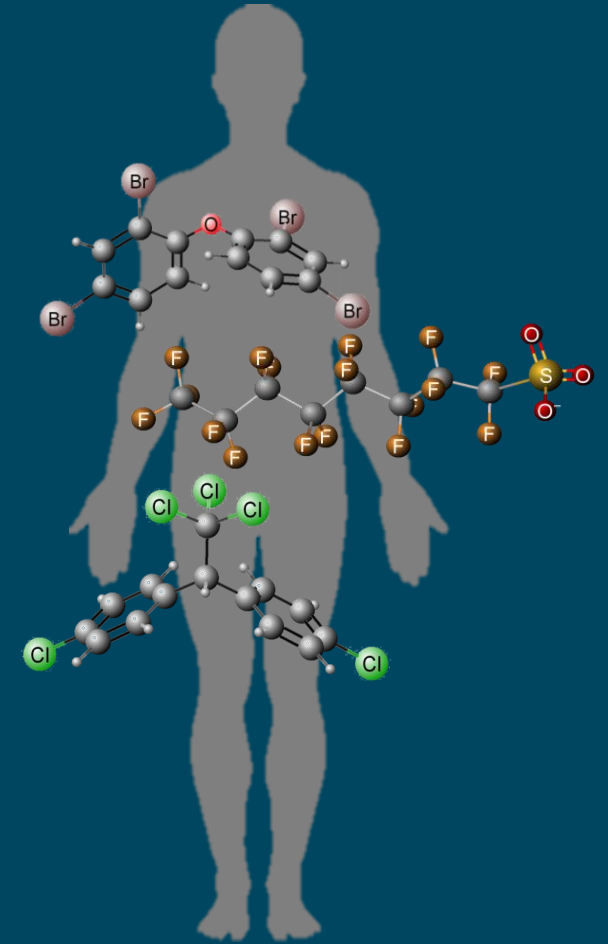
Therese Haugdahl Nøst, PhD

Associate Professor, Department for Community Medicine,

UiT The Arctic University of Norway

Researcher, HUNT Research Centre,

NTNU Norwegian University of Science and Technology



Acknowledgements

- Long-term collaborations representing also work lead by colleagues
- Made possible due to that surveys started collecting and biobanking blood in the 1970s
- NILU's environmental chemistry research group in Tromsø in 1994
- A continuum of research projects

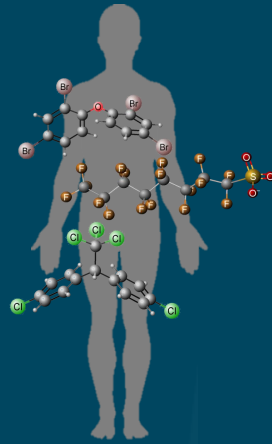


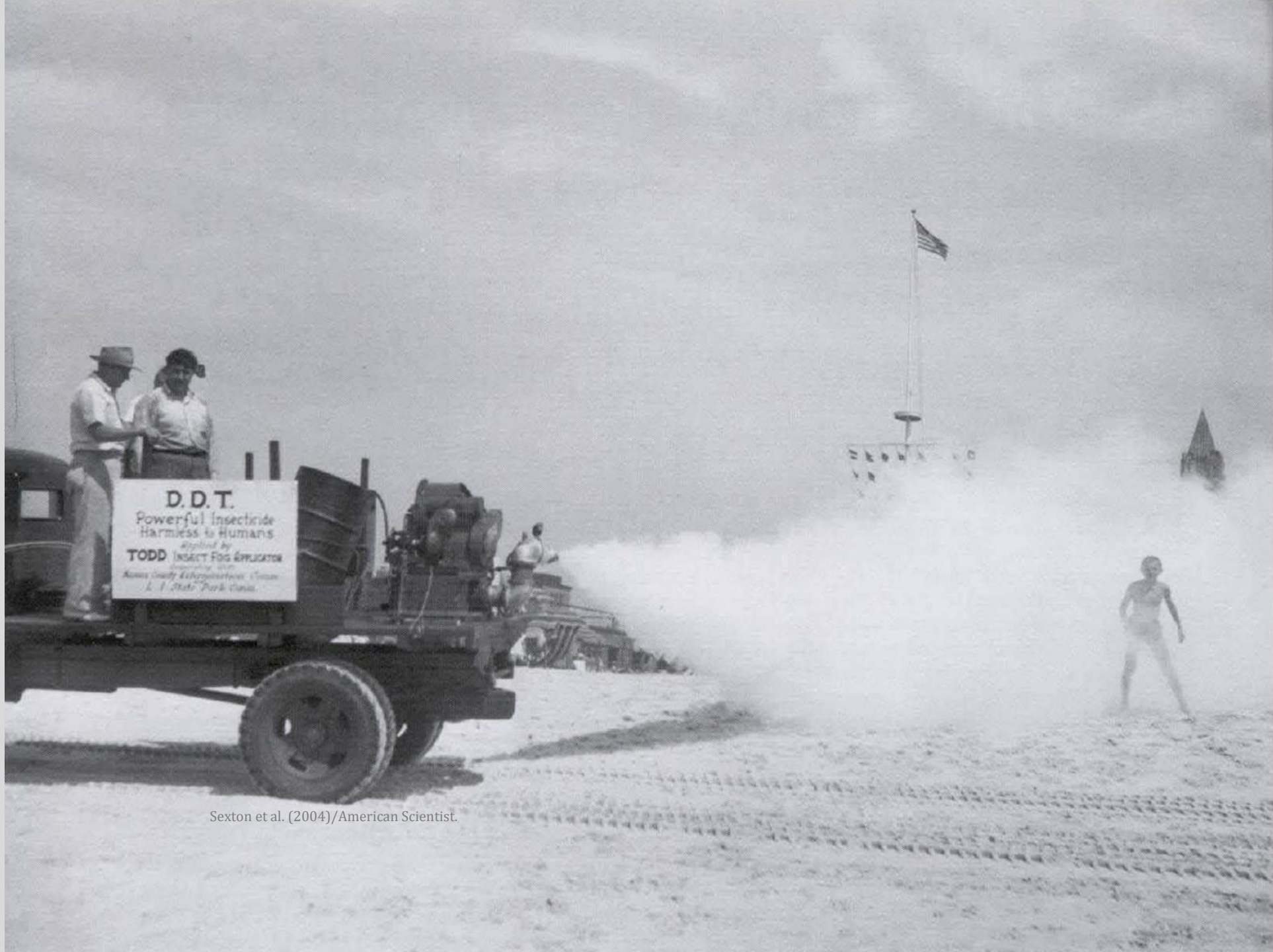
Fram Centre

nilu



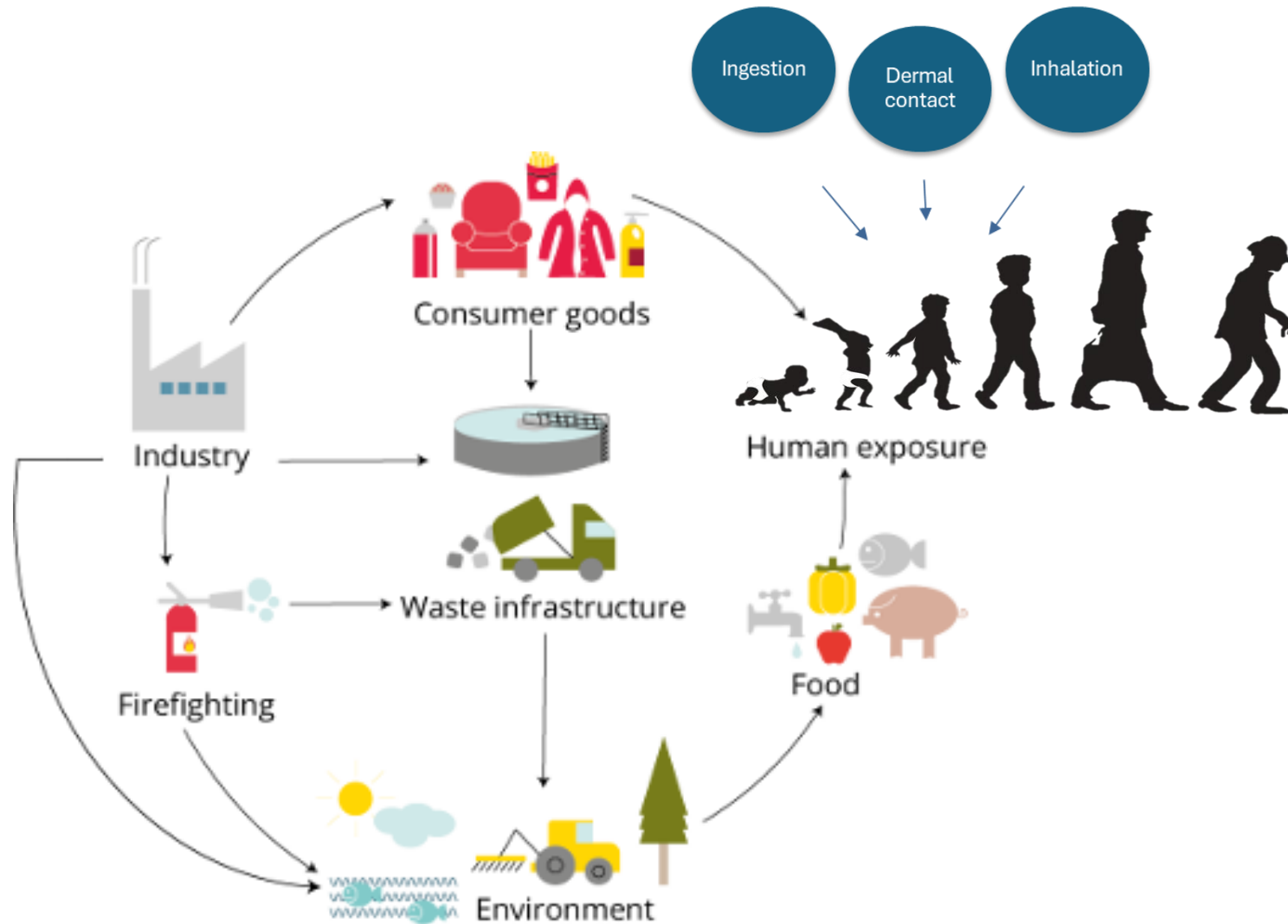
Human exposures to environmental contaminants





Sexton et al. (2004)/American Scientist.

Exposure – who and how?



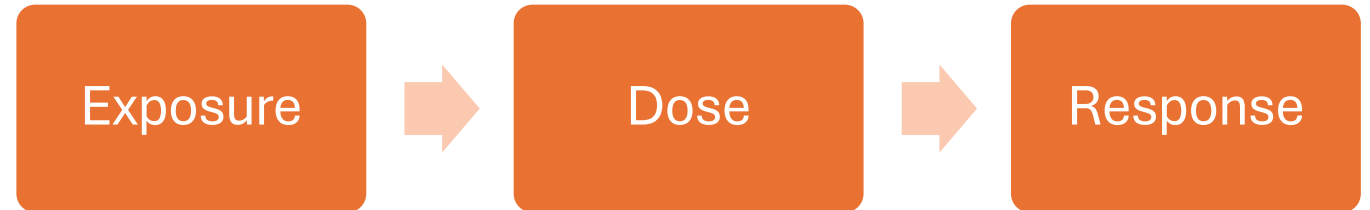
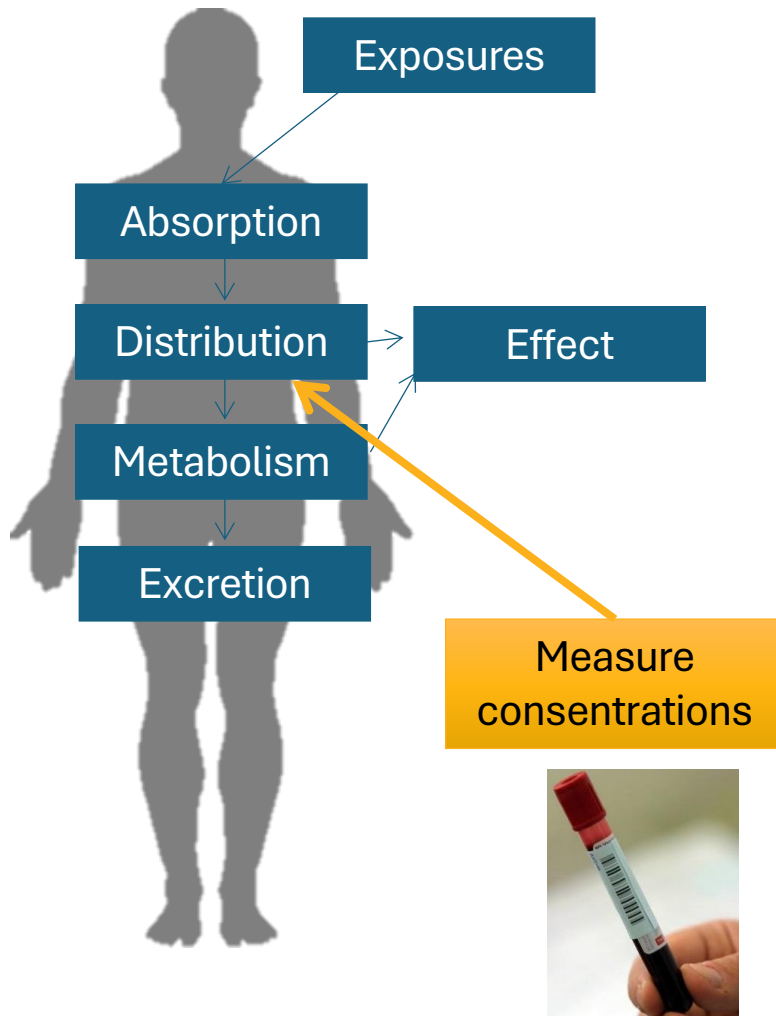
In the past



Now



Human monitoring studies

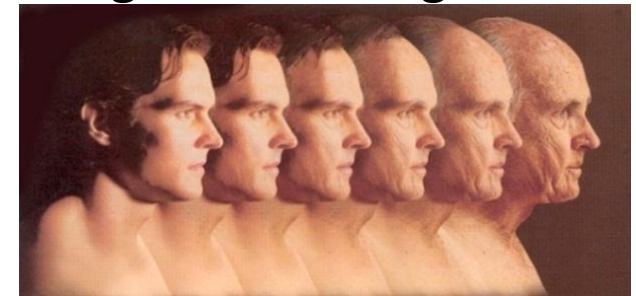


Cross sectional design:



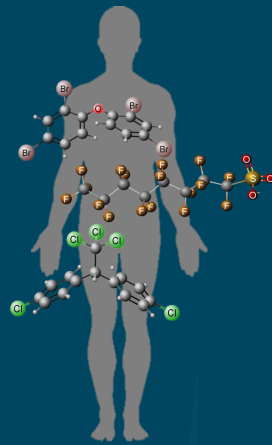
Time

Longitudinal design:



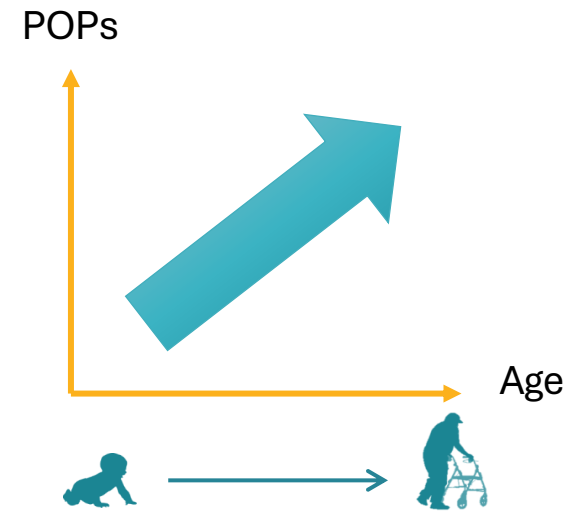
Time

Time trends of environmental contaminants in Northern Norway



Human exposures and age

- The beginning: Investigate trends of POPs in humans with respect to time
 - «Increase with age»
 - «Effect of age»
 - «Positively correlates with age»



- We could design a longitudinal study - Intraindividual changes
- Analyze a broad range of POPs in blood samples in the period 1979-2007
- Examine trends with respect to age, birth cohort and exposure history
- Validation of human exposure model

Population-based surveys at UiT



The Tromsø Study

Tromsø, 45000 at least once, 8th survey ongoing

1974 1979-80 1986-87 1994-95 2001 2007-08 2015-06 2025-26

The Norwegian Women and Cancer Study

National, 174000

1991 - 2017 2025

The Saminor Study

Sami municipalities, 28000 at least once, 3rd surveys ongoing

2003-04 2012-14 2023-25

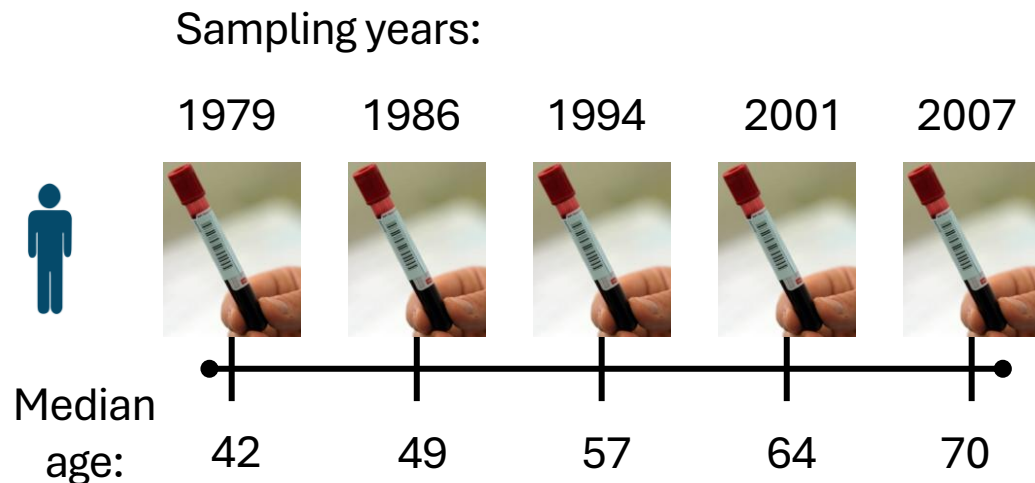
The Fit Futures Study

Troms county, youth, 1200, 3 surveys

2010-11 2012-13 2021-22

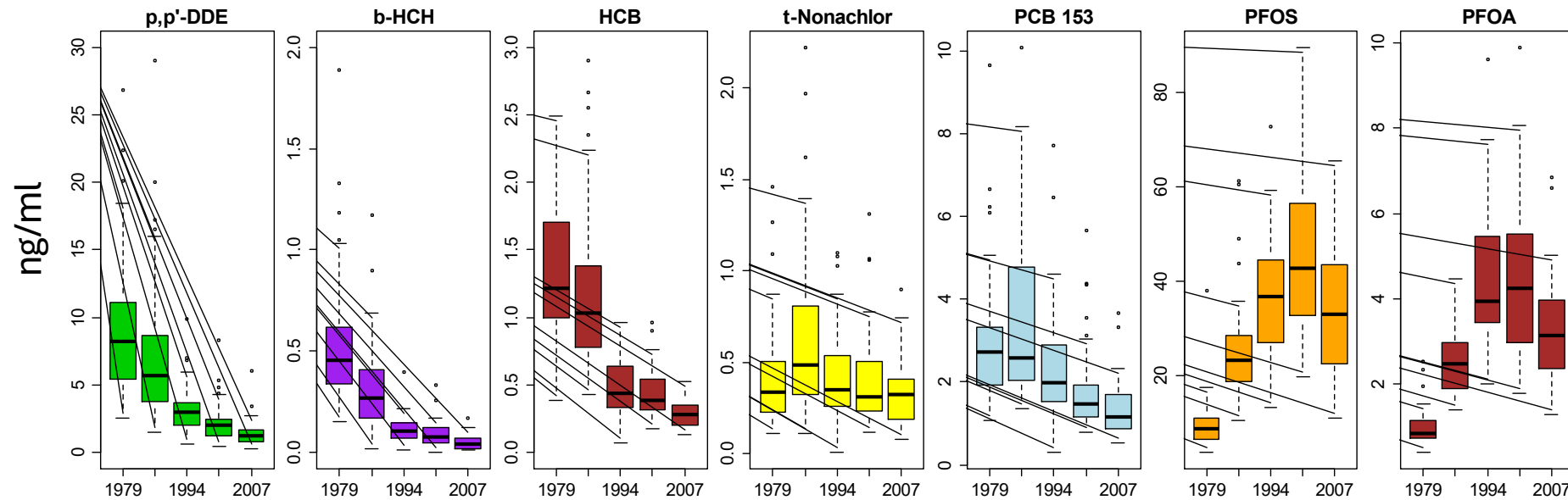
The Tromsø Study 1979-2007

- Archived serum samples of high quality in the Tromsø Study
- 5 repeated blood samples from 54 men over a 30 year period



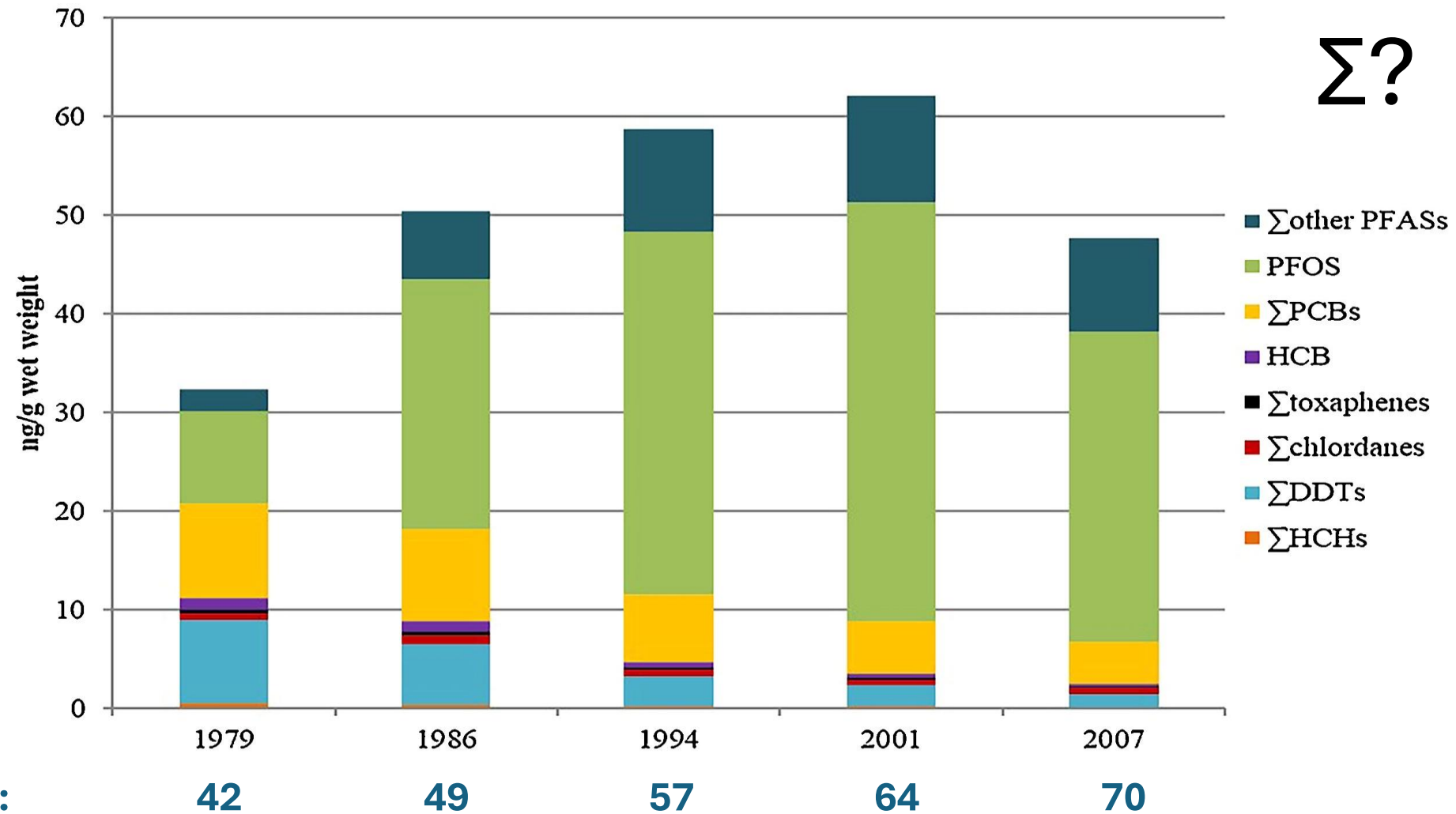
Time trends for over 50 components

- Different trends for different components



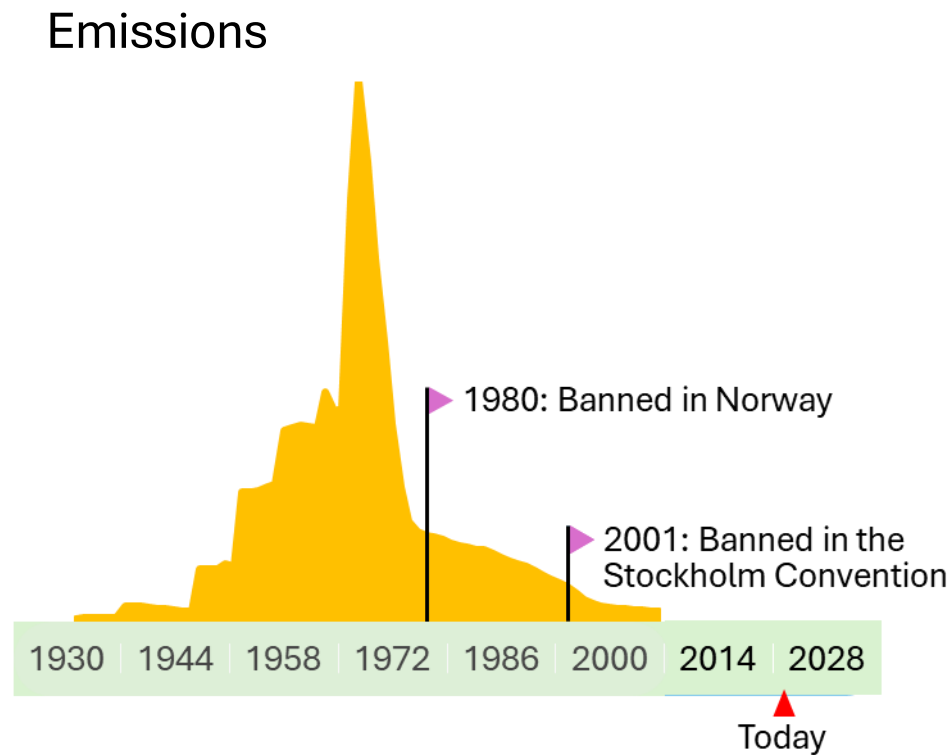
Dynamic serum POP burden

$\Sigma?$

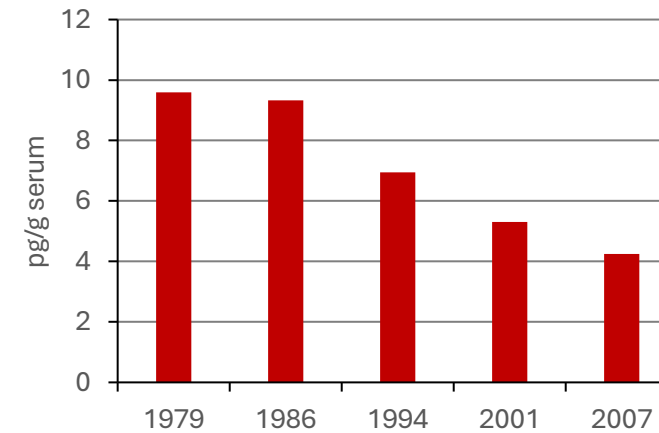


Good news about old sins

- PCB-153

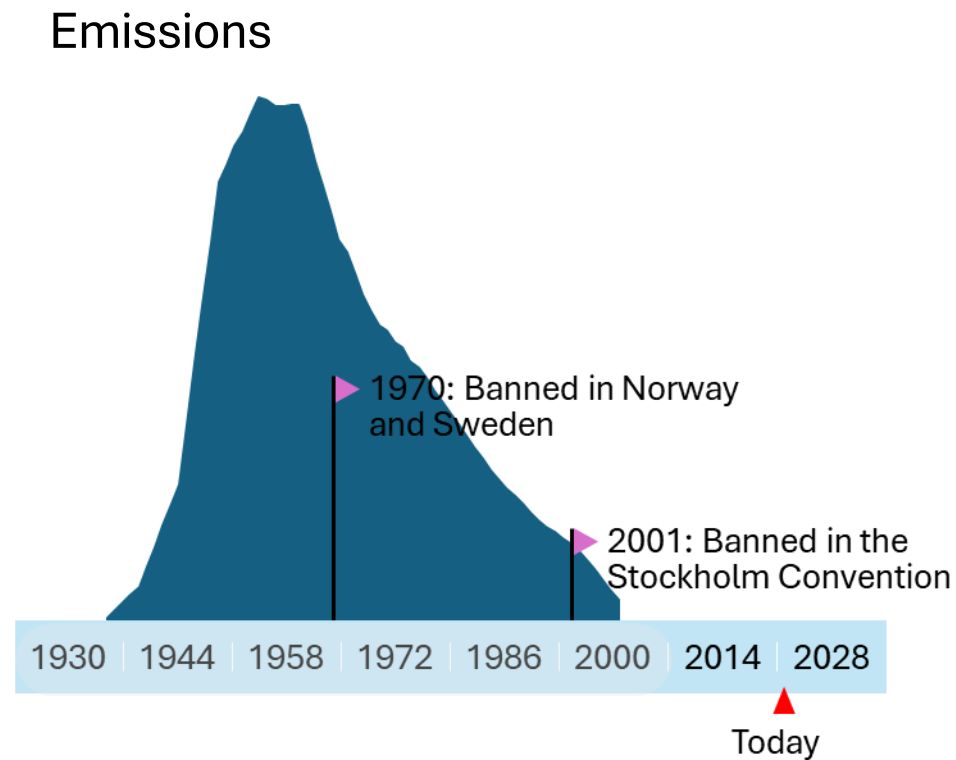


The Tromsø Study blood samples

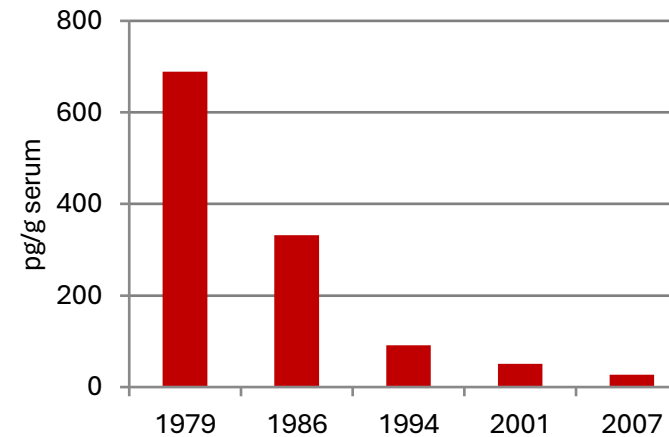


Good news about old sins

- DDT

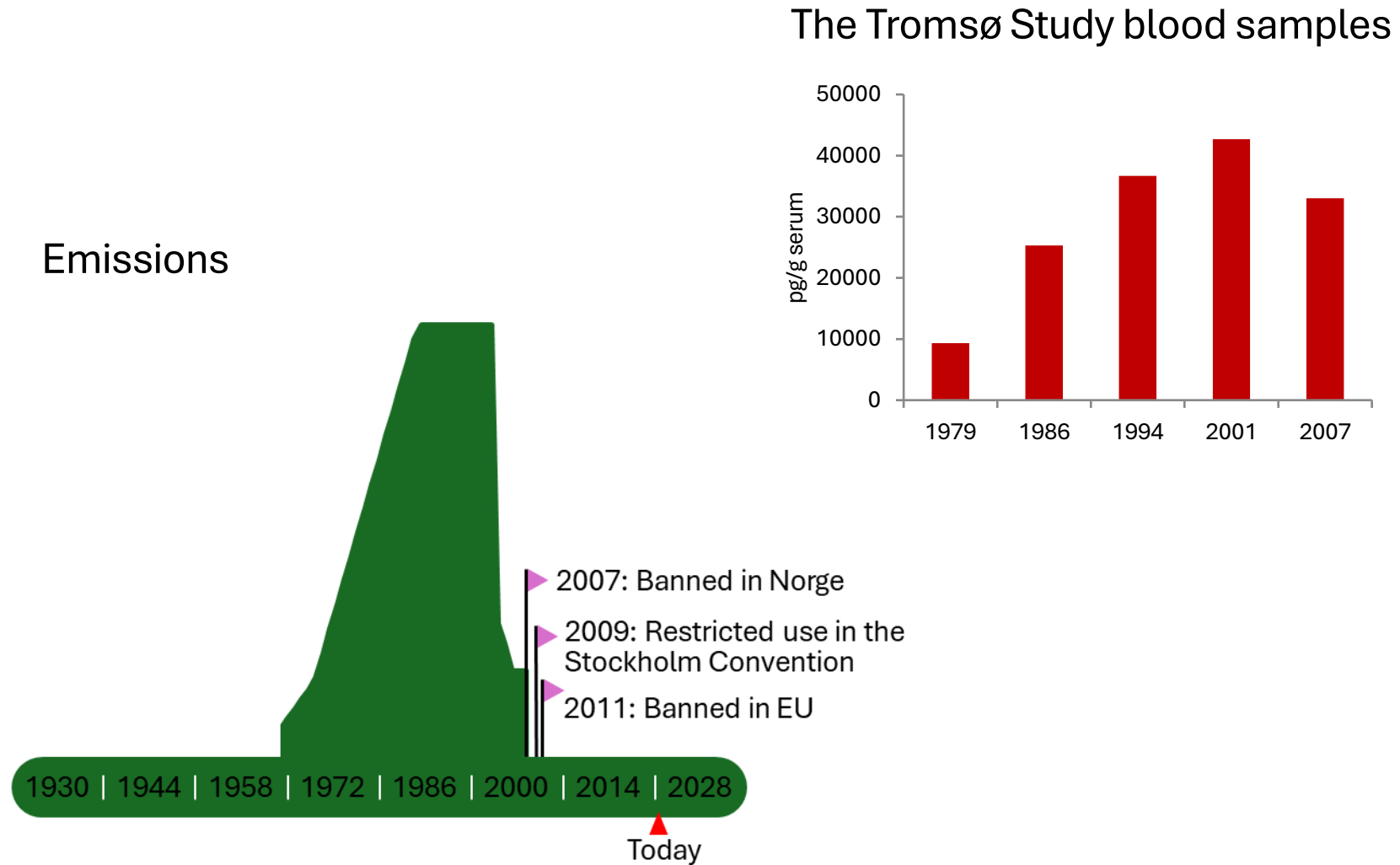


The Tromsø Study blood samples



Good news also about more recent sins

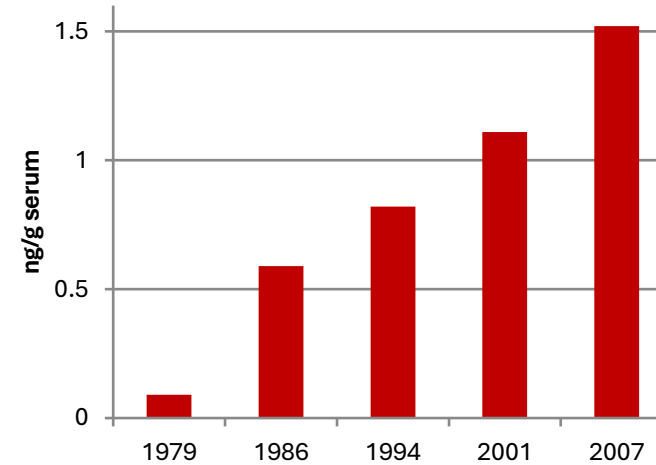
- PFOS



Not good news for newer sins

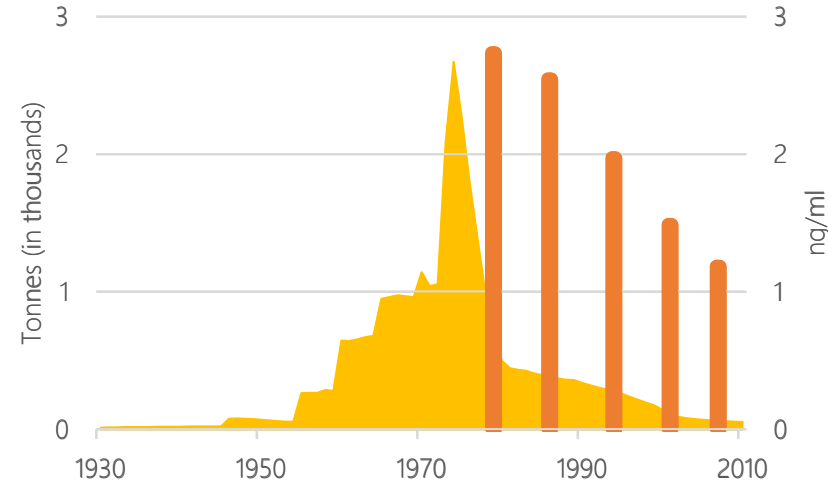
- PFNA
- Emission history not quantified
- Not banned at the time

The Tromsø Study blood samples

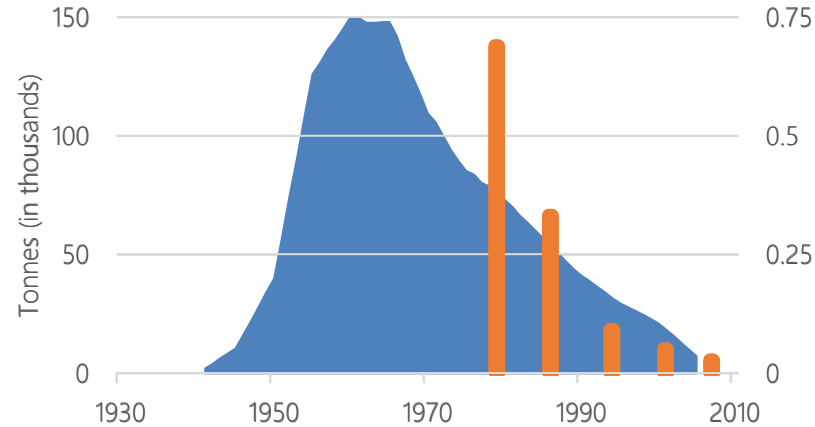


Dynamic and complex burden reflects trends in emissions?

