

# Äggstockscancer med epitelial histologi

Utdrag ur nationellt vårdprogram:  
Kapitel 6, Screening och ärftlighet  
(och referenser)

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## KAPITEL 6

# Screening och ärftlighet

## 6.1 Screening av asymtomatiska kvinnor utan påvisad ärftlig risk

### Rekommendationer

- Screening av asymtomatiska kvinnor i den allmänna befolkningen för äggstockscancer rekommenderas inte.
- Bakgrunden är att screening av asymtomatiska kvinnor med låg risk för äggstockscancer
  - inte leder till minskad total dödlighet (⊕⊕⊕)
  - inte leder till färre kvinnor som upptäcks med avancerat stadium (⊕⊕)
  - är associerat med onödiga kirurgiska ingrepp med risk för allvarliga komplikationer (⊕⊕⊕).

Flera kriterier behöver uppfyllas innan en screeningmetod för en viss sjukdom kan införas i en befolkning. Bland annat krävs evidens att testet är tillräckligt känsligt för att upptäcka sjukdomen tidigt (sensitivitet) och tillräckligt specifikt för att inte orsaka skada på friska människor. Därutöver krävs att randomiserade studier visar en minskning av dödligheten i sjukdomen för de som screenats jämfört med de som inte screenats.

En systematisk översikt visar att screening (CA 125 och ultraljud) av asymtomatiska, huvudsakligen postmenopausala, kvinnor med låg risk för att insjukna i äggstockscancer (ovarialcancer), inte signifikant minskar dödligheten jämfört med kvinnor som inte genomgått screening [45]. Översikten identifierade 4 randomiserade studier som utvärderade effekten av screening med CA 125 och ultraljud, varav de 2 största studierna är den amerikanska PLCO-studien och den engelska UKCTOCS-studien.

I PLCO-studien randomiserades 78 216 kvinnor i åldern 55–74 år till screening med årliga CA 125 (i 6 år) och vaginala ultraljud (i 4 år) jämfört med rutinvård [46]. Resultaten visar ingen skillnad i cancerspecifik dödlighet vare sig vid användning av CA 125 med cut-off på 35 eller en algoritm, ROCA (risk of ovarian cancer algorithm), av CA 125-värden över tid, i kombination med ålder [47]. Vid en utvidgad uppföljning på 15 år, kvarstod resultaten att screening med CA 125 och vaginalt ultraljud inte resulterade i minskad äggstockscancerdödlighet [48].

I UKCTOCS-studien randomiserades 202 638 postmenopausala kvinnor mellan årlig screening bestående av en multimodal metod med CA 125 och ultraljud versus enbart ultraljud versus rutinvård [49]. I denna studie användes ROCA för att bedöma risken för äggstockscancer och vid användning av algoritmen upptäcktes dubbelt så många fall av invasiv äggstockscancer. Man har dock inte kunnat visa att dödligheten i sjukdomen minskat.

Sammantaget visade resultaten från dessa 2 studier att screeningen ledde till onödig kirurgi hos 1 % av kvinnorna i ROCA-gruppen och 3 % av de som genomgått ultraljud, med eller utan CA 125. Kirurgi som utfördes på grund av falskt positivt screeningresultat ledde till allvarliga komplikationer i 3 % respektive 15 % av de opererade fallen [45]. Evidens för psykologisk

sjuklighet på grund av screening var begränsad förutom för en grupp av patienter i UKCTOCS-studien som, på grund av screeningresultat, fick genomgå upprepade undersökningar under uppföljningen [50-52].

