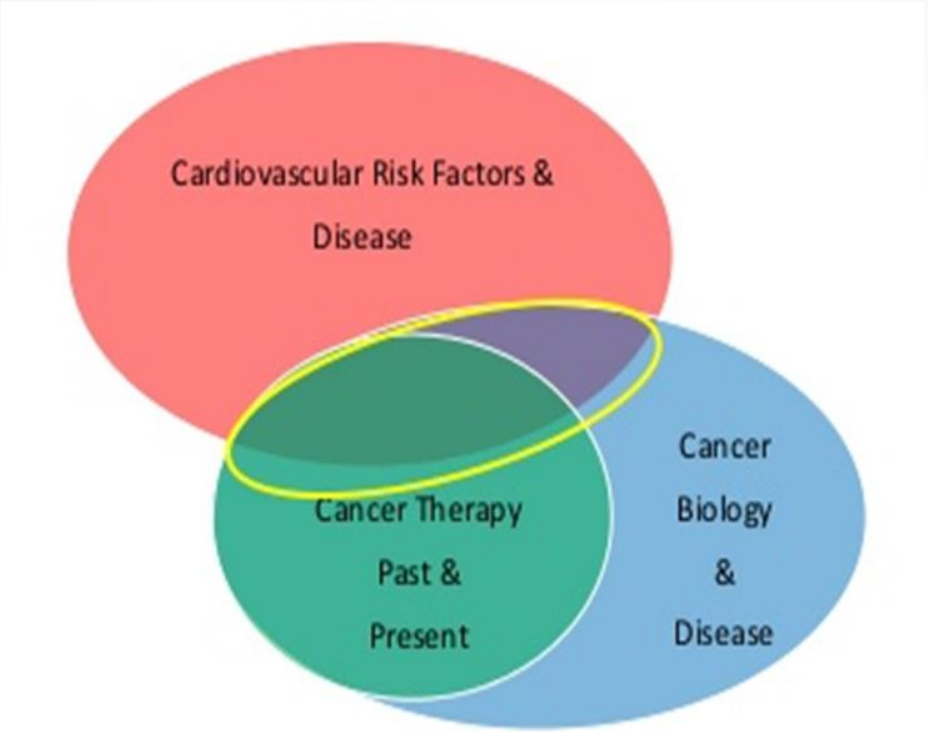
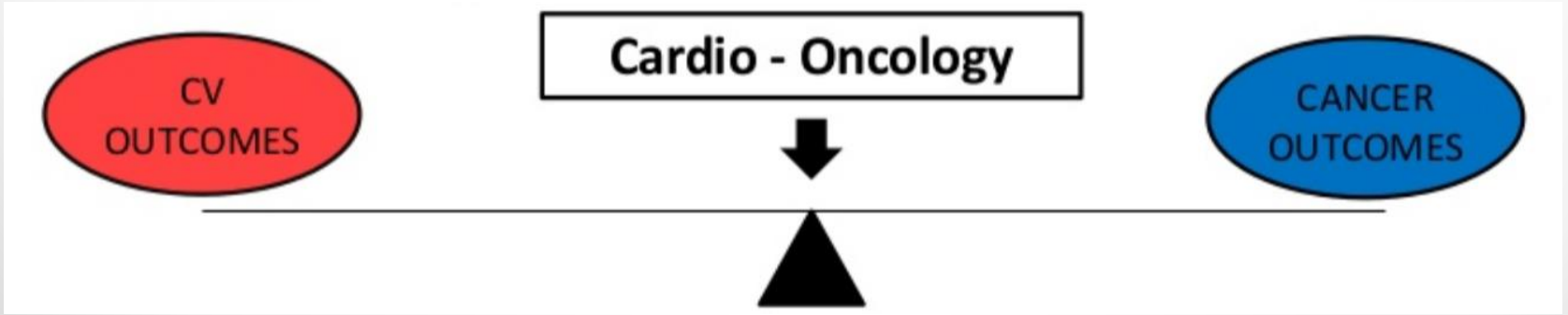
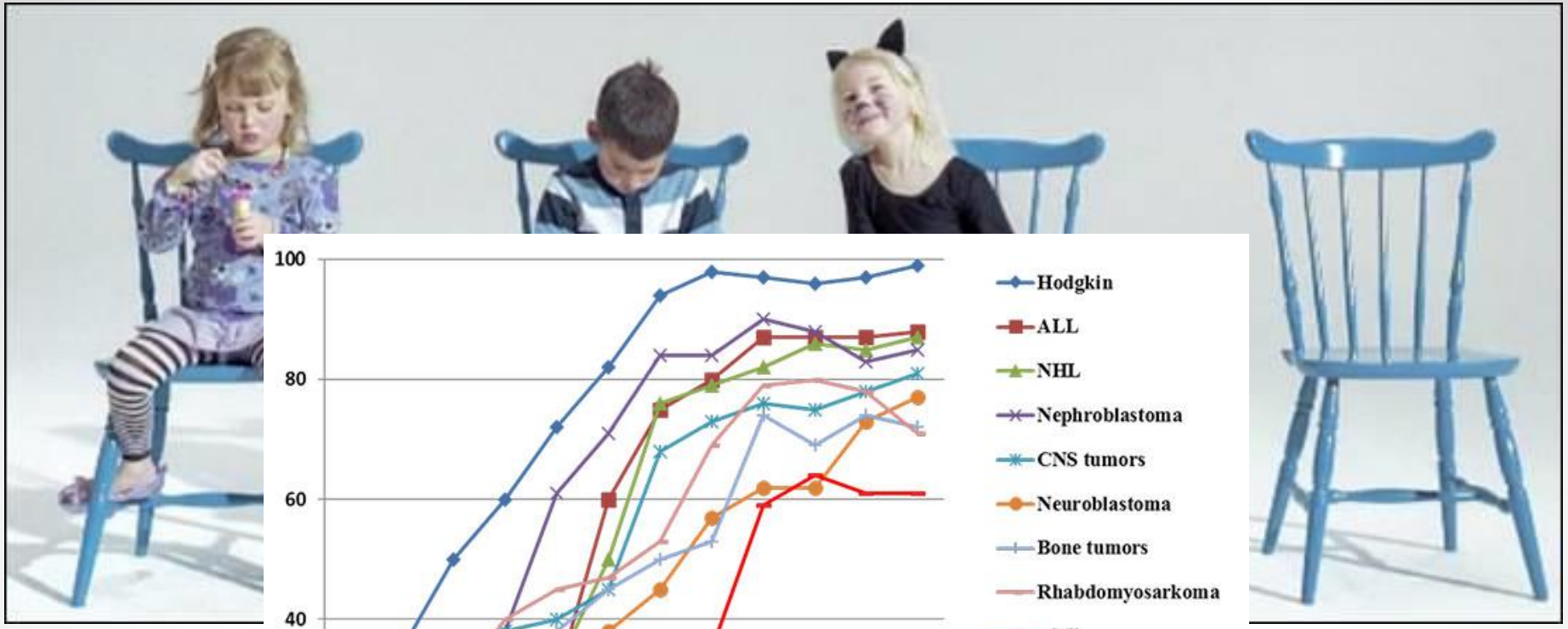
När hjärtat sviktar av cancerbehandling


Cardiovascular Risk Factors & Disease
Cancer Therapy Past & Present
Cancer Biology & Disease


Cardio - Oncology
CV OUTCOMES
CANCER OUTCOMES



Källa: Svenska Barncancerregistret (Margareta af Sandeberg 2015-11-24)