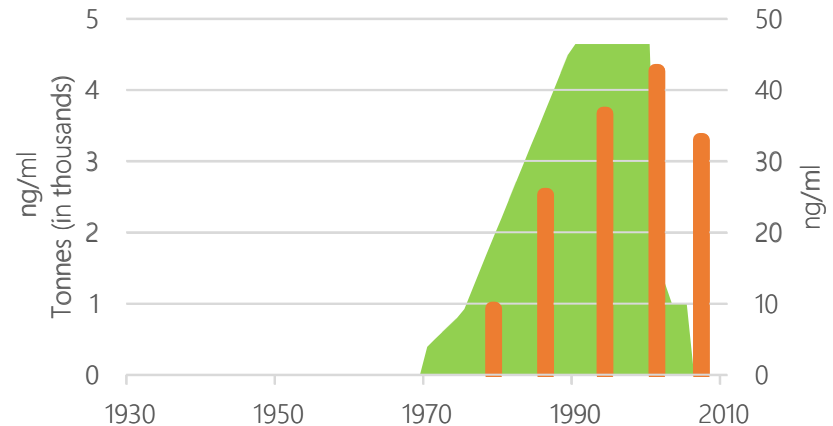
PCB-153



DDT

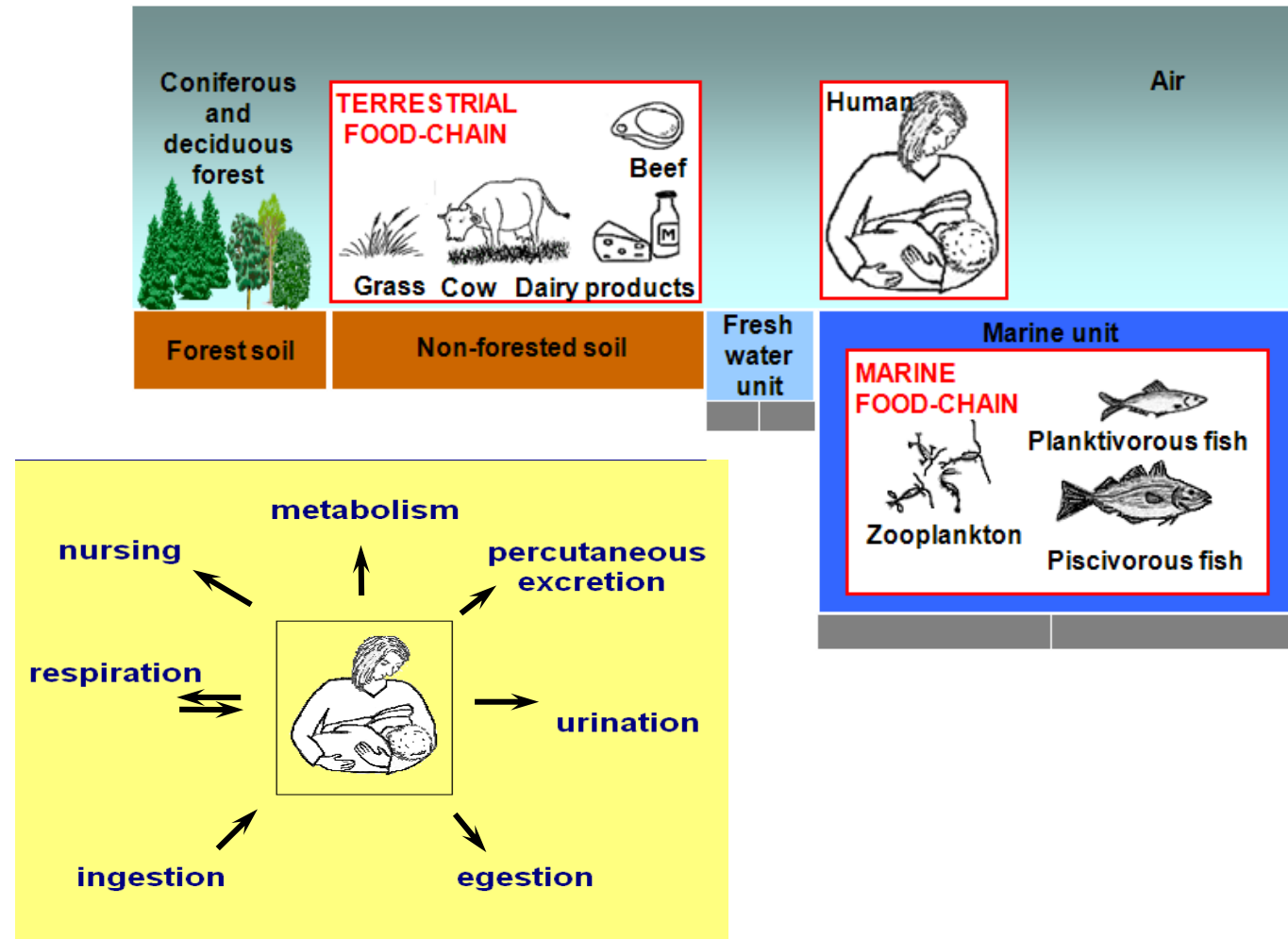


PFOS

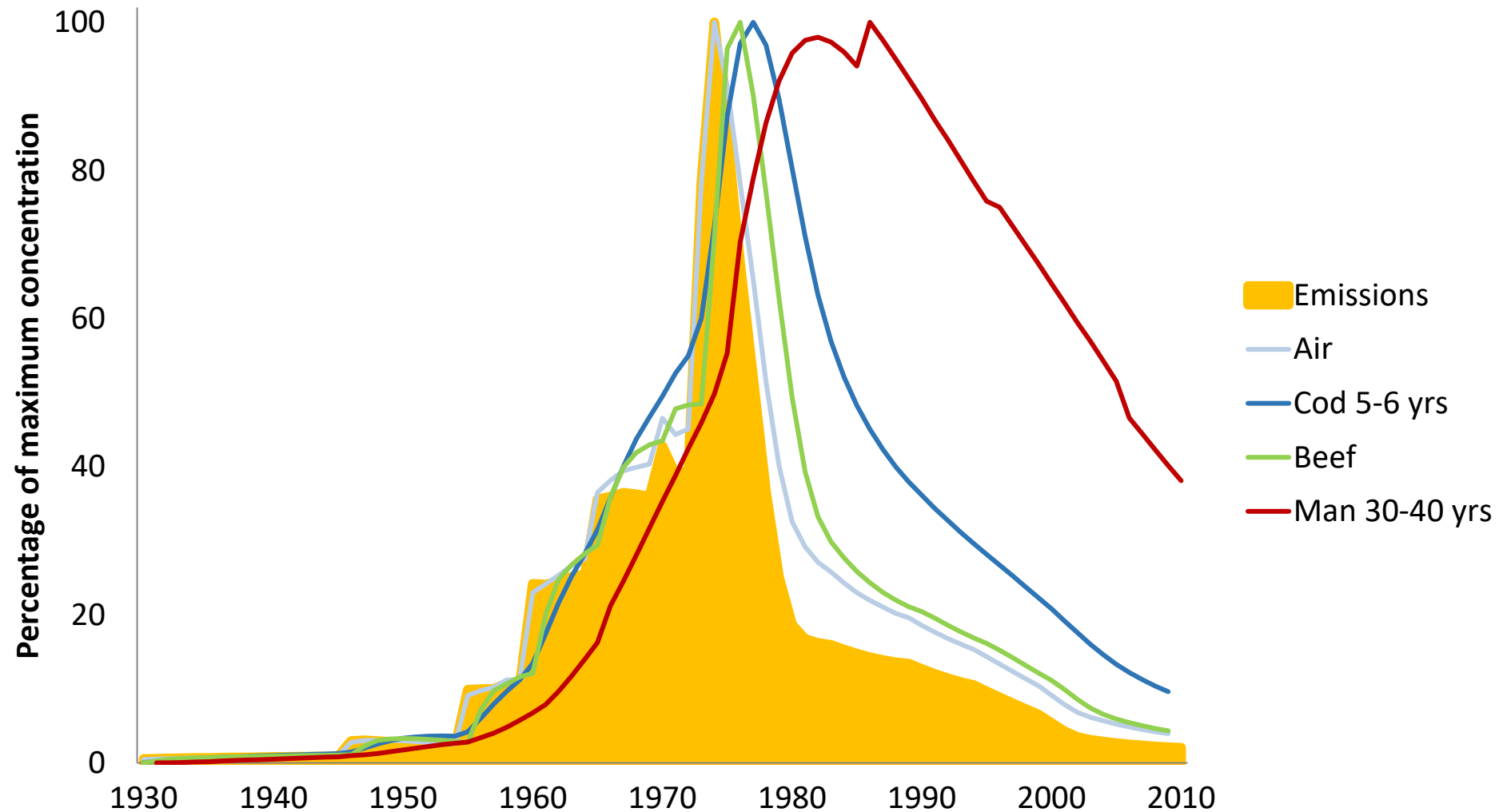


Emission-based modelling

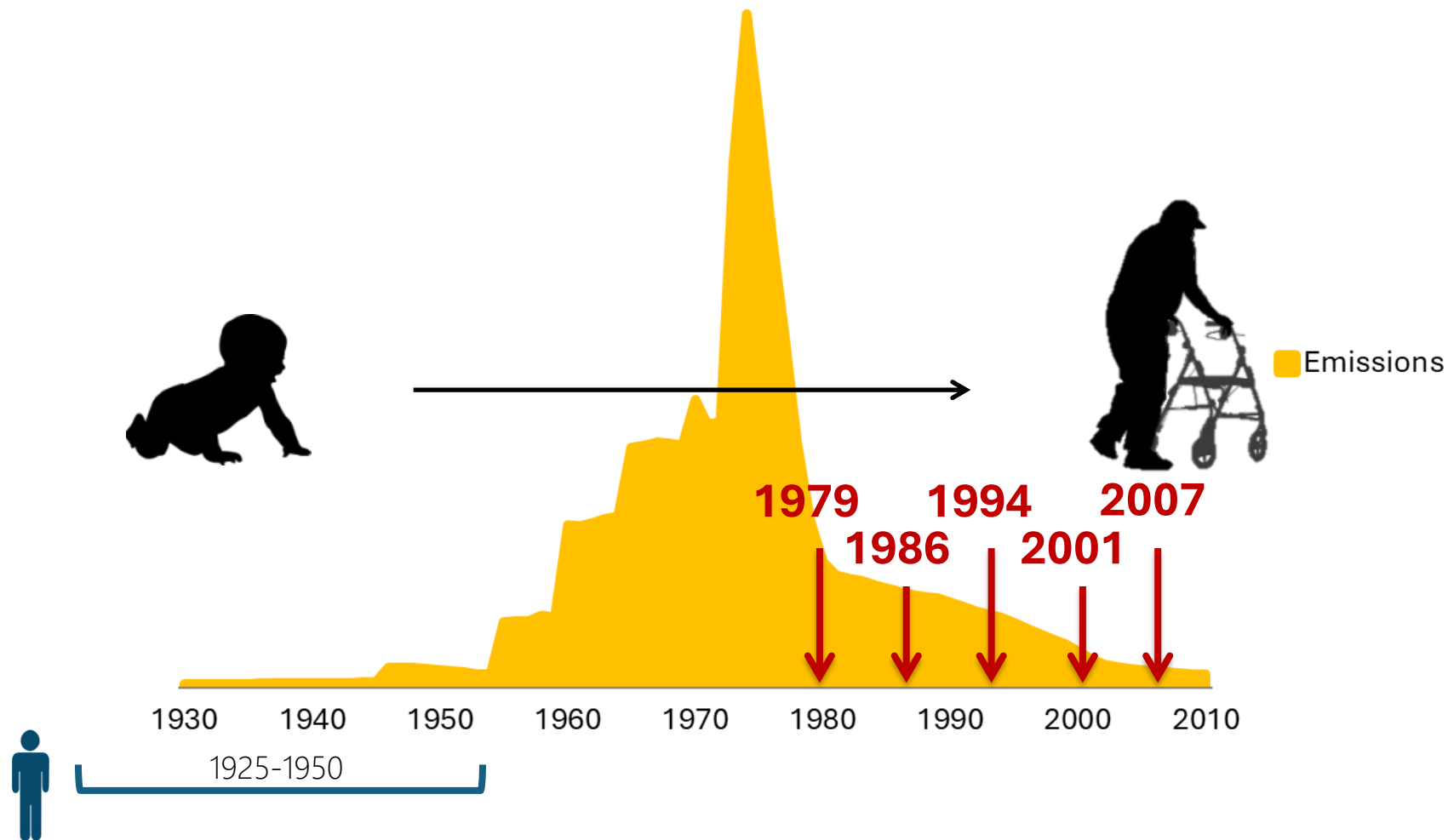
- CoZMoMAN, NILU
- Inventories of global emissions



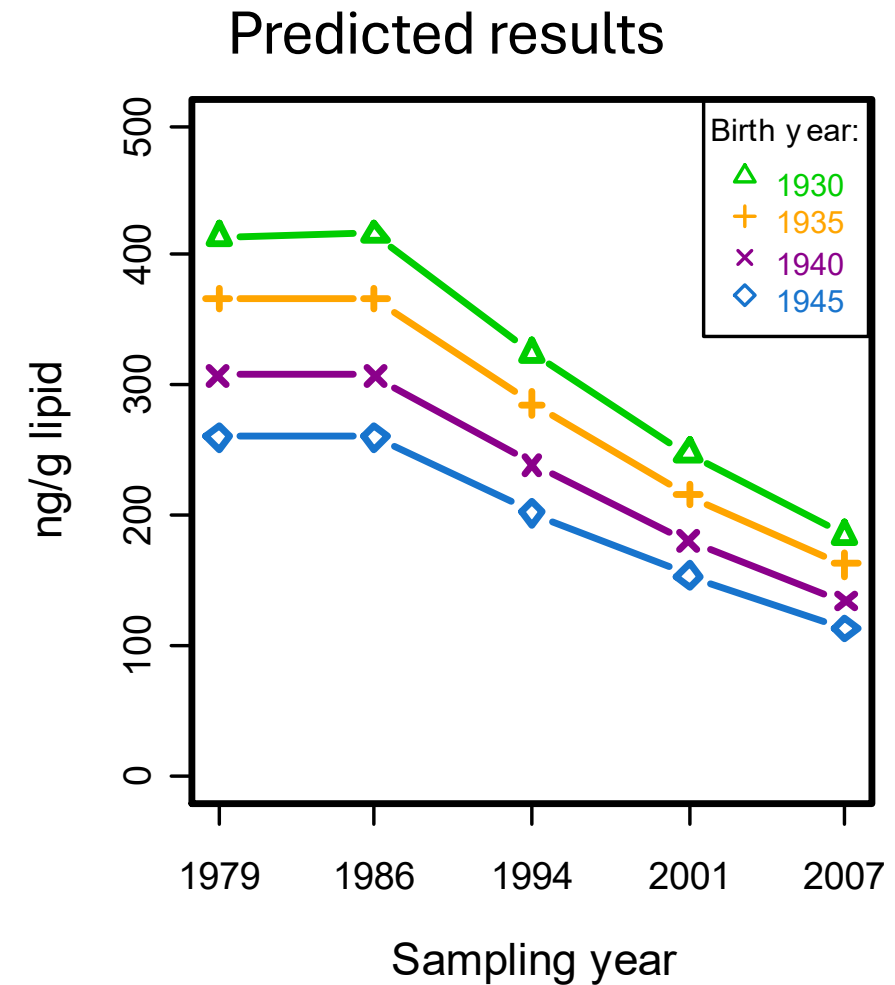
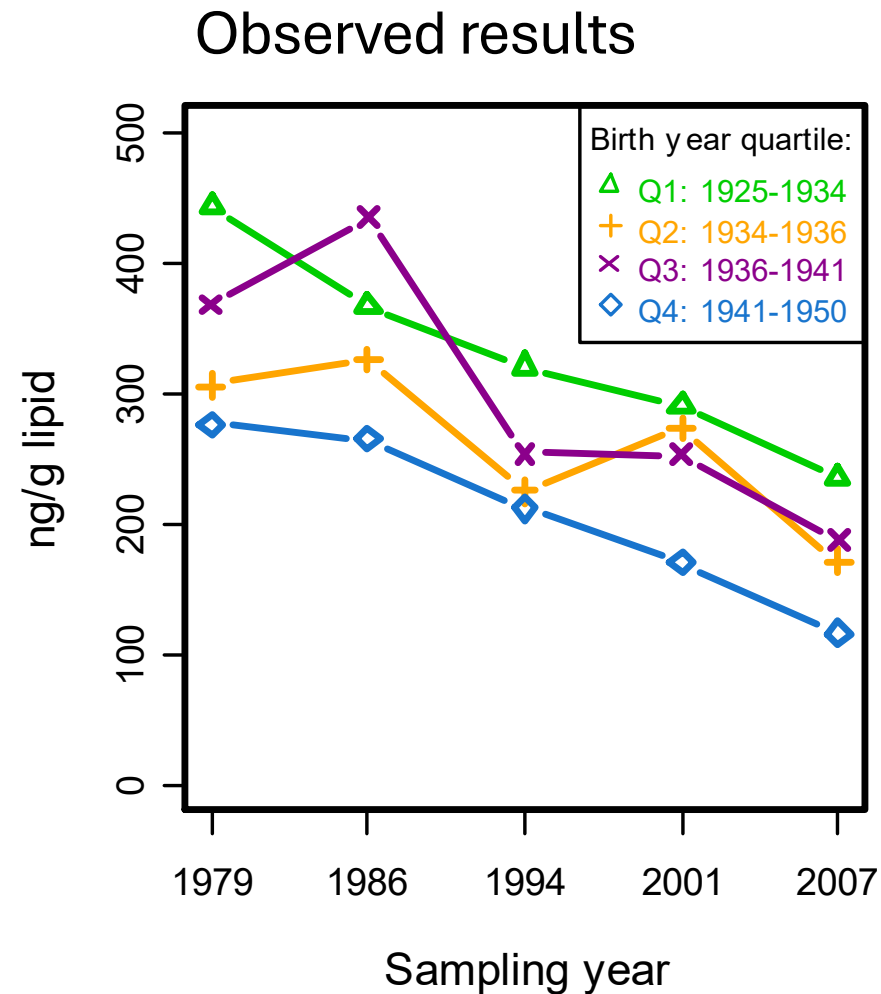
Environmental concentrations follow emissions



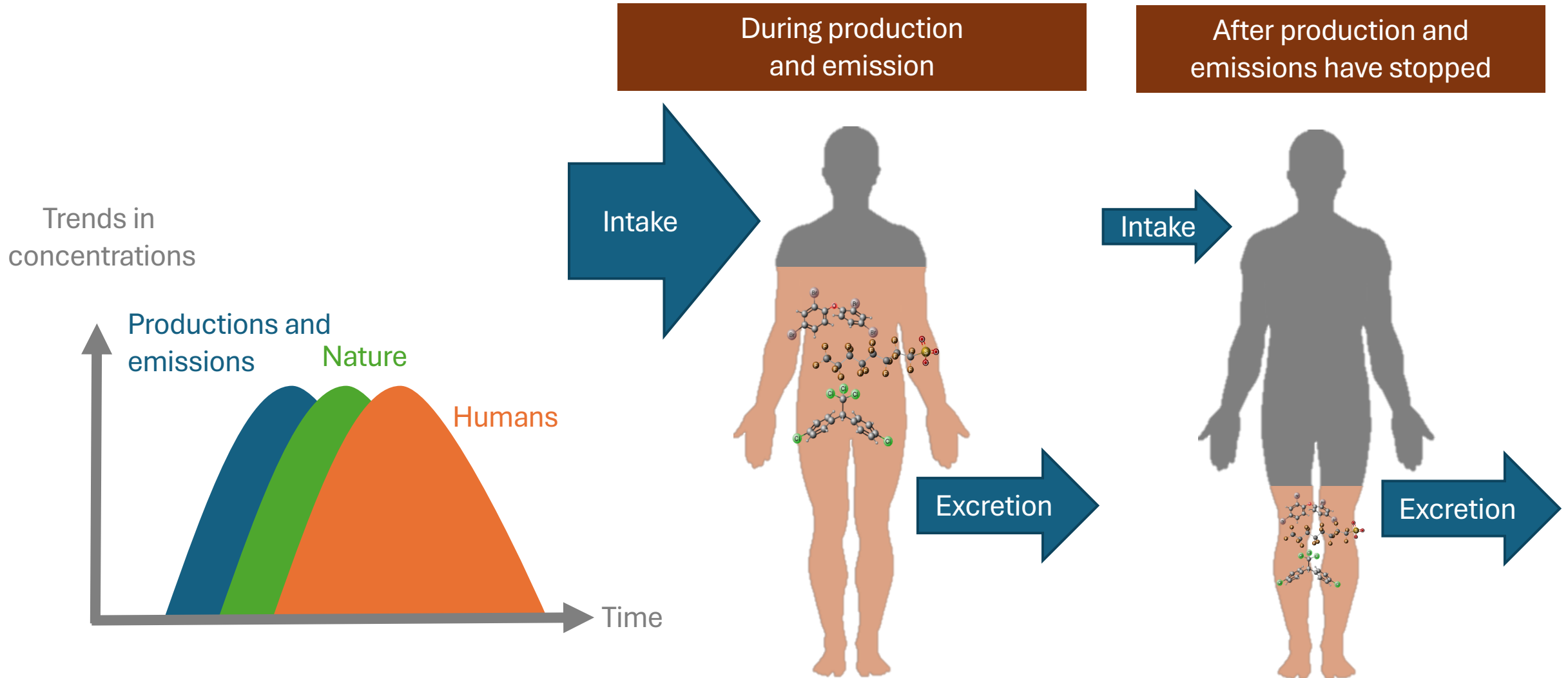
Time trends, lifetime exposure and age



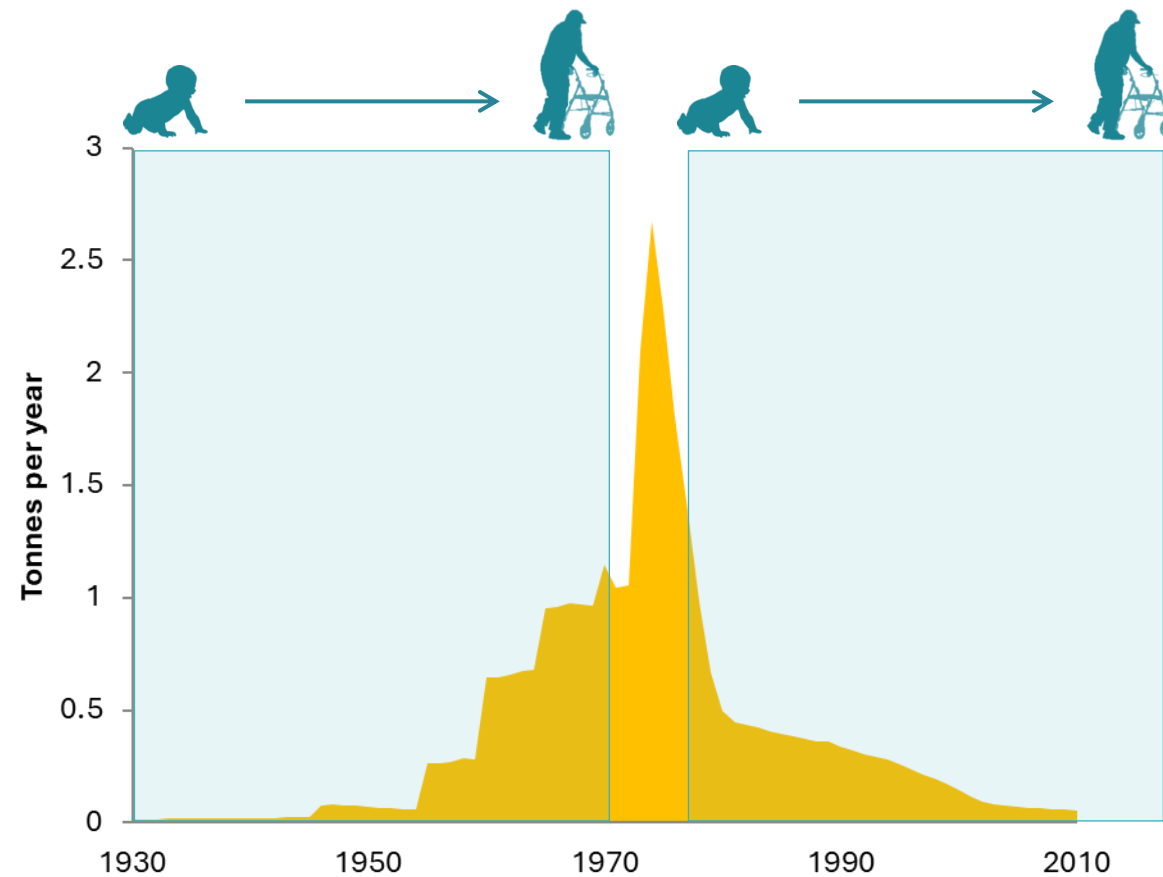
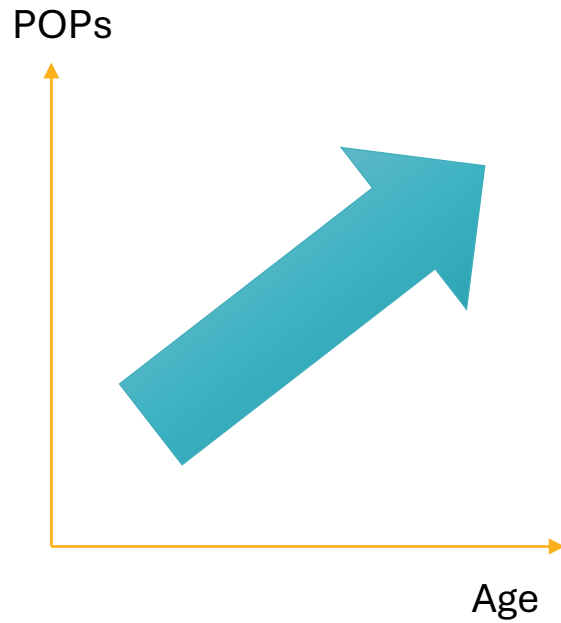
Model predictions for the Tromsø men



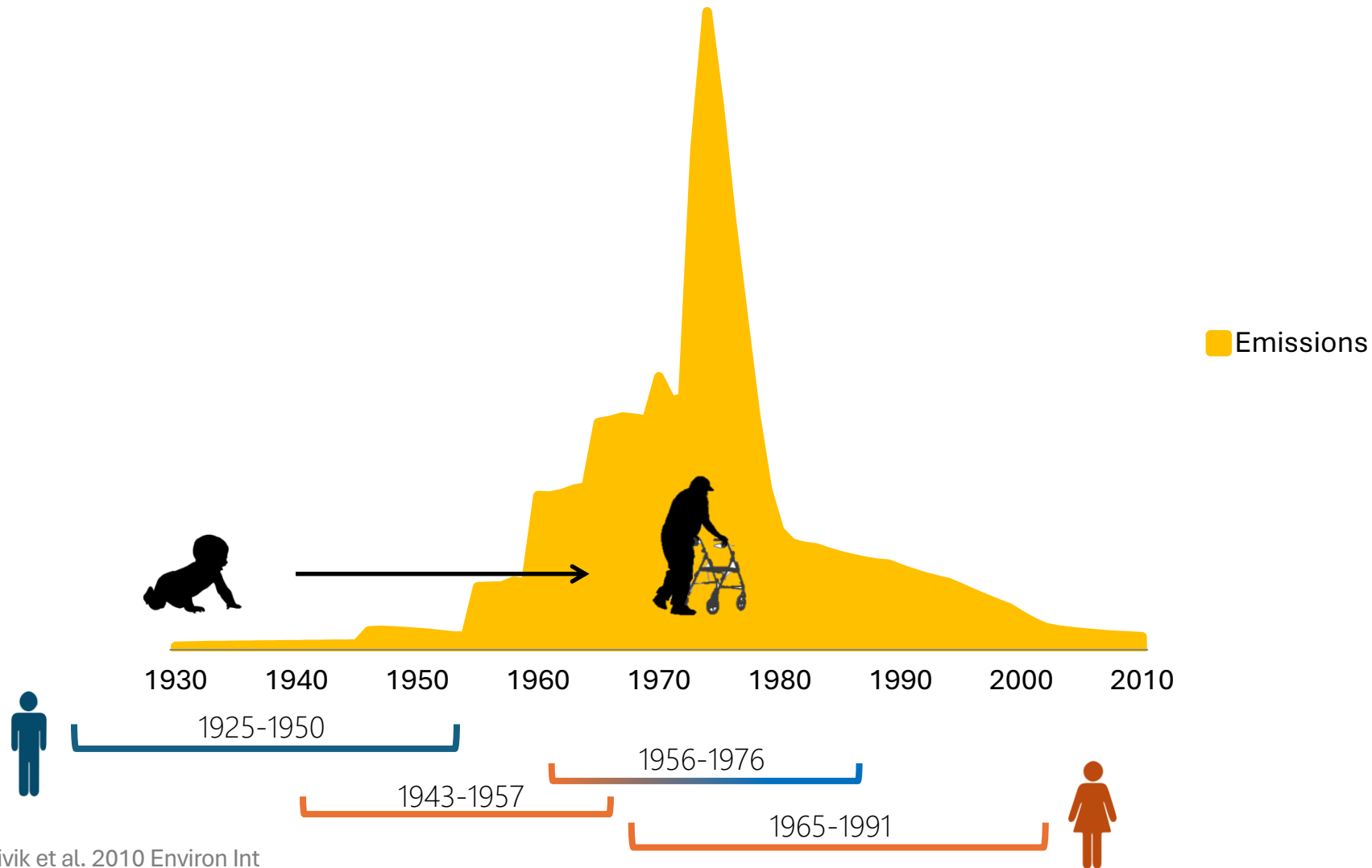
Human burden reflects trends in emissions



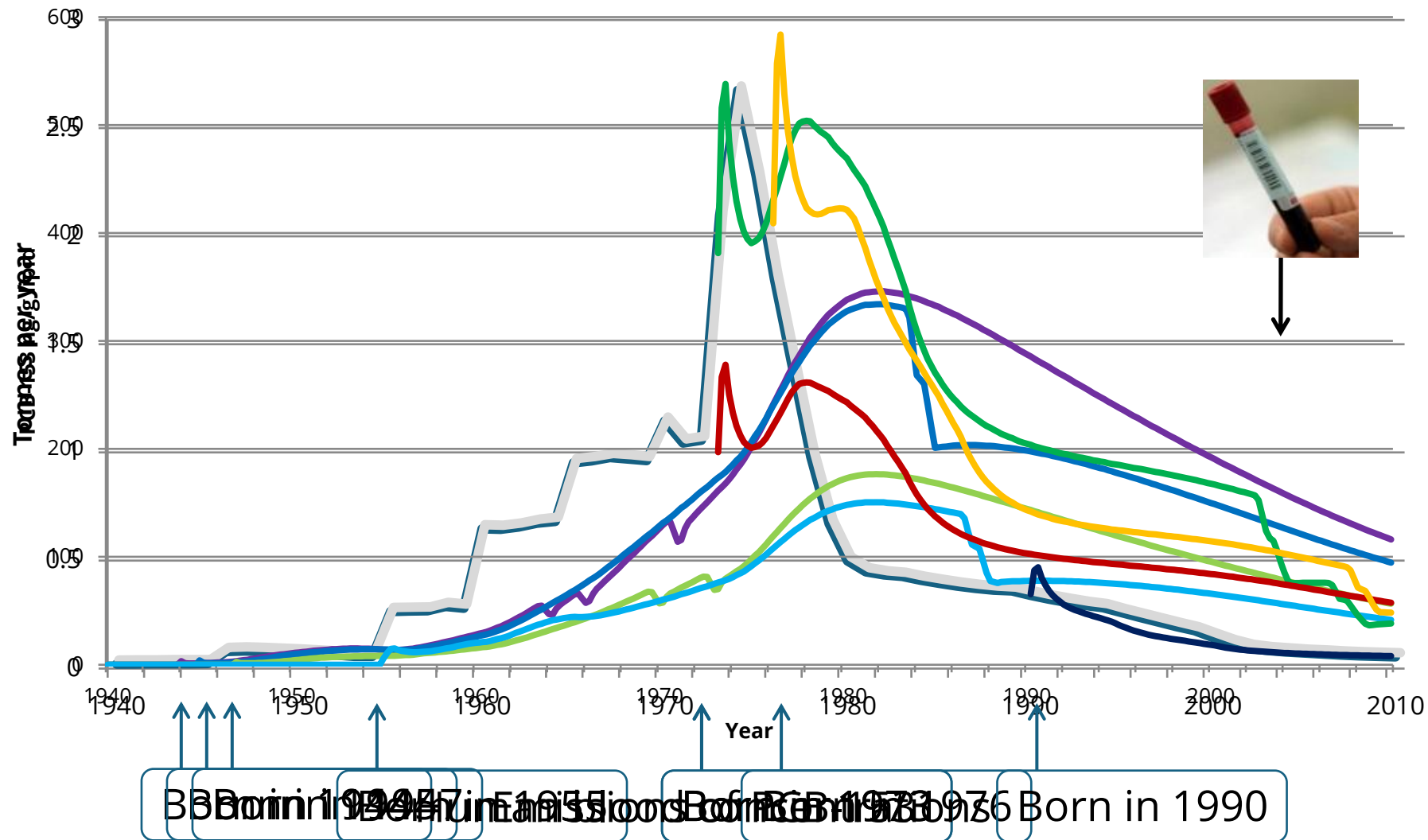
Time trends, lifetime exposure and age



Time trends, lifetime exposure and age

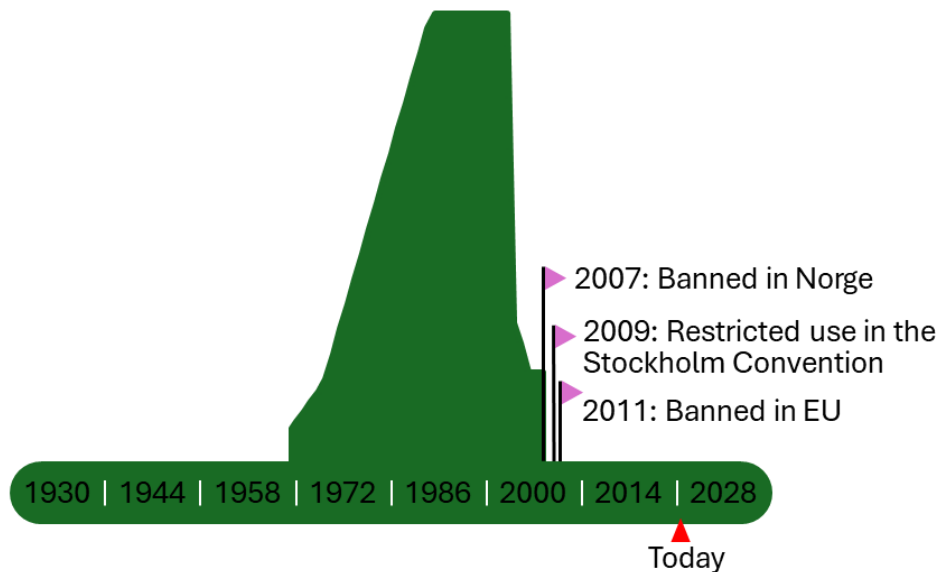


Time trends, lifetime exposure and age

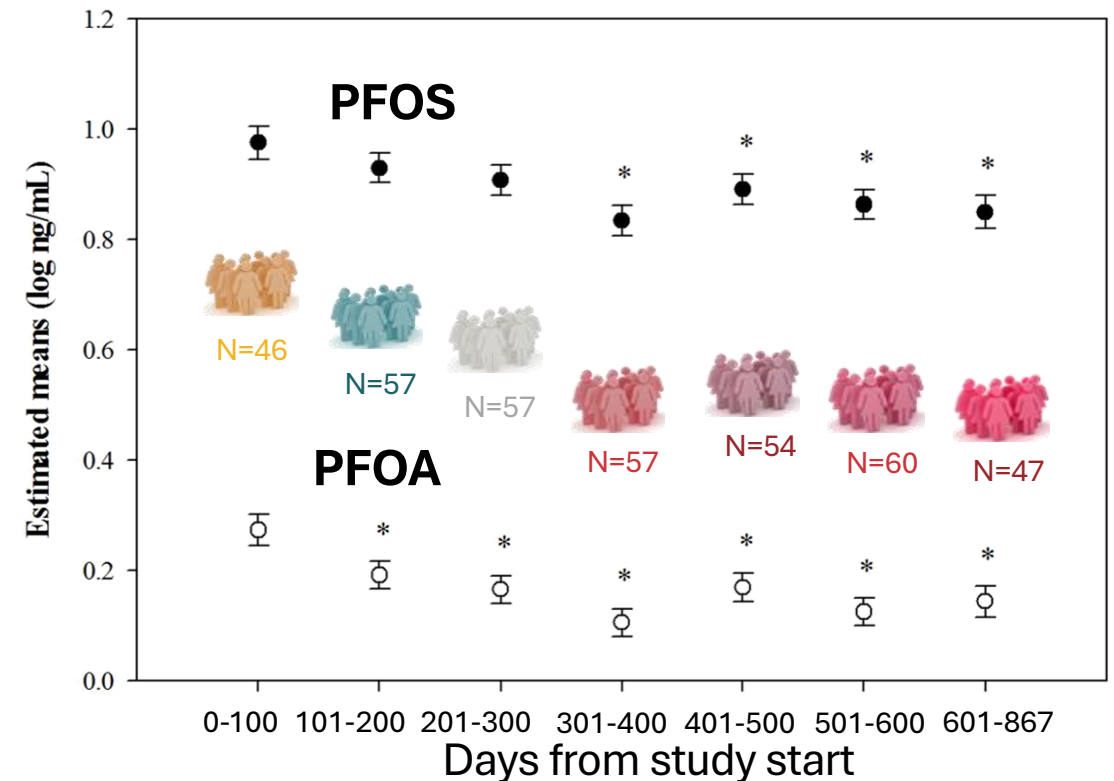


Time trends in cross-sectional studies?