## 6.2 Ärftlighet

### Sammanfattning och rekommendationer

- Profylaktisk salpingooforektomi minskar risken att insjukna i äggstockscancer och rekommenderas för kvinnliga BRCA1- och BRCA2-bärare efter avslutad reproduktion, för BRCA1-bärare vid cirka 35–40 års ålder och för BRCA2-bärare vid cirka 40–50 års ålder.
- Samtliga kvinnor med äggstockscancer (ej borderline) bör erbjudas genetisk analys avseende BRCA1 och BRCA2.
- Kvinnor med äggstockscancer kan därutöver också erbjudas analys för MLH1, MSH2, MSH6, PMS2, EPCAM, BRIP1, RAD51C och RAD51D.
- Provtagning av tumörvävnad från primärtumören rekommenderas för genetisk screening avseende BRCA1 och BRCA2. En påvisad patogen variant betyder att den antingen är ärftlig eller förvärvad. Om sådan påvisas ska patienten remitteras till cancergenetisk mottagning för fortsatt utredning. Ärftlighet ska alltid bekräftas med analys av normalvävnad (t.ex. blod).
- Vid stark misstanke om ärftlig äggstockscancer bör patienten erbjudas remiss till en cancergenetisk mottagning för utredning även om ingen patogen variant påvisats.
- Friska bärare av ärftlig patogen variant bör få information om profylaktisk kirurgi och andra riskminskande åtgärder.
- Det saknas evidens för att regelbundna kontroller med ultraljud och CA 125 minskar dödligheten i äggstockscancer hos mutationsbärare. I första hand rekommenderas därför profylaktisk bilateral salpingooforektomi.
- Efter premenopausal riskreducerande salpingooforektomi bör HRT erbjudas upp till cirka 50 års ålder om det inte föreligger tidigare bröstcancerdiagnos.
- Kvinnor med ärftlig riskökning för äggstockscancer har en påtaglig skyddseffekt av kombinerade p-piller och bör erbjudas detta om de inte genomgått profylaktisk kirurgi.

Cirka 15–20 % av alla fall med äggstockscancer är ärftliga. Epidemiologiska studier anger en knappt 3 % livstidsrisk att insjukna om en kvinna har en förstegradssläkting med äggstockscancer, med högre risk vid starkare familjehistoria [53].

Hos de kvinnor med ärftlig äggstockscancer där man kan påvisa en ärftlig patogen variant kan 65–85 % härledas till BRCA1- och BRCA2-generna, medan patogena varianter i andra gener orsakar 10–15 % av all ärftlig äggstockscancer [54].

### 6.2.1 Utredning av misstänkt ärftlighet

Genetisk screening vid nydiagnostiserad äggstockscancer bör omfatta BRCA1 och BRCA2 och kan även omfatta MLH1, MSH2, MSH6, PMS2, EPCAM, BRIP1, RAD51C och RAD51D. Teknikutvecklingen tillåter att man utan större merkostnad kan testa samtliga dessa gener inom en multigenpanel. Sensitiviteten och specificiteten vid screening skattas till över 90 % på de laboratorier som rutinmässigt utför analysen, men påverkas av utgångsmaterial för analysen. Om

screening sker på tumörvävnad bör utförande laboratorium därför säkerställa att man har en godtagbar sensitivitet att påvisa ärftliga varianter i samtliga gener man analyserar, jämfört med om analysen hade utgått från blodprov.

Om multigenpanel utnyttjas så ökar sannolikheten att påvisa varianter av oklar signifikans (VUS) vid analysen, d.v.s. genetiska varianter som med rådande kunskapsläge varken kan tolkas som benigna eller patogena. För att inte försvåra klinisk handläggning så bör VUS i allmänhet inte rapporteras ut till inremitterande kliniker. VUS bör däremot registreras och systematiskt följas upp på laboratoriet så att det finns en möjlighet att omvärdera dessa när ny kunskap tillkommer.

Om tidigare utförd immunhistokemi eller mikrosatellitinstabilitet (MSI) har indikerat misstanke om Lynchs syndrom utförs mutationscreening endast av relevanta MMR-gener (MLH1, MSH2, MSH6, PMS2, EPCAM).

Beslutet om att erbjuda kvinnor utökade kontroller eller förebyggande operationer vid misstanke om ärftlig cancerrisk bör baseras på en kvalificerad bedömning av släkträdets och resultatet av molekylärgenetisk testning. Dessa frågor bör utredas på de cancergenetiska mottagningar som finns inom varje sjukvårdsregion.