Kardiotoxicitet

Direkt hjärtskada

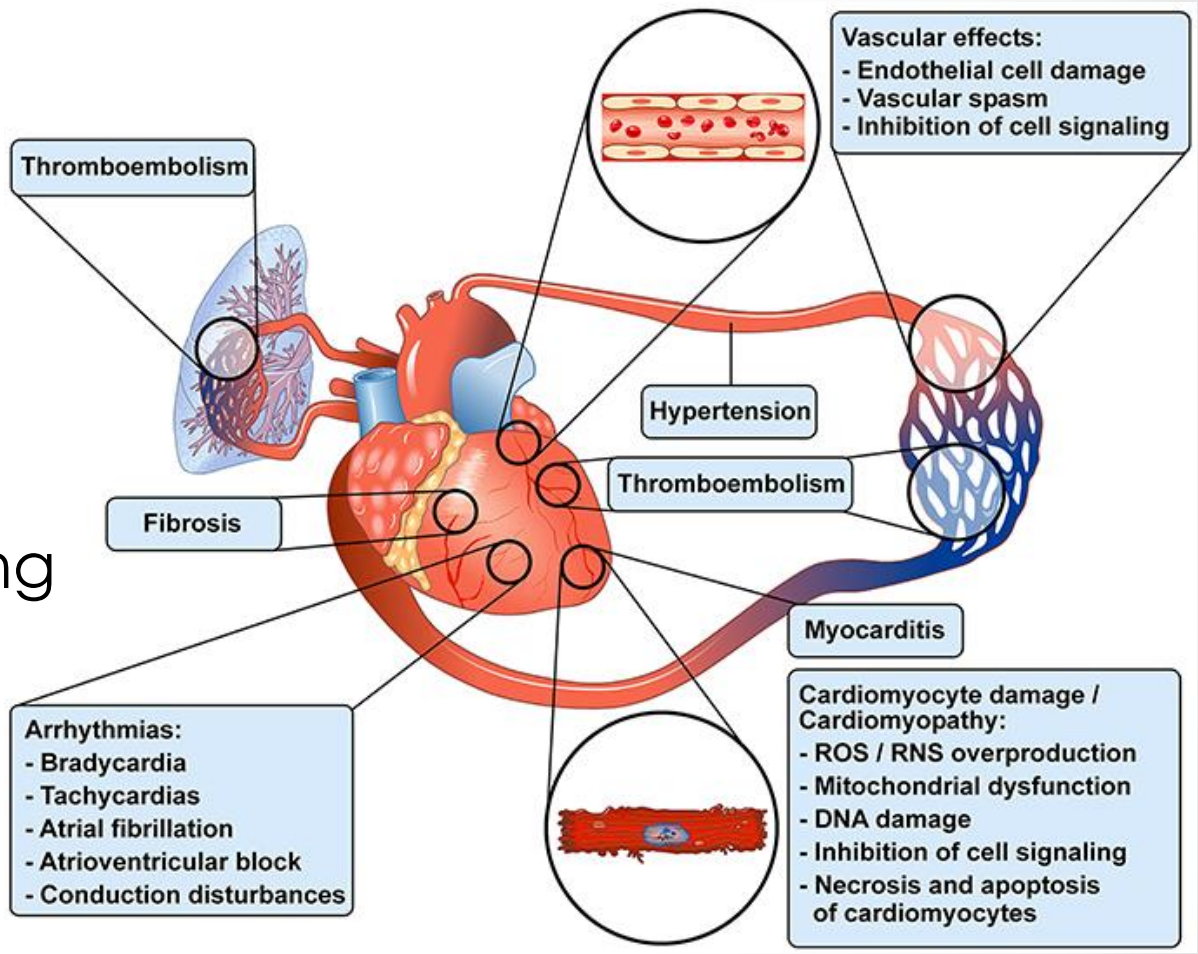
- Akut/subakut cellskada
- Muskel/kranskärl/klaffar

Koagulationsrubbning

Endotelskada

Högt blodtryck

Arytmier



European Heart Journal (2016) 37, 2768–2801
doi:10.1093/eurheartj/ehw211

ESC CPG POSITION PAPER

2016 ESC Position Paper on cancer treatments and cardiovascular toxicity developed under the auspices of the ESC Committee for Practice Guidelines

The Task Force for cancer treatments and cardiovascular toxicity of the European Society of Cardiology (ESC)

Table 6 Proposed diagnostic tools for the detection of cardiotoxicity

Technique	Currently available diagnostic criteria	Advantages	Major limitations
Echocardiography: **- 3D-based LVEF** **- 2D Simpson's LVEF** **- GLS**	• LVEF: >10 percentage points decrease to a value below the LLN suggests cardiotoxicity. • GLS: >15% relative percentage reduction from baseline may suggest risk of cardiotoxicity.	• Wide availability. • Lack of radiation. • Assessment of haemodynamics and other cardiac structures.	• Inter-observer variability. • Image quality. • GLS: inter-vendor variability, technical requirements.
Nuclear cardiac imaging (MUGA)	• >10 percentage points decrease in LVEF with a value <50% identifies patients with cardiotoxicity.	• Reproducibility.	• Cumulative radiation exposure. • Limited structural and functional information on other cardiac structures.
Cardiac magnetic resonance	• Typically used if other techniques are non-diagnostic or to confirm the presence of LV dysfunction if LVEF is borderlines.	• Accuracy, reproducibility. • Detection of diffuse myocardial fibrosis using T1/T2 mapping and ECVF evaluation.	• Limited availability. • Patient's adaptation (claustrophobia, breath hold, long acquisition times).
Cardiac biomarkers: **- Troponin I** **- High-sensitivity Troponin I** **- BNP** **- NT-proBNP**	• A rise identifies patients receiving anthracyclines who may benefit from ACE-Is. • Routine role of BNP and NT-proBNP in surveillance of high-risk patient needs futher investigation.	• Accuracy, reproducibility. • Wide availability. • High-sensitivity.	• Insufficient evidence to establish the significance of subtle rises. • Variations with different assays. • Role for routine surveillance not clearly established.

ACE-Is = angiotensin converting enzyme inhibitors; BNP = B-type natriuretic peptide; ECVF = extracellular volume fraction; GLS = global longitudinal strain; LV = left ventricular; LLN = lower limit of normality; LVEF = left ventricular ejection fraction; MUGA = multigated radionuclide angiography; NT-proBNP = N-terminal fragment B-type natriuretic peptide.

Incidence of left ventricular dysfunction associated with chemotherapy drugs

Chemotherapy agents	Incidence (%)
Anthracyclines (dose dependent)	
Doxorubicin (Adriamycin) 400 mg/m² 550 mg/m² 700 mg/m²	 3–5 7–26 18–48
Idarubicin (>90 mg/m²)	5–18
Epirubicin (>900 mg/m²)	0.9–11.4
Mitoxanthone >120 mg/m²	2.6
Liposomal anthracyclines (>900 mg/m²)	2
Alkylating agents	
Cyclophosphamide	7–28
Ifosfa <10 g 12.5-	
Antimetabolites	
Clofarabine	27
Antimicrotubule agents	
Docetaxel	2.3–13
Paclitaxel	<1

Chemotherapy agents	Incidence (%)
Monoclonal antibodies	
Trastuzumab	1.7–20.1
Bevacizumab	1.6–4
Pertuzumab	0.7–1.2
Small molecule tyrosine kinase inhibitors	
Sunitinib	2.7–19
Pazopanib	7–11
Sorafenib	4–8
Dasatinib	2–4
Imatinib mesylate	0.2–2.7
Lapatinib	0.2–1.5
Carfilzomib	11–25
Bortezomib	2–5
Miscellanous	
Everolimus	<1
Temsirolimus	<1

Antracyklin + Trastuzumab 7-34%

European Heart Journal doi:10.1093/eurheartj/ehw211 - EHJ 2016;37:2768-2801

Cancer drug agents associated with cardiac arrhythmias

Type of arrhythmia	Causative drug
Bradycardia	Arsenic trioxide, bortezomib, capecitabine, cisplatin, cyclophosphamide, doxorubicine, epirubicine, 5-FU, ifosfamide, IL-2, methotrexate, mitoxantrone, paclitaxel, rituximab, thalidomide.
Sinus tachycardia	Anthracyclines, carmustine.
Atrioventricular block	Anthracyclines, arsenic trioxide, bortezomib, cyclophosphamide, 5-FU, mitoxantrone, rituximab, taxanes, thalidomide.
Conduction disturbances	Anthracyclines, cisplatin, 5-FU, imatinib, taxanes.
Atrial fibrillation	Alkylating agents (cisplatin, cyclophosphamide, ifosfamide, melphalan), anthracyclines, antimetabolites (capecitabine, 5-FU, gemcitabine), IL-2, interferons, rituximab, romidepsin, small molecule TKIs (ponatinib, sorafenib, sunitinib, ibrutinib), topoisomerase II inhibitors (amsacrine, etoposide), taxanes, vinca alkaloids.
Supraventricular tachycardias	Alkylating agents (cisplatin, cyclophosphamide, ifosfamide, melphalan), amsacrine, anthracyclines, antimetabolites (capecitabine, 5-FU, methotrexate), bortezomib, doxorubicin, IL-2, interferons, paclitaxel, ponatinib, romidepsin.
Ventricular tachycardia/ fibrillation	Alkylating agents (cisplatin, cyclophosphamide, ifosfamide), amsacrine, antimetabolites (capecitabine, 5-FU, gemcitabine), arsenic trioxide, doxorubicin, interferons, IL-2, methothrexate, paclitaxel, proteasome inhibitors (bortezomib, carfilzomib), rituximab, romidepsin.
Sudden cardiac death	Anthracyclines (reported as very rare), arsenic trioxide (secondary to torsade de pointes), 5-FU (probably related to ischaemia and coronary spasm), interferons, nilotinib, romidepsin.