- The Northern Norway mother and child contaminant cohort study
 - Recruitment period: 2007-2009
 - 2nd trimester (n=391)
 - Blood sampling period: 2.3 years

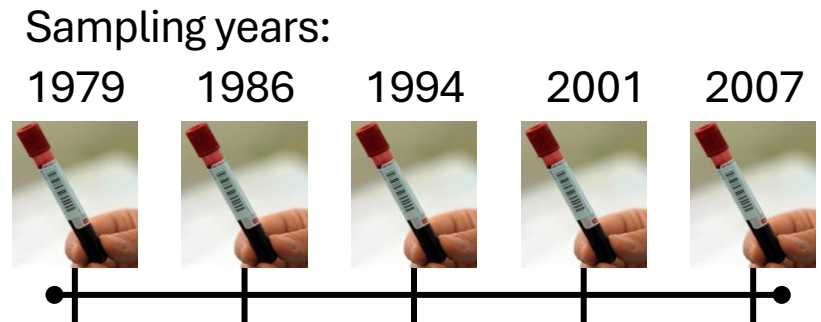


Berg et al. 2014 Env Int / Illustration by Vivian Berg, UiT.



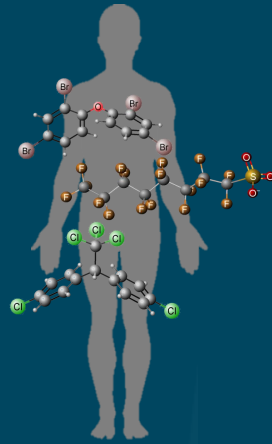
Monitoring and study designs

- Components of time in longitudinal and cross-sectional studies



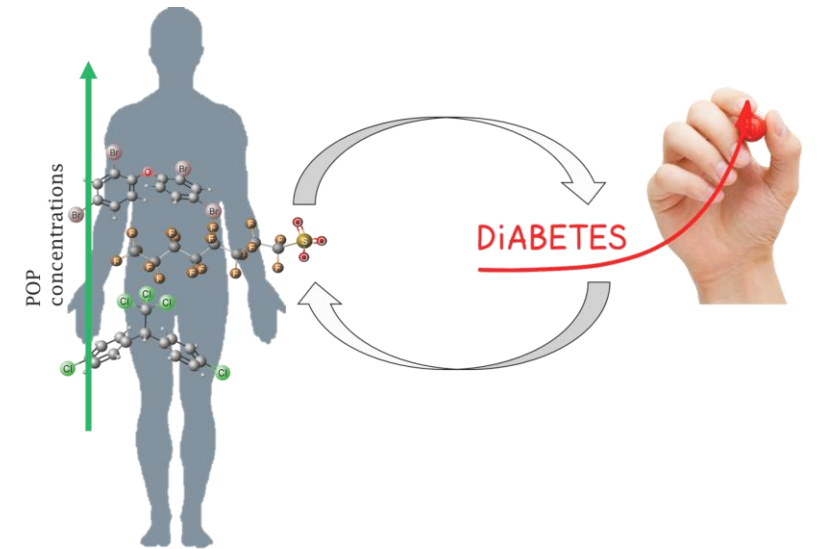
- **Emissions** are time-variant and compound-specific

Human health effects of environmental contaminants



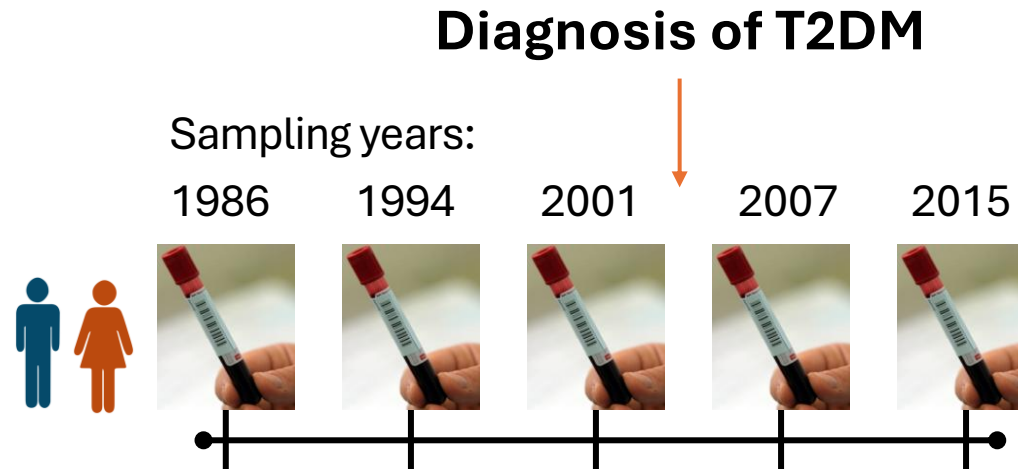
T2DM studies in the Tromsø Study

- An example of research on the relation of POP exposure and risk of Type 2 Diabetes Mellitus (T2DM)
- Rapid increase in T2DM and obesity globally
- T2DM prevalence in Norway: 4.8%
- Strong effect sizes related to POPs in previous studies
- Designed longitudinal study using repeated measurements from the same persons

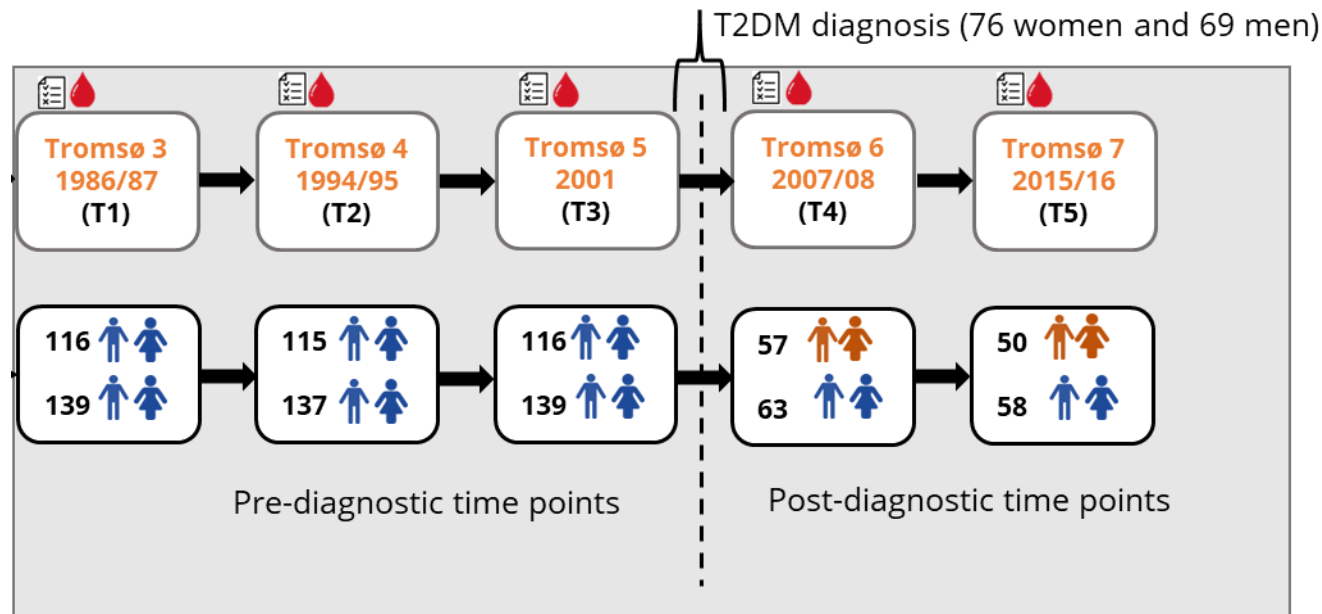


Longitudinal design - T2DM

- A longitudinal, nested case-control study with samples before diagnosis
- Examine the associations with T2DM prospectively and cross-sectionally in the same individuals
- Compare the time trends of PCBs, OCPs, PFAAs, and PBDEs between T2DM cases and controls



Longitudinal design - T2DM

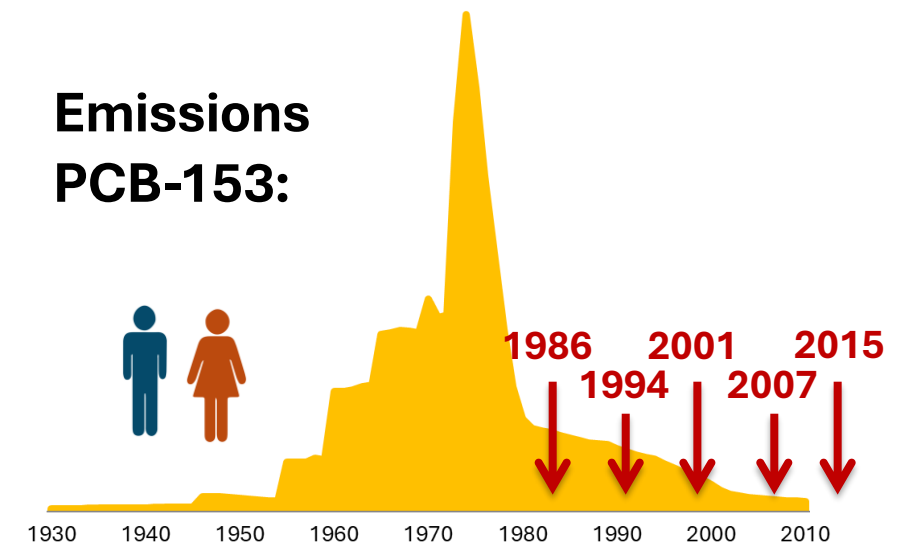


Total serum samples
N=990

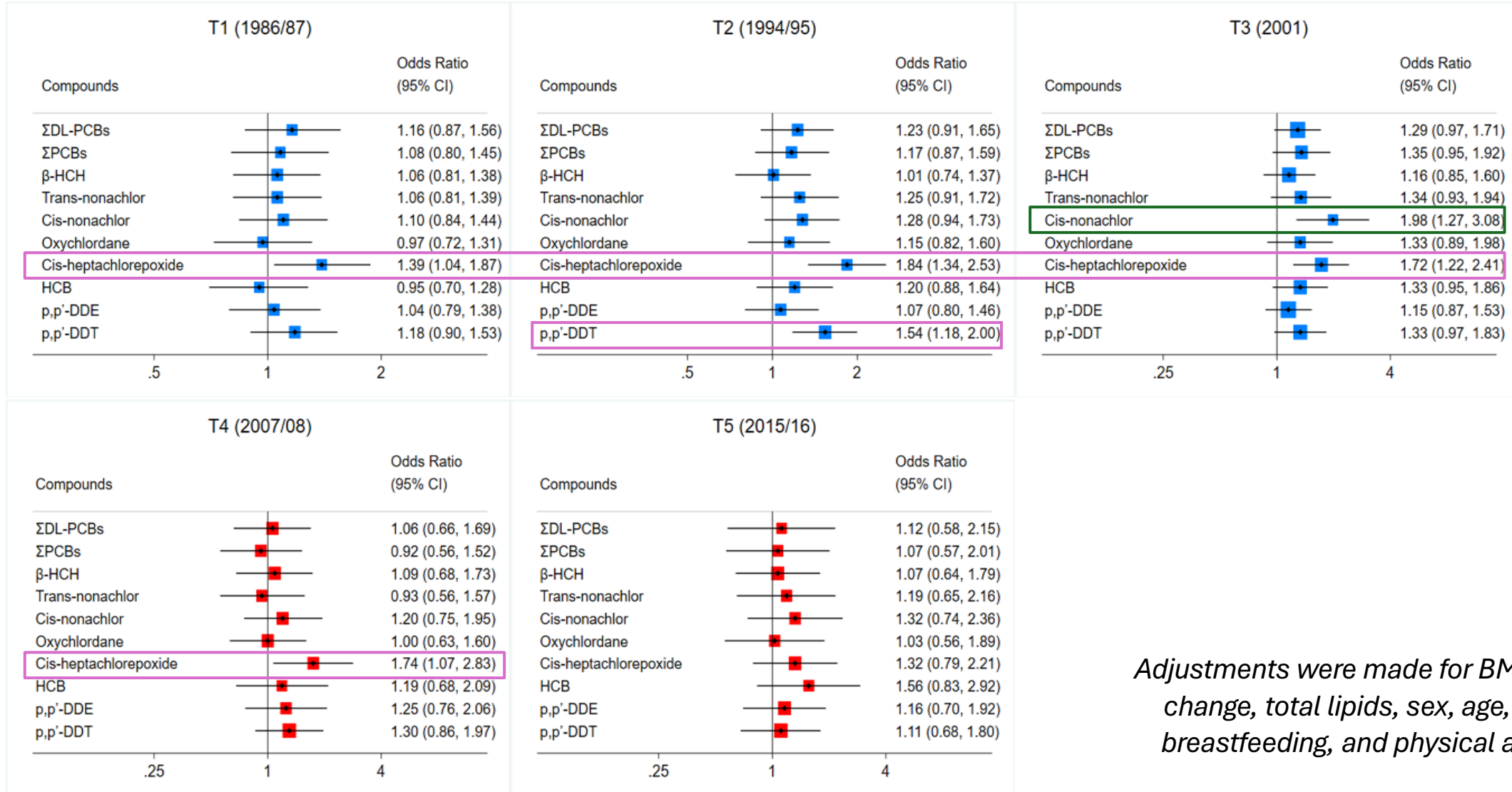
Diabetes status:

- T2DM free
- Diagnosed with T2DM

Emissions
PCB-153:

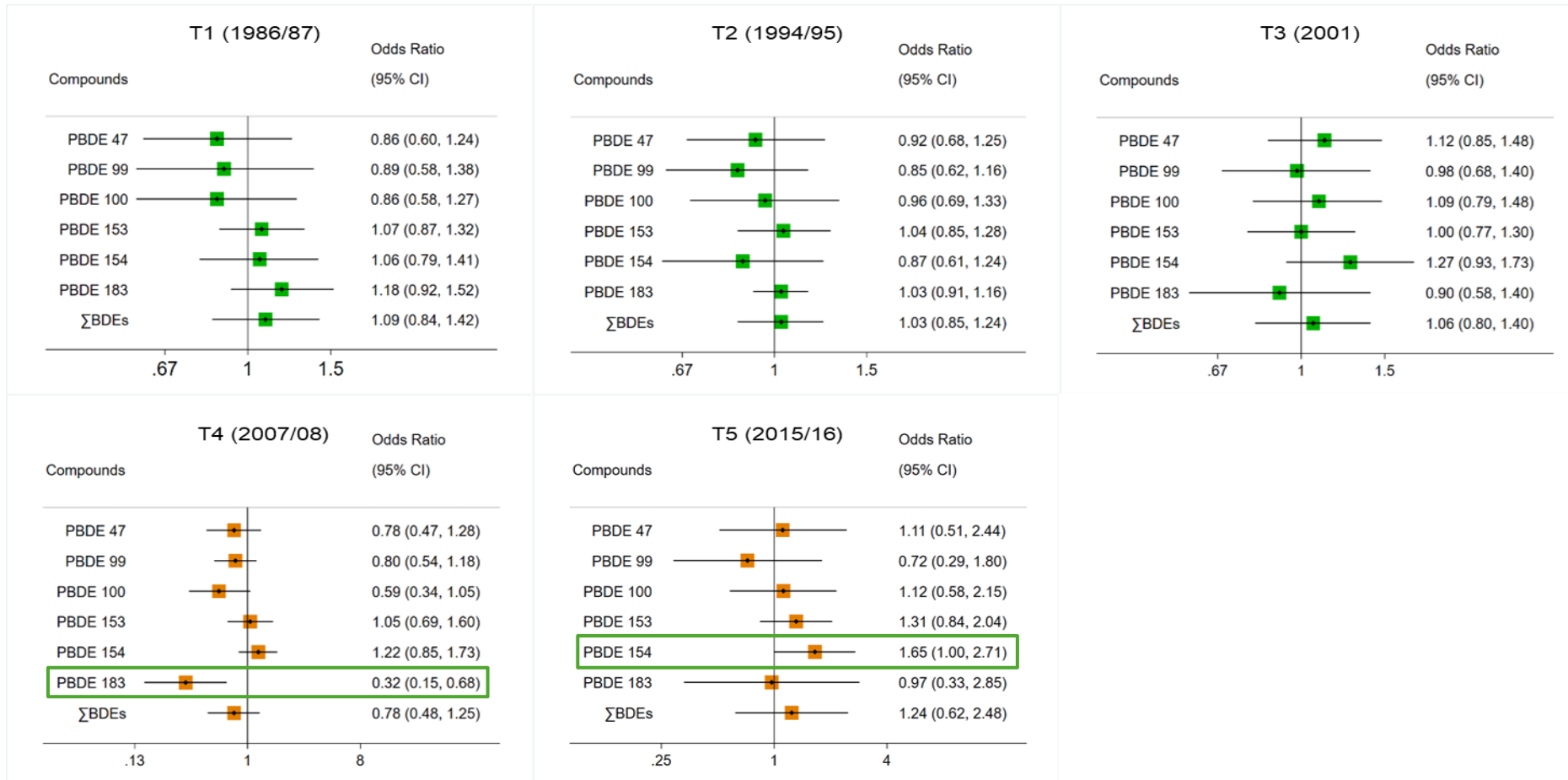


Associations to T2DM

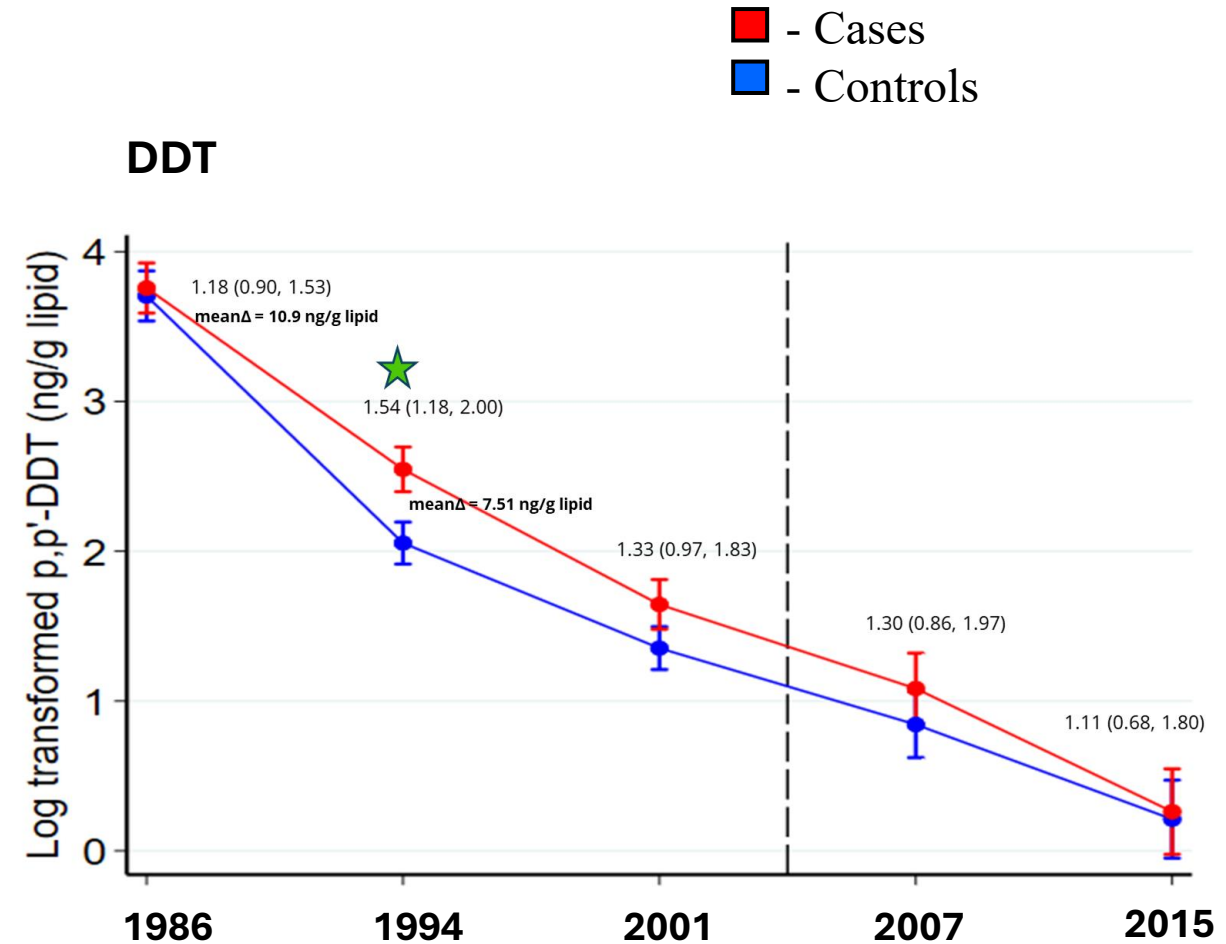
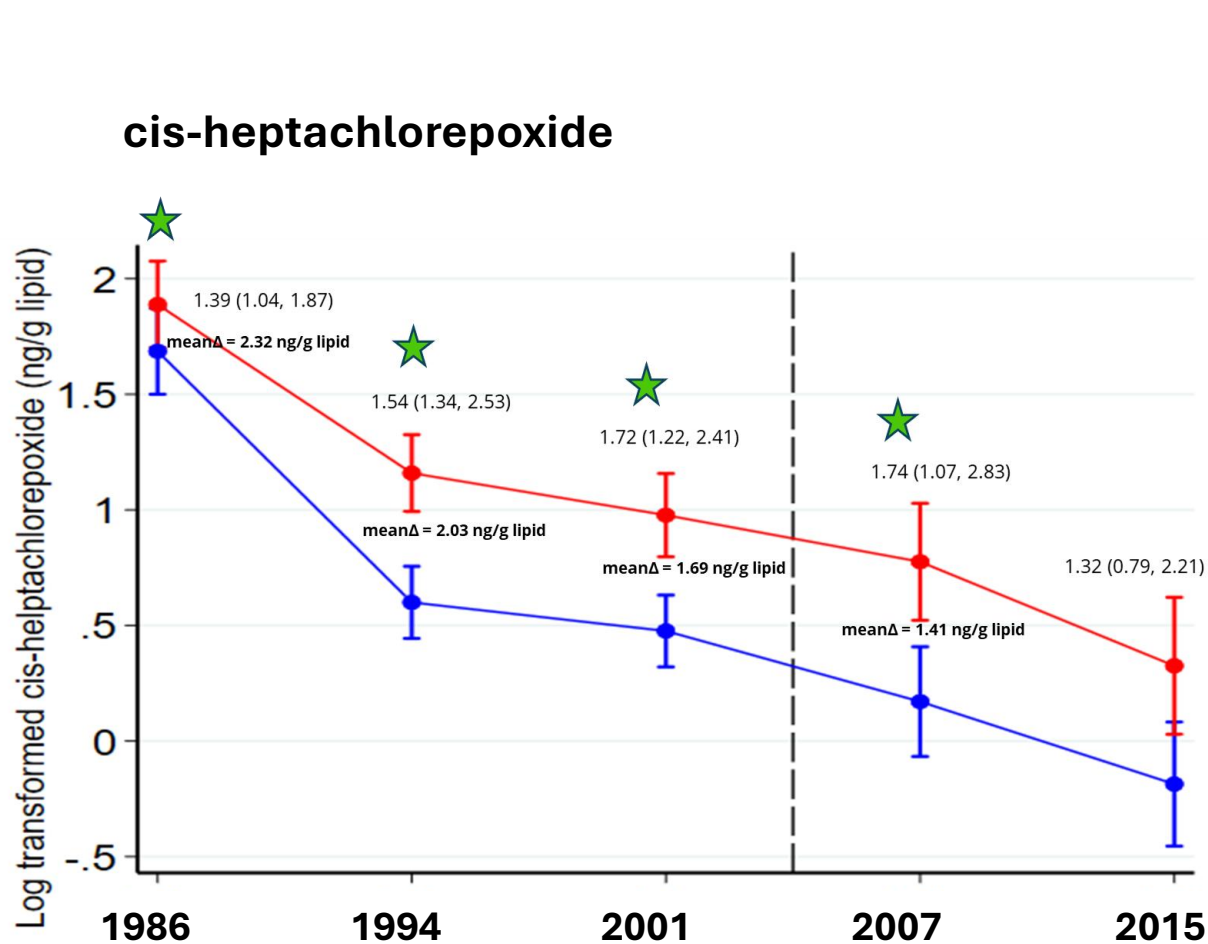


Adjustments were made for BMI, weight change, total lipids, sex, age, parity, breastfeeding, and physical activity.

Associations to T2DM

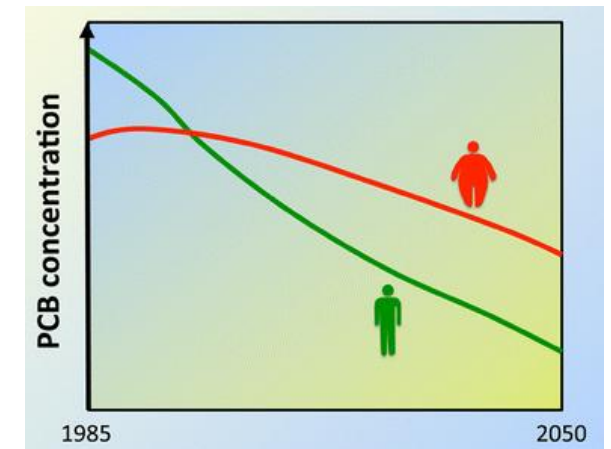
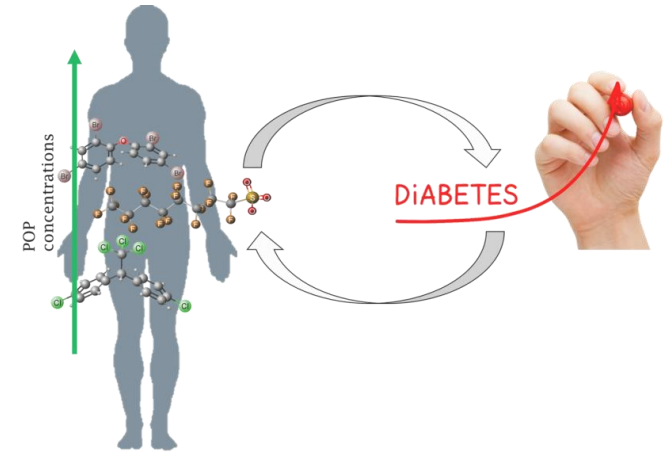


Time trends in cases and controls

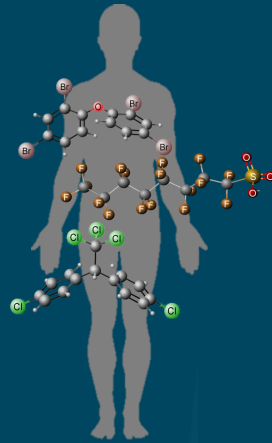


T2DM studies

- Study designs:
 - Cross-sectional study – one time point
 - Longitudinal study – repeated observations
- Up to three measurements before clinical diagnosis of T2DM in cases.
- Consistently strong associations between cis-heptachlor epoxide and T2DM
 - Higher in cases compared to controls and slower declines in cases in pre-diagnostic time points

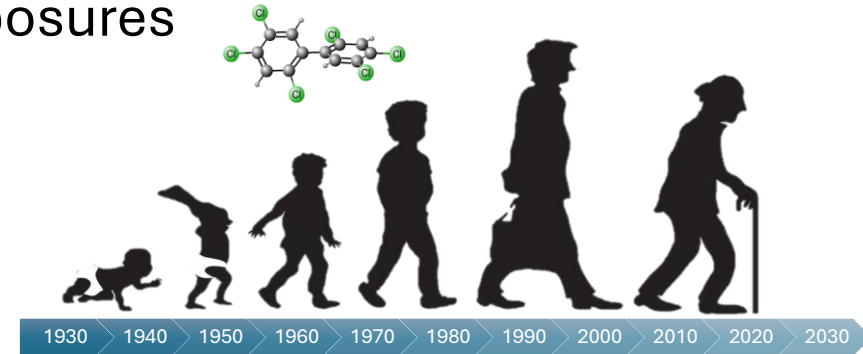


A summary



Investigating human exposures

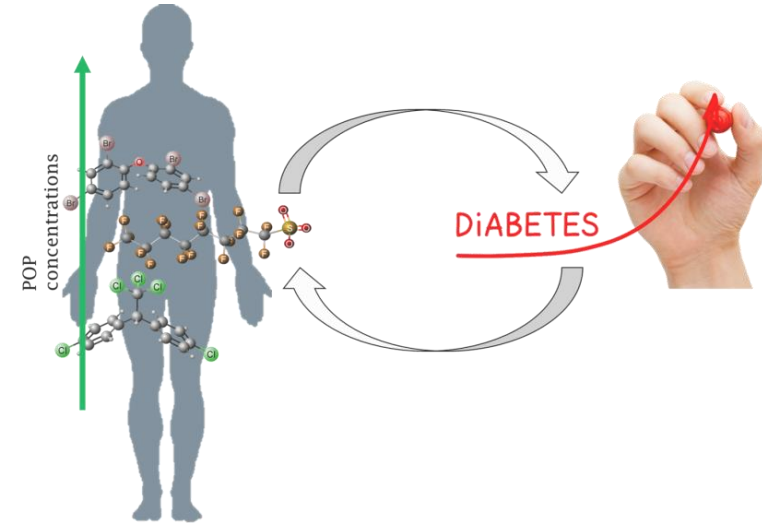
- Aspects of time when investigating human long-term exposures
 - Sampling year and emissions
 - Birth cohort
 - Age (inter-individually and intra-individually)



- Longitudinal studies: repeated measurements, long study period
- Cross-sectional studies: good range of birth years
- How much of lifetime exposure is reflected in a blood sample depends on compound-specific past emission histories relative to birth years

Effect studies using repeated measurements

- Prospective associations in relation to environmental contaminants have rarely been studied



- PCBs and OCPs - slower declines in T2DM cases, strong positive associations with T2DM for some OCPs
- Slower decline → increased POP concentrations → positive associations?
- Complex health endpoints and causality difficult to evaluate

Acknowledgements

- Other PIs of research projects: Torkjel Manning Sandanger, Charlotta Rylander and Vivian Berg
- Dolley Charles for slides from her work
- Many co-authors
- Study participants from Tromsø Study and the NOWAC Study
- Funders: EU 7th FW, Helse Nord, the Fram Centre, Odd Berg Medical Research Fund, the Tromsø Study, Department of Laboratory Medicine at UNN
- Illustrations cited or from Mostphotos.com



Fram Centre

nilu





UiT The Arctic University of Norway

Thank you for your attention!

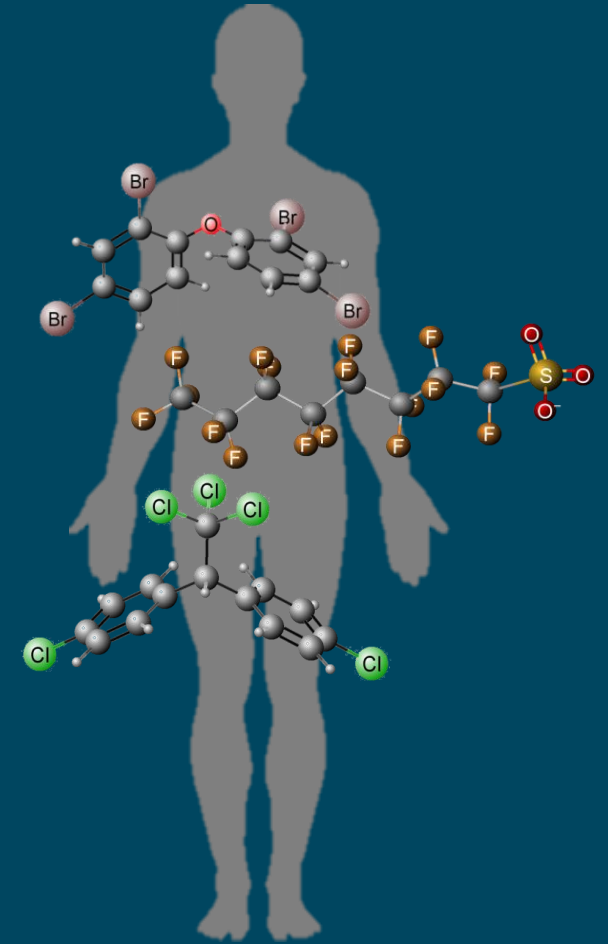
Therese Haugdahl Nøst, PhD

Associate Professor, Department for Community Medicine,

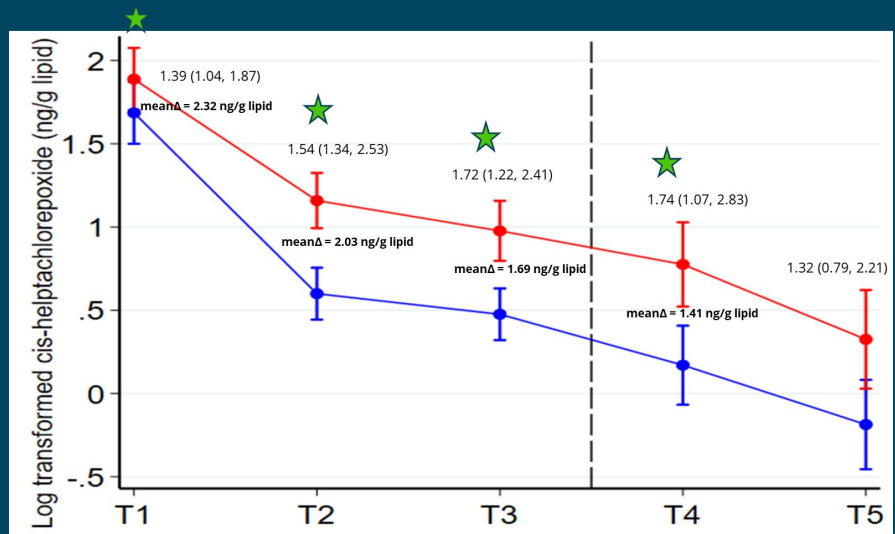
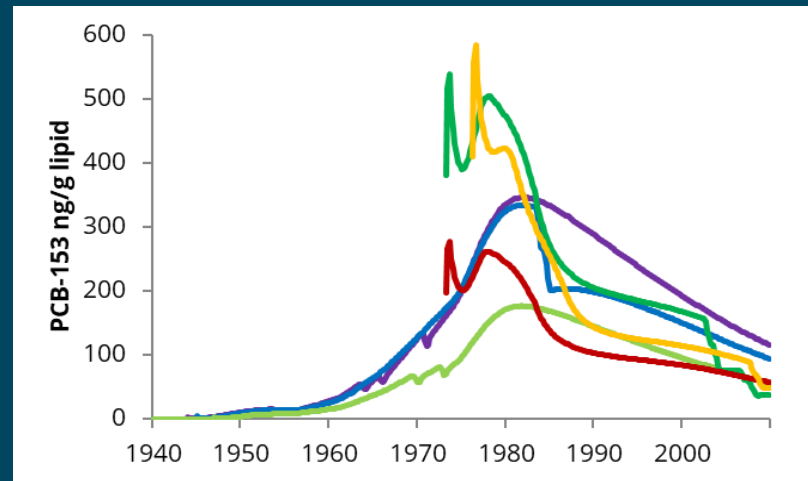
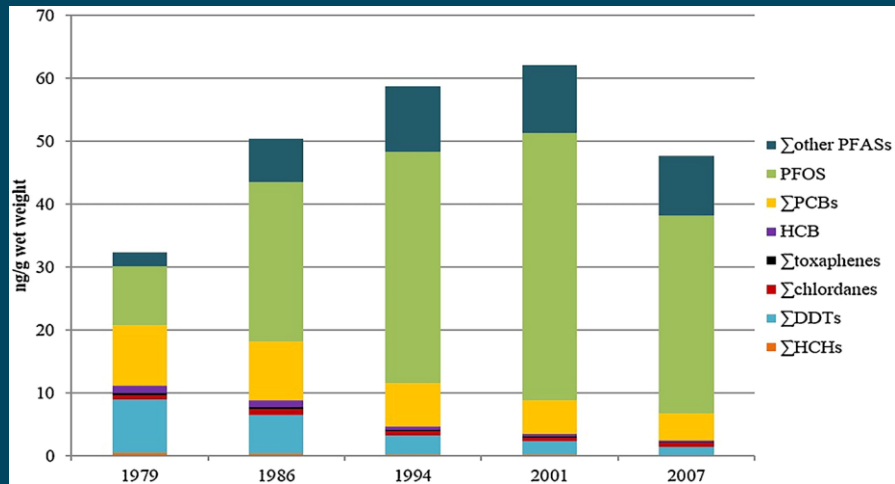
UiT The Arctic University of Norway