## 6.2.2 Ärftliga varianter

### 6.2.2.1 Hereditära bröst- och ovarialcancersyndromet (HBOC) – BRCA1 och BRCA2

I familjer med många fall av bröstcancer eller äggstockscancer (ovarialcancer) samt ett dominant nedärvningsmönster kunde man i mitten av 1990-talet lokalisera och identifiera två gener: BRCA1-genen på kromosom 17q21 och BRCA2-genen på kromosom 13q16 [55]. Det är rimligt att ange livstidsrisken för kvinnlig bröstcancer vid patogen variant i BRCA1 eller BRCA2 till 50–80 %. Livstidsrisken för äggstockscancer vid patogen variant i BRCA1 kan anges till 30–60 % respektive 10–25 % för BRCA2, jämfört med strax under 2 % i den svenska befolkningen [56]. Insjuknandeåldern för äggstockscancer hos BRCA-bärare är i genomsnitt högre än för bröstcancer. För BRCA1 ses en genomsnittlig insjuknandeålder mellan 40 och 60 år, och för BRCA2 mellan 50 och 70 år [57].

Utöver ökad risk för bröstcancer och äggstockscancer har kvinnor med BRCA1-mutation en ökad risk för såväl tubarcancer som primär peritonealcancer. Avseende BRCA2 så är risken även förhöjd för pankreascancer, manlig bröstcancer och prostatacancer.

Man kan inte histologiskt urskilja BRCA-associerad äggstockscancer. I likhet med de sporadiska äggstockscancerfallen är HGSC den absolut vanligaste histologin vid BRCA-associerad äggstockscancer. Patogena germline-varianter i BRCA1/2 förekommer hos drygt 20 % av HGSC-fallen, och i upp till drygt 8 % vid andra histologiska undergrupper såsom klarcellig respektive endometrioid cancer [58]. Mucinös äggstockscancer och borderlinetumörer förekommer mycket sällan bland BRCA-bärare [59–61]. Familjehistoria för ärftlighet saknas hos drygt 40 % av kvinnorna med BRCA-germline-varianter. Mot denna bakgrund rekommenderas att alla kvinnor med äggstocks-, tubar- eller primär peritonealcancer (ej borderline), oavsett familjehistoria, ska erbjudas genetisk screening. Då det av behandlingsskäl är viktigt att identifiera både somatiska och ärftliga varianter i BRCA1/2 så görs screeningen bäst genom provtagning av tumörvävnad från primärtumören, eventuellt i kombination med blodprov. Beroende på utgångsmaterial och analysmetod kan tumöranalysen ha en lägre sensitivitet att detektera ärftliga varianter jämfört med analys utgående från blodprov. Ett positivt testresultat i tumörvävnad betyder att påvisad variant är antingen ärftlig eller förvärvad, och av de BRCA-varianter som

påvisas i tumören är cirka 75 % ärftliga [62]. Patienter med påvisad patogen variant bör remitteras till cancergenetisk mottagning för rådgivning och kompletterande blodprovstagning (om detta ej skett tidigare).

Studier av BRCA-bärare som genomgått profylaktisk bilateral salpingooforektomi (SOEB) har visat att BRCA-associerad äggstockscancer oftast har sitt ursprung i äggledaren (se även avsnitt 4.2 Orsaker). BRCA-bärare med äggstockscancer har bättre prognos jämfört med sporadiska fall [63]. I återfallssituationen svarar BRCA-bärare oftare på såväl platinum- som icke-platinum-innehållande regimer jämfört med mutationsnegativa patienter [58]. Poly(ADP-Ribose)polymeras-hämmare (PARP-hämmare) är en typ av målriktad behandling som kan erbjudas kvinnor med patogena varianter i BRCA1 eller BRCA2 och återfall i platinumkänslig serös äggstockscancer (se avsnitt 11.3.4 PARP-hämmare).

### 6.2.2.2 Lynchs syndrom – MLH1, MSH2, MSH6, PMS2 och EPCAM

Individer med Lynchs syndrom har en patogen variant i någon av DNA mismatch repair (MMR)-generna (MLH1, MSH2, PMS2, MSH6 eller EPCAM). De Lynch-associerade tumörerna karakteriseras av mikrosatellitinstabilitet (MSI) samt av defekt MMR-protein i tumörvävnaden påvisat genom immunhistokemi. Analys av MSI och immunhistokemisk färgning för MMR-proteiner kan därför användas som första screeningmetod vid misstänkt Lynchs syndrom [64, 65]. Med billigare och mer lättillgängliga genetiska analyser så blir det allt vanligare att direkt utföra sekvensering av Lynch-generna.