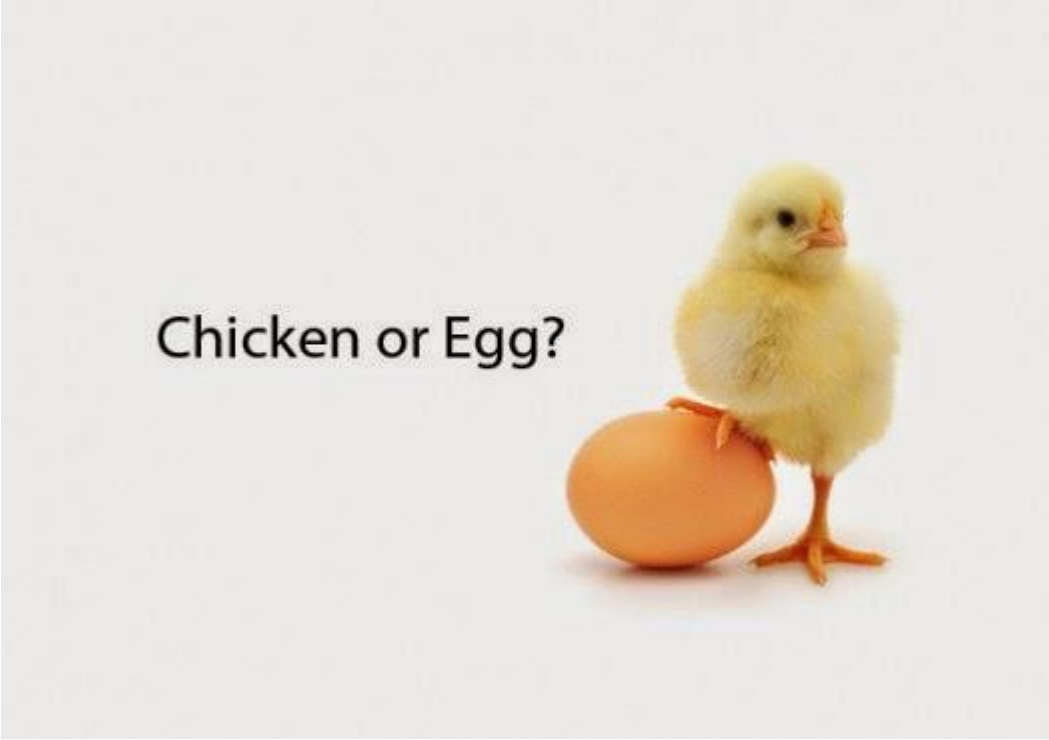
European Heart Journal doi:10.1093/eurheartj/ehw211 - EHJ 2016;37:2768-2801

Cancer drug agents associated with QT prolongation and Torsade de Pointes

Cancer drug agents	Average QT Prolongation (ms)	Increase in QTc >60 ms (%)	QTc >500 ms (%)	Torsade de pointes (%)
Anthracyclines				
Doxorubicin	14	11-14	NA	NA
Histone deacetylase inhibitors				
Depsipeptide	14	20-23.8	NA	NA
Vorinostat	<10	2.7-6	<1	NA
Tyrosin				
Axitinib				
Bosutini				
Cabozar				
Crizotinib	9-13	3.5	1.3	NA
Dasatinib	3-13	0.6-3	<1.4	NA
Lapatinib	6-13	11	6.1	NA
Nilotinib	5-15	1.9-4.7	<1.2	NA
Pazopanib	NA	NA	2	<0.3
Ponatinib	<10	NA	NA	NA
Sorafenib	8-13	NA	NA	NA
Sunitinib	9.6-15.4	1-4	0.5	<0.1
Vandetanib	36	12-15	4.3-8	Described, % NA
Vemurafenib	13-15	1.6	1.6	Described, % NA
Others				
Arsenic trioxide	35.4	35	25-60	2.5

European Heart Journal doi:10.1093/eurheartj/ehw211 - EHJ 2016;37:2768-2801

cancer ⟹ hjärtsvikt
hjärtsvikt ⟹ cancer?



Risken för att utveckla kardiotoxicitet vid onkologisk behandling ökar med:

- Ålder o ärftlighet

- Tidigare hjärtsjukdom
- Njursjukdom

- Strålbehandling över vänstra thorax
- Given kumulativ dos av cytostatika.

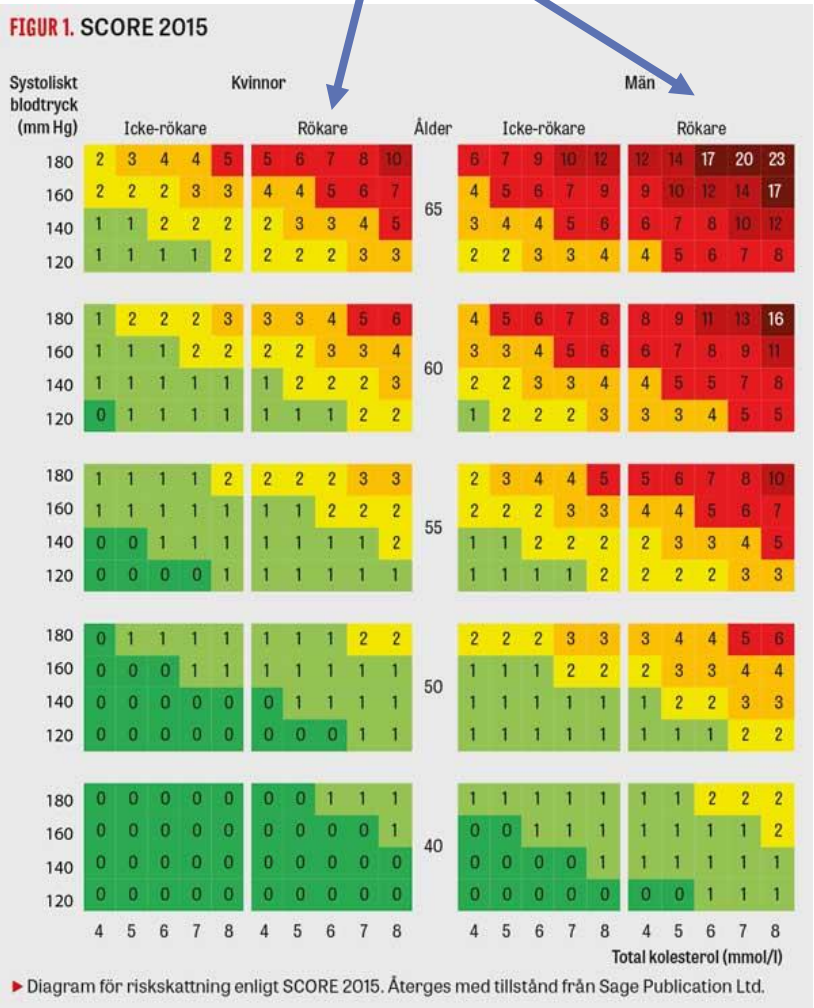
- Kardiovaskulära Riskfaktorer
  - Rökning
  - Obesitas
  - Hypertoni
  - Diabetes
  - Hyperkolesterolemi
  - Fysisk aktivitet

Vanligare med kardiovaskulära riskfaktorer hos cancersjuka.

Kvinnor

Män

Rökare



syst. Blodtryck

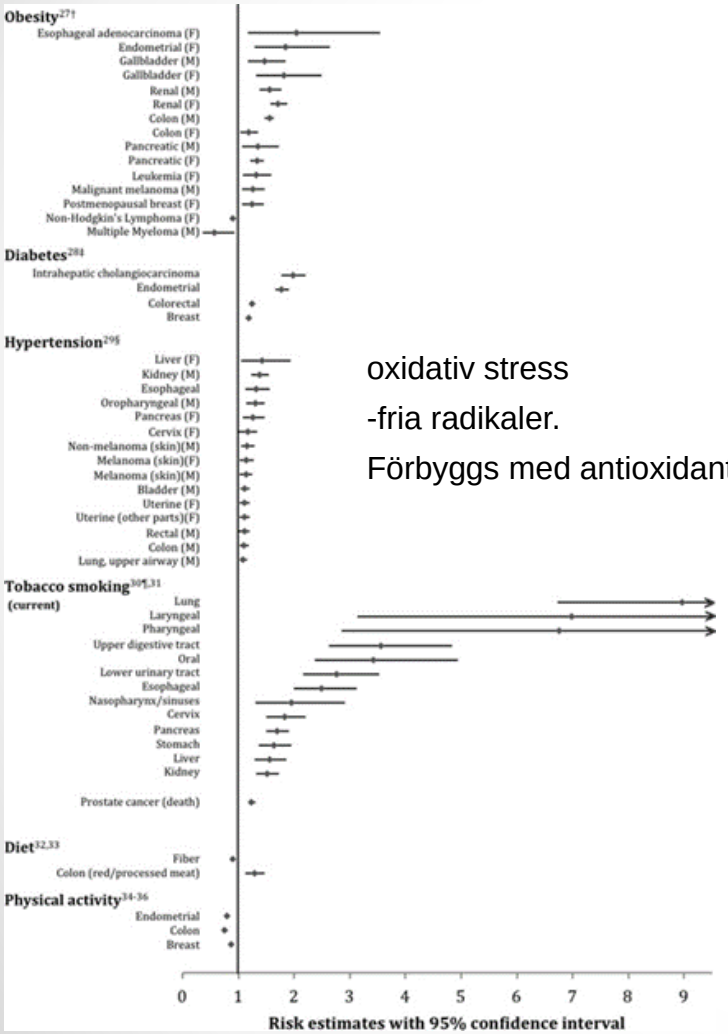
Ålder

Total kolesterol

Gemensamma riskfaktorer

Cardio-oncology Related to Heart Failure
Common Risk Factors Between Cancer and Cardiovascular Disease

Anne Blaes, MD, MS[a,*], Anna Prizment, PhD[b], Ryan J. Koene, MD[c], Suma Konety, MD, MS[d]



oxidativ stress

-fria radikaler.