Researcher, HUNT Research Centre,

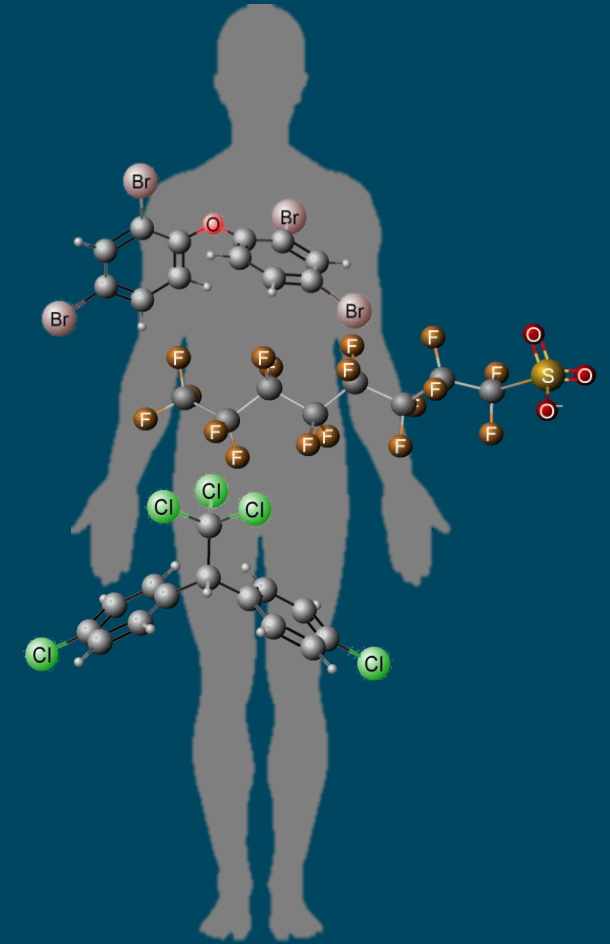
NTNU Norwegian University of Science and Technology



Take home visuals:



Questions?
therese.h.nost@uit.no



Forever Chemicals in Occupational and Environmental Contexts

Marcin Wojewodzic, PhD
Senior Researcher

Cancer Registry & Department of Chemical Toxicology
Norwegian Institute of Public Health

Oslo 25.09.2025

Marcin.Wojewodzic@fhi.no



Cancer Registry





DDT



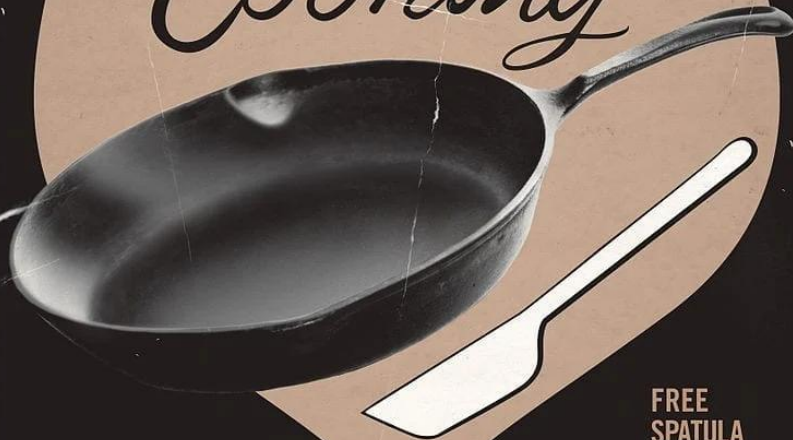
<https://drapiezniki.pl>





AMAZING NEW
CONCEPT IN

Cooking



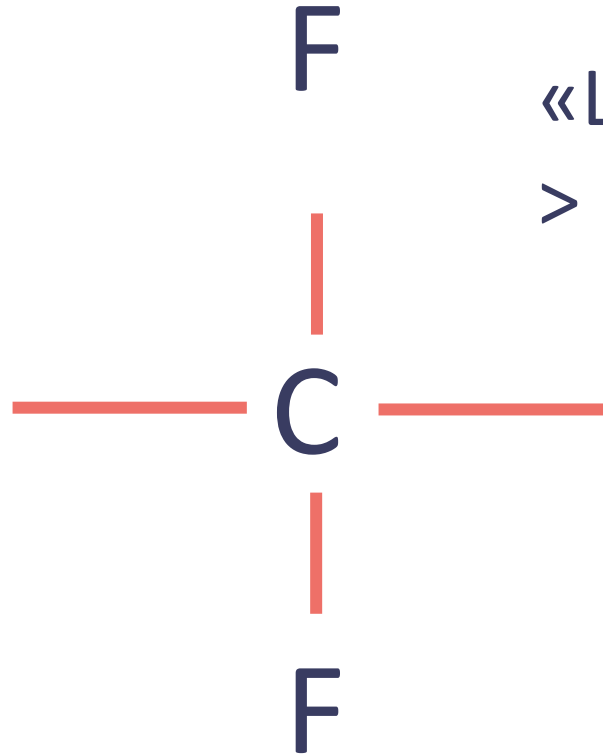
FREE
SPATULA
WITH EACH
"HAPPY PAN"

NOTHING STICKS TO
"HAPPY PAN"

A cast iron skillet sealed with DuPont TEFLON®

Credit: pickpik

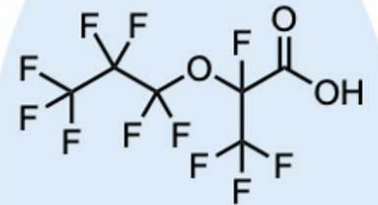
PFAS - Per- og polyfluoroalkyl substances



«Lego» of chemical industry
> 10 000 substances



GenX

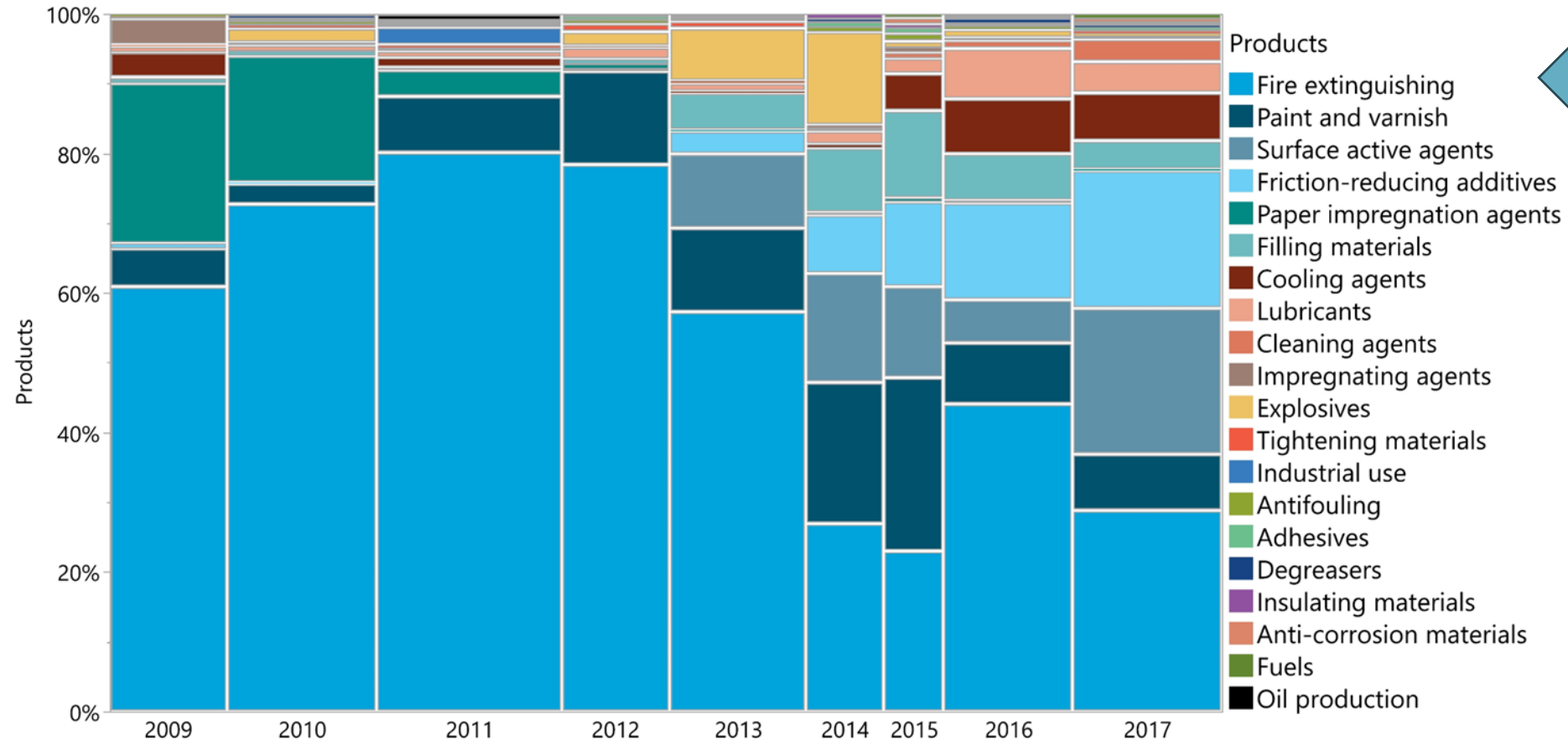


Hexafluoropropylene
oxide dimer acid



Commercial use of PFAS in Norway

PFAS > 10 000 chemicals



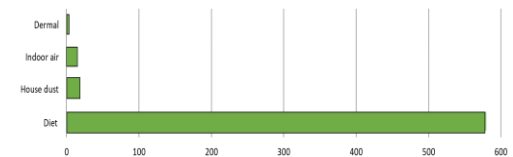
250 tonn

Where can Line find PFAS?



Exposure pathways:

- Dermal
- Inhalation
- **Ingestion**



Poothong et al. *Environment International* 134 (2020) 105244



Line Småstuen Haug

Foundation
Sunscreen
Dental floss
Toothpaste
Contact lenses
Toilet paper

Food
Drinking water
Cookware
Food contact material
Floor polish

Waterproof cloths and shoes
Climbing ropes
Fishing lines
Ski wax

Wax Electronics Interior

PFAS Cycle

PFAS Production/
Using Industries

Household products with PFAS:
fast food wrappers, non-stick cookware,
shampoo, paint, detergent, etc.

Homes & Offices

Landfill

Food Products

Soil / Farmland

Waste Water
Treatment Plant
(WWTP)

Wastewater discharge
to stream

Leachate to
WWTP

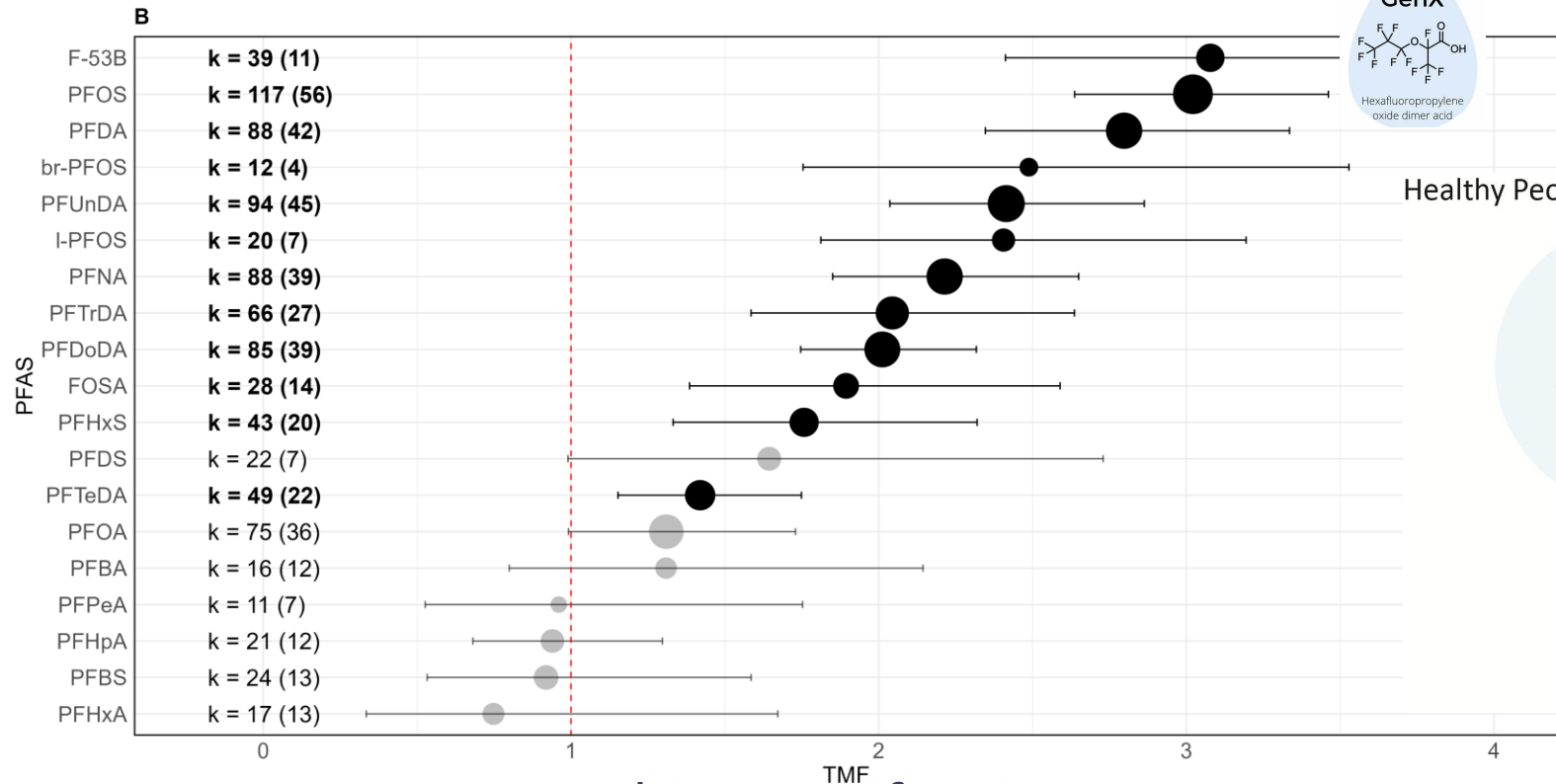
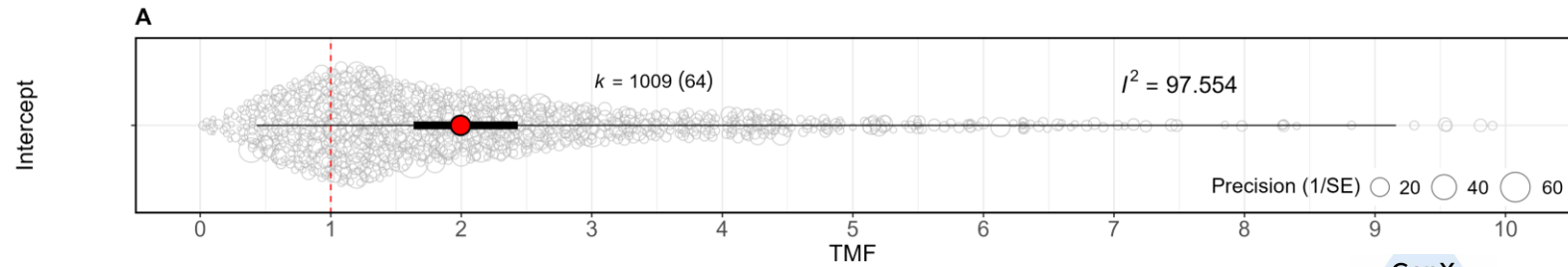
Biosolids

Wastewater discharge
to stream

Firefighting foam



A meta-analysis reveals **PFAS concentrations double with each trophic level** across aquatic and terrestrial food webs



Healthy People

Healthy Environment

The One Health

Healthy Animals



IARC assessment

PFOA: Carcinogenic to humans

PFOS: Possibly carcinogenic to humans



It has not been assessed how much exposure is required to increase the risk



International Agency for Research on Cancer
World Health Organization

IARC MONOGRAPHS VOL. 135
PERFLUOROOCTANOIC ACID (PFOA) AND
PERFLUOROOCTANESULFONIC ACID (PFOS)
(7–14 NOVEMBER 2023)

PFOA

PFOS

IARC GROUP

Group 1
Carcinogenic to humans

Sufficient evidence for cancer in animals and strong mechanistic evidence in exposed humans:

Epigenetics Immunosuppression

Limited evidence for cancer in humans (for renal cell carcinoma and testicular cancer)

Group 2B
Possibly carcinogenic to humans

Strong mechanistic evidence in exposed humans:

Epigenetics Immunosuppression

THE LANCET Oncology
Carbogenicity of perfluorooctanoic acid and perfluorooctanesulfonic acid
Shuo Zhai • Jens Peter Rode • Weizhuo A. Chiu • Jane Hoppe • Jan Kram • Mohamed Abdelhak • et al.
Published: November 20, 2023 • DOI: [https://doi.org/10.1016/S1473-3099\(23\)00002-1](https://doi.org/10.1016/S1473-3099(23)00002-1)



Cancer in Norway: kidney and testis



Figure 9.1-O: Kidney (excl. renal pelvis) (ICD-10 C64)

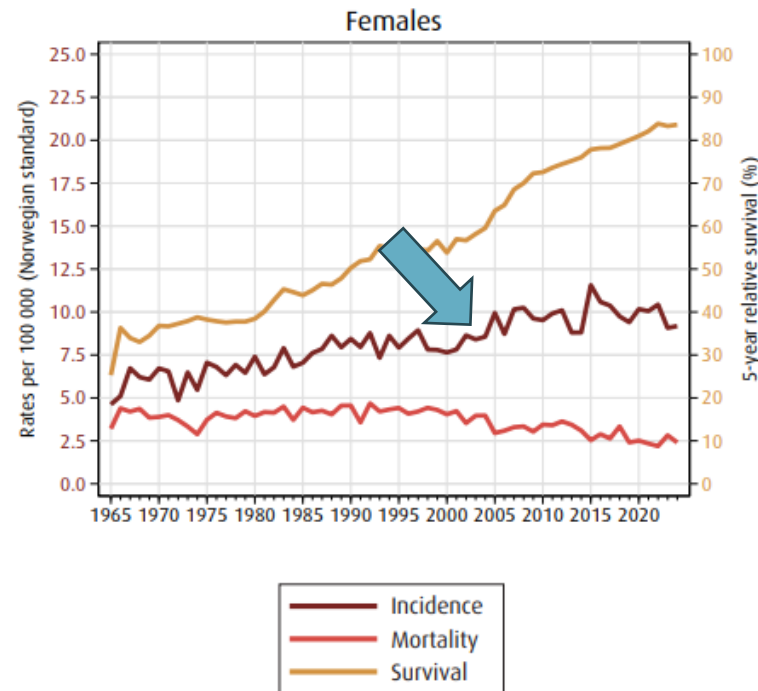
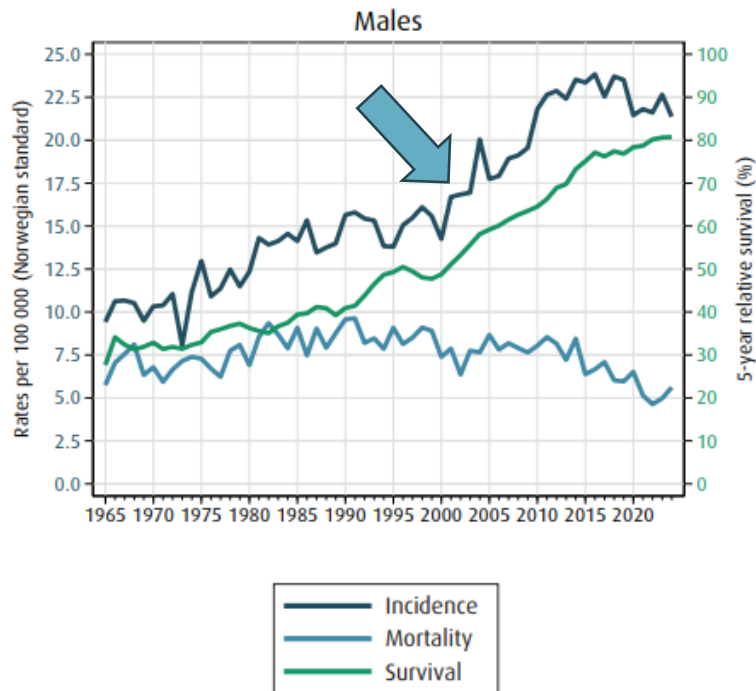
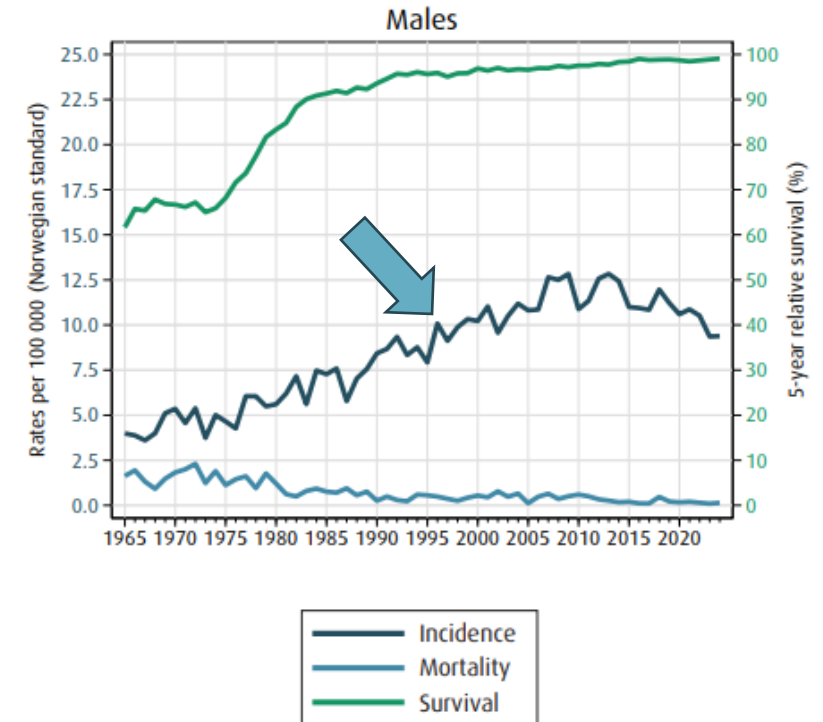


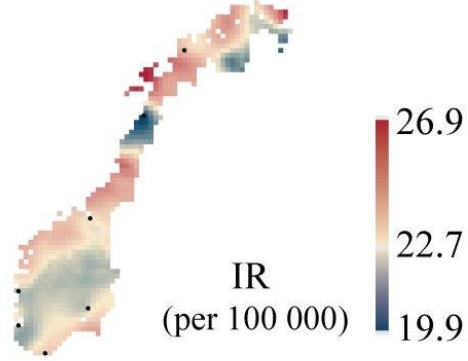
Figure 9.1-T: Testis (ICD-10 C62)





Employing the population-based Janus Serum Bank as 'Time Machine', to explore **kidney** and testicular cancer and their relationship with exposure to **PFAS 'Forever Chemicals'**

A) ICD-10: C64, 2013-2022



B) ICD-10: C62, 2013-2022

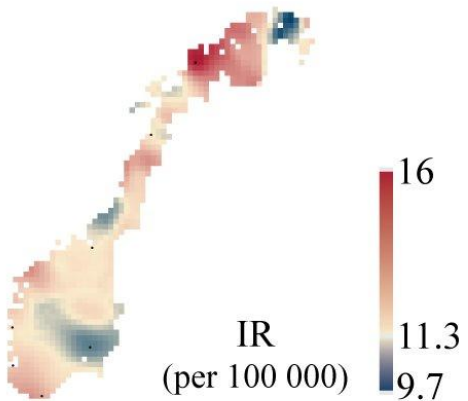
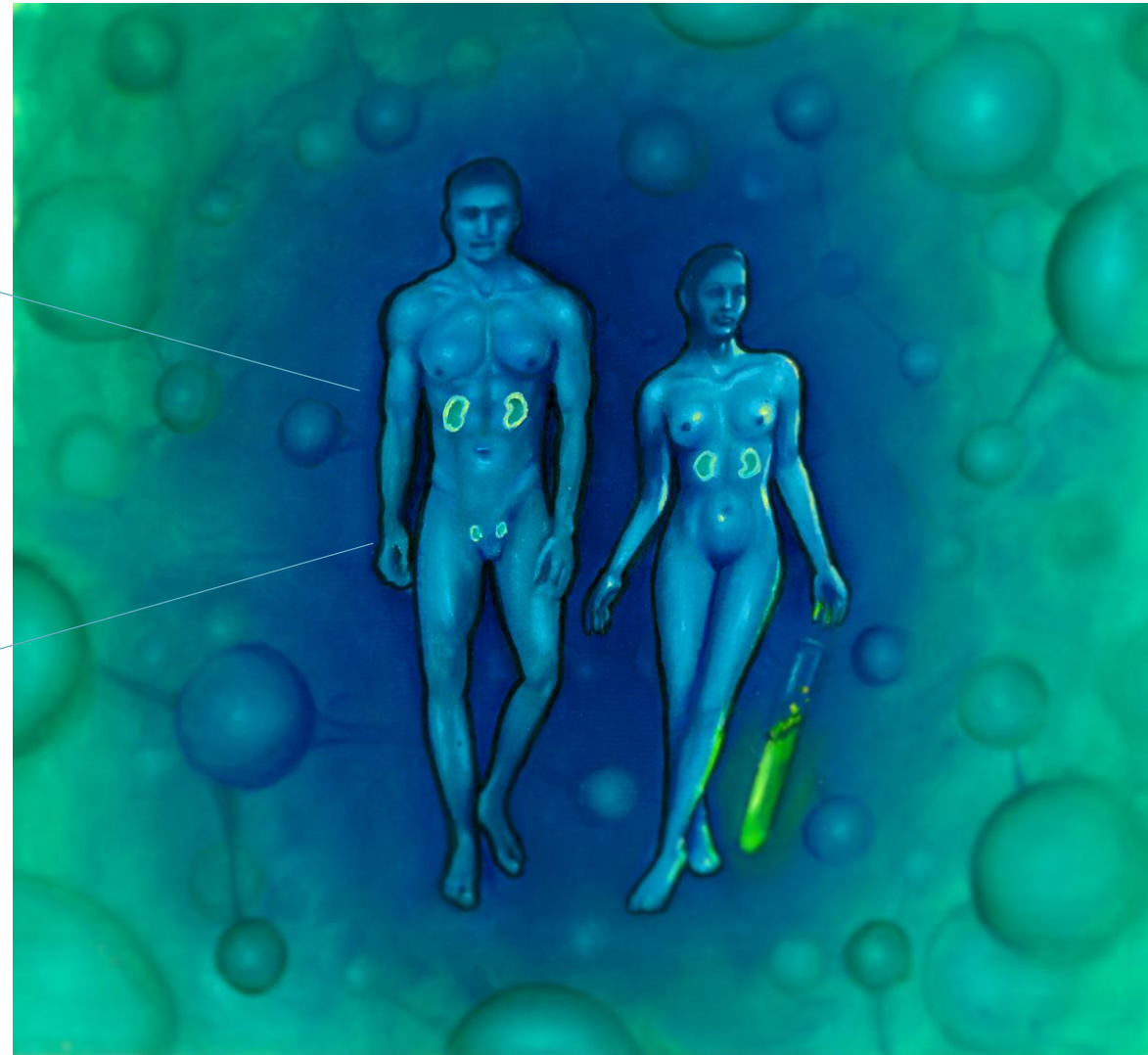
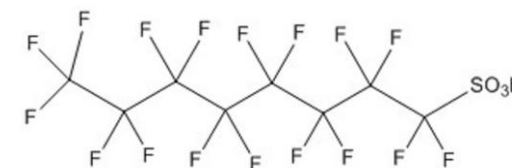


Figure1 Geographical distribution of incidence rates (IR) for kidney, C64 (top) and testicular, C62, (bottom) cancer in Norway for period 2013-2022 (only men).

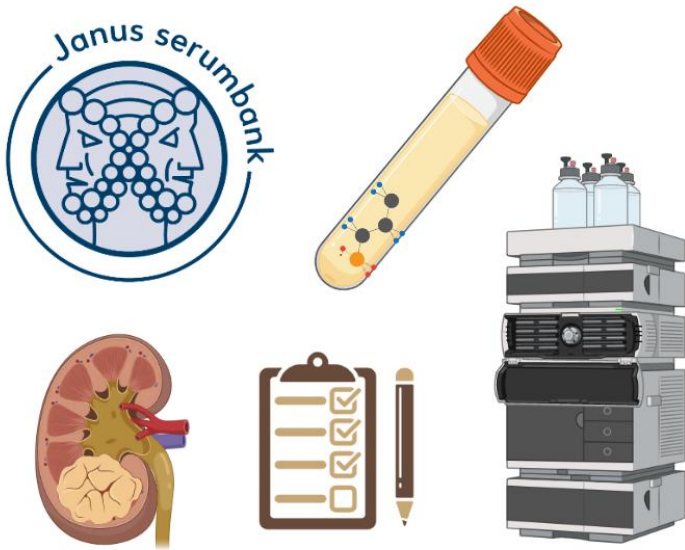






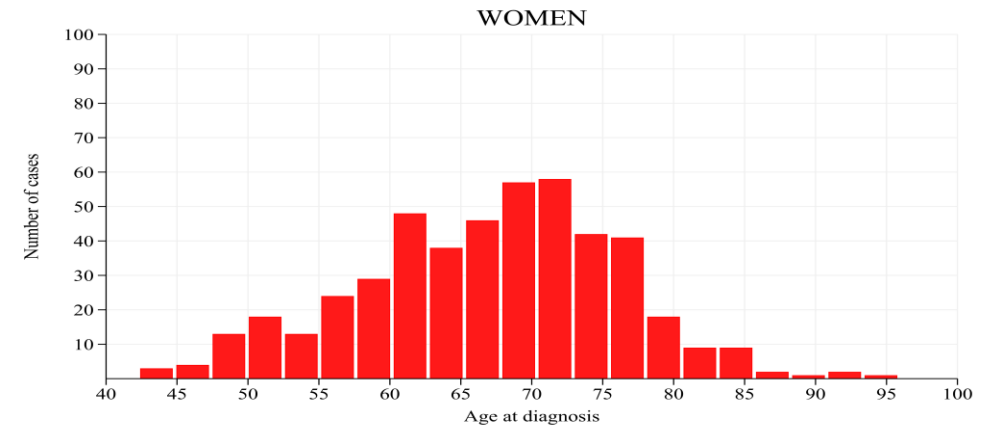
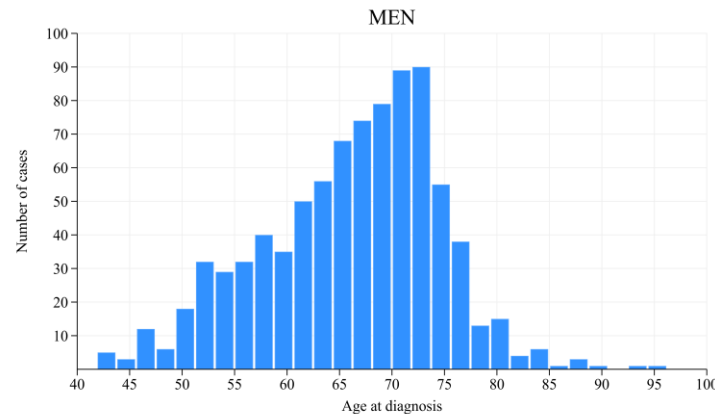
RCC - renal cell carcinoma

Risk estimation



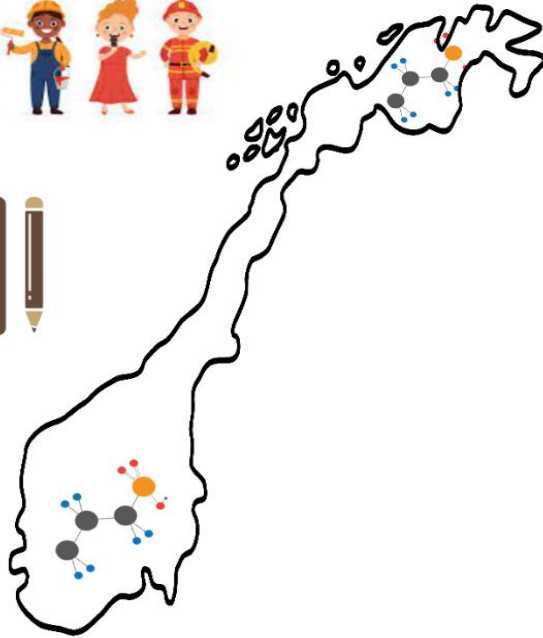
Link the RCC and TGCT risk with PFAS exposure using archived serum samples. We hypothesize that:

- PFAS serum **levels are higher** in cohort members who develop RCC, compared to controls when accounting for known covariates. In addition, risk of developing **RCC is dose dependent**.
- Some of the measured PFAS will have **stronger associations** to RCC compared to others.
- Increased PFAS concentration** in serum is positively correlated with death rates (exploratory).




1000 Control x 1000 Cancer

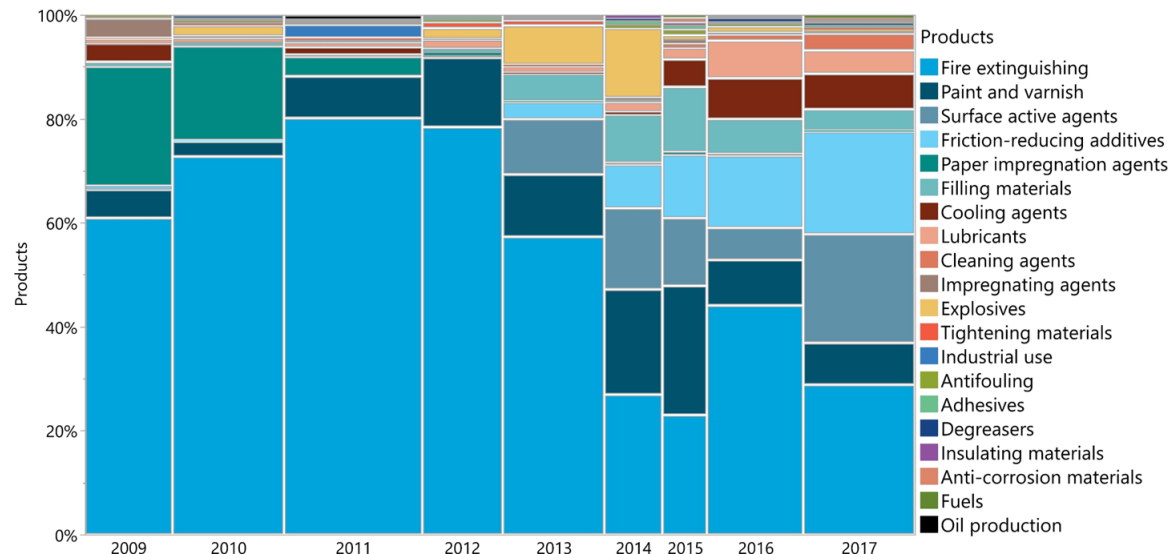
Environmental exposure



Workers can be exposed to PFAS (per- and polyfluoroalkyl substances) in certain industries:

Identify geographic and occupational variations in PFAS levels. We hypothesize that:

 PFAS serum levels are higher in cohort members with lifestyle-related exposure, such as place of residence, and occupation.



Industry specific:



- Firefighters
- Ski waxers
- Textile, **paper**, and food-packaging
- Chemical industry workers
- Electronics and plating industries





Firefighters and exposure?