De flesta beskrivna ärftliga patogena varianter vid Lynchs syndrom finns i generna MLH1 eller MSH2 (cirka 90 % av alla med Lynchs syndrom). För dessa två gener skattas livstidsrisken att insjukna i kolorektalcancer till 40–80 %, och risken för endometrieccancer och äggstockscancer hos kvinnor uppgår till 25–60 % respektive 4–24 %. För MSH6-bärare så är risken för endometrieccancer jämförbar, medan risken för kolorektalcancer och äggstockscancer sannolikt är något lägre. PMS2-bärare har lägre livstidsrisker för samtliga Lynch-associerade tumörformer. Patogena varianter i EPCAM orsakar hypermetylering av promotorn i MSH2, och medför därför samma cancerrisker som för MSH2-bärare [66]. Tumörer i andra organ såsom magsäck, tunntarm och övre urinvägar kan också vara associerade med syndromet. För detaljerad beskrivning inklusive kriterier för testning gällande Lynchs syndrom, se dokument om ärftlig kolorektalcancer på [Svensk förening för Medicinsk Genetik och Genomik](#)

Jämfört med sporadisk äggstockscancer är åldern vid insjuknandet låg, med en medianålder på 42 år. Histologiskt är den endometrioida celltypen vanligast (29 %) vid Lynch-associerad äggstockscancer efterföljt av mucinös (19 %) och klarcellig (18 %) celltyp. Synkron endometrieccancer förekommer i cirka 20 % av fallen [65, 67].

Kvinnor med misstänkt Lynchs syndrom bör remitteras till en cancergenetisk mottagning.

### 6.2.2.3 Gener associerade med måttligt förhöjd äggstockscancerriksk – BRIP1, RAD51C och RAD51D

Ett flertal gener har identifierats de senaste åren där patogena varianter kopplats till en måttligt förhöjd risk att insjukna i äggstockscancer. För många av dessa gener är evidensläget fortfarande oklart, och i dagsläget bör flertalet av dessa gener inte ingå i kliniska genpaneler [53]. Avseende generna BRIP1, RAD51C och RAD51D finns ett flertal studier som anger en förhöjd risk [54, 68-72]. Den genomsnittliga risken för äggstockscancer upp till 80 års ålder för kvinnliga bärare av patogen variant i BRIP1 har angivits vara 4–13 %, för RAD51C 6 % och för RAD51D 14 %. Liksom för andra cancerassocierade gener modifieras dock risken av familjebilden (till stor del på

grund av samverkande vanliga genetiska polymorfier, SNPs), så att en kvinna som har både en stark familjehistoria och en patogen variant i BRIP1, RAD51C eller RAD51D kan ha en risk som är högre än genomsnittsrisk. Hos BRIP1-, RAD51C- och RAD51D-bärare uppträder riskökningen främst efter 50 års ålder.

Det finns i nuläget inga säkra evidens för att patogena varianter i BRIP1, RAD51C eller RAD51D medför kliniskt relevant förhöjda risker för andra cancerformer än äggstockscancer.

## 6.3 Uppföljning och omhändertagande vid ärftlighet för äggstockscancer

### 6.3.1 Bakgrund

En engelsk prospektiv kohortstudie (the UK familial ovarian cancer screening study, UK FOCSS) av kvinnor över 35 år med betydande familjeanamnes av äggstockscancer har utförts [58]. Över 5 000 kvinnor med hög risk för äggstockscancer bjöds under åren 2002–2009 in att delta i studien för att utvärdera effekten av ultraljud och serum CA 125. I en första fas av studien, med 3 563 kvinnor, utfördes vaginalt ultraljud och S-CA 125 en gång per år. I andra fasen kortades intervallen av S-CA 125-bestämning till var 4:e månad och omfattade ROCA. Vaginalt ultraljud utfördes årligen vid normal CA 125 eller inom 2 månader vid onormalt värde [59]. Resultaten från denna studie, och en liknande amerikansk studie, talar för en potentiell stage-shift till tidigare stadier när en ROCA-baserad screening används för kvinnor med ärftlig risk för äggstockscancer [60]. Det är dock fortfarande okänt om ett sådant förfarande förbättrar överlevnaden.