Förbyggs med antioxidanter

Ålder o kön
10%/10år

Fetma 20%
övriga riskfaktorer adipokiner,
Cytokiner, metainflammation

Diabetes (typ II)
oxidativ stress, IGF-1

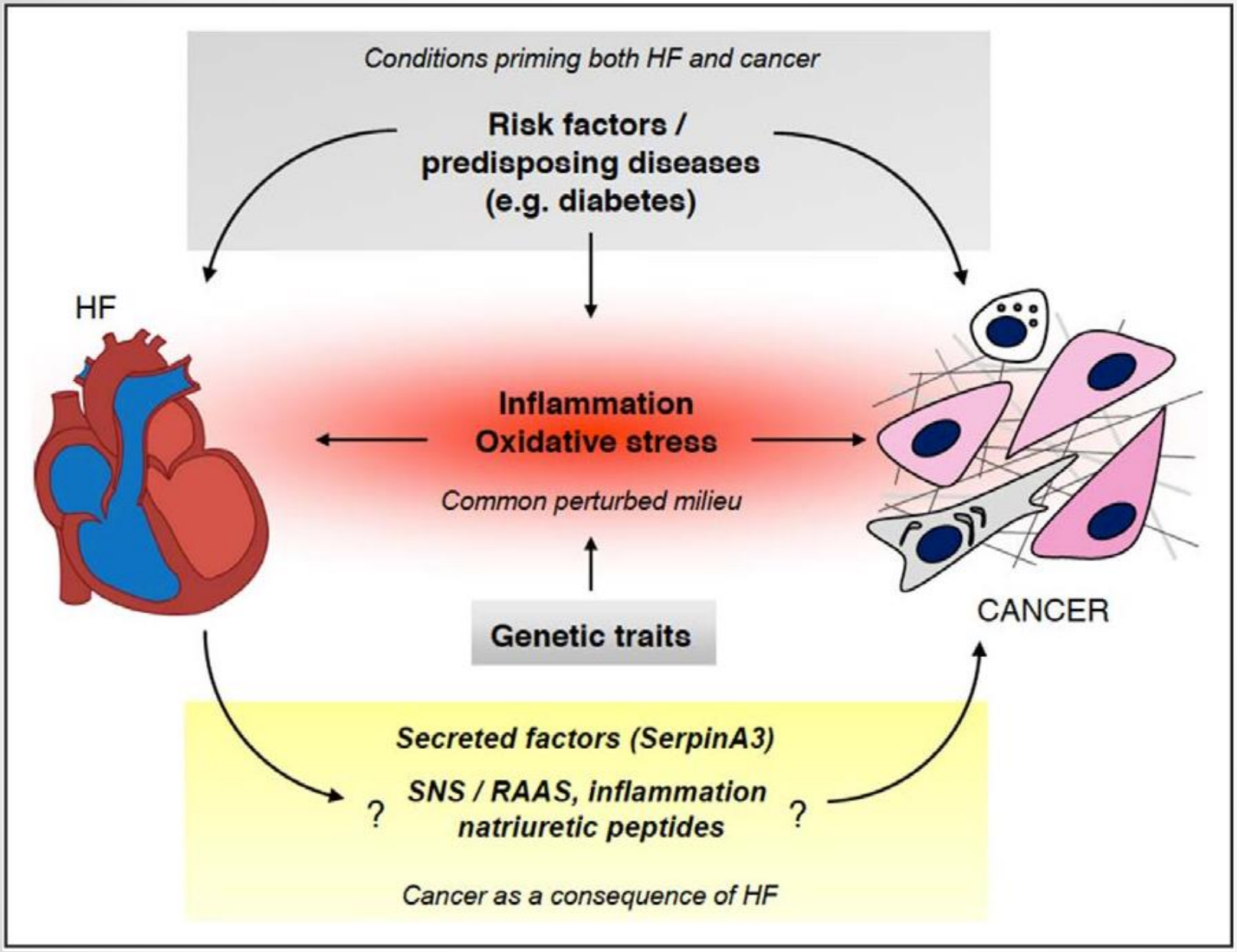
Hypertension
hemodynamisk påverkan
oxidative stress och inflammation.

Rökning 30%
carcinogen o inflammation

Mat (colon) fibrer + rött kött -

Fysisk aktivitet +
minskar övriga riskfaktorer
1% riskminskning/15 min/dag

Heart Failure Clin 13 (2017) 367–380



Circulation. 2018;138:735–742.

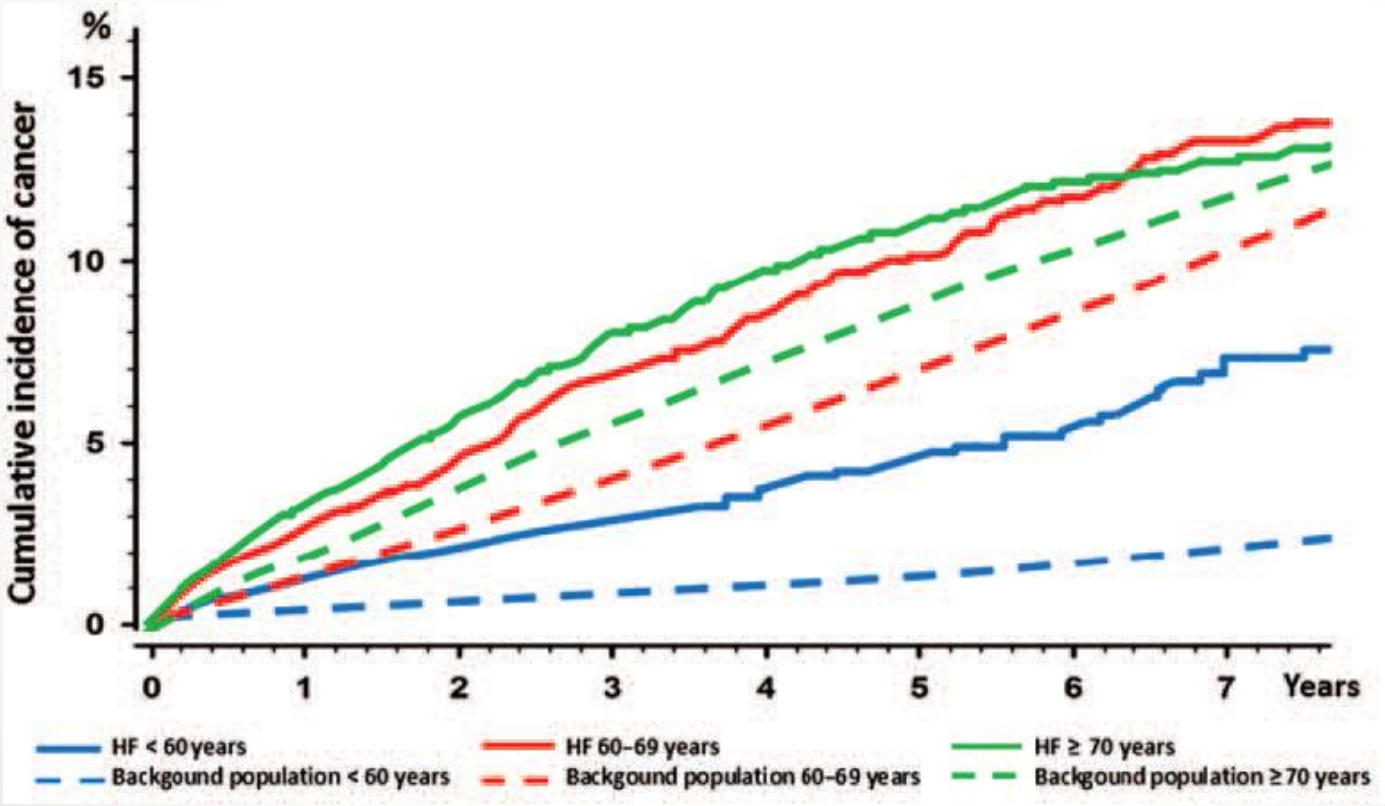
Incidence of cancer in patients with chronic heart failure: a long-term follow-up study

Ann Banke[1]*, Morten Schou[2], Lars Videbæk[1], Jacob E. Møller[1], Christian Torp-Pedersen[3], Finn Gustafsson[4], Jordi S. Dahl[1], Lars Køber[4], Per R. Hildebrandt[5], and Gunnar H. Gislason[6]

[1]Department of Cardiology, Odense University Hospital, Odense, Denmark; [2]Department of Cardiology, Herlev University Hospital, Herlev, Denmark; [3]Department of Health, Science and Technology, Aalborg University, Aalborg, Denmark; [4]Department of Cardiology, Rigshospitalet, Copenhagen, Denmark; [5]Department of Cardiology, Frederiksberg University Hospital, Frederiksberg, Denmark; and [6]Department of Cardiology, Gentofte University Hospital, Hellerup, Denmark

hjärtsvikt ➡ cancer?