- Aqueous Film Forming Foam (AFFF)
- Dust
- Protective clothing



Firefighters spraying foam, The Yellowstone Fires



Norwegian firefighters



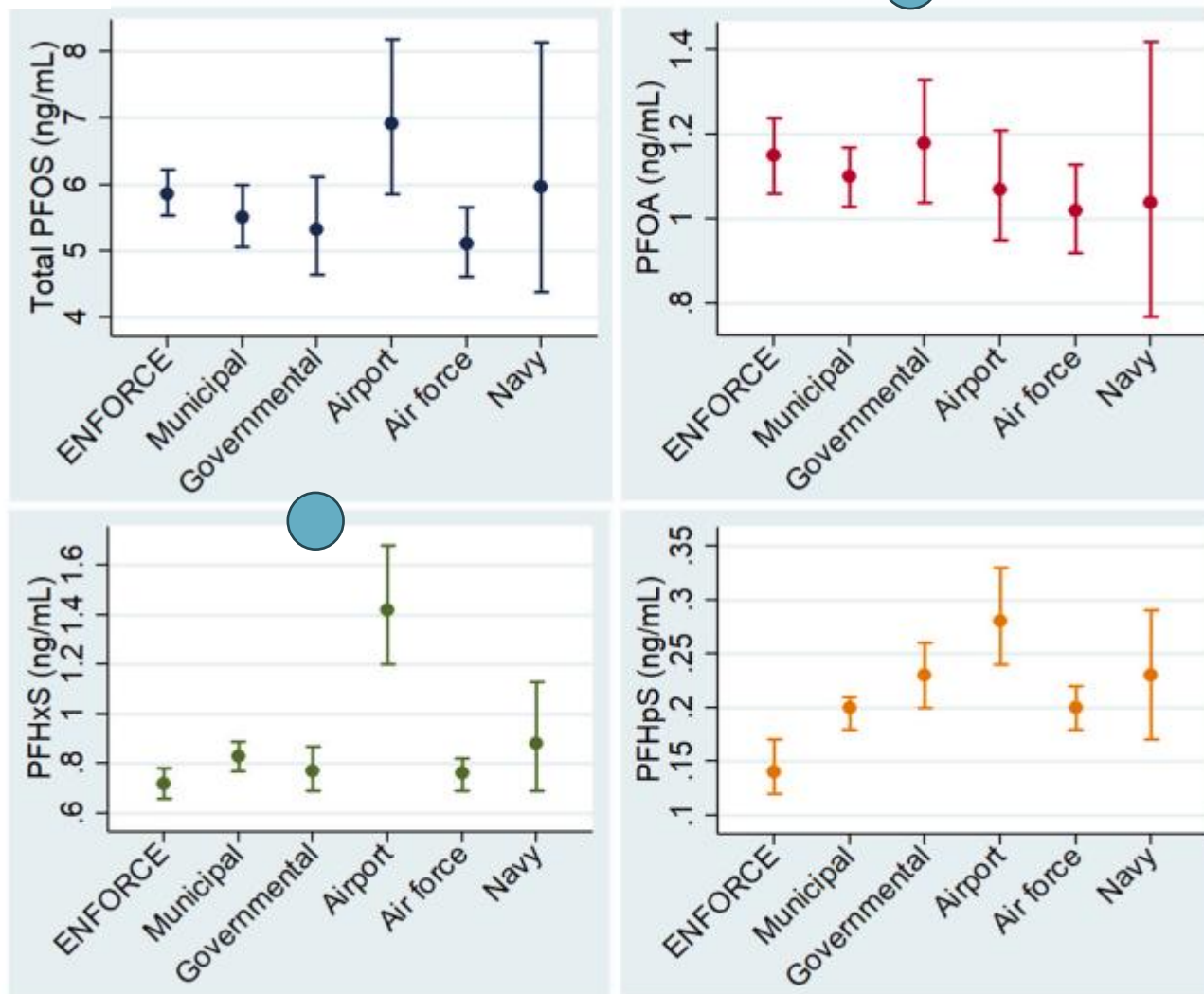
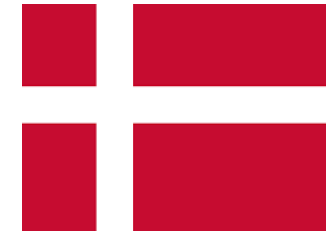
- PFAS in blood samples from 20 experienced firefighters who had worked at least 20 years as smoke divers and were still working as smoke divers (2019)
- Levels in Norwegian firefighters compared with other studies

	Firefighters			General Norwegian population			EFSA
	Min	Maks	Median	Min	Maks	Median	Median
PFOA	0,66	4,0	2,0	0,38	25	1,9	1,9
PFNA	0,36	3,7	0,82	0,19	2,7	0,94	0,61
PFHxS	0,43	1,9	1,3	0,23	2,3	0,95	0,67
PFOS	3,6	16	8,6	1,28	20	5,2	7,7

ng PFAS/mL serum



Danish firefighters



Past occupational exposure to firefighting foam

2025

Serum concentrations of per- and polyfluoroalkyl substances (PFAS) among men from the Danish fire services and Armed Forces

Kajsa Ugelvig Petersen^{a,*}, Dorthe Furstrand Lauritzen^a, Regitze Sølling Wils^a, Anne Thoustrup Saber^b, Ulla Vogel^b, Niels Erik Ebbehøj^c, Johnni Hansen^d, Julie Elbæk Pedersen^d, Tina Kold Jensen^e, Maria Helena Guerra Andersen^b

^a Department of Occupational and Environmental Medicine, Copenhagen University Hospital - Bispebjerg and Frederiksberg, Bispebjerg Bakke 23, 2400, Copenhagen, NW, Denmark

^b The National Research Centre for the Working Environment, Lersø Parkallé 105, 2100, Copenhagen E, Denmark

^c The Danish Society for Occupational and Environmental Medicine, Denmark

^d Danish Cancer Institute, The Danish Cancer Society, Strandboulevarden 49, 2100, Copenhagen E, Denmark

^e Department of Clinical Pharmacology, Pharmacy and Environmental Medicine, University of Southern Denmark, Campusvej 55, 5230, Odense M, Denmark

Age-adjusted geometric means for serum concentrations (ng/mL) of total PFOS, PFOA, PFHxS and PFHpS among men from the Danish fire services and Armed Forces (n = 429, 2023–2024) compared to national measurements from the ENFORCE study (n = 496 men, 2021)

Mapping environmental contamination of PFAS in Norway



Map PFAS contamination
in Norwegian freshwater



Examine the extent to
which airports influence
contamination in Norway

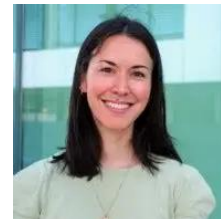
- Database from the Norwegian Environment Agency with all reported PFAS measurements
- Python programming used to retrieve and analyze data



Norges miljø- og
biovitenskapelige
universitet



Kathrine Bekkadal, Master of Science, August 2025
Supervisors: Marcin Wojewodzic, Niki Marjerrison



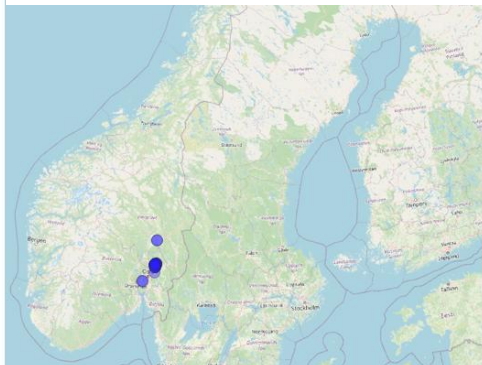
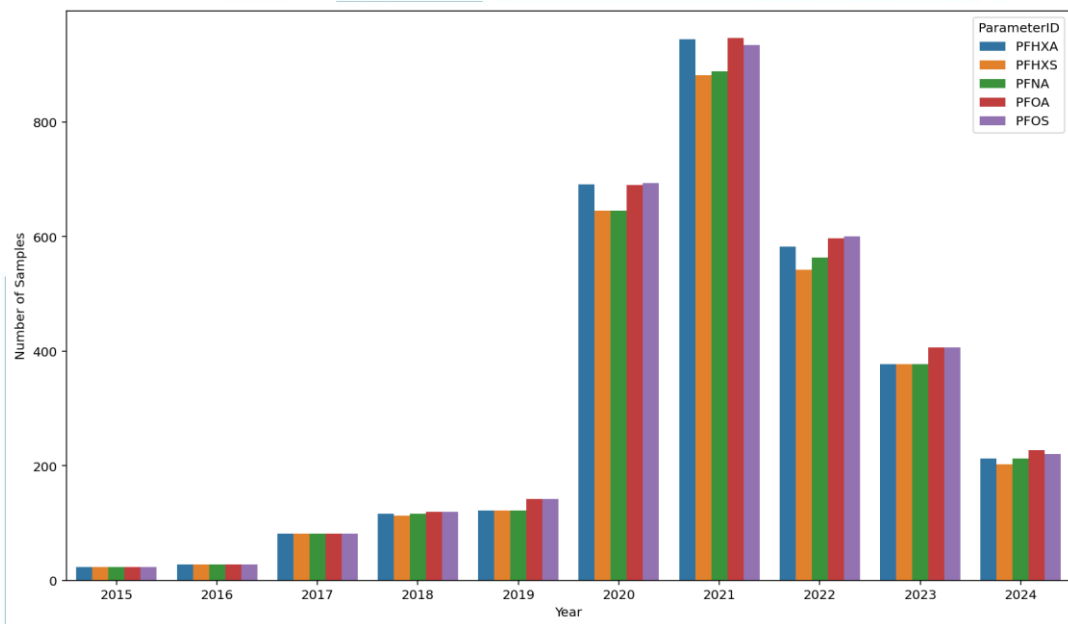


- PFAS measured across all media
 - 1993–2024
 - Total samples 6,483
 - Total analyses conducted: 264,173
- Samples distributed across 1,703 unique locations suspected of contamination
- 309 sites sampled only once
 - **Monitoring programs** applied varying sampling intervals across different time periods



Spatial distribution of **PFNA** in Freshwater (2015-2024). PFNA samples reported at two-year intervals.

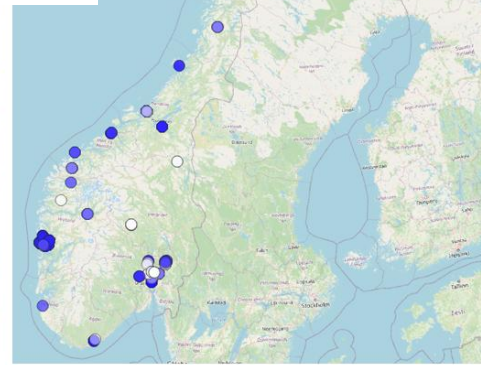
The colour intensity corresponds to the PFNA concentration (ng/L).



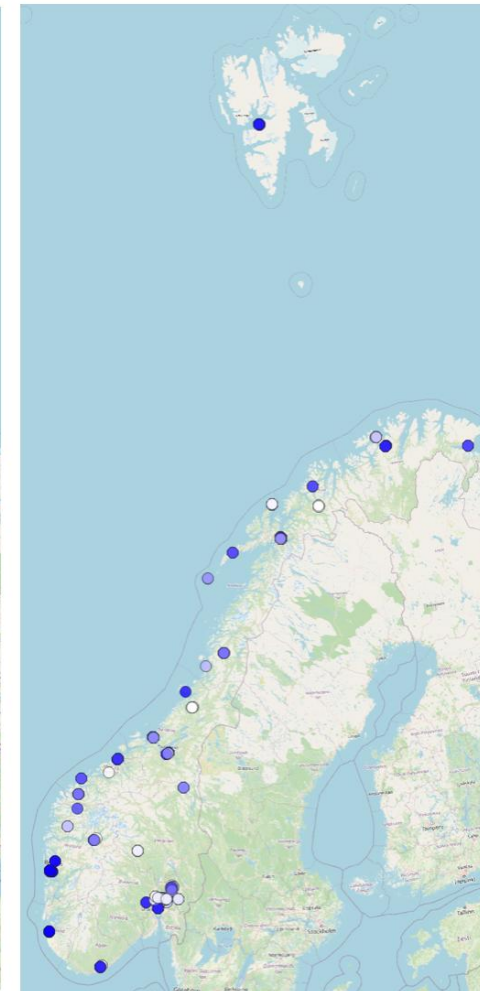
2015-2016



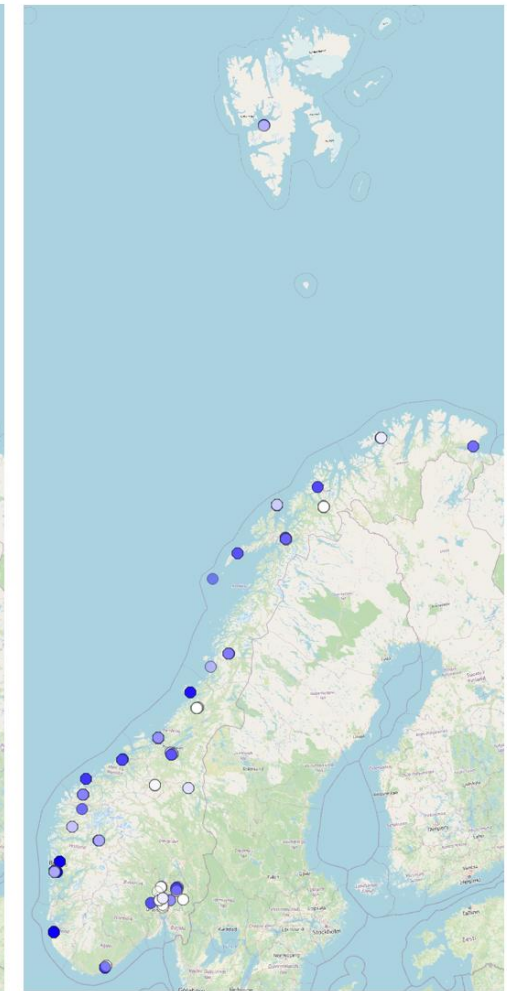
2017-2018



2019-2020

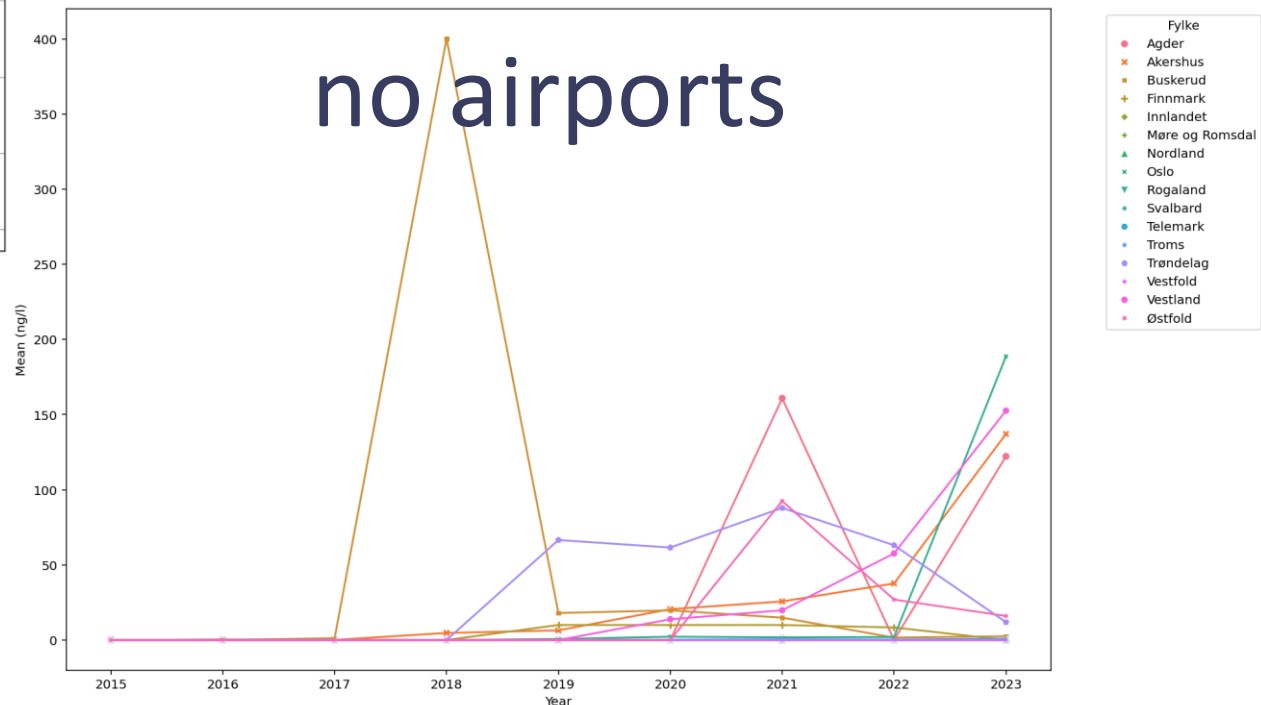
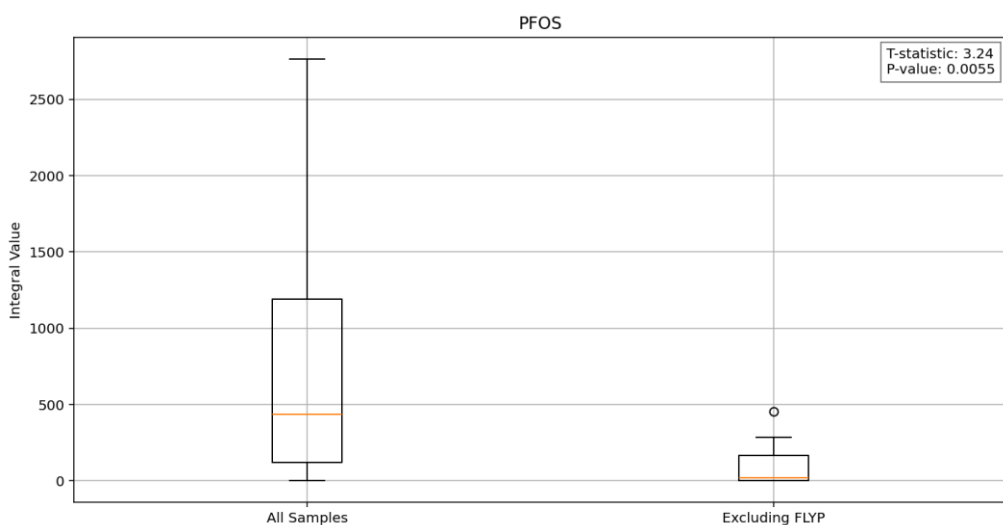


2021-2022



2023-2024

Contamination of airports with PFOS



One Health Perspective

Healthy People

Healthy Environment

The One Health

Healthy Animals



Acknowledgment



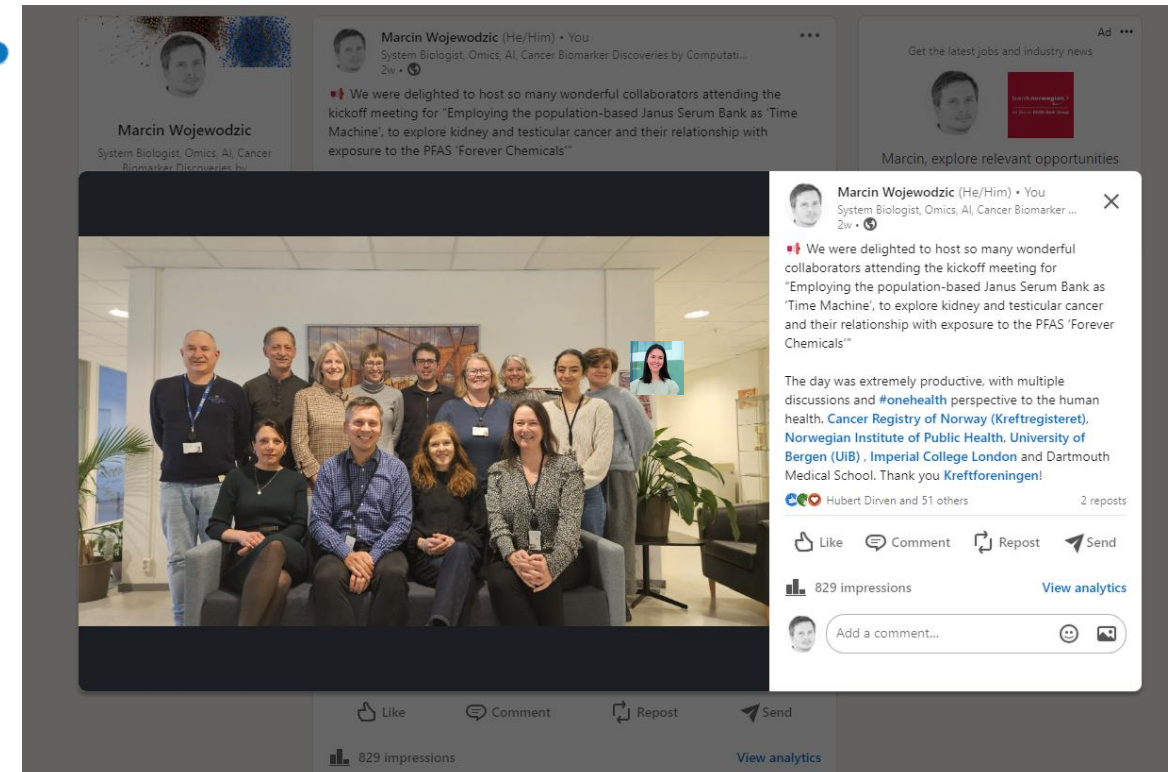
NORWEGIAN CANCER SOCIETY



NATIONAL
CANCER
INSTITUTE



- Line Småstuen Haug, Dorte Herzke, Niki Marjerrison, Hilde Langseth, Trude E. Robsahm, Trine Husøy, Tom Grimsrud, Cathrine Thomsen, and Laboratory
- Susanna Röblitz, Angeline S. Andrew; Oliver Robinson; Michal Chovanec
- Jo Stenehjem, Renée T. Forner
- Marianne Lauritzen, Kristin Haugan, Marie Udneseter Lie, Jan Ivar Martinsen, Mieke Louwe, Marianne Fismen, Hanna L. Skerven



European Researcher's Night



NORWEGIAN CANCER SOCIETY

Tomorrow,
DROP IN
17-20
Bjørsvika
Deichman Library

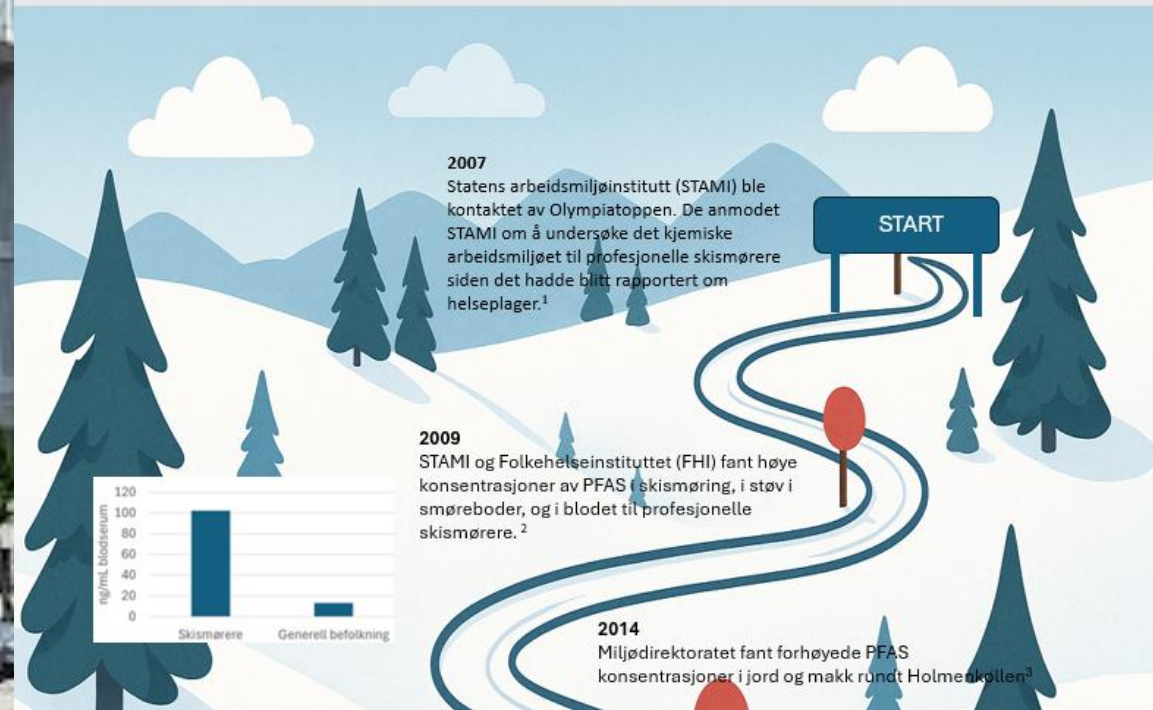


EUROPEAN
RESEARCHERS'
NIGHT 2025



Ski med god gli – er det bare positivt?

Historien om PFAS i skismøring



ADOPTED: 9 July 2020

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Risk to human health related to the presence of perfluoroalkyl substances in food

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Abstract

The European Commission asked EFSA for a scientific evaluation on the risks to human health related to the presence of perfluoroalkyl substances (PFASs) in food. Based on several similar effects in animals, toxicokinetics and observed concentrations in human blood, the CONTAM Panel decided to perform the assessment for the sum of four PFASs: PFOA, PFNA, PFHxS and PFOS. These made up half of the lower bound (LB) exposure to those PFASs with available occurrence data, the remaining contribution being primarily from PFASs with short half-lives. Equal potencies were assumed for the four PFASs included in the assessment. The mean LB exposure in adolescents and adult age groups ranged from 3 to 22, the 95th percentile from 9 to 70 ng/kg body weight (bw) per week. Toddlers and 'other children' showed a twofold higher exposure. Upper bound exposure was 4- to 49-fold higher than LB levels, but the latter were considered more reliable. 'Fish meat', 'Fruit and fruit products' and 'Eggs and egg products' contributed most to the exposure. Based on available studies in animals and humans, effects on the immune system were considered the most critical for the risk assessment. From a human study, a lowest BMDL₁₀ of 17.5 ng/mL for the sum of the four PFASs in serum was identified for 1-year-old children. Using PBPK modelling, this serum level of 17.5 ng/mL in children was estimated to correspond to long-term maternal exposure of 0.63 ng/kg bw per day. Since accumulation over time is important, a tolerable weekly intake (TWI) of 4.4 ng/kg bw per week was established. This TWI also protects against other potential adverse effects observed in humans. Based on the estimated LB exposure, but also reported serum levels, the CONTAM Panel concluded that parts of the European population exceed this TWI, which is of concern.

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Keywords: PFAS, food, exposure, mixtures, immune system, PBPK, risk assessment

Requestor: European Commission

Question number: EFSA-Q-2017-00549

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* Sandra Ceccatelli was a member of the Working Group until 7 April 2018.

EFSA's tolerable limit

The exposure level that can be sustained over a lifetime without risk of adverse health effects.

More than half of the European population exceeds the tolerable intake level for the sum of four PFAS (PFOA, PFOS, PFNA, PFHxS).

Nickel and cancer in green transition times

Tom K Grimsrud, Senior Consultant, MD, PhD

Cancer Registry of Norway, part of Norwegian Institute of Public Health (NIPH)

Seminar arranged on Thursday 25. September 2025, at Cancer Registry/NIPH, Oslo:

“Environment, occupation and cancer: etiology and prevention

Session 2: Chemicals, environmental toxins and cancer: Etiology and prevention”

In our application for funding, we called the new (present) project:

**Nickel, cobalt, and respiratory cancer:
Unmasking the relationship with lower occupational
exposures, potential impact on public health,
and biological mechanisms**

Indeed, it is a chapter from a long story – starting in the early 1900s

The nickel refinery started 1910 in an orchard ...

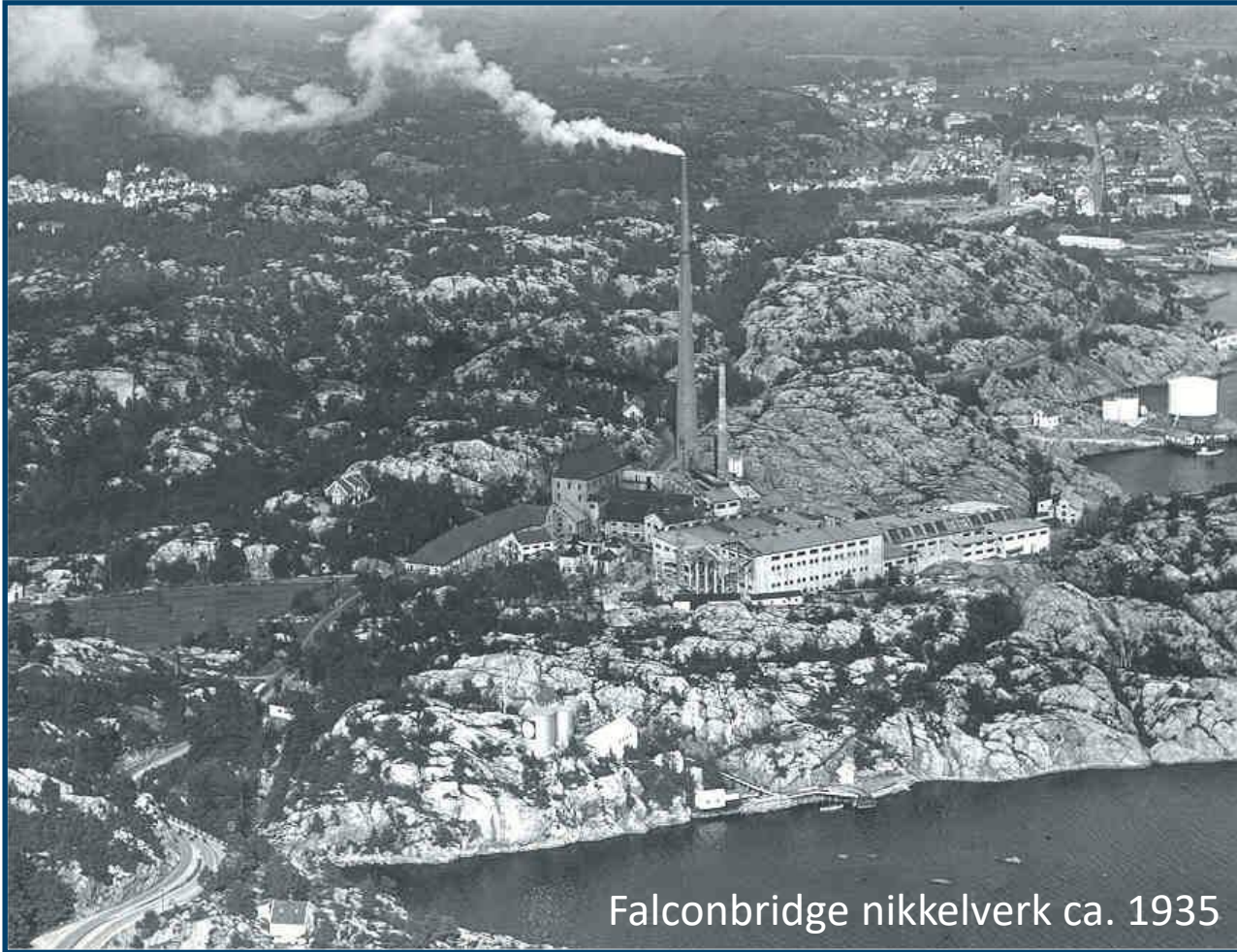


An apple garden it was, indeed,
but not the Garden of Eden
and – alas – there was no tree of wisdom

The founders included (among others)

- a rich man (Sam Eyde),
- a chemist (ingenious Victor Hybinette), and
- a guy from Sunnmøre (Norway), (Anton Grønningsæter), making things happen

Long story abbreviated ...



Falconbridge nikkelverk ca. 1935

By 1930:

- Canadian owners
- A modern factory
- Highest chimney in Scandinavia

In the surrounding you can see why they needed the chimney: Local outlets (sulphur dioxides, some metals), vegetation destroyed

Early 1930s: Alarming cancer reports from a nickel refinery at Clydach, South Wales (UK)

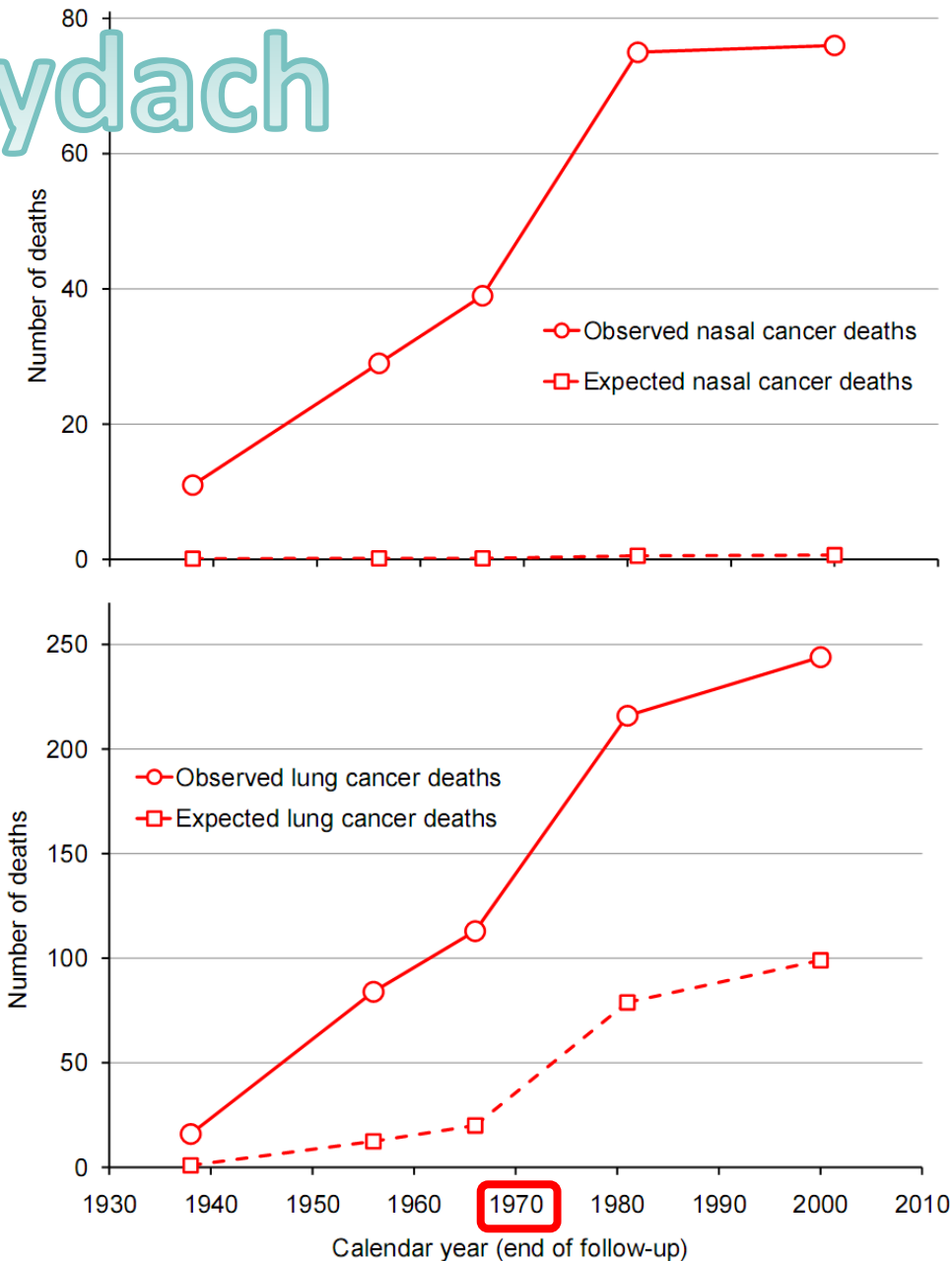


Mond Nickel Refinery, Clydach, 1930

Nickel refineries and respiratory cancer

Clydach

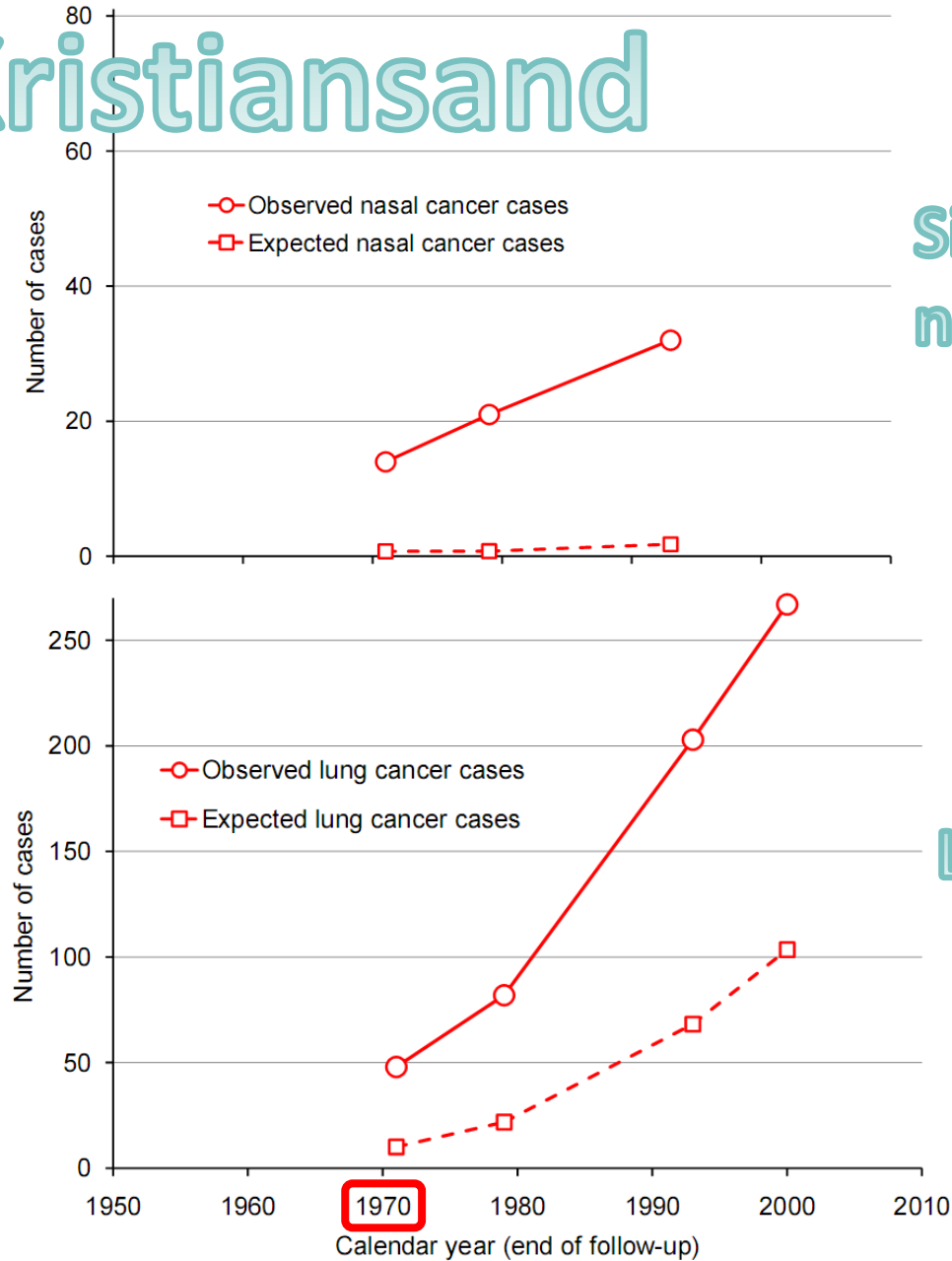
Sino-nasal



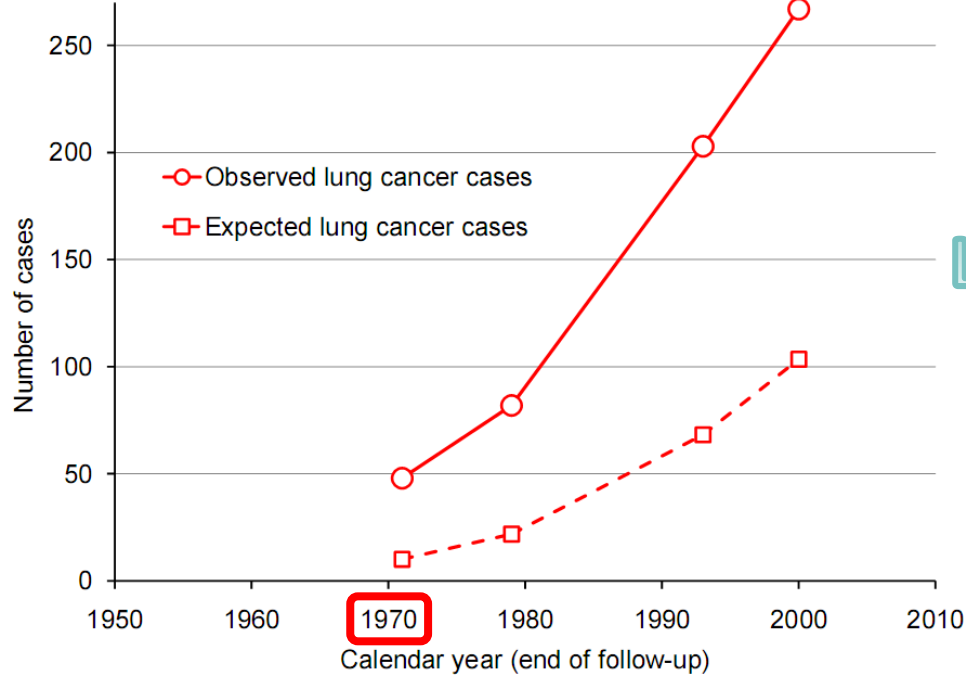
Lung

Kristiansand

Sino-nasal



Lung



A 'modern epidemiology' aetiological study

Pedersen et al 1973 – an important contribution to international research, and for Norwegian attention towards occupational carcinogens

Among 57 workers with 15 years work in the electrolysis:

Cancer form	Observed cases	Expected cases	SIR	95% CI
Sinonasal	5	0.02	250.0	81.0–583
Lung	11	0.47	23.4	11.7–41.9

These were times when dust was considered a main problem in industrial hygiene: Asbestosis was believed to cause (lead to) lung cancer



Falconbridge Nikkelverk 1967

Nickel – international understanding and classification

The Pedersen 1973 results added to earlier data from

- South Wales (UK) 1932–1970
- Canada 1959–1967
- ...

Many candidates had been suggested as ‘the carcinogen’; even ‘nickel in some form’

IARC classified ***nickel compounds*** as a Group 1 human carcinogens in 1990

Ni^{2+} . What about metallic nickel (Ni^0 , 2B)?



The first study was a great shock, unexpected

What did they/ we do (in addition to workers' economical compensation)?

- Continuous environmental improvements by changes in process and technology
- Regularly performed measurements (personally born samplers, filter pumps)
- Assessment and re-assessment of exposures, confounders and cancer incidence:
 - 1973 First SIR analyses among workers employed for 3 years +
 - 1982 Extended follow-up and analyses with smoking data
 - 1990 International Committee on Nickel Carcinogenesis in Man, with qualitative exposure estimates, workers employed for 1 year + (after WW2, only)
 - 1996 Qualitative exposure data translated to quantitative
 - 2002 Measurements 1973–1994 used for a department-time-exposure matrix addressing 4 forms of nickel
 - 2003–2005 Case-control analyses with collected life-time smoking habits
 - 2003 Cohort analyses with SIR-analyses, regressions, exposure data, smoking habits
- Continuous efforts for cessation of smoking among employees

Historical changes in exposure

1910–2025 process changes, from primitive to high-tech, electronically monitored production

Still, problems do arise, demanding manual repair, potentially harmful exposures, and need of personal protective equipment

2025: New copper electrolysis, energy efficient, encapsulated tanks



Nickel
(mg/m³)

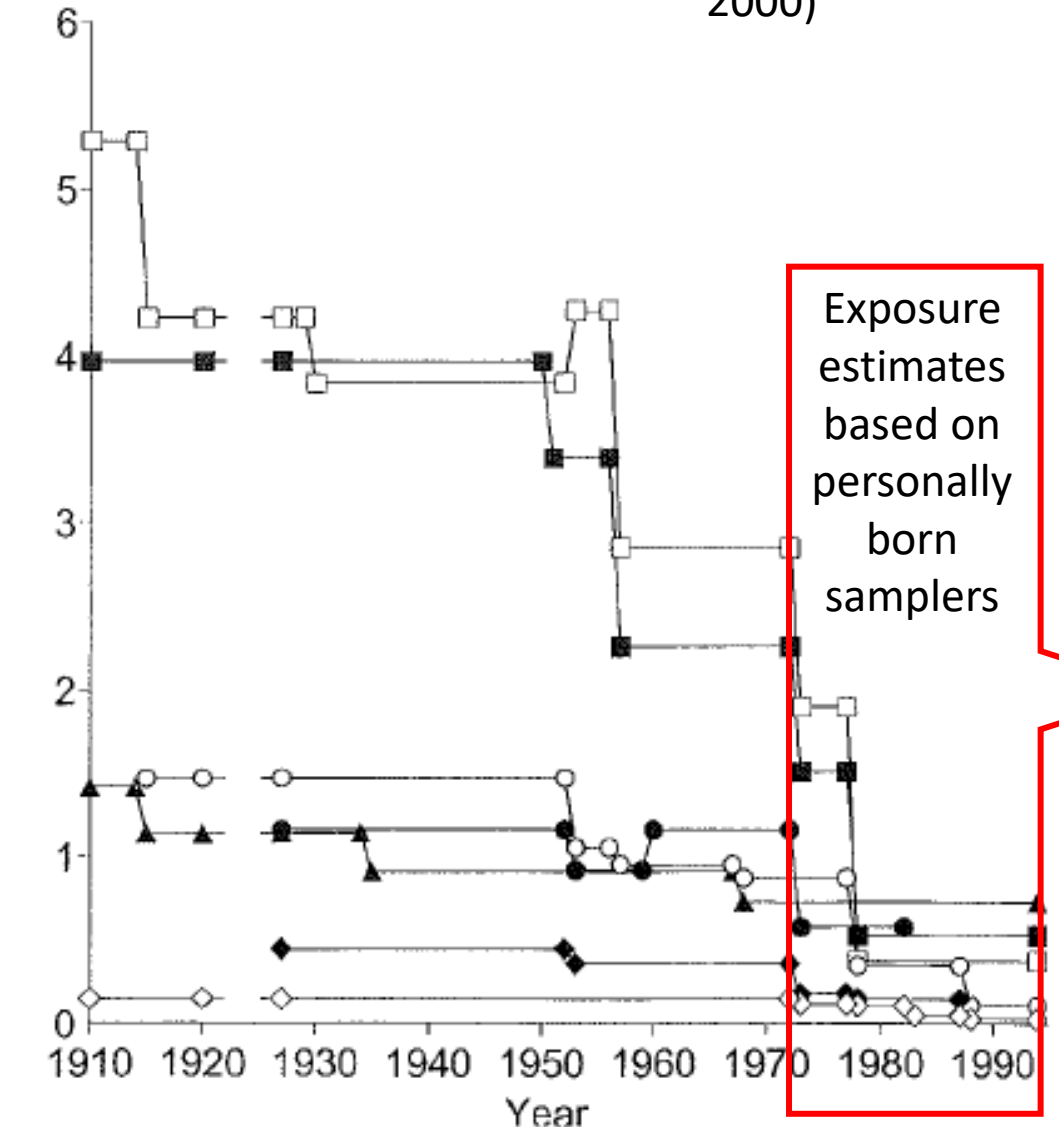
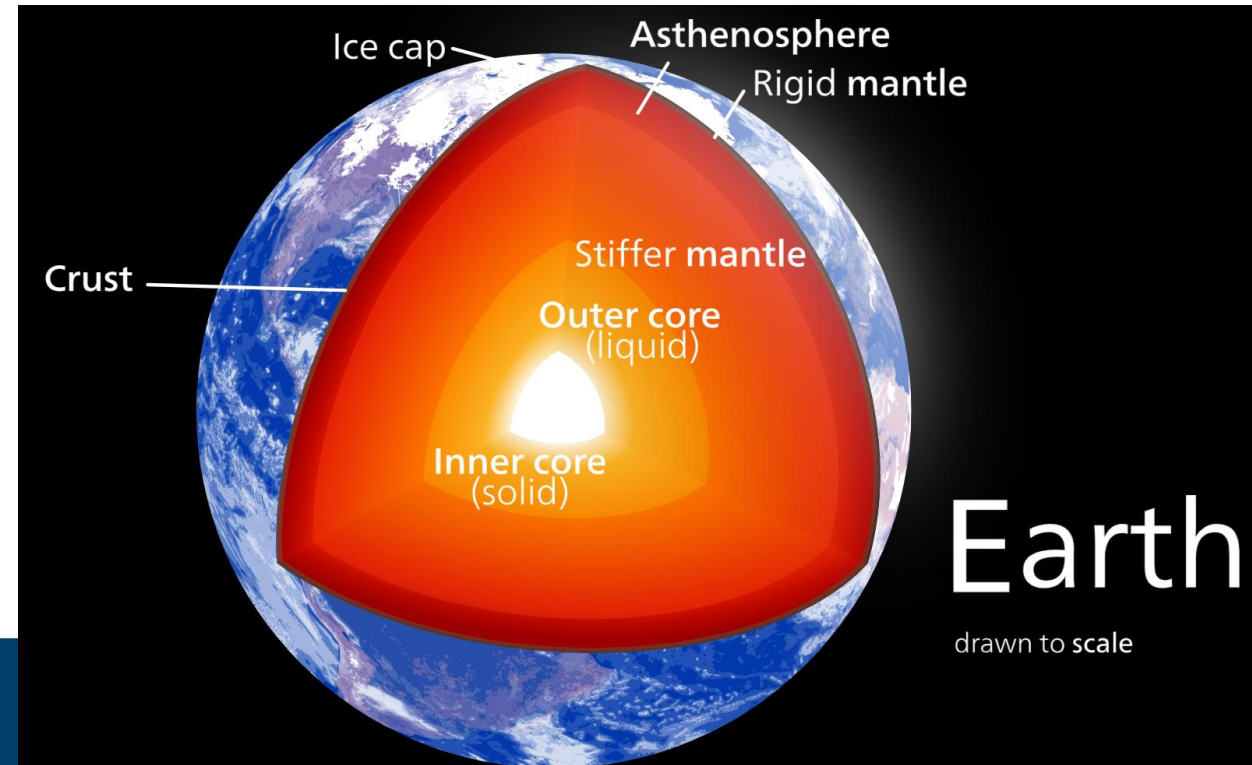


Figure 1. Time trends for the concentration of total nickel in air in selected departments.

Nickel (Ni) – an essential element for modern technology

- Nickel is used for stainless steel (75%), high-strength alloys, and plating
- Used more and more for rechargeable batteries (<5%)
- A ubiquitous element (emissions from volcanoes, forest fires, combustion and other human activity)
 - ≈ 10% nickel in Earth's inner core (unaccessible)
 - In nickel mines, usually 1–4% Ni in the ore
 - Concentrate («matte» from smelting of sulphidic ore) contains some 30–40% Ni
 - Refineries can produce «pure» nickel, 99.9%



What did the last follow-up study show?

Equally high or higher risks of lung cancer in the electrolysis and electrolysis-related departments compared with the dusty roasting and smelting departments

The total cohort, first employment 1910–1989, showed a falling lung cancer risk by increasing (= more recent) employment year, between 5 and 2 times the expected numbers

For the most recently employed there were only 5 cases of lung cancer (still suggestively too much):

First employed	Duration 1–2.9 years	3–14.9 years	15 years +	Totalt
1979–1989	Observed: 2 Expected: 0.34 SIR: 5.9 (95% CI 0.7–21)	Observed: 3 Expected: 0.91 SIR 3.3 (95% CI 0.7–9.7)	Observed: 0 Expected: 0.1 (95% KI 0.0–37)	Observed: 5 Expected: 1.35 SIR 3.7 (95% CI 1.2–8.7)

(Grimsrud et al 2003)

New studies on metals and respiratory cancer

A focus on low exposures (reduced levels) and short-term workers with long follow-up

Sinonasal cancer, a very rare cancer, nickel a potential environmental cause at low levels

Lung cancer rates mainly driven by historical smoking habits, still, 'air pollution' is carcinogenic

- Historical cohort of refinery workers
- Methylation of DNA in present-day workers
- Ecological studies of residents near nickel industry

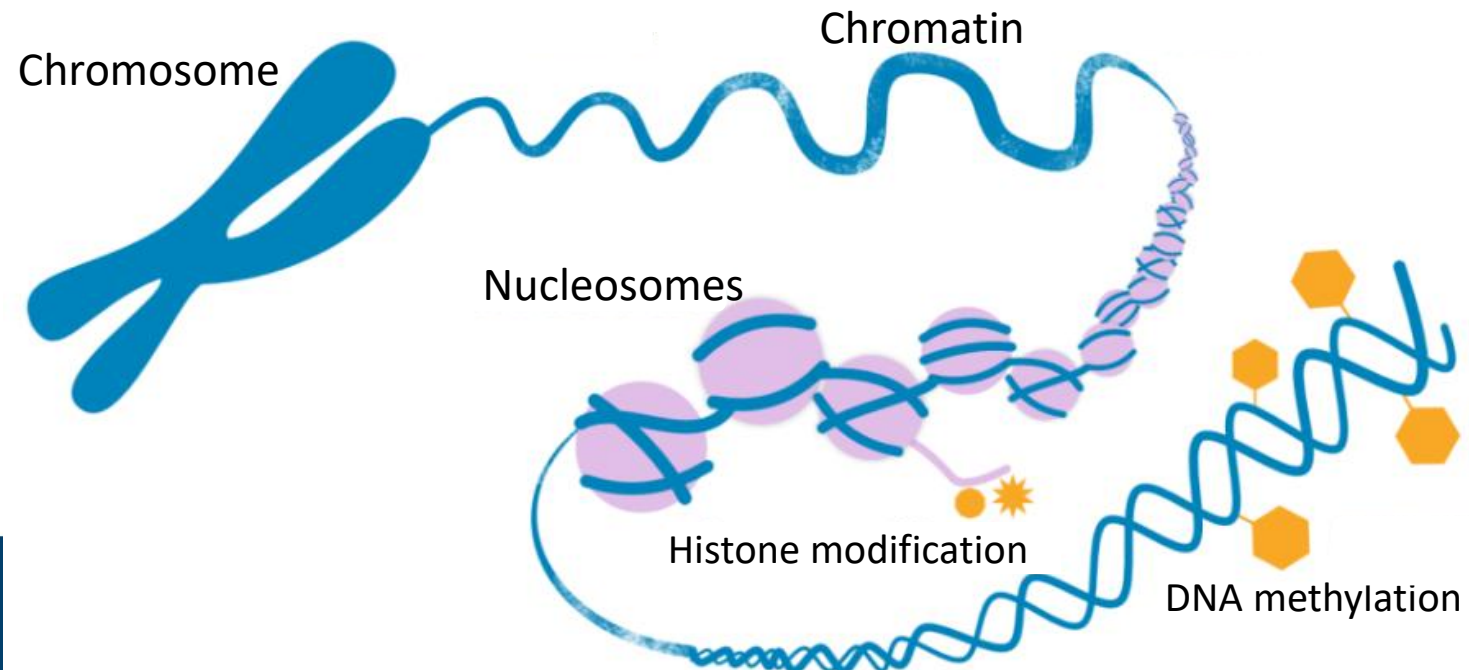
Funded by the

Norwegian Cancer Society



Biomolecular studies in refinery workers

- Nickel (Ni) compounds are known as only weakly genotoxic agents
- Ni carcinogenesis works mainly by epigenetic mechanisms:
 - DNA methylation, or histone modification, turning gene transcription on and off
 - Found in *in vitro* and *in vivo* experiments and in some human studies
- New epigenetic studies on blood samples from active, healthy workers
- Exposure biomarkers? Evidence of biological effect?

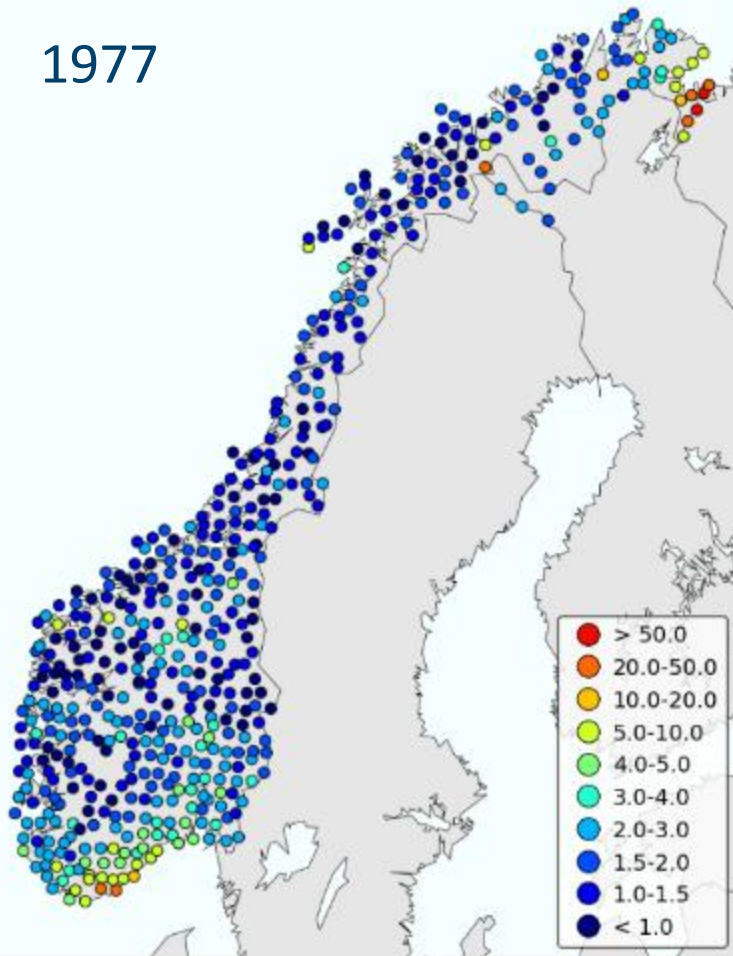


Nickel in ambient air

Mainly “local” industrial emissions, or from combustion
Monitored by measurements in moss

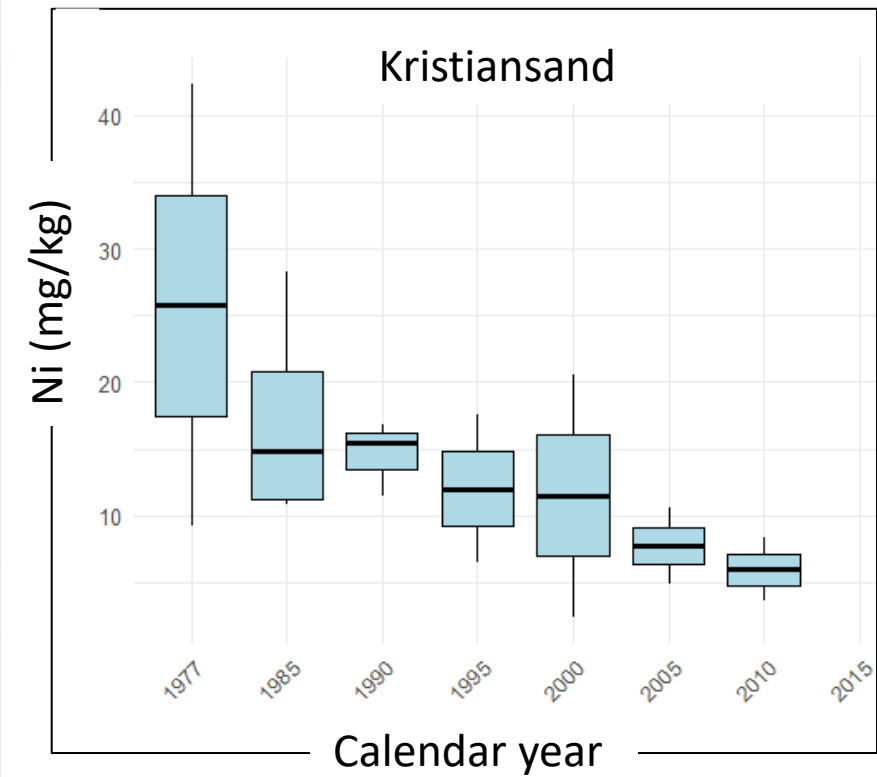
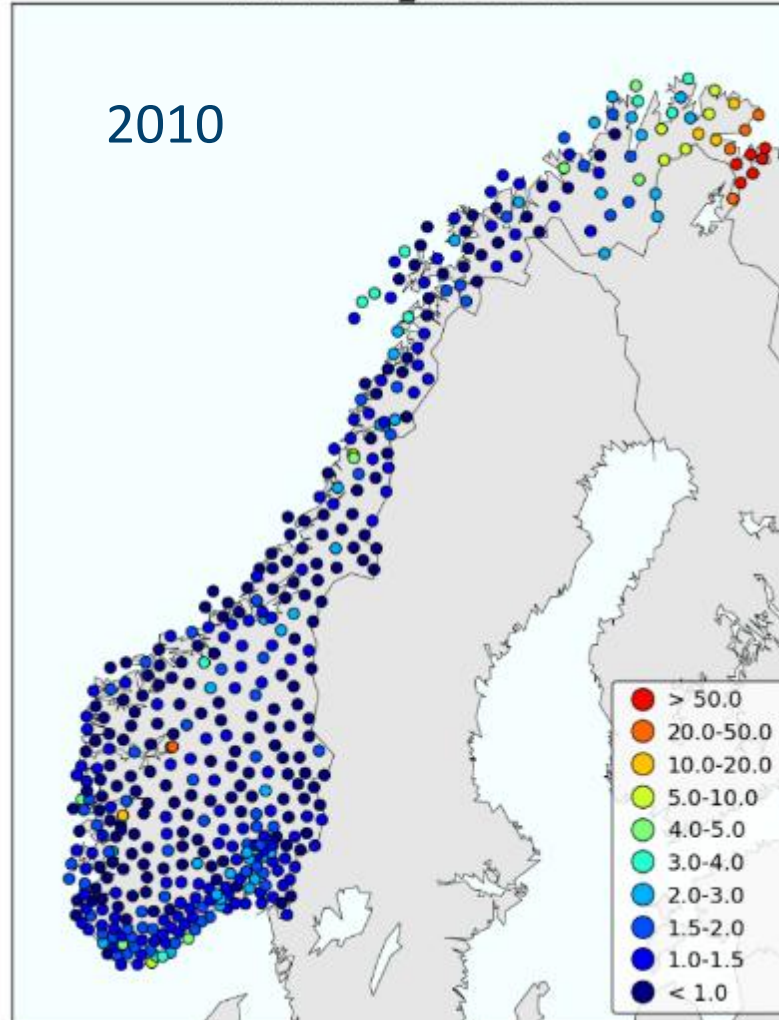
nickel dried_moss 1977

1977



nickel dried_moss 2010

2010

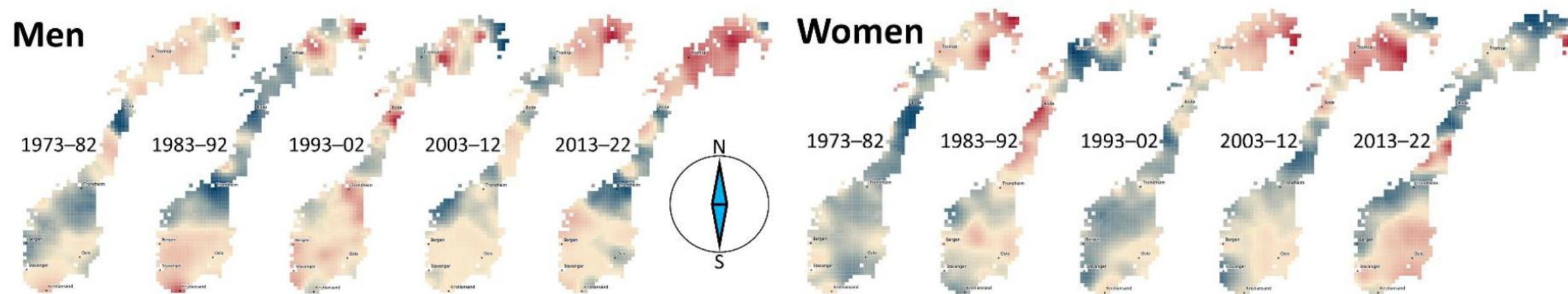


Ecological study: Respiratory cancer among residents

- In Kristiansand (refinery) and in surrounding municipalities (adjusted for incident cases among resident refinery workers derived from the cohort study)
- In Finnmark county (northernmost), neighboring Soviet, later Russian, smelters (Nikel, Zapoljarnyj)
- Lung cancer and sinonasal cancer incidence rates 1953–2024

Sinonasal cancer rates:

Heat map depicting age-standardised incidence rates during consecutive 10-year periods by gender. Red = high; Blue = low.



(https://sb.kreftregisteret.no/?sub=incidence_map&lang=no)

Session 3 – Occupational cancer: Current situation and future risk

13.45 – 14.45 **Keynote: Paolo Vineis** – Professor, Imperial College London, UK

Climate change and occupation

Niki Marjerrison - Researcher, University of Oslo, Cancer Registry of Norway-NIPH

Firefighters' cancer risk

Leon A McLaren Berge – Researcher, University of Oslo, Cancer Registry of Norway-NIPH

Exposure to dust and fibres, and risk of respiratory cancers in the Norwegian Offshore Petroleum workers cohort

Break

14.45 – 15.00

Session 3

Occupational cancer: Current situation and future risk

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14.45 – 15.00

Paolo Vineis
Imperial College London

Climate Change and Occupational Health

Oslo, 25 September 2025

MRC-HPA Centre for Environment & Health

**Imperial College
London**

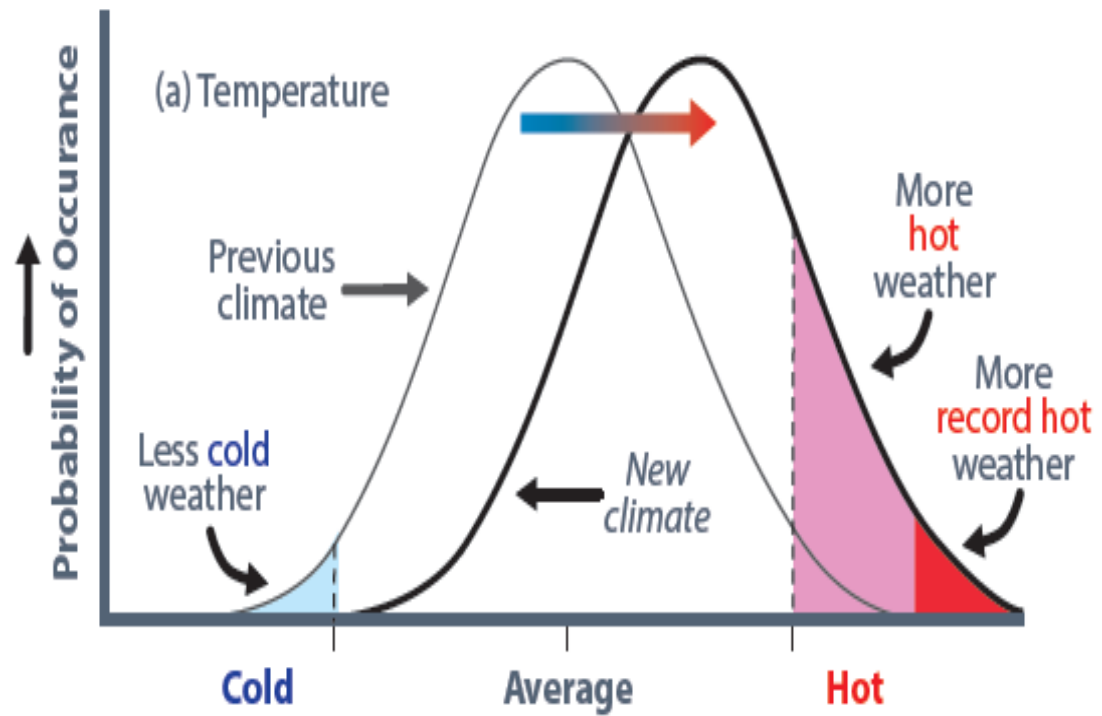


**KING'S
College
LONDON**

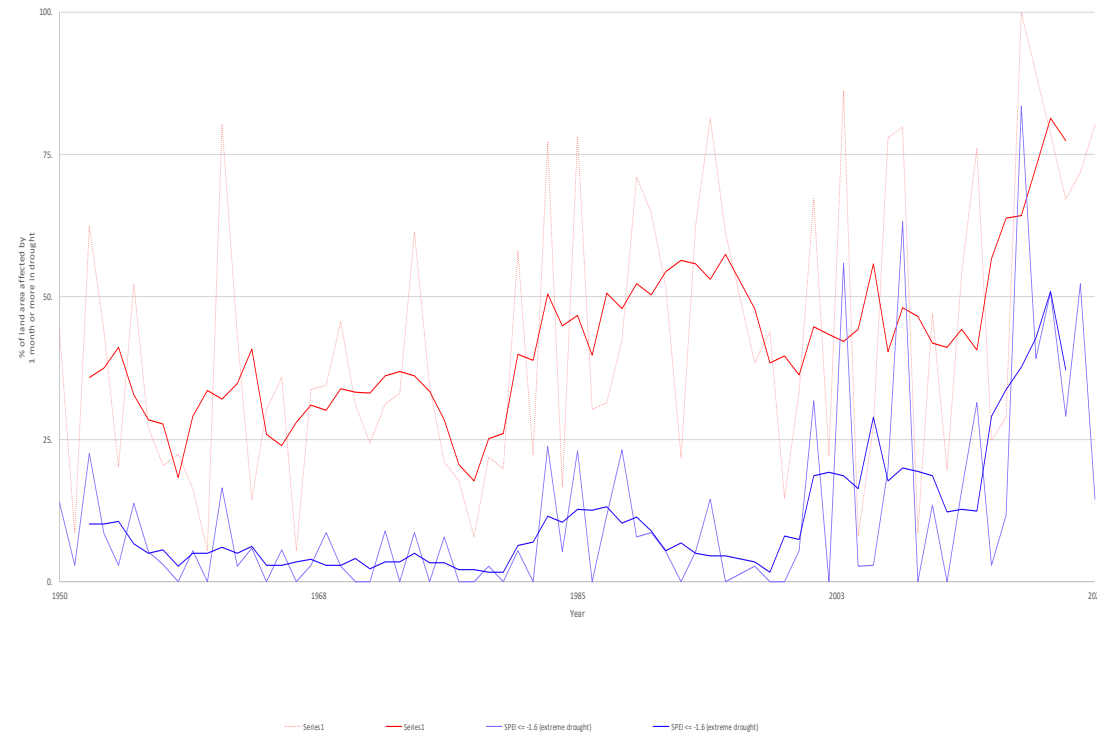


**Peter Paul Rubens: Faedon sets the earth on fire for his
arrogance**

Extreme events: the exception becomes common



Drought in Italy. Percentage of land area affected by at least 1 month of severe (red) and extreme (blue) drought. Thin dashed and continuous lines represent the annual percentage of affected land area. Thick lines represent the centred 5-year moving averages (2 years forward and 2 years backward) (source Lancet Countdown 2022)



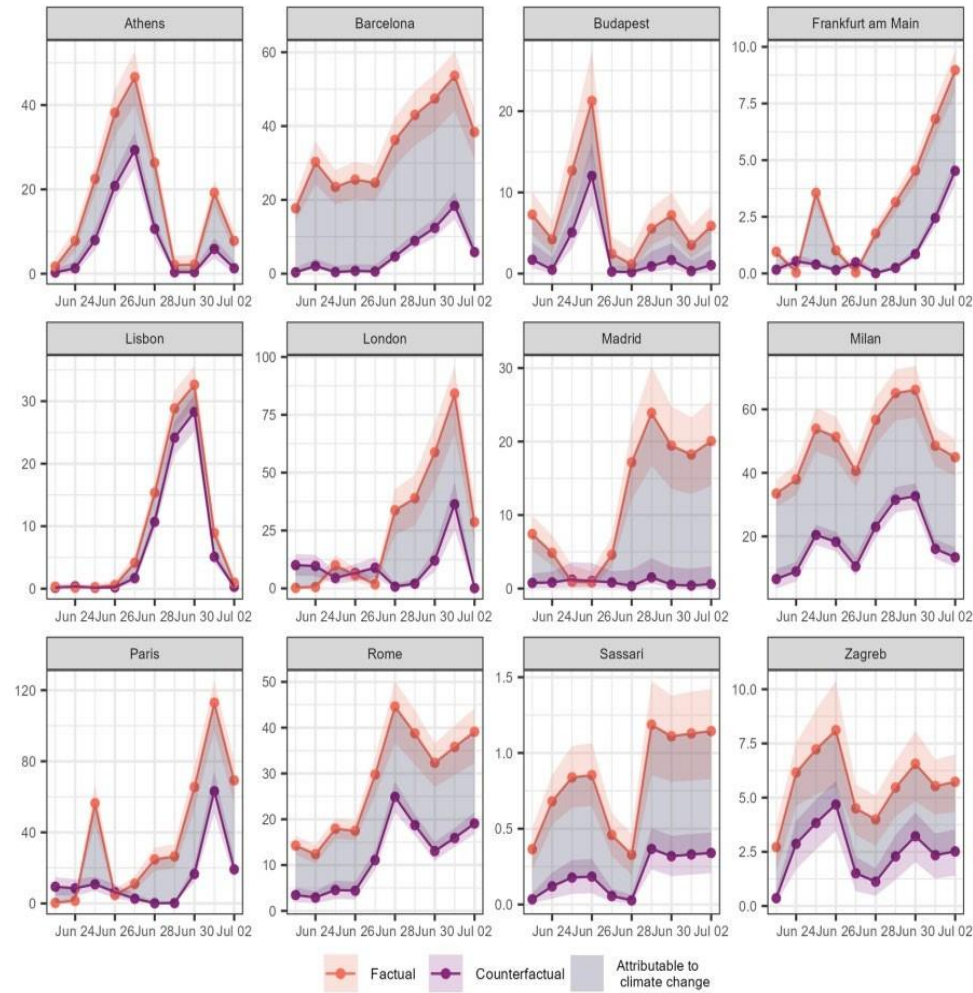


Climate change tripled heat-related deaths in early summer European heatwave

2025

The study uses a well-established methodology to estimate the total number of heat-related deaths in 12 European cities during the recent heatwave (2025) and to calculate the proportion of these deaths attributable to climate change. In total, we estimate that temperature changes caused by climate change resulted in 1,504 excess deaths (95% confidence intervals: 1,262 to 1,709) in the 12 cities..

Excess heat-related deaths



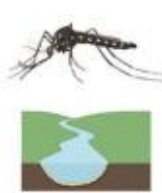
Median estimates and 95% empirical confidence intervals of heat-related deaths for the 12 major cities. The gray shaded area represents the estimated number of heat-related deaths attributed to human-induced climate change.

Climate change: major biological and health impacts

Climate change factors



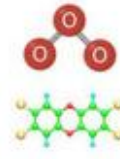
Heat
UV light



infectious
agents



pollens



air
pollutants
chemicals



extreme
weather



anxiety
mourning

Associated biological events

- temperature regulation
- dehydration
- metabolism

- inflammation, cytokines
- tissue damage
- microbiome

- allergic immune reaction

- oxidative stress
- inflammatory cytokines
- microbiome

- injury
- psychological stress

- neurological & mood disorders

Associated health effects

kidney, heart,
brain diseases
skin cancers

infectious
diseases

allergies

lung, heart
diseases

injuries;
mental
diseases

mental health
effects

From R.Barouki Frontiers in Public Health 2024

Impacts of climate on existing jobs

Reviews:

Fatima et al. Extreme heat and occupational injuries in different climate zones: A systematic review and meta-analysis of epidemiological evidence *Environ Int.* 2021 Mar;148:106384.

Binazzi et al. [Evaluation of the impact of heat stress on the occurrence of occupational injuries: Meta-analysis of observational studies.](#) *Am J Ind Med.* 2019 Mar;62(3):233-243.

Advancing the framework for considering the effects of climate change on worker safety and health

P.A. Schulte^a, A. Bhattacharya^a, C.R. Butler^b, H.K. Chun^c, B. Jacklitsch^a, T. Jacobs^d, M. Kiefer^b, J. Lincoln^e, S. Pendergrass^a, J. Shire^d, J. Watson^f, and G.R. Wagner^g

JOURNAL OF OCCUPATIONAL AND ENVIRONMENTAL HYGIENE
2016, VOL. 13, NO. 11, 847–865
<http://dx.doi.org/10.1080/15459624.2016.1179388>

Occupational health in the era of climate change and the green transition: a call for research



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- ^fErasmus MC, Rotterdam, the Netherlands
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- ^lCatalan Institution for Research and Advanced Studies (ICREA), Barcelona, Spain
- ^mLondon School of Hygiene and Tropical Medicine (LSHTM), London, UK
- ⁿDepartment of Environment, Climate Change and Health, World Health Organization (WHO), Geneva, Switzerland
- ^oInternational Labour Organization (ILO), Geneva, Switzerland
- ^pInternational Agency for Research on Cancer (IARC), Lyon, France
- ^qHalmstad University, Sweden
- ^rMRC Centre for Environment and Health and NIHR GHRC on NCDs and Environmental Change, Imperial College, London, UK

Summary

Work and working conditions are fundamental social determinants of health. Climate change poses an urgent and growing threat to workers' health, through both direct exposure to environmental hazards and indirect exacerbation of social and health inequalities. Occupational health, which focusses on the promotion of mental and physical health and well-being of workers, is a key but often overlooked area in this context. Research at the intersection of climate change and occupational health remains limited. At the same time, climate change mitigation and adaptation efforts are driving rapid transformations in the workplace, including shifts towards sustainability and circular economy models. These transitions are creating new occupational hazards, including in renewable energy and circular economy sectors. We argue for increased investment in occupational health research and surveillance to address the evolving impacts of both climate change and the green transition, to better promote and protect workers' health and rights.