10 533 outpatients with verified HF and 4 959 275 citizens (background pop)



European Journal of Heart Failure (2016) **18**, 260–266

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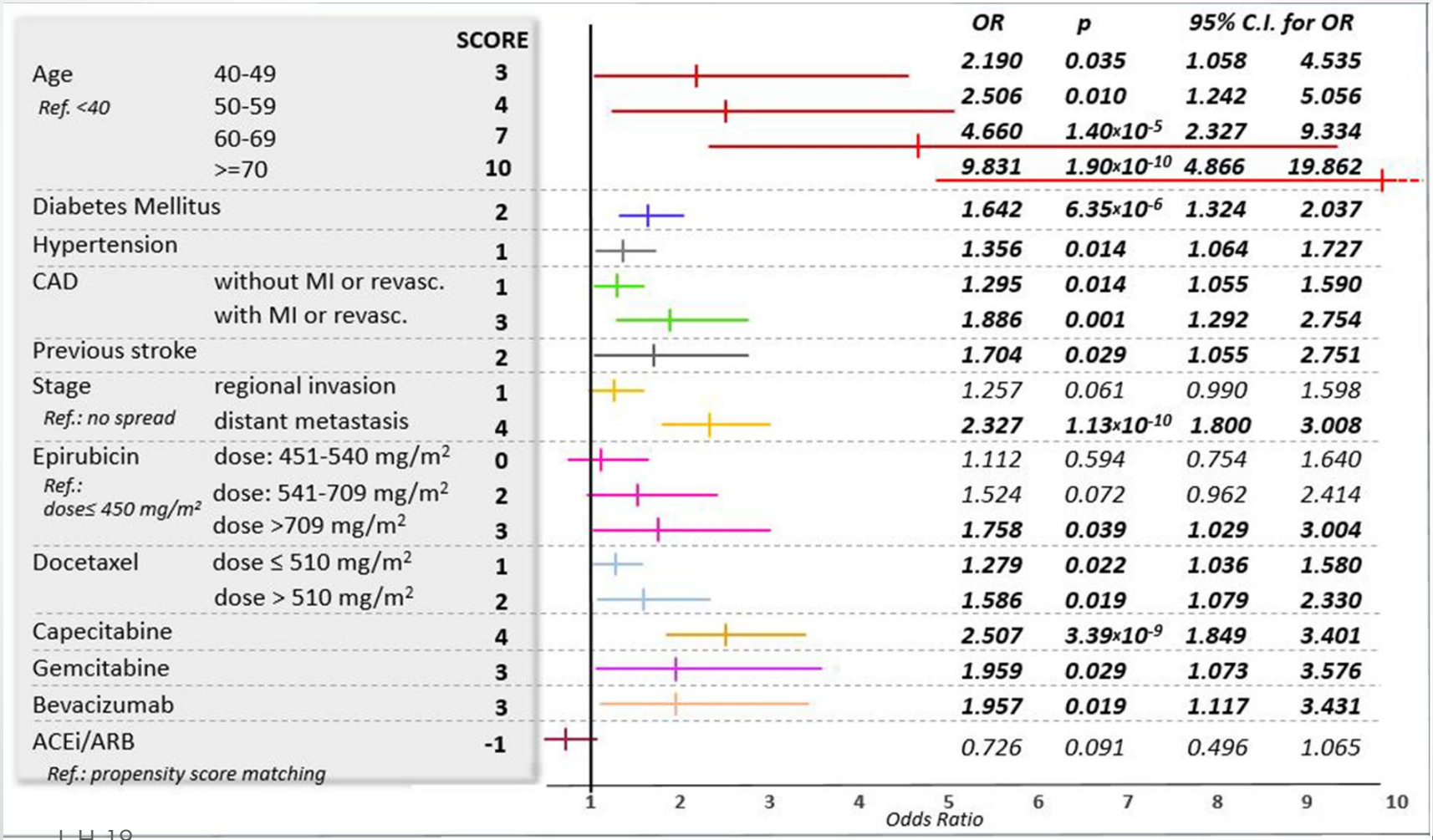
Risk prediction model for long-term heart failure incidence after epirubicin chemotherapy for breast cancer – A real-world data-based, nationwide classification analysis

György Fogarassy [a,*,1], Ágnes Vathy-Fogarassy [b,1], István Kenessey [c,2], Miklós Kásler [d,3], Tamás Forster [e,4]

Retrospektiv
Bröstcancer 8068 pat
antracyklin
3-10 år efter behandling



Int J Cardiol. 2019 Jun 15;285:47-52..

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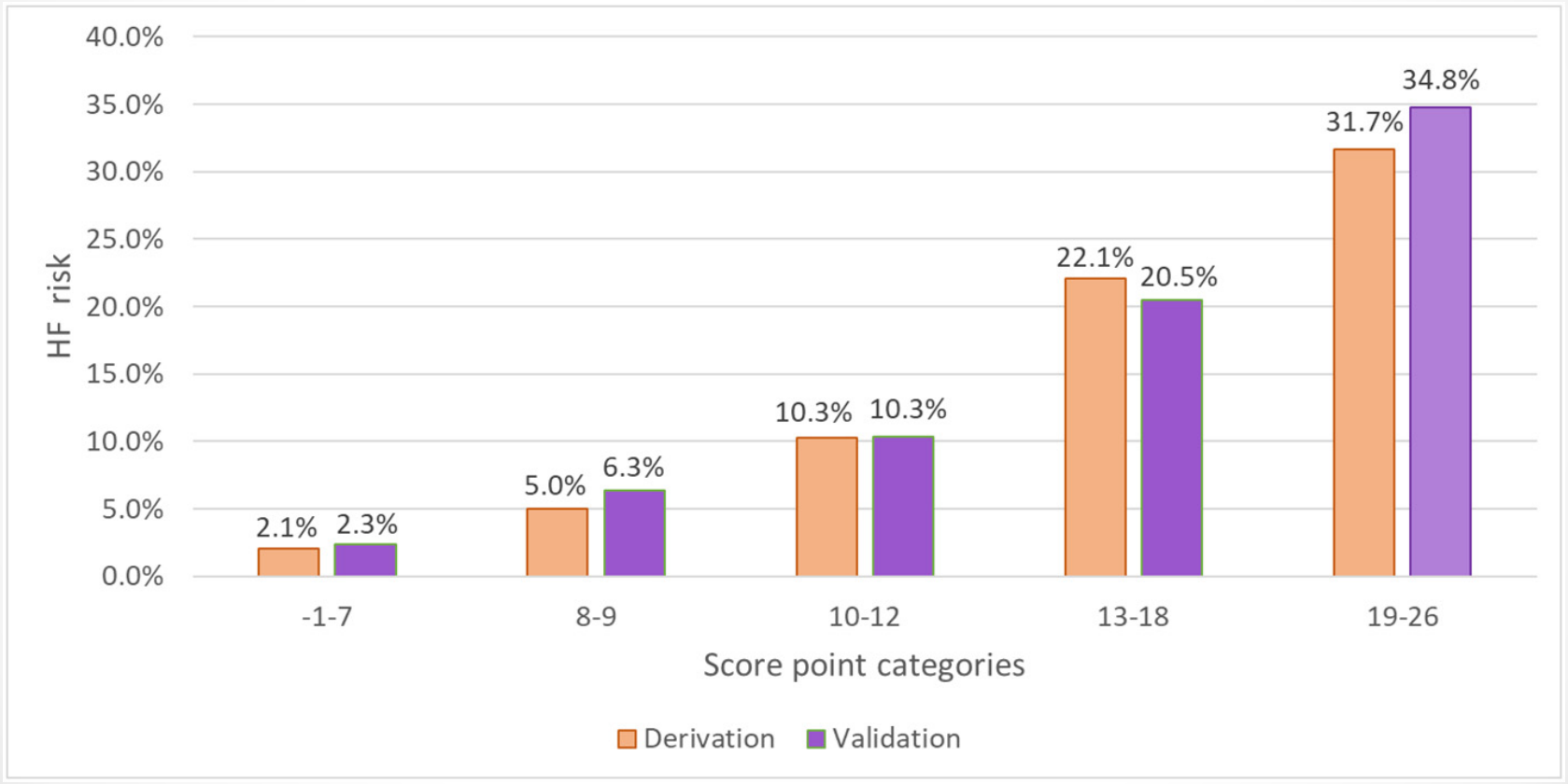


Derivation poäng i riskvärderingen
Validation 30% av patienterna som validering

6.9% HF

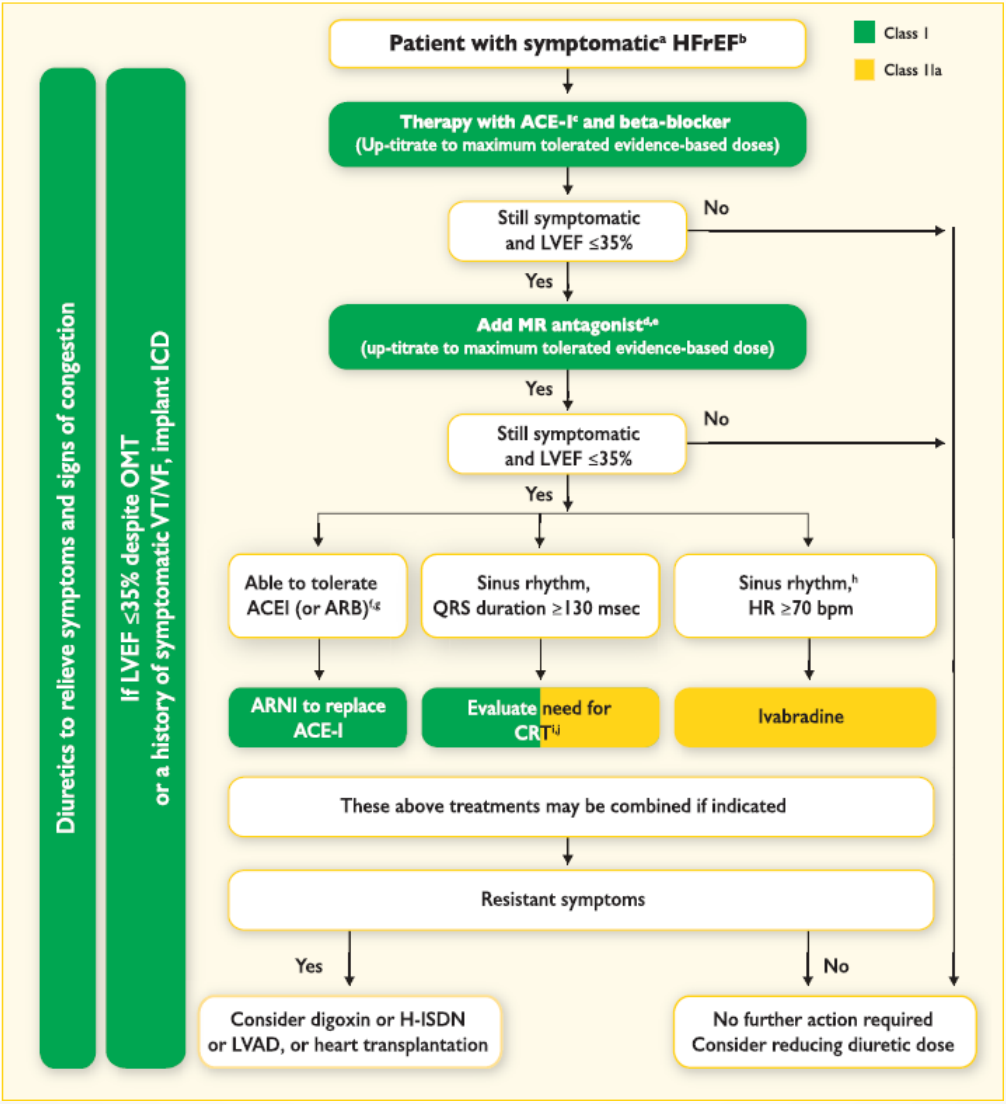
Strategies to reduce chemotherapy-induced cardiotoxicity

Chemotherapy drug	Potential cardioprotective measure
All chemotherapy drugs	Identify and treat cardiovascular risk factors
	Treat comorbidities (CAD, HF, PAD, HTN)
	QTc prolongation and torsade de pointes: - Avoid QT prolonging drugs - Manage electrolyte abnormalities
	Minimize cardiac irradiation
Anthracyclines and analogues	Limit cumulative dose (mg/m^2): - Daunorubicin <800 - Doxorubicin <360 - Epirubicin <720 - Mitoxantrone <160 - Idarubicin <150
	Altered delivery systems (liposomal doxorubicin) or continuous infusions
	Dexrazoxane as an alternative
	ACE-Is or ARBs
	β-blockers
	Statins
	Aerobic exercise
Trastuzumab	ACE-Is
	β-blockers

Overcome-betabl ACE
Prada-betabl/ARB
Manticor-betabl

European Heart Journal doi:10.1093/eurheartj/ehw211 EHJ 2016;37:2768-2801

ACE-I is recommended in patients with asymptomatic LV systolic dysfunction without a history of myocardial infarction, in order to prevent or delay the onset of HF.	I	B



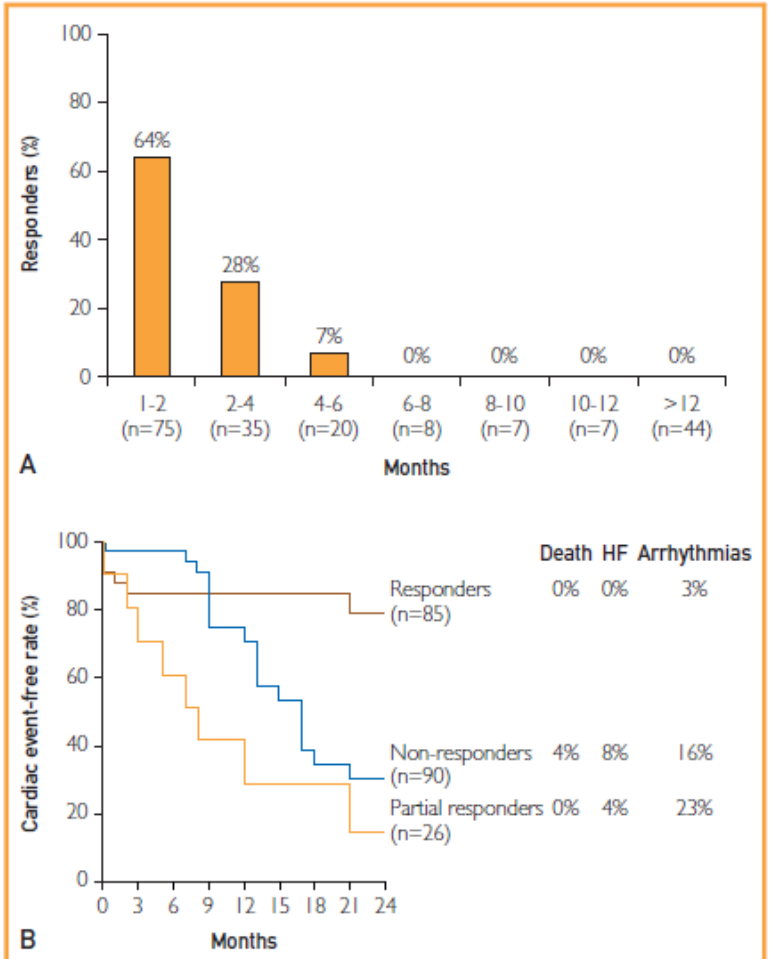


FIGURE 1. Illustration of the percentage of patients with an improvement in left ventricular ejection fraction to greater than 50%, depending on the timing of the initiation of heart failure therapy after the diagnosis of anthracycline-induced cardiomyopathy (A) and survival free of cardiac events for patients with an improvement in left ventricular ejection fraction less than (partial responders) or greater than 50% (responders) (B). From Cardinale et al,[25] with permission.

Snabb 50% förbättring av EF

MAYO CLINIC

Evaluation and Management of Patients With Heart Disease and Cancer: Cardio-Oncology

Joerg Herrmann, MD; Amir Lerman, MD; Nicole P. Sandhu, MD, PhD; Hector R. Villarraga, MD; Sharon L. Mulvagh, MD; and Manish Kohli, MD

Heart failure in breast cancer patients: findings from a Swedish register-based study

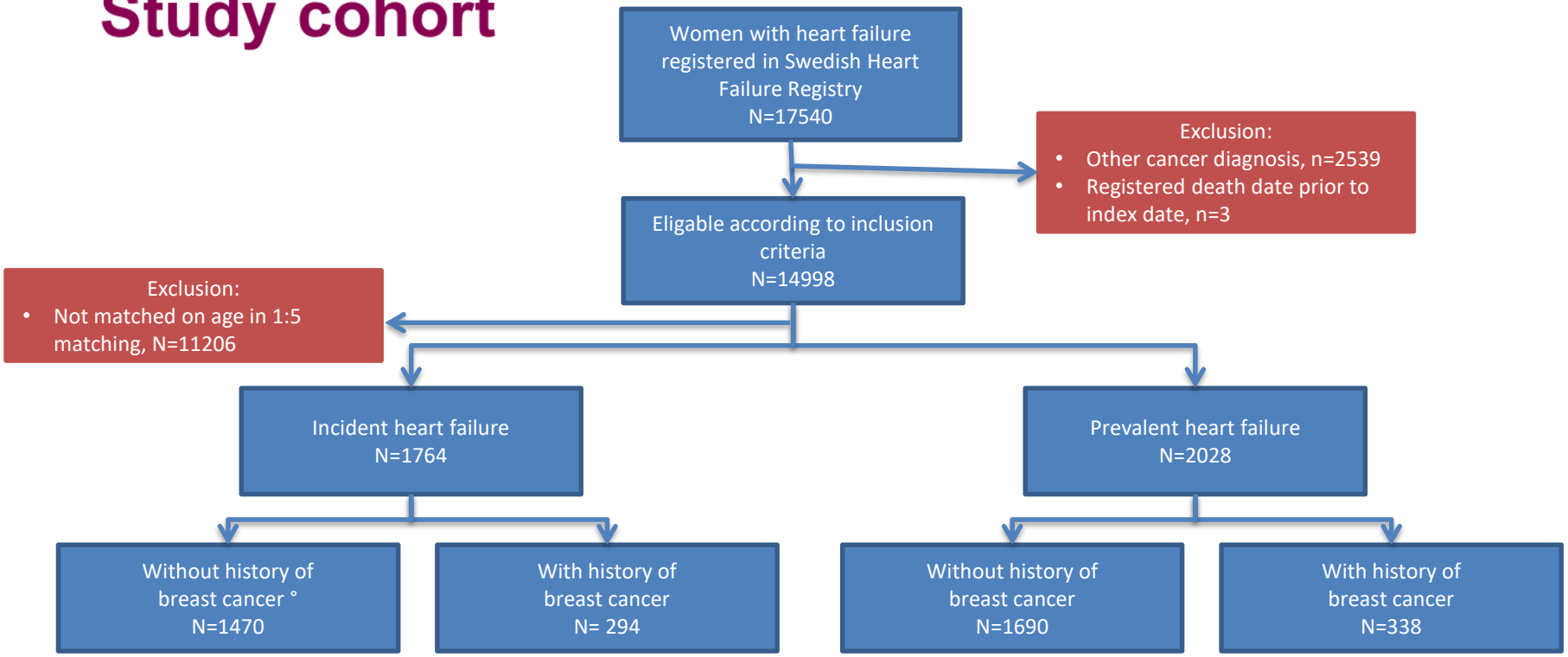
Elham Hedayati, A. Papakonstantinou, SAM Gernaat, R Altnea, JS Brand, J Alfredsson, N Bhoo-Pathy, J Herrmann, C Linde, U Dahlstrom, J Bergh, L Hubbert

Aims

1. To study if heart failure patients with breast cancer have a higher risk of death compared to heart failure patients without breast cancer.

2. To study if patients with breast cancer have a different heart failure presentation and treatments than patients without breast cancer.

Study cohort



Swedish Registries

- **Swedish Heart Failure Register:**
  - Founded in 2000 with 10 000 registrations every year, with 80 variables per patient
  - Incident HF: ≤1 month since diagnosis, Prevalent HF: >1 month since diagnosis
- **Swedish Cancer Register**: breast cancer diagnosis
- **Swedish Cause of Death Register**: death from any cause & cardiovascular disease
- **Prescribed Drug Register**

Statistics

1. Chi-square test for differences in characteristics for categorical data
2. T-test for differences in characteristics for continuous data
3. Kaplan-Meier plots for survival estimates of death from any cause, cardiovascular disease and heart failure
4. Cox proportional hazard models

Results

Table I **Baseline characteristics at enrolment in SwedeHF of 294 women with incident HF and BC compared to 1470 age-matched women with incident HF without BC; and 338 women with prevalent HF and BC compared to 1690 age-matched women with prevalent HF without BC**

Characteristics	Incident HF			Prevalent HF		
	Without BC (*N* = 1470)	With BC (*N* = 294)	*P*-value	Without BC (*N* = 1690)	With BC (*N* = 338)	*P*-value
Age (years), median (IQR)	77 (68–85)	77 (68–85)		79 (73–86)	79 (73–86)	
HF diagnosis, *n* (%)			<0.01			<0.01
Outpatient clinic	467 (31.8)	117 (39.8)		781 (46.2)	162 (47.9)	
Inpatient department	1003 (68.2)	177(60.2)		909 (53.8)	176 (52.1)	
Time between HF diagnosis and SwedeHF enrolment (years), median (IQR)	0.0 (0.0–0.0)	0.0 (0.0–0.0)	0.36	2.1 (0.4–5.5)	1.8 (0.4–5.0)?	0.12
Year of SwedeHF enrolment, *n* (%)			0.20			0.20
2008	297 (20.2)	65 (22.1)		396 (23.4)	89 (26.3)	
2009	224 (15.2)	52 (17.7)		298 (17.6)	52 (15.4)	
2010	251 (17.1)	39 (13.3)		248 (14.7)	44 (13.0)	
2011	273 (18.6)	50 (17.0)		294 (17.4)	63 (18.6)	
2012	243 (16.5)	41 (13.9)		198 (11.7)	43 (12.7)	
2013	182 (12.4)	47 (16.0)		256 (15.1)	47 (14.0)	
Specialty enrolling, *n* (%)			0.62			0.63
Cardiology	674 (50.8)	145 (53.1)		812 (53.7)	156 (51.8)	
Internal medicine	641 (48.3)	127 (46.5)		684 (53.7)	142 (47.2)	
Geriatrics	12 (0.9)	1 (0.4)		15 (1.0)	3 (1.0)	
Follow-up time (years), median (IQR)	2.2 (0.9–2.8)	2.0 (0.8–4.0)		1.8 (0.7–3.4)	2.1 (0.7–3.5)	
Mean	2.0	2.0		2.0	2.0	
Maximum	6.0	6.0		6.0	6.0	
Time between BC and HF (years), median (IQR)	NA	6.2 (3.3–8.4)	NA	NA	6.2 (2.8–8.4)	NA
<1 year, *n* (%)	NA	25 (8.5)		NA	24 (7.1)	
1–5 years, *n* (%)	NA	95 (32.3)		NA	114 (33.7)	
>5 years, *n* (%)	NA	174 (59.2)		NA	200 (59.2)	
New BC or metastatic disease after HF, *n* (%)			<0.01			<0.01
BC	12 (0.8)	55 (19)	<0.01	10 (0.6)	72 (21.3)	<0.01
Metastases	24 (1.6)	13 (4.4)	<0.01	17 (1.0)	15 (4.4)	<0.01

BC, breast cancer; HF, heart failure; IQR, interquartile range; NA, not applicable.

Table 2 **Cardiovascular risk factors, comorbidities, clinical presentation, medical and device therapy at enrolment in SwedeHF of 294 women with incident HF and BC compared to 1470 age-matched women with incident HF without BC; and 338 women with prevalent HF and BC compared to 1690 age-matched women with prevalent HF without BC**

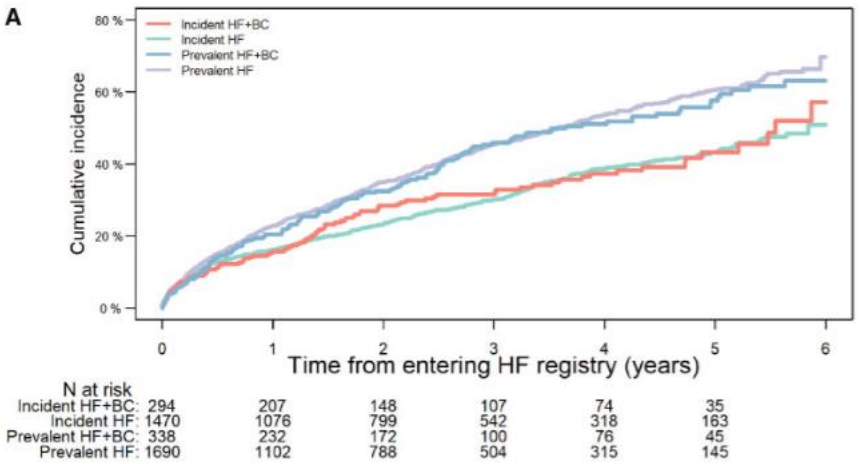
	Incident HF			Prevalent HF		
	Without BC (*N* = 1470)	With BC (*N* = 294)	*P*-value	Without BC (*N* = 1690)	With BC (*N* = 338)	*P*-value
Cardiovascular risk factors						
Body mass index (kg/m^2), mean (SD)	27.6 (6.4)	26.8 (5.5)	0.12	27.2 (6.1)	27.1 (5.6)	0.89
Smoking, *n* (%)			0.25			0.22
Never	541 (36.8)	120 (40.8)		645 (38.2)	140 (41.4)	
Former (>6 months cessation)	335 (22.8)	52 (17.7)		436 (25.8)	83 (24.6)	
Current (<6 months cessation)	175 (11.9)	34 (11.6)		114 (6.7)	21 (6.2)	
Unknown	419 (28.5)	88 (29.9)		495 (29.3)	94 (27.8)	
Hypertension, *n* (%)	829 (56.4)	153 (52.0)	0.26	922 (54.6)	184 (54.4)	0.24
Unknown	25 (1.7)	3 (1.0)		47 (2.8)	9 (2.7)	
Diabetes, *n* (%)			0.90			0.89
Type 1	15 (1.0)	3 (1.0)		26 (1.5)	5 (1.5)	
Type 2	247 (16.8)	43 (14.6)		382 (22.6)	67 (19.8)	
Unknown	15 (1.0)	10 (3.4)		26 (1.5)	9 (2.7)	
Pre-existing comorbidities, *n* (%)						
Myocardial infarction	268 (18.2)	43 (14.6)	0.15	484 (28.6)	73 (21.6)	<0.01
Unstable angina	83 (5.6)	9 (3.1)	0.08	141 (8.3)	28 (8.3)	1.00
Stable angina	188 (12.8)	31 (10.5)	0.33	446 (26.4)	88 (26.0)	0.95
Atrial fibrillation/flutter	492 (33.5)	93 (31.6)	0.59	964 (57.0)	186 (55.0)	0.51
Stroke	199 (13.5)	37 (12.6)	0.71	281 (16.6)	52 (15.4)	0.63

Table 2 ***Continued***

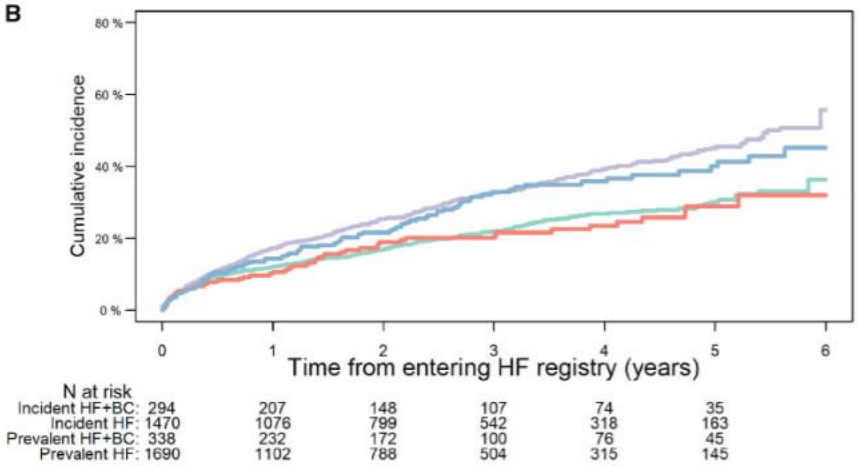
	Incident HF			Prevalent HF		
	Without BC (*N* = 1470)	**With BC (*N* = 294)**	***P*-value**	**Without BC (*N* = 1690)**	**With BC (*N* = 338)**	***P*-value**
Treatment						
Beta-blocker, *n* (%)	921 (62.7)	184 (62.6)	1.00	1462 (86.5)	298 (88.2)	0.48
Agents acting on the renin–angiotensin system, *n* (%)	871 (59.3)	184 (62.6)	0.30	1393 (82.4)	290 (85.8)	0.15
Angiotensin-converting enzyme inhibitor	615 (41.8)	126 (42.9)	0.75	987 (58.4)	199 (58.9)	0.90
Angiotensin receptor blocker	328 (22.3)	70 (23.8)	0.59	561 (33.2)	122 (36.1)	0.31
Mineral corticoid receptor antagonist, *n* (%)	166 (11.3)	36 (12.2)	0.62	623 (36.9)	118 (34.9)	0.54
Calcium channel blockers, *n* (%)	374 (25.4)	77 (26.2)	0.83	438 (25.9)	81 (24.0)	0.50
Diuretics, *n* (%)	877 (59.7)	176 (59.9)	>0.99	1506 (89.1)	301 (89.1)	>0.99
Statins, *n* (%)	539 (36.7)	96 (32.7)	0.21	765 (45.3)	149 (44.1)	0.72
Aspirin, *n* (%)	636 (43.3)	120 (40.8)	0.48	932 (55.1)	161 (47.6)	0.01
Coronary revascularization, *n* (%)	166 (11.3)	24 (8.2)	0.12	274 (16.2)	40 (11.8)	<0.01
Device therapy, *n* (%)			>0.99			0.03
Implantable cardioverter-defibrillators	5 (0.3)	2 (0.7)		27 (1.6)	2 (0.6)	
Cardiac resynchronization therapy/+implantable cardioverter-defibrillators	4 (0.3)	1 (0.3)		50 (3.0)	3 (0.9)	

BC, breast cancer; HF, heart failure.

All cause mortality

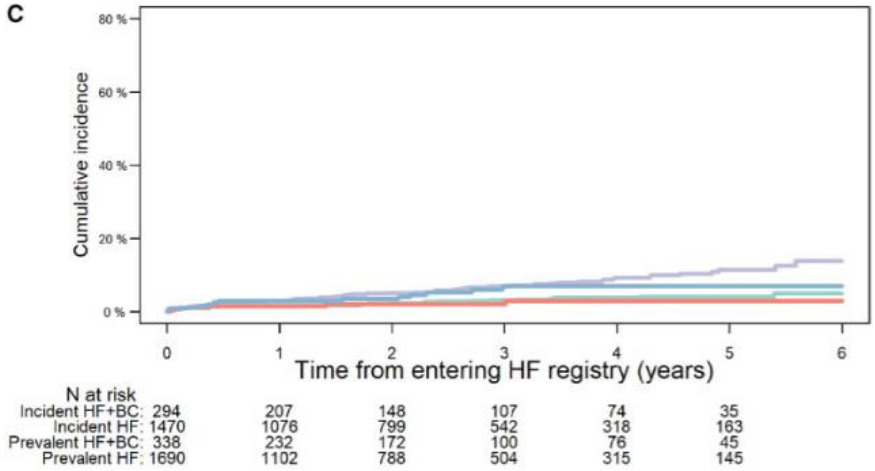


Cardiovascular disease mortality



Incident HF+BC
Incident HF
Prevalent HF+BC
Prevalent HF

Heart failure mortality



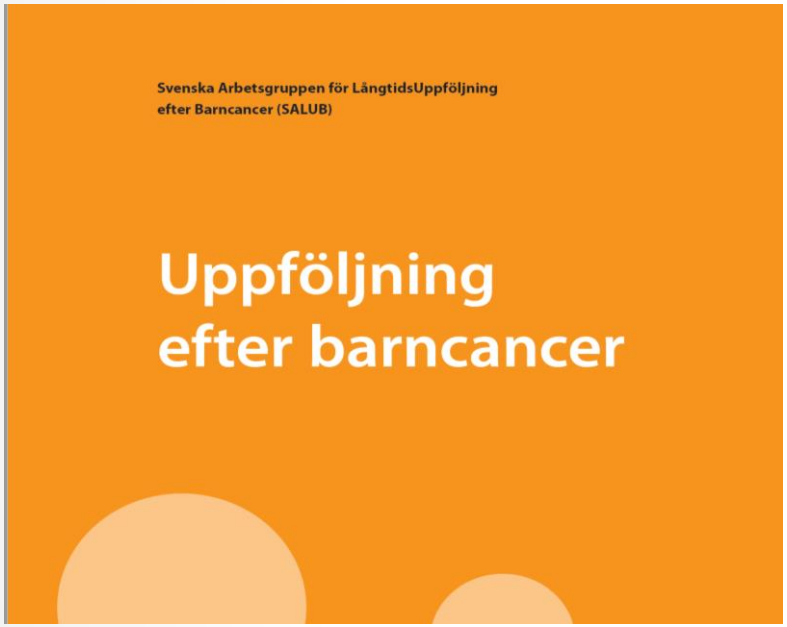
There are no differences between the groups on survival.

Limitations

- No information on breast cancer characteristics or treatments
- Death from heart failure may be underestimated
- Some patients in the non-breast cancer group developed breast cancer and metastasis after SwedeHF enrolment during follow-up
- SwedeHF does not register advanced heart failure treatments such as left ventricular assist device or heart transplantation

Conclusions

1. Risk of all-cause mortality, cardiovascular mortality and heart failure mortality did not differ by breast cancer status.
2. Prevalent heart failure patients with breast cancer had less often myocardial infarction and were less often treated with aspirin and coronary revascularization than those without breast cancer.



Uppföljning

Undersökning görs med **EKG och ekokardiografi** som visar hjärtfunktionen i systole och diastole. Fynden relateras till normalvärden för åldern.

Som komplement till undersökningarna görs riktad anamnes med avseende på subjektiv fysisk funktionsnivå och kardiella symptom, framför allt arytmier.

Utifrån idag befintliga behandlingsprotokoll får cirka 70% av de antracyklinbehandlade patienterna en ackumulerad dos på ≤210 mg/m², vilket betyder att 30% får >210 mg/m².

Antracykliner och strålbehandling mot hjärtat ges till en begränsad grupp patienter. Den grupp som enbart strålbehandlats med hjärtat i strålfältet är också begränsad.

Grupp 1

(Antracyklinbehandling ≤210 mg/m², ej strålbehandling mot hjärtat):

1) Ekokardiografi inom 6 månader efter avslutad Antracyklinbehandling.
2) Ekokardiografi i tidig pubertet samt vid 18 års ålder (före överföring till vuxenklinik/seneffektmottagning). Visst beaktande av tidsintervallet mellan undersökning 1 och 2 beroende på insjuknandeålder.
3) Ekokardiografi under vuxenlivet rekommenderas ej rutinmässigt.
4) För flickor görs hjärtbedömning i samband med graviditet.
5) För bägge könen sker regelbunden hjärtbedömning vid tävlingsidrott på elitnivå.

Grupp 2

(Antracyklinbehandling >210 mg/m², ej strålbehandling mot hjärtat):

1) Ekokardiografi inom 6 månader efter avslutad Antracyklinbehandling.
2) Ekokardiografi efter 5 år, i tidig pubertet samt vid 18 års ålder. Beroende på insjuknandeålder anpassas kontrollerna men 2 ekokardiografier bör ha genomförts inom en 10 års period.
3) Ekokardiografi regelbundet vart 5:e år under vuxenlivet rekommenderas.
4) Se punkt 4 och 5 för grupp 1 ovan.

Hur undviker vi hjärtsvikt efter cancerbehandling?

Behandla riskfaktorer
Kardioprotektion med ACE/ARB och/eller betablockad för högriskpatienter
Samarbete och tät monitorering