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<https://doi.org/10.1016/j.lanepe.2025.101353>

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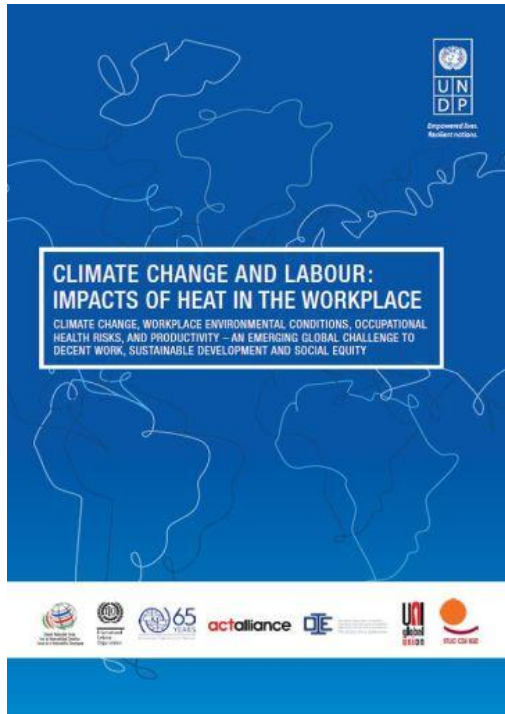
Keywords: Circular economy; Climate change; Environment; Green transition; Occupation

Introduction

Work and working conditions are fundamental underlying social determinants of health, and climate change is a rapidly emerging challenge impacting mental and

physical health of workers on an unprecedented scale.¹ Globally, 2024 was the warmest year on record, approximately 1.55 °C above pre-industrial levels.^{2,3} However, research on climate change and

Abbreviations: DALYs, Disability-adjusted life years; EDAS, Eco-driving assistance system; EU, European Union; GHG, Greenhouse gas; IARC, International Agency for Research on Cancer; ILO, International Labour Organization; KCCs, Key characteristics of carcinogens; LMIC, Low- and middle-income country; PM_{2.5}, Particulate matter; UR, Uncertainty range; UV, Ultraviolet; WHO, World Health Organization
*Corresponding author. Barcelona Institute for Global Health (ISGlobal), Doctor Aiguader, 88, Barcelona, Spain.
E-mail address: michelle.turner@isglobal.org (M.C. Turner).
Translation For the Translated abstracts see [Supplementary Materials](#) section.



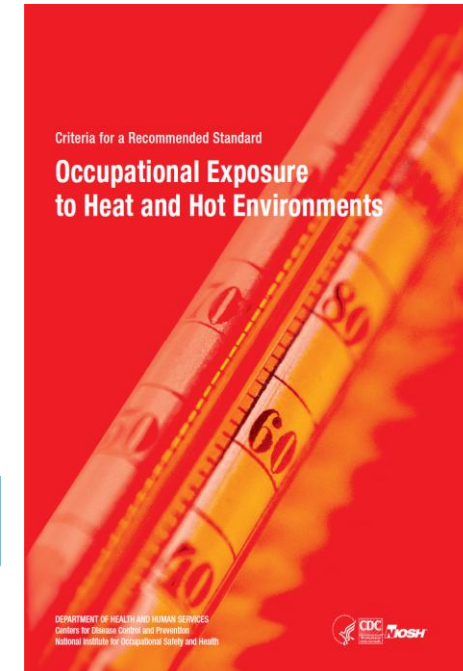
❶ The lowest income-bracket work – heavy labour and low-skill agricultural and manufacturing jobs – are among the most susceptible to climate change.

❷ Actions are needed to protect workers and employers now and in the future, including low cost measures such as assured access to drinking water in workplaces, frequent rest breaks, and management of output targets, carried out with protection of income and other conditions of Decent Work.



Heat stress in the workplace

A brief guide



Climate Vulnerable Forum, UNDP, ILO, WHO, IOM, IOE, UNI Global Union, NIOSH, HSE

Extreme temperatures and risk of work accidents in Italy



Nationwide epidemiological study for estimating the effect of extreme outdoor temperature on occupational injuries in Italy

Alessandro Marinaccio^{a,*}, Matteo Scortichini^b, Claudio Gariazzo^a, Antonio Leva^a, Michela Bonafede^a, Francesca K. de' Donato^b, Massimo Stafoggia^b, Giovanni Viegi^c, Paola Michelozzi^b, BEEP Collaborative Group (Ancona Carla, Angelini Paola, Argentini Stefania,

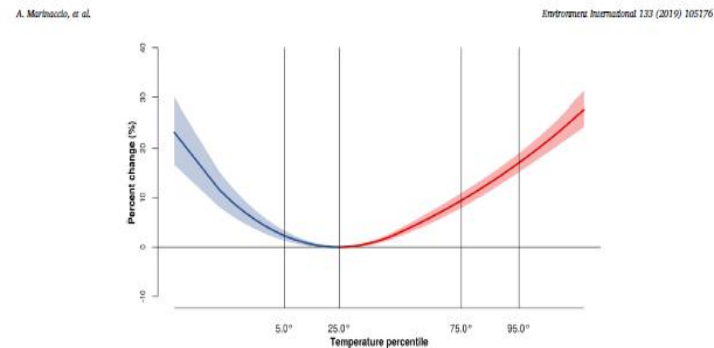


Fig. 2. Dose-response relationship. Percent change in work related injuries by temperature percentile. Blue and red areas correspond to cold and hot temperature effects. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Marinaccio A, Environ Int. 2019 Dec;133(Pt A):105176. doi: 10.1016/j.envint.2019.105176. Epub 2019 Oct 22. PMID: 31654985.

Heat (>75° percentile)

Age

15-34

35-60

>60

Relative Risk

1.25 (1.19-1.30)

1.14 (1.10-1.80)

0.91 (0.78-1.08)

Firm size

<10 workers

10-49

50-250

>250

1.20 (1.15-1.25)

1.19 (1.11-1.27)

1.20 (1.10-1.31)

1.06 (1.00-1.18)

Sector

Construction
1.38)

1.30 (1.22-

More vulnerable occupational segments









International Journal of
*Environmental Research
and Public Health*



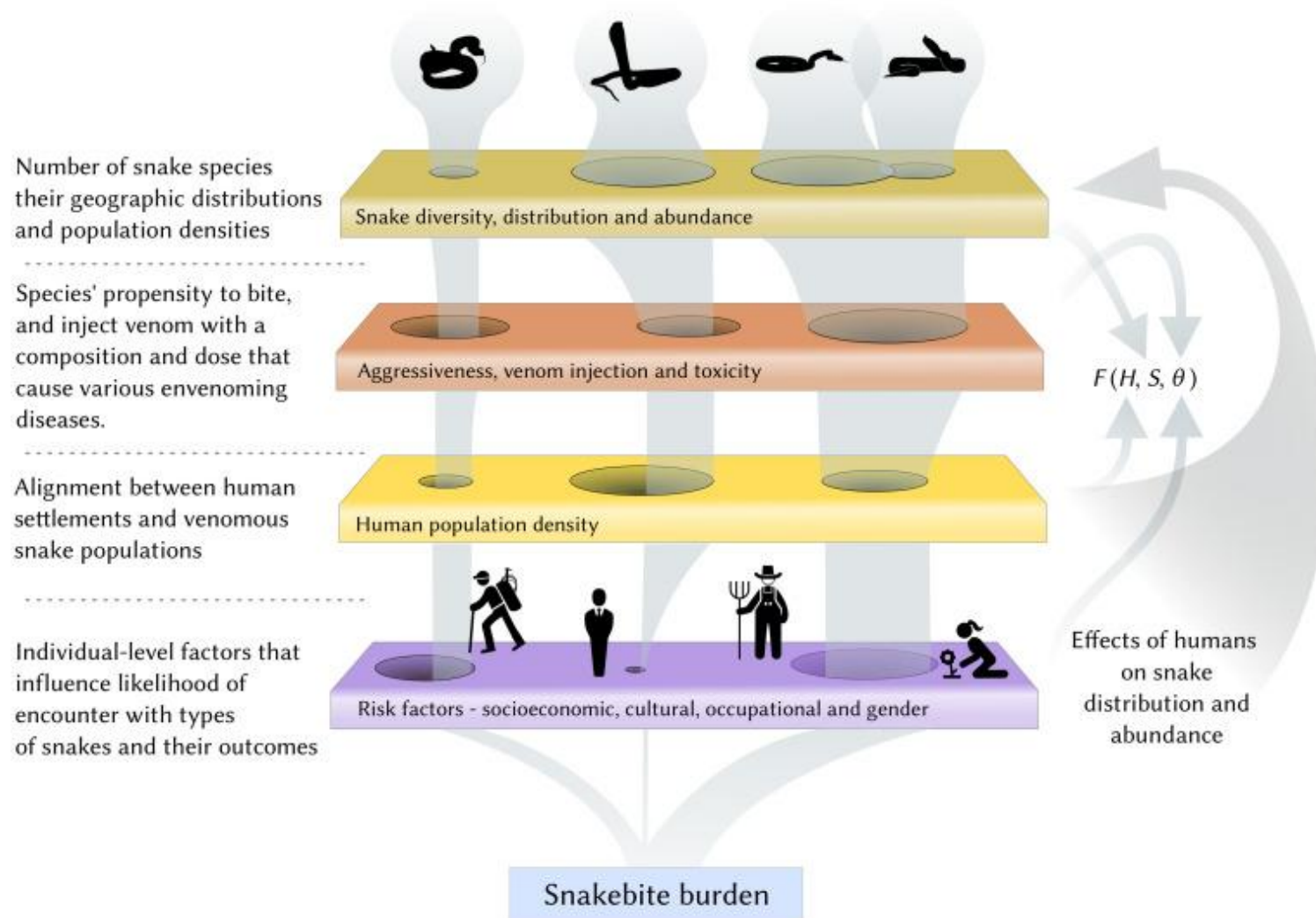
Article

Heat Stress Perception among Native and Migrant Workers in Italian Industries—Case Studies from the Construction and Agricultural Sectors

Alessandro Messeri ^{1,2,*}, Marco Morabito ^{1,3}, Michela Bonafede ⁴, Marcella Bugani ⁴,
Miriam Levi ⁵, Alberto Baldasseroni ⁵, Alessandra Binazzi ⁴, Bernardo Gozzini ⁶,
Simone Orlandini ^{1,2}, Lars Nybo ⁷ and Alessandro Marinaccio ⁴



The spread of some hazards is almost unpredictable: snake bites (courtesy K Murray)



Research needs (from Turner et al)

Official estimates and indicators of the burden of disease from occupational exposures to climate change risk factors is a **global priority** for workers' health monitoring.

Data and evidence are needed on: occupational exposures to climate change risk factors; mechanisms linking them to relevant health outcomes for hazard identification; and their effect on these health outcomes for risk quantification.

Although there is extensive literature on hot and cold temperatures and occupational injury, **less is known regarding occupational disease**. Only 19% of the total 1.9 million work-related deaths in 2016 were due to injuries, while 81% were due to disease.

Research needs (from Turner et al)

The Lancet Countdown Europe monitors and quantifies health impacts of climate change and health co-benefits of accelerated mitigation and adaptation using over forty indicators, adding nine new indicators from 2022.

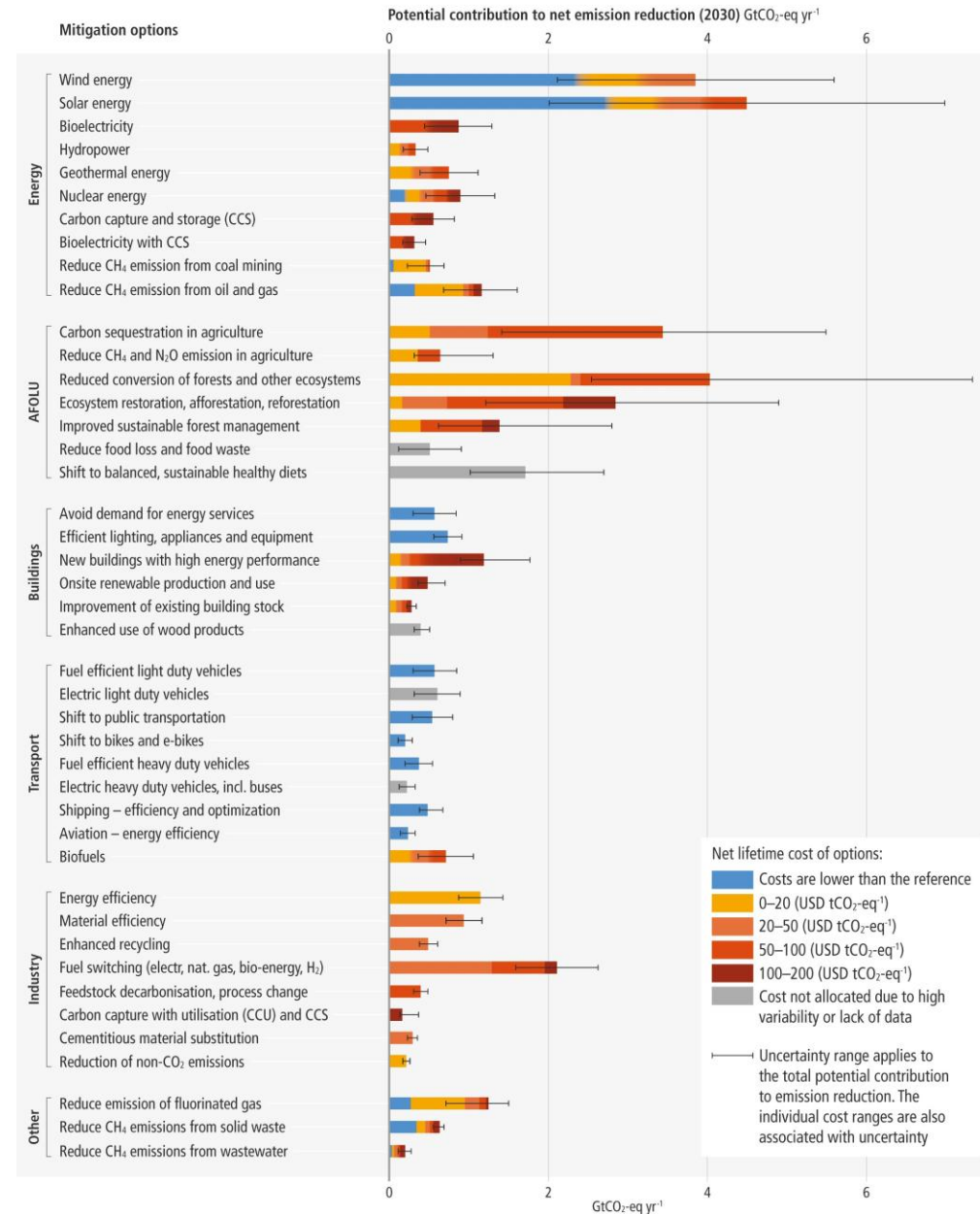
However, there is **lack of systematic approaches**.

Indicators often depend on publicly available data collected for other purposes. As a result, absence of (standardised) disaggregated health burden and population data often forms a barrier to uncovering climate-related health disparities and inequalities.

The changing landscape of jobs as a consequence of
mitigation actions

Which jobs will be more impacted?

Many options available now in all sectors are estimated to offer substantial potential to reduce net emissions by 2030. Relative potentials and costs will vary across countries and in the longer term compared to 2030.



Impact of occupations on greenhouse gas emissions

The job sectors that most directly contribute to greenhouse gas emissions are:

- Agriculture/forestry
- Fishing
- Energy
- Resource-intensive manufacturing
- Waste management
- Construction
- Transportation

These sectors are the best targets of policies designed to mitigate climate change, and together they employ more than **1.5 billion people, or about half the global workforce** (ILO, 2012)

Source: IMF

<http://www.imf.org/external/pubs/ft/fandd/2015/12/poschen.htm>

The Green Deal: what does it imply?

With the Green Deal, the EU establishes new and more stringent rules and measures aimed at:

- exploit available low-carbon and low-pollutant solutions across the entire production and value chain

- implement new technologies in energy production, distribution and end use

- adapt industrial and agricultural activities and consumption patterns to a circular economy

...

- support the transition with massive investment in research, development and innovation

- use the leverage of public procurement (14 percent of European GDP) to support and accelerate the green and digital transition and strategic autonomy.

However:

IEA 2023

Our scenarios model orderly processes of change in which markets are always in equilibrium, with investment rising and falling in different sectors to allow for a balance of supply and demand. **However, today's energy crisis has underscored that, in practice, the future of energy markets is likely to be disjointed, subject to geopolitical friction and prone to regular market imbalances.**

OECD Outlook 2023

To address the climate emergency requires nothing short of a total transformation of energy, agrifood, and mobility systems. Science, technology and innovation (STI) have essential roles in these transformations, but governments must be more ambitious and act with greater urgency in their STI policies to meet these challenges.

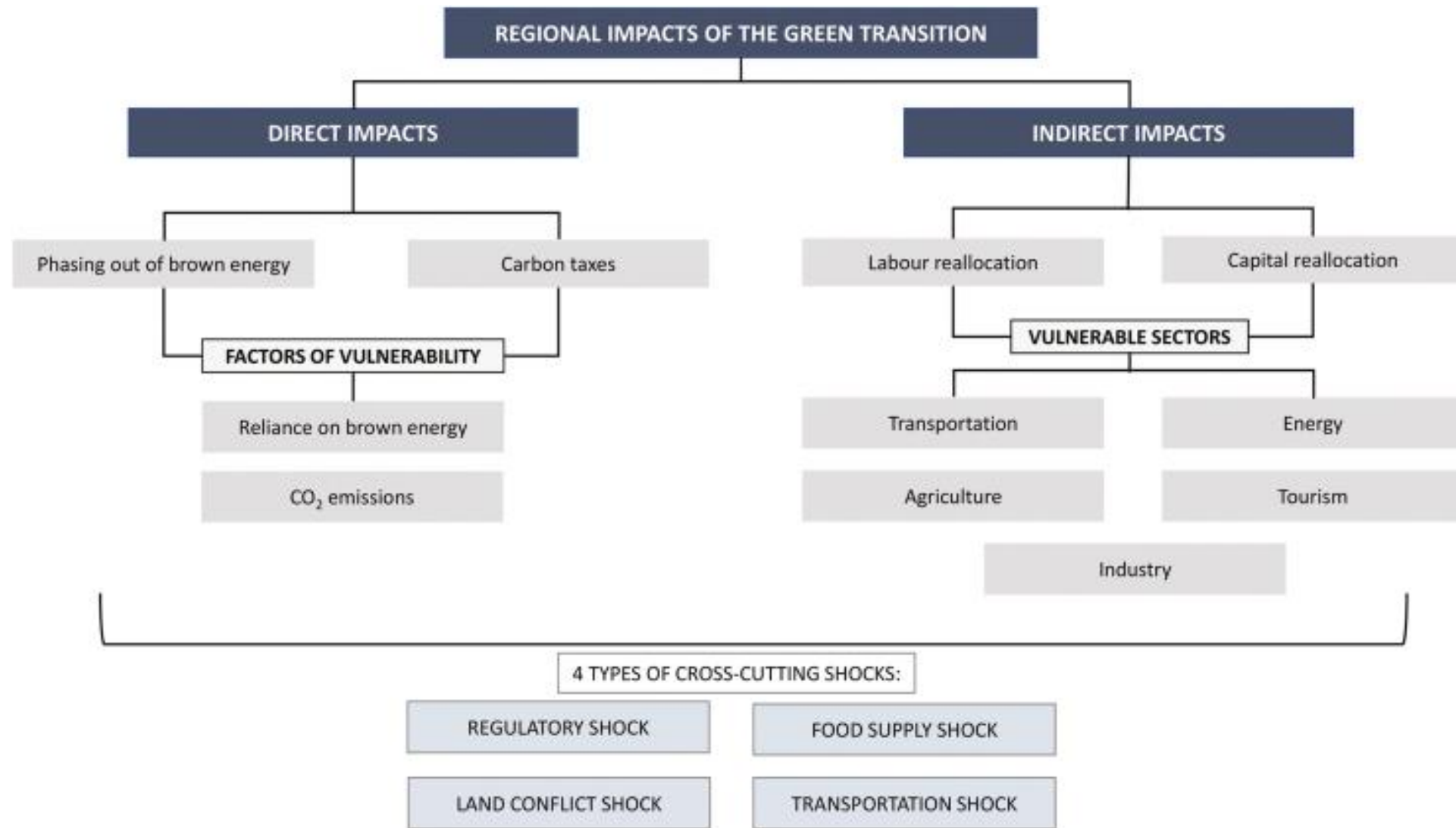


JOURNAL OF OCCUPATIONAL AND ENVIRONMENTAL HYGIENE
2016, VOL. 13, NO. 11, 847–865
<http://dx.doi.org/10.1080/15459624.2016.1179388>

Green technologies are not exempt from accidents and toxicity

What actions will be attempted to mitigate climate change cannot be predicted. Actions suggested by the Green Deal and IEA are very ambitious but little has been done so far.

If taken at face value they imply a subversion of many productive sectors. Priorities are not clearly defined, though an attempt can be made on the basis of the IPCC reports



Strong political barriers

Case studies of animal breeding: Netherlands, Ireland

Netherlands: <https://www.theguardian.com/environment/2023/jan/16/netherlands-european-union-regulations-livestock>:

«Like the other 2,500-plus farmers whose environmental permission was suddenly invalid, she wants a future where she can earn a living and farm legally again.»

The government says this means reducing local nitrogen compound emissions from between 12% and 70%, including slashing the Netherlands' 118 million farmed animals by 30% by 2030, according to Netherlands Environmental Assessment Agency projections.

There are currently far more livestock on the planet than wild animals, and more than three times the human population. Livestock production is forecast to continue increasing, as diets transition across the world to include more animal products.

Occupational health in the context of co-benefits

CLIMATE ACTION CO-BENEFIT 1: Healthier Air to Breathe

Air pollution is a major environmental hazard that contributes to an estimated 6.7 million deaths per year

Estimated ratio of air quality health co-benefit to GHG mitigation cost ranges from 1.4 to 2.45.

CLIMATE ACTION CO-BENEFIT 2: Increased Energy Security

About 1 billion people worldwide rely on health facilities that lack access to electricity.

Local renewable sources, such as from solar panels, can improve access to quality healthcare and promote a healthier quality of life.

CLIMATE ACTION CO-BENEFIT 3: Healthier Food

Livestock raised for animal-based food account for about 14.5% of global human-induced GHG emissions

Reducing meat consumption in our diets would cut water use, slow global warming by cutting emissions of methane (the most powerful GHG) and improve health.

**Policy Committee of the International
Society for Environmental Epidemiology
(ISEE)**

MRC
Centre for Environment & Health



**Imperial College
London**

Just focusing on respiratory diseases, the Global Burden of Disease (GBD) study estimated that, in 2013, coal worker's pneumoconiosis (a chronic progressive interstitial lung disease caused by exposure to coal dust) caused a loss of 600,200 (447,600 to 838,600) disability-adjusted life-years (DALYs), and a reduction in healthy life expectancy (HALE) of 9.2 (6.9 to 12.8) years.

In the period 1995-2006, a rapid positive trend in 'black lungs' has been reported by the National Institute for Occupational Safety and Health (NIOSH), from a prevalence of about 4% among US coal mine workers in the late 1990s, up to 9% in 2006.



- * Reducing coal mining/combustion will reduce both climate change and occupational illness and death
- * War on fossil fuels is not war on jobs: investing in green jobs may produce a net employment gain
- * More generally we need to establish in which sectors the transition will contribute more to improve the workers' health
- * In the case of carcinogens, occupational studies opened the way to the identification of environmental hazards (generally at lower levels). The same may happen for climate
- * Research on climate, like in other sectors, has led to preventive measures by policy-makers: e.g. in Italy the Workclimate action by a number of Regions

Thank you

Firefighters' cancer risk

Niki Marjerrison, PhD

Cancer Registry of Norway, Norwegian Institute of Public Health
Oslo Center for Biostatistics and Epidemiology, University of Oslo

Seminar – Environment, occupation and cancer: etiology and prevention
25.9.2025

Presentation

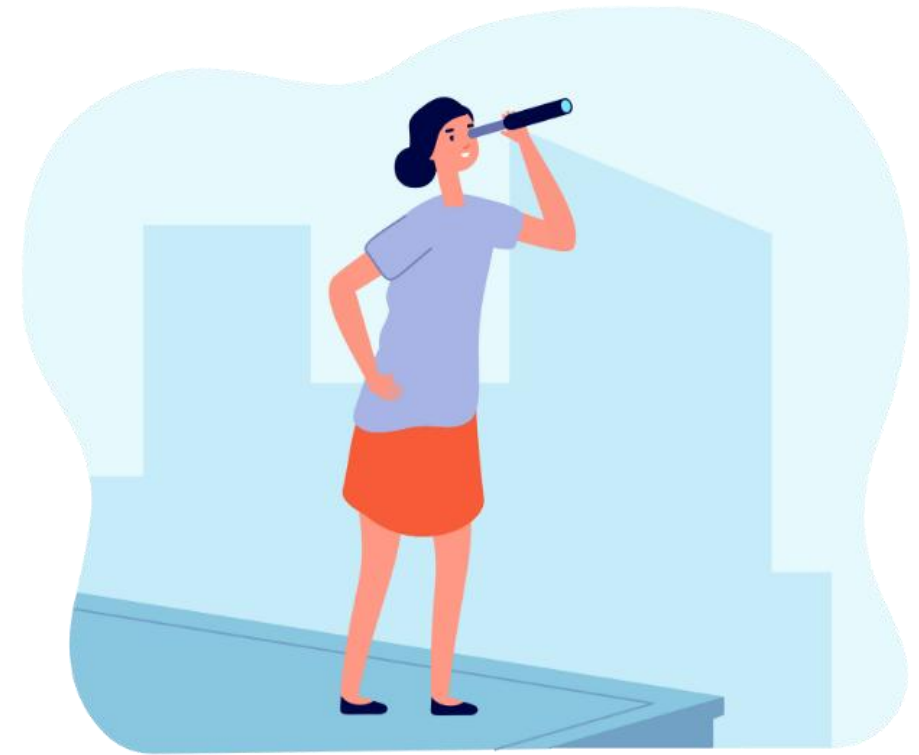
Session: Current situation and future risk

Current findings of past situation

- Norwegian Fire Departments Cohort
- IARC 2022 Monograph

Current situation and future risk

- Today's exposures
- Future risk



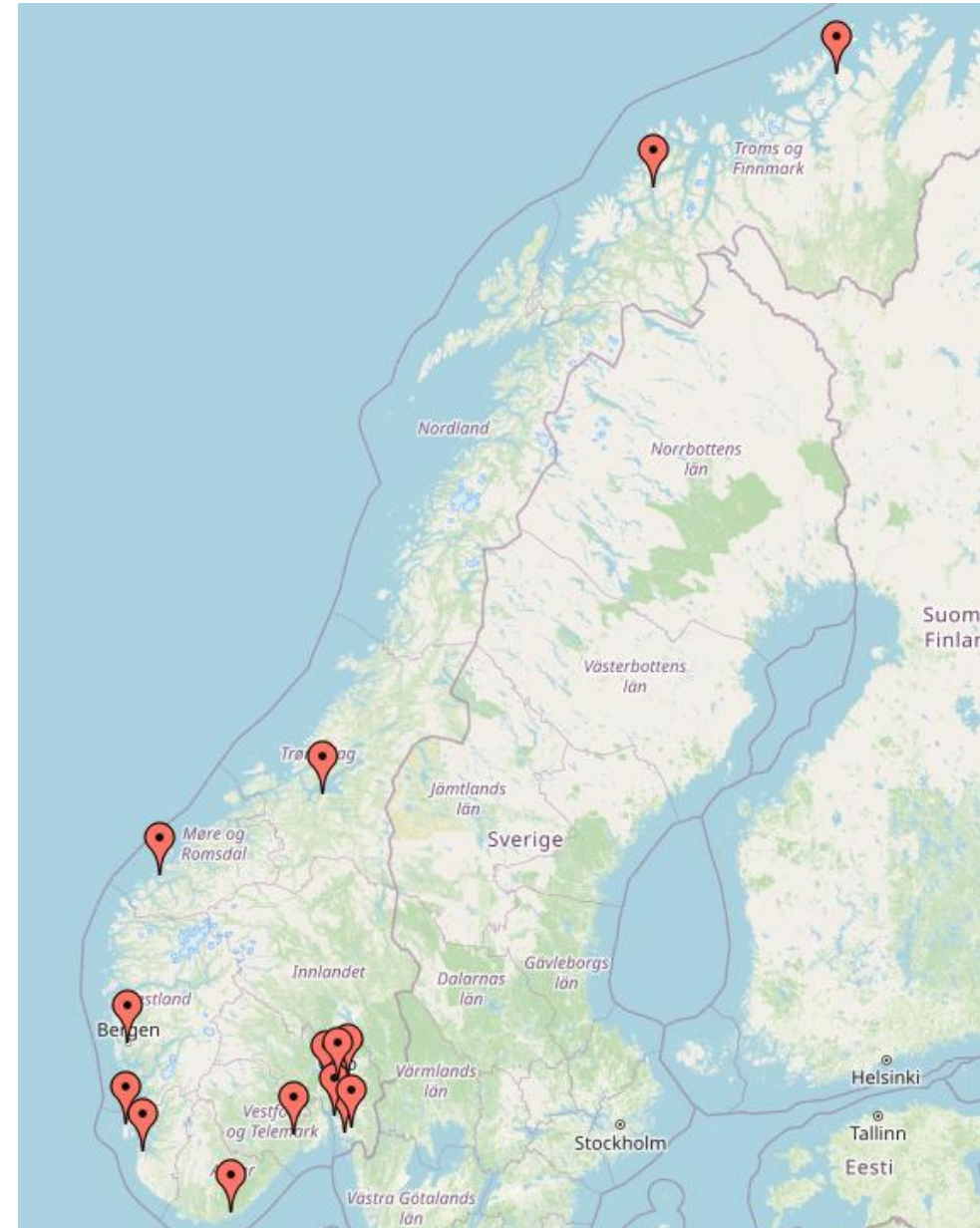
Background

- Firefighters exposed to many known and potential carcinogens
- Previous studies found elevated risk of several cancers
- Findings mixed

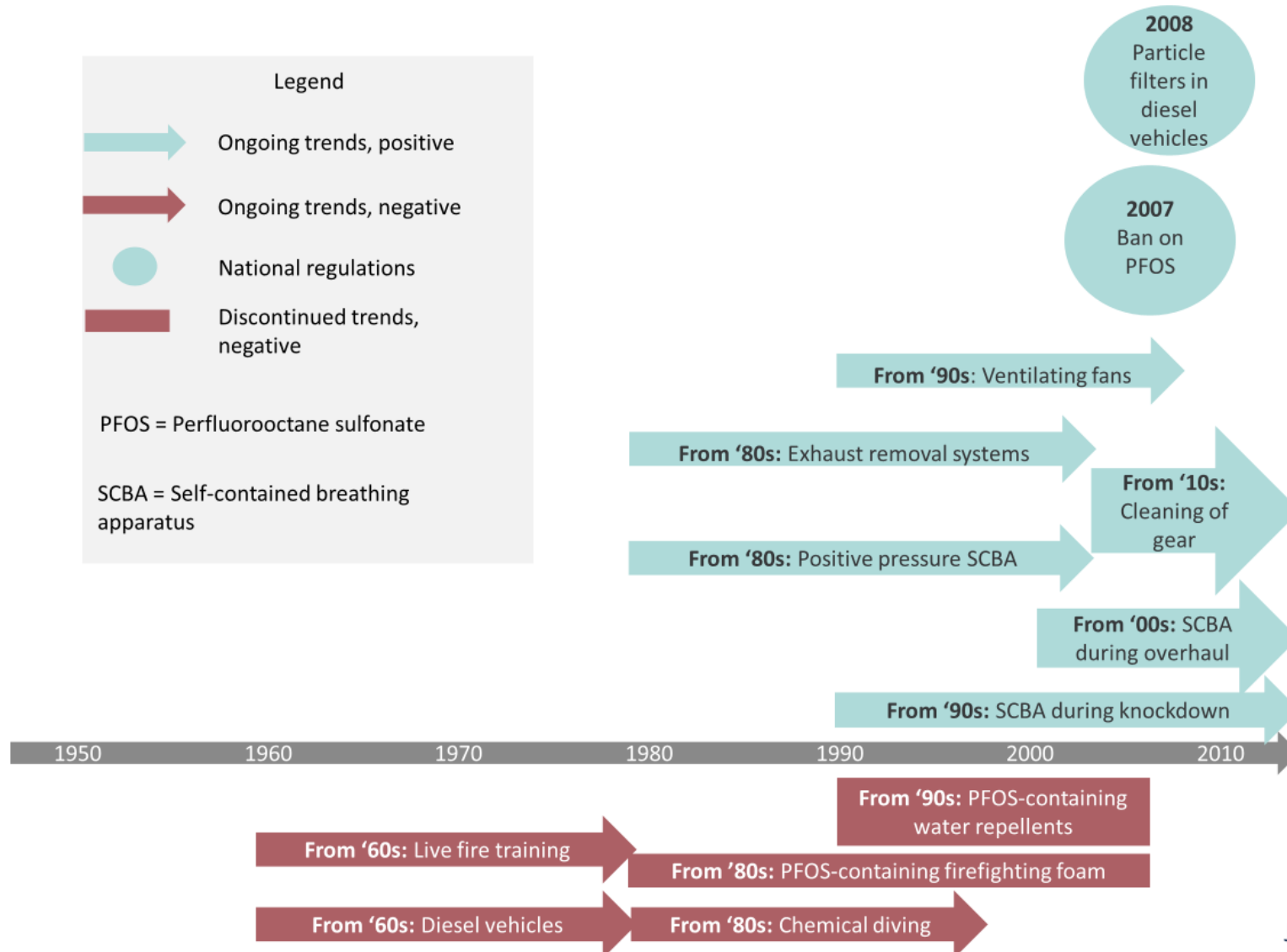


The Norwegian Fire Departments Cohort

- Registration by fire departments
- 15 fire departments
- 1950–2018
- $n = 4627$



Working conditions at Norwegian fire departments, 1950–2018



Jakobsen et al. (2020)

Findings

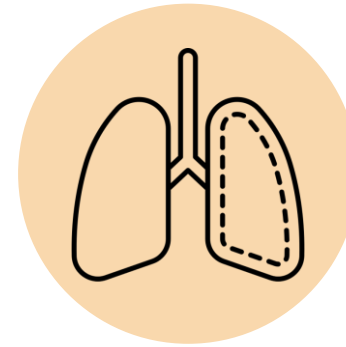


Urinary tract cancer

SIR 1.25 (0.97–1.58), 73 *cases*

SMR 1.13 (0.64–1.83), 16 *deaths*

Benzo[a]pyrene, diesel exhaust



Mesothelioma

SIR 2.59 (1.12–5.11), 8 *cases*

SMR 2.77* (0.90–6.47), 5* *deaths*

Asbestos

- Elevated risks observed associated with firefighters' occupational exposures
- Period-based differences may reflect changes in exposures from personal protective equipment (PPE)

Findings



Larynx cancer

SIR 1.77 (0.94–3.03)

13 cases

SMR 2.22 (0.72–5.17)

5 deaths



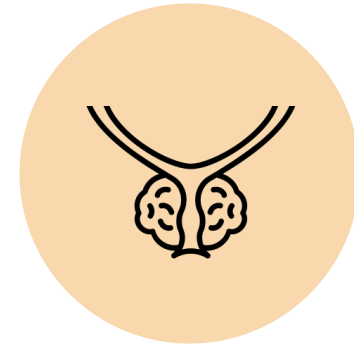
Colon cancer

SIR 1.27 (1.02–1.58)

82 cases

SMR 1.20 (0.84–1.67)

35 deaths



Prostate cancer

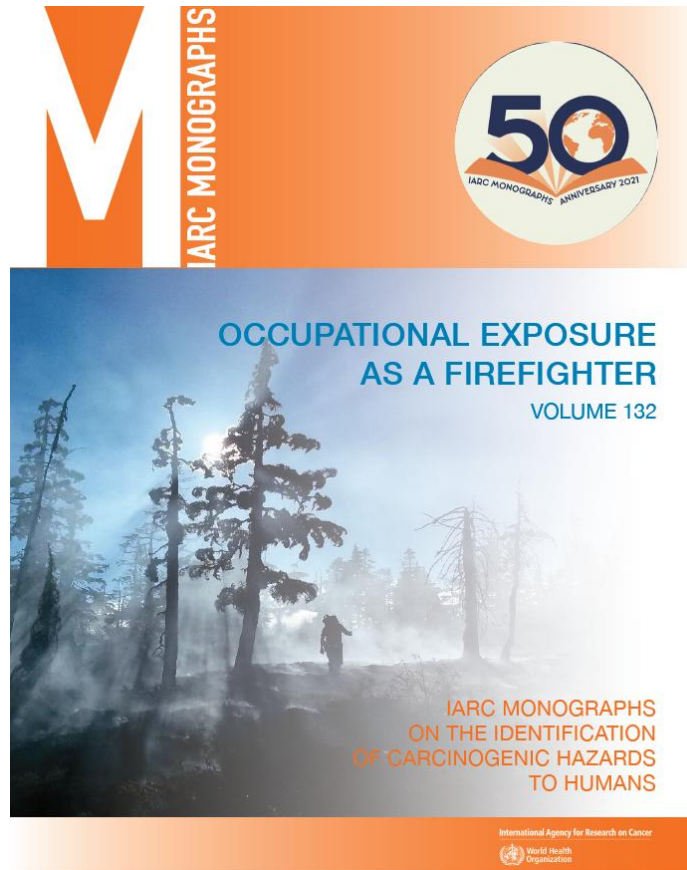
SIR 1.18 (1.03–1.34)

231 cases

SMR 1.01 (0.76–1.31)

55 deaths

IARC Monograph v. 132



International Agency for Research on Cancer
World Health Organization

IARC MONOGRAPHS VOL. 132: OCCUPATIONAL EXPOSURE AS A FIREFIGHTER

Occupational exposure as a firefighter is **carcinogenic to humans (Group 1)** on the basis of **sufficient evidence** for cancer in humans

GROUP 1 **GROUP 2A** **GROUP 2B** **GROUP 3**

The IARC Monographs classification indicates the level of certainty that an agent can cause cancer (*hazard identification*)

Higher level of certainty Lower level of certainty

Cancer types with **sufficient evidence** for cancer in humans:

Mesothelioma Bladder cancer

Cancer types with **limited evidence** for cancer in humans:

Colon cancer Prostate cancer Testicular cancer Melanoma of the skin Non-Hodgkin lymphoma

Strong mechanistic evidence in exposed firefighters

Genotoxicity Epigenetic alterations Oxidative stress Chronic inflammation Modulation of receptor-mediated effects

Exposures of firefighters include combustion products, diesel exhaust, building materials, asbestos, chemicals, shift work, ultraviolet radiation

Firefighters respond to various types of fire

Structure Wildland Vehicle

Exposures today



Annals of Work Exposures and Health, 2025, **69**, 765–776
<https://doi.org/10.1093/annweh/wxaf031>
Advance access publication 28 June 2025
Original Article



The Chartered
Society for Worker
Health Protection

OXFORD

Contamination of firefighters' merino wool and mixed fibre sweater and hood undergarments with polycyclic aromatic hydrocarbons

Cecilie Rosting, Hilde P. Notø*, Dag G. Ellingsen, Thea H. Johansen, Raymond Olsen

National Institute of Occupational Health, Department of Chemical Work Environment, P.O. Box 5330, Gydas vei 8, OSLO N-0304, Norway

*Corresponding author: Email: Hilde.Noto@stami.no

- Trace levels of PAHs found on undergarments
- Highest = forehead, upper chest



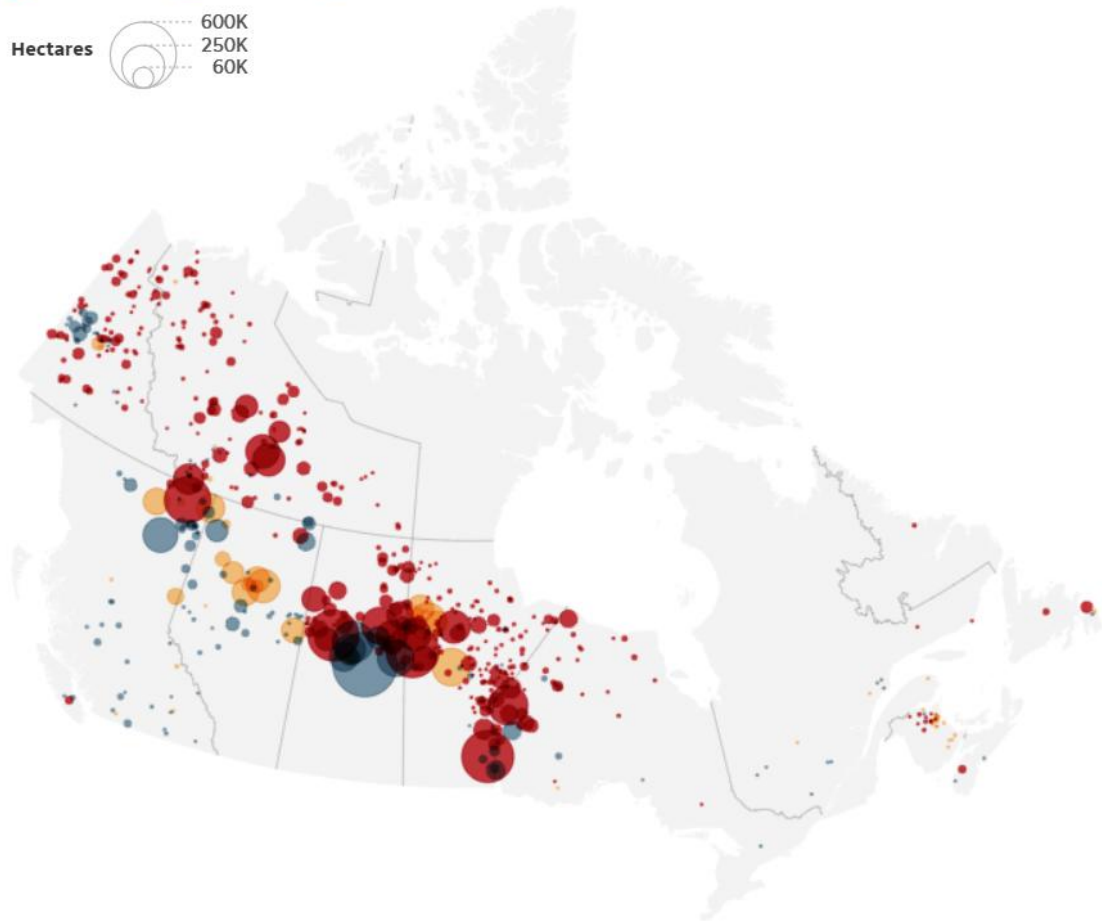
Photo: OBRE/Flickr

Canada, July 2025

Wildfires in Canada

■ Out of control ■ Being held ■ Under control

Hectares
600K
250K
60K



Toronto, Ontario

Take home messages

- Firefighters' cancer risk →
 - Urinary tract cancer, mesothelioma risk associated with occupational exposures
- Occupational exposures today are lower
- Hazards, risks are changing
- Importance of risk identification, reduction, and management for effective cancer prevention – for firefighters, us, and the planet



Thank you

Exposure to dust and fibres, and risk of respiratory cancers in the Norwegian Offshore Petroleum workers cohort

Leon Alexander McLaren Berge, PhD

Oslo Center for Biostatistics and Epidemiology, University of Oslo

Department of Research, Cancer Registry of Norway,
Norwegian Institute of Public Health

l.a.m.berge@medisin.uio.no



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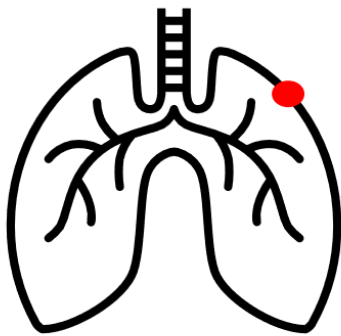


The Research
Council of Norway



Pleural mesothelioma

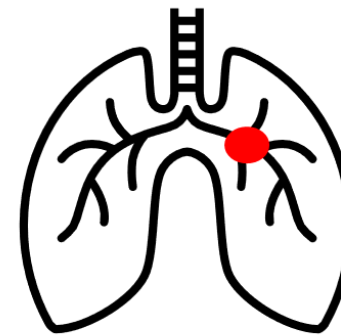
- Uncommon respiratory cancer, but poor prognosis.
- Asbestos inhalation is the primary etiological factor.
- A 138% increased risk in offshore workers vs Norwegian population.



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Lung cancer

- Second-most common malignancy in Norway, with wide array of risk factors (primarily tobacco)
- Dose-dependent associations with many exposures common to occupational settings (asbestos, silica dust etc.)



Cancer
Registry of Norway



Part of the Norwegian
Institute of Public Health

Asbestos

Classification:

Group 1 carcinogen (IARC)

Description: Small mineral fibres used in insulation, building materials, brakes etc. Replaced with refractory ceramic fibres (RCF) in 1985.

Increases risk of: Pleural mesothelioma, Lung cancer, asbestosis etc.

Offshore exposure:

- Additives in drilling mud (mud mixing)
- Insulation for living quarters and gaskets
- Brake bands

Crystalline silica dust

Classification:

Group 1 carcinogen (IARC)

Description: Small mineral particles produced from processing stone, concrete etc.

Increases risk of: Lung cancer, COPD, silicosis etc.

Offshore exposure:

- Dry additives in drilling mud (mud mixing)
- Cementing
- Sandblasting

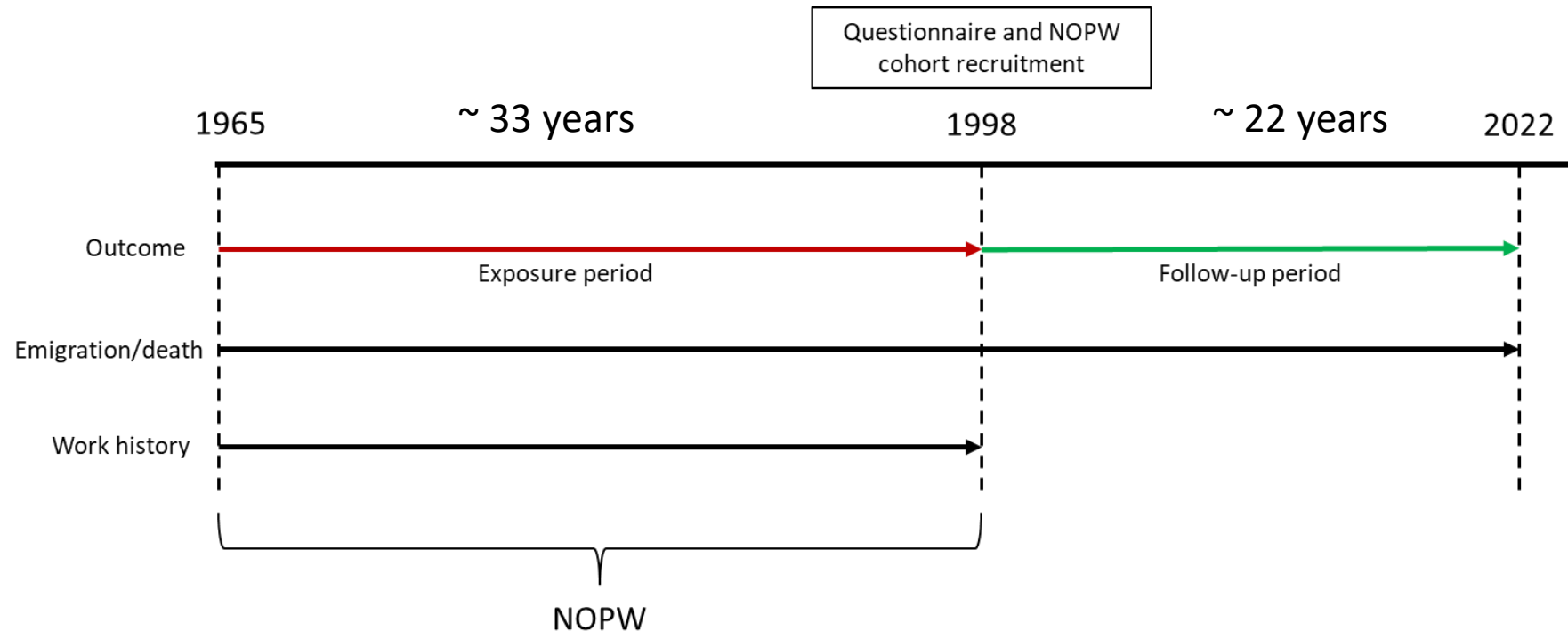
Study design

4/11

Case-cohort study:

Pleural mesothelioma: 43 cases and 2138 randomly drawn non-cases from the NOPW cohort

Lung cancer: 475 cases, and 2015 randomly drawn non-cases from the NOPW cohort



Person-years generated from start of follow-up (1 July 1999) to date of pleura/lung cancer diagnosis, emigration, death or end of follow-up (31 December 2021)



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Pleural mesothelioma

Statistical analyses:

Weighted Cox regression to estimate hazard ratios (HRs) and 95% confidence intervals (CIs).

Adjusted for: Age at baseline, and pre-offshore employment with potential asbestos exposure.

Multiple imputation of missing variables

Lung cancer

Statistical analyses:

Cause-specific cumulative incidence ratios with death as a competing event.

Adjusted for: Age at baseline, smoking, first year of offshore employment, CSD, asbestos, and welding fume exposure using inverse probability of treatment weights.

Pleural mesothelioma – Avg. int. of asbestos

6/11

Average intensity of asbestos exposure	No. cases/non-cases	HR (95% CI)
0	3/460	0.29 (0.08 – 1.07)
T1 (>0 – 0.9)	13/554	1.00 (reference)
T2 (0.9 – 1.3)	11/539	0.86 (0.38 – 1.93)
T3 (≥ 1.3)	16/542	1.16 (0.55 – 2.47)
P-trend		0.034



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Pleural mesothelioma – Pre-offshore employment

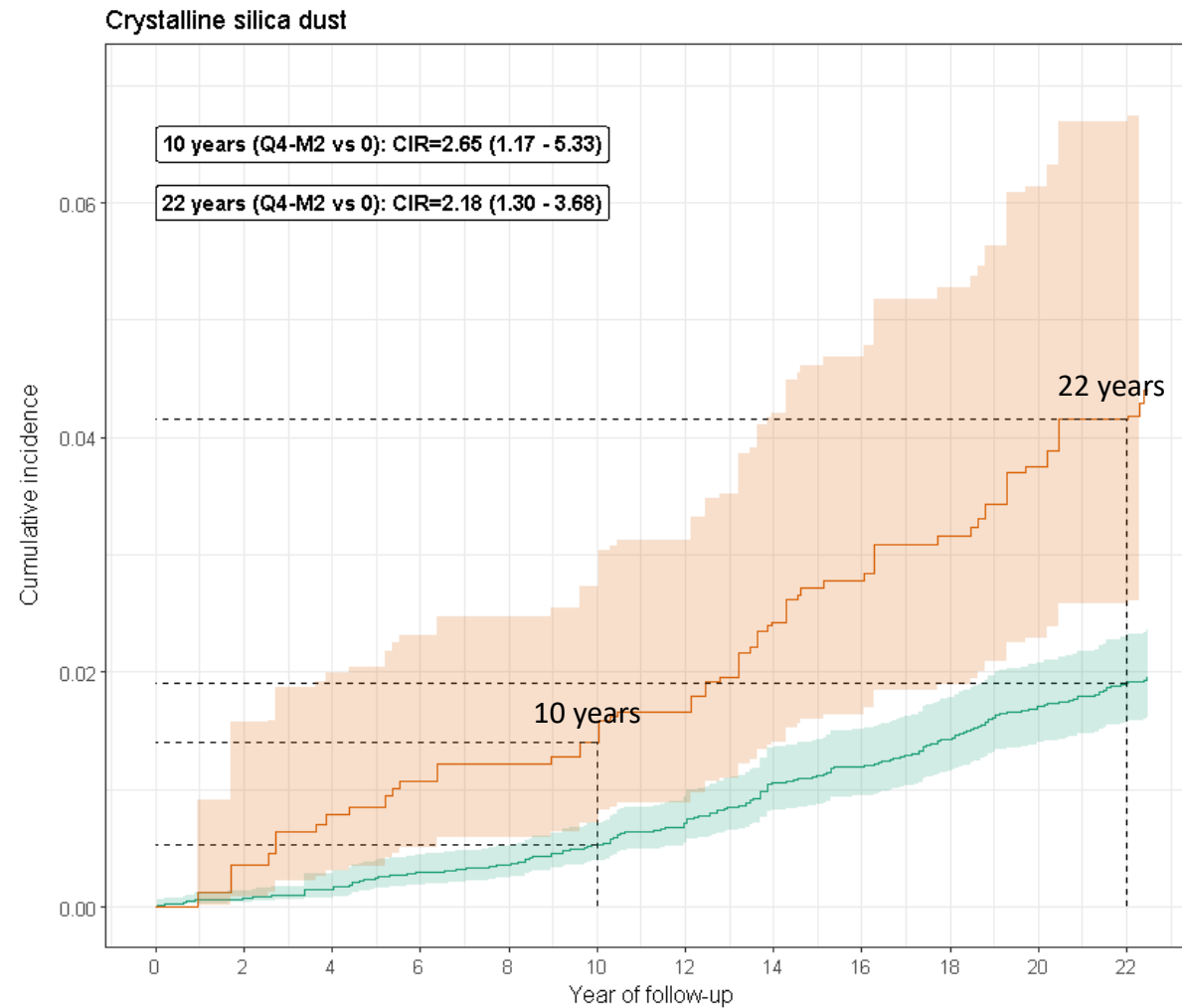
7/11

Pre-offshore employment ^{a, b}	Cases/Non-cases	HR (95% CI) ^c
No	21/1415	1.00 (reference)
Yes	22/680	2.24 (1.22 – 4.10)

Pre-offshore employment with asbestos exposure ^a				
	No		Yes	
Average intensity of asbestos exposure ^{b, d}	Cases/Non-cases	HR (95% CI) ^c	Cases/Non-cases	HR (95% CI) ^c
Unexposed	1/383	0.28 (0.03 – 2.60)	2/77	0.49 (0.10 – 2.36)
T1 (>0 – 0.9)	3/347	1.00 (reference)	10/207	1.00 (reference)
T2 (0.9 – 1.3)	6/350	1.98 (0.49 – 7.97)	5/189	0.51 (0.17 – 1.56)
T3 (≥1.3)	11/335	3.48 (0.99 – 12)	5/207	0.46 (0.15 – 1.44)
P-trend ^e		<0.001		0.551

Lung cancer – Cumulative CSD exposure

8/11

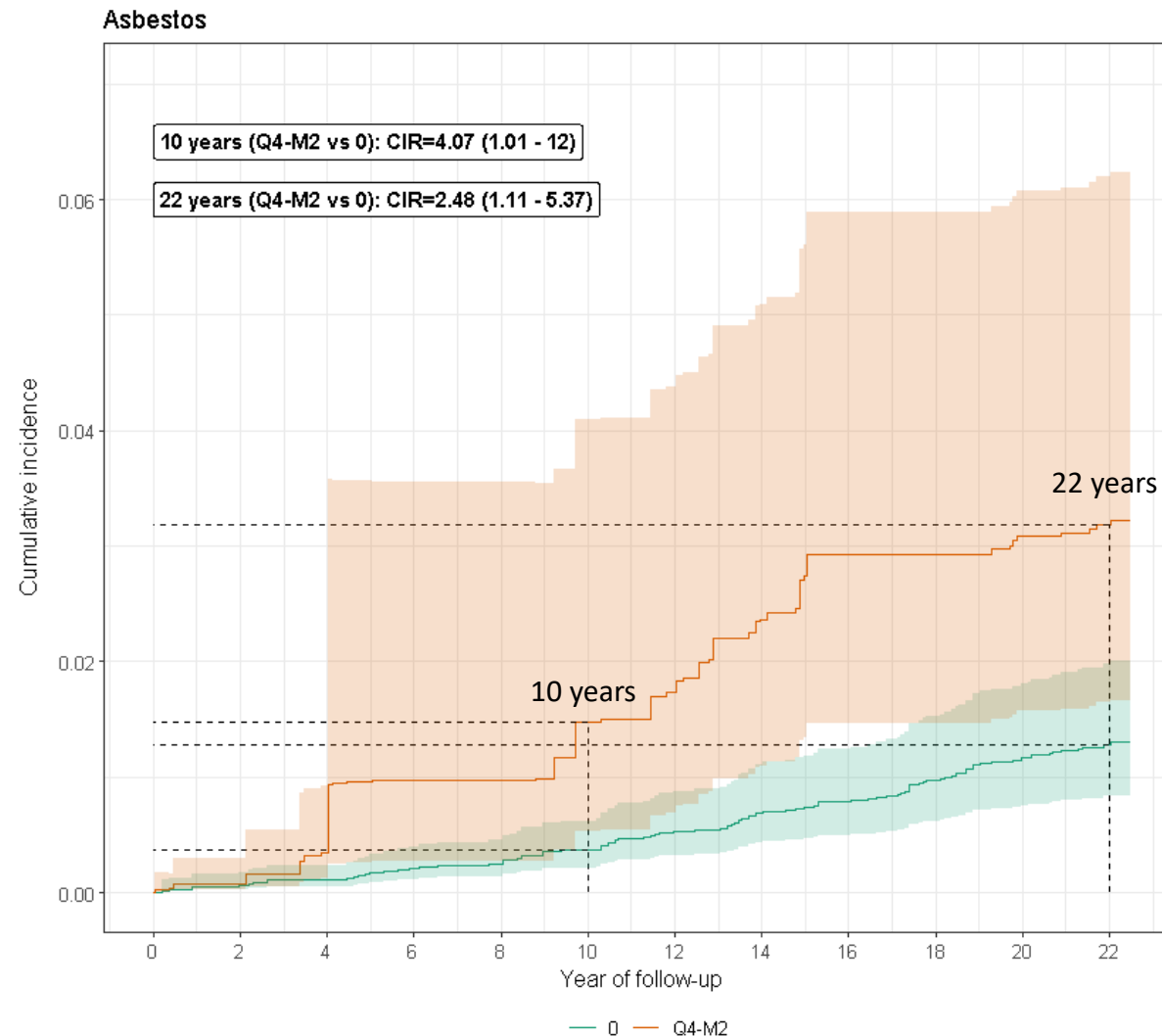


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At risk (cumulative events)							
0	1252 (0)	1189 (28)	1097 (63)	959 (121)	774 (195)	706 (219)	
Q4-M2	141 (0)	132 (7)	117 (14)	95 (29)	78 (38)	70 (42)	

Lung cancer – Cumulative asbestos exposure

9/11



At risk (cumulative events)

0	521 (0)	491 (13)	450 (28)	395 (56)	310 (87)	287 (96)
Q4-M2	237 (0)	216 (10)	199 (14)	168 (31)	143 (38)	129 (41)



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- Workers with increasing average intensity of asbestos exposure offshore showed increased risk of pleural mesothelioma.
- No association between offshore asbestos exposure and pleural mesothelioma in workers with pre-offshore jobs.
- High cumulative exposure to CSD and asbestos may increase the probability of lung cancer in offshore petroleum workers.

A special thanks

11/11

- Jo S Stenehjem ^{1,2}
- Nita K Shala ^{1,2}
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- Niki Marjerrison^{1,2}
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- Jorunn Kirkeleit ^{4,5}
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- **8.** Occupational and Environmental Epidemiology Branch, Division of Cancer Epidemiology and Genetics, National Cancer Institute, Bethesda, MD, United States.



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Results – Duration in high-intensity jobs

7/10

Duration of asbestos exposure in high-intensity jobs ^{a, b, c}	No. cases/non-cases	HR (95% CI)
0	3/460	0.26 (0.04 – 1.68)
>0 – 4.2	3/111	1.00 (reference)
≥4.2	5/108	1.98 (0.37 – 11)
P-trend ^d		0.030

a Categorized into unlikely exposed (0) and by median among higher exposed based on a 1970–1985 ranking of job-categories in the asbestos JEM.

b Defined as employment in the following job categories: Derrick employees, Machinists, and Insulators.

c Adjusted for age at baseline (cont.) and other employment prior to offshore work (yes/no).

d Cox regression adapted to a case cohort design with calendar time as the time scale.

e Modelled as a continuous variable to test for linear trend.

Compared to <median duration of asbestos exposure in high-intensity jobs, there was an increased risk of pleural mesothelioma for:

- ≥median duration of asbestos exposure
- Positive linear trend

Results – Asbestos (Exposed job categories and JEM-based metrics)

Duration (years) in offshore job categories with high/low asbestos exposure

Duration (years) in offshore job categories with asbestos exposure	Cases/Non-cases	HR (95% CIs)
Job categories with higher exposure		
0	3/460	0.26 (0.04 – 1.68)
>0 – 4.2	3/111	1.00 (reference)
>=4.2	5/108	1.98 (0.37 – 11)
<i>P</i> -trend		0.030
Job categories with lower exposure		
0	3/460	0.32 (0.08 – 1.25)
>0 – 7.2	10/445	1.00 (reference)
>=7.2	11/447	1.04 (0.43 – 2.50)
<i>P</i> -trend		0.191
Higher and lower exposed job categories combined		
0	3/460	0.29 (0.07 – 1.10)
>0 – 8.1	12/502	1.00 (reference)
>=8.1	15/498	1.19 (0.54 – 2.62)
<i>P</i> -trend		0.071

JEM-based (cumulative, duration, avg. int.) asbestos exposure

JEM-based asbestos exposure	Cases/Non-cases	HR (95% CIs)
Cumulative asbestos exposure		
0	3/460	0.23 (0.06 – 0.83)
>0 – <7.2	16/543	1.00 (reference)
7.2 – <17.0	11/547	0.66 (0.30 – 1.44)
>17.0	13/545	0.75 (0.35 – 1.59)
<i>P</i> -trend		0.598
Duration (years) of asbestos exposure		
0	3/460	0.27 (0.07 – 1.02)
>0 – <7.0	14/544	1.00 (reference)
7.0 – <15.1	15/544	1.06 (0.51 – 2.24)
>15.1	11/547	0.77 (0.35 – 1.72)
<i>P</i> -trend		0.434

Results – Asbestos (Lagged analyses)

Duration (years) in offshore job categories with asbestos exposure

Job categories with higher exposure	Person-years	Cases/Non-cases	HR (95% CI)
10-year lag			
0	8893.04	3/460	0.26 (0.04 – 1.64)
>0 – 4.2	2329.67	3/111	1.00 (reference)
>=4.2	1998.67	5/108	2.01 (0.38 – 11)
P-trend			0.026
15-year lag			
0	9049.06	3/460	0.26 (0.04 – 1.62)
>0 – 4.2	2339.03	3/113	1.00 (reference)
>=4.2	1833.30	5/106	2.13 (0.41 – 11)
P-trend			0.020
20-year lag			
0	9435.24	3/461	0.25 (0.04 – 1.44)
>0 – 4.2	2334.35	3/113	1.00 (reference)
>=4.2	1451.79	5/105	2.49 (0.49 – 13)
P-trend			0.010

Average intensity of asbestos exposure

Average intensity of exposure			
10-year lag	Person-years	Cases/Non-cases	HR (95% CI)
0	9600	3/462	0.30 (0.08 – 1.10)
>0 – <0.9	10 604	12/545	1.00 (reference)
0.9 – 1.3	10 423	12/545	1.01 (0.46 – 2.23)
>=1.3	10 704	16/543	1.23 (0.57 – 2.62)
P-trend			0.031
15-year lag	Person-years	Cases/Non-cases	HR (95% CI)
0	11 455	3/463	0.24 (0.07 – 0.84)
>0 – <0.9	9213	13/545	1.00 (reference)
0.9 – 1.3	9396	11/533	0.84 (0.37 – 1.90)
>=1.3	11 267	16/554	1.02 (0.48 – 2.20)
P-trend			0.027

Pre-offshore job

POSITIONS BEFORE/AFTER OFFSHORE AND IN OFF-DUTY PERIODS

35. What type of positions outside the offshore sector have you held? Please classify each position according to the corresponding industry sectors:

- | | | | |
|----------------------------------|---------------------------------------|------------------------------|-------------------------------|
| 1 = Shipping, <i>bridge/deck</i> | ● 6 = Chemical industry | ● 11 = Eletrical occupations | 16 = Academics |
| ● 2 = Shipping, <i>machinist</i> | ● 7 = Heavy industry/works/mech. ind. | 12 = Health/social services | 17 = Military |
| 3 = Shipping, <i>other</i> | ● 8 = Other industry | 13 = Trade/office services | 18 = Transport |
| 4 = Fishing industry | ● 9 = Building and construction | 14 = Hotel and restaurant | 19 = Other (specify in table) |
| 5 = Farming/forestry | ● 10 = Painting/surface treatment | 15 = Housework | |

In the table below, please type the *number* corresponding to the industrial sector for each position, and add information on start and stop for this position, and specify occupational title or type of work. You may merge several short-term positions within the same sector when one position followed the other, and no more than a one-year gap was in-between. State whether you worked full-time or part-time, or if you worked during off-duty periods.



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Lung cancer results – CSD Cumulative dose

8/12

Cumulative CSD exposure	No. cases/non-cases	HR (95% CI)
0	224/1028	1.00 (reference)
Q1 (>0 – <2.0)	54/237	1.10 (0.77 – 1.57)
Q2 (2.0 – <4.71)	50/241	0.73 (0.50 – 1.07)
Q3 (4.71 – <9.48)	60/230	0.83 (0.56 – 1.22)
Q4-M1 (9.48 – <13.1)	28/116	0.64 (0.37 – 1.13)
Q4-M2 (≥13.1)	46/95	1.31 (0.81 – 2.11)
P-trend		0.469



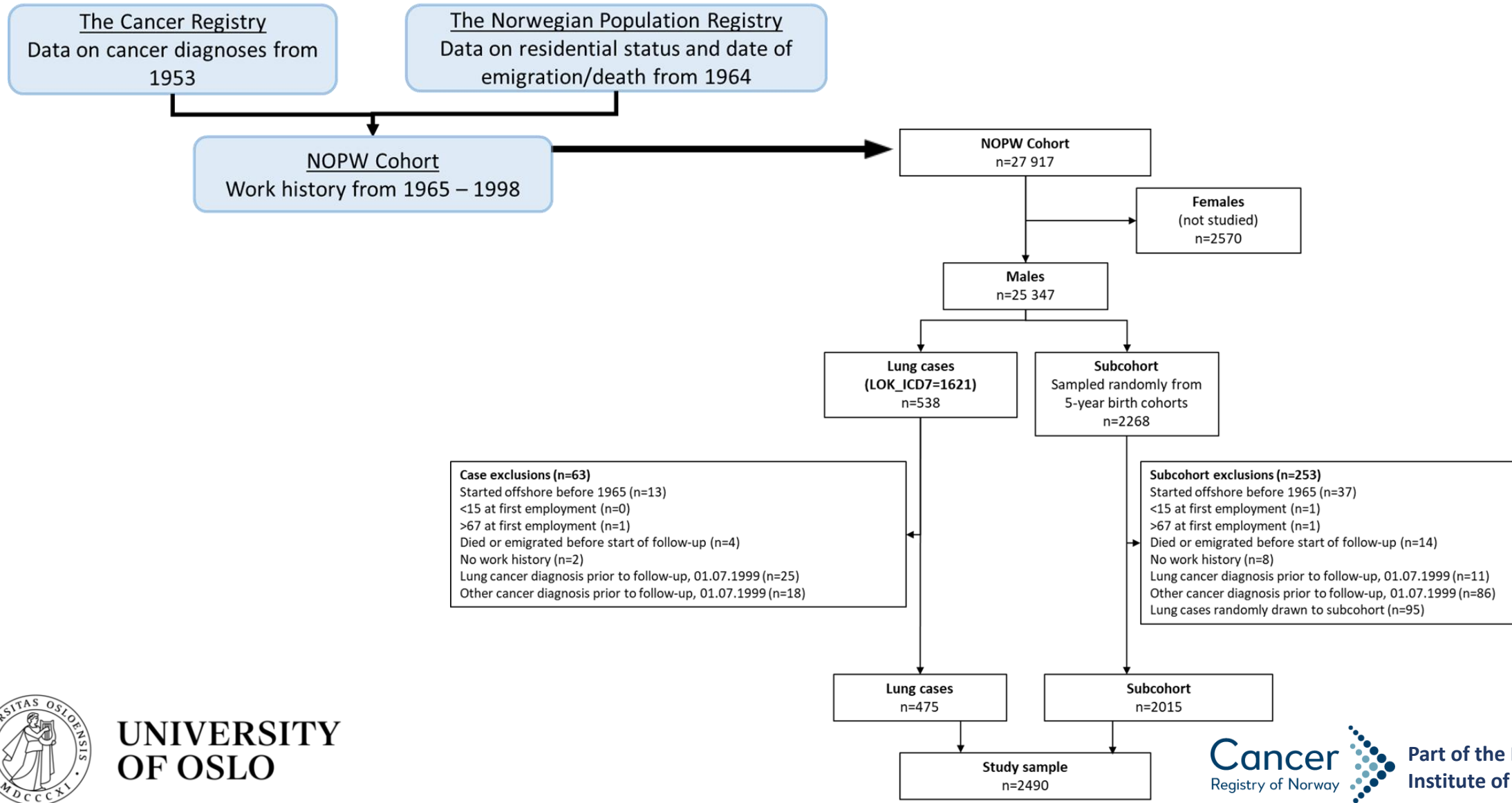
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Results – Duration in high-intensity jobs

Table S4 Hazard ratios (HR) and 95% confidence intervals (95% CI) of lung cancer in the Norwegian Offshore Petroleum Workers cohort (n=27 917), according to a job-exposure matrix of crystalline silica dust, asbestos and refractory ceramic fibres, 1999–2021.

		Model 1 ^a			Model 2 ^b			Model 3 ^c
Duration in job categories with high CSD exposure ^{d, e}	Cases/non-cases	HR (95% CIs) ^f	Cases/non-cases	HR (95% CIs) ^f	Cases/non-cases	HR (95% CIs) ^f	Cases/non-cases	HR (95% CIs) ^f
0	229/1060	1.00 (reference)	224/1028	1.00 (reference)	224/1028	1.00 (reference)		
T1 (>0 – <2.2)	10/52	0.70 (0.35 – 1.41)	10/50	0.55 (0.26 – 1.18)	10/50	0.52 (0.24 – 1.16)		
T2 (>2.2 – <7.4)	20/43	1.66 (0.95 – 2.90)	20/42	1.62 (0.87 – 3.01)	20/42	1.54 (0.82 – 2.93)		
T3-M1 (>7.4 – <13.5)	7/24	1.14 (0.48 – 2.70)	7/23	1.07 (0.43 – 2.70)	7/23	0.99 (0.40 – 2.47)		
T3-M2 (≥13.5)	19/13	7.03 (3.46 – 14)	18/10	7.74 (3.65 – 16)	18/10	6.74 (3.08 – 15)		
P-trend ^g		<0.001		<0.001		<0.001		
Duration in job categories with high asbestos exposure ^{d, h}								
0	99/441	1.00 (reference)	97/424	1.00 (reference)	97/424	1.00 (reference)		
T1 (>0 – <2.5)	18/73	0.96 (0.55 – 1.69)	17/73	0.68 (0.36 – 1.25)	17/73	0.85 (0.38 – 1.89)		
T2 (>2.5 – <8.6)	23/65	1.45 (0.86 – 2.46)	21/62	1.29 (0.72 – 2.32)	21/62	1.35 (0.64 – 2.83)		
T3-M1 (>8.6 – <15.0)	9/36	1.14 (0.53 – 2.45)	9/36	1.06 (0.47 – 2.40)	9/36	0.96 (0.40 – 2.30)		
T3-M2 (≥15.0)	8/36	1.09 (0.49 – 2.43)	8/35	1.38 (0.59 – 3.26)	8/35	1.33 (0.58 – 3.07)		
P-trend ^g		0.566		0.356		0.541		
Duration in job categories with high RCF exposure ^{d, i}								
Never	63/329	1.00 (reference)	62/324	1.00 (reference)	62/324	1.00 (reference)		
Ever	6/11	2.36 (0.82 – 6.74)	6/11	2.40 (0.71 – 8.06)	6/11	1.00 (0.20 – 5.09)		
P		0.110		0.157		1.000		

Study design – Lung cancer



Panel debate - Prevention

15.00 – 15.30 Bridging past and future: translating historical learnings into cancer prevention strategies in modern workplaces

The panel – Kurt Straif, Therese Haugdal Nøst, Paolo Vineis
Moderator: Hilde Langseth

Summary and future perspectives

15.30 – 16.00 **Jo S Stenehjem** - Senior Researcher, Cancer Registry of Norway-NIPH, University of Oslo

Panel debate

Bridging past and future: translating historical learnings into cancer prevention strategies in modern workplaces

Panel debate - Prevention

15.00 – 15.30 Bridging past and future: translating historical learnings into cancer prevention strategies in modern workplaces

The panel – Kurt Straif, Therese Haugdal Nøst, Paolo Vineis
Moderator: Hilde Langseth

Summary and future perspectives

15.30 – 16.00 **Jo S Stenehjem** - Senior Researcher, Cancer Registry of Norway-NIPH, University of Oslo

Panel

Summary and future perspectives

Environment, occupation and cancer: etiology and prevention

Summary and future perspectives

Jo S. Stenehjem, PhD

Senior Researcher

Cancer Registry of Norway, NIPH

Dept of Biostatistics, UiO

Summary: Session 1: Occupational Cancer – Historical Experiences and Current Challenges

Kurt Straif

- Reflected on global efforts to classify carcinogens; emphasized translating evidence into prevention and policy.
- Highlighted the importance of IARC Monographs and international collaboration in shaping occupational cancer research and regulation.

Jo S. Stenehjem

Benzene exposure linked to multiple cancers in offshore workers; associations found for AML, MM, and cancers of the, bladder, lung and colorectal.

Hilde Langseth

Farmers show lower overall cancer rates; Janus Agricultural Cohort enables detailed exposure-cancer analyses.

Summary: Session 2: Chemicals, Environmental Toxins and Cancer – Etiology and Prevention

Therese Haugdahl Nøst

- Longitudinal biomonitoring shows shifting contaminant trends; slower decline of OCPs in T2DM cases suggests disease linkage.
- Demonstrated how birth year, diet, and reproductive history influence contaminant body burden, using advanced modeling and repeated sampling

Marcin Wojewodzie

PFAS exposure studied in occupational and environmental settings; highlighted persistence, bioaccumulation, and regulatory challenges, examples with skiwaxers and firefighters

Tom K. Grimsrud

Nickel exposure historically linked to sinonasal and lung cancer; new studies explore epigenetic mechanisms and low-level risks.

Bente Oftedal

Ecological study of Air pollution (PM_{2.5}, NO₂, BC) increases lung cancer risk even at low levels; findings support stricter air quality standards.

Summary Session 3: Occupational Cancer – Current Situation and Future Risk

Paolo Vineis

- Climate change increases occupational risks; green transition introduces new hazards needing research and regulation (heat induced injuries, snake bites in farmers).
- Called for investment in occupational health surveillance and emphasized co-benefits of climate action for worker health and equity ('Research Needs' as in Turner et al.).

Niki Marjerrison

Firefighters face elevated cancer risks (bladder and mesothelioma) due to smoke and chemical exposure; research focuses on long-term health outcomes and prevention.

Leon A. McLaren Berge

Strong dose-response between silica dust and lung cancer; asbestos exposure linked to mesothelioma in offshore workers.

Future perspectives

- Integration of environmental and occupational epidemiology through – soil, air, water pollution
- Investigate cancer site with largely unknown etiology – also encompassing new methods, exposome
- Collect data on loss of biodiversity – rapid acceleration
- Smart study design – repeated measurements in individuals
- Still need for classic occupational cohort studies and extended follow-up – think new to fund old
- Exposure assessment and data collection in environmental epidemiology
- Integrate biological samples like those in Tromsø, Janus and UK biobank, others
- Network for collaboration and recruitment of master and PhD students to occupational and environmental epidemiology. → Collaboration across institutions in Norway? Is there a need and interest to meet more often?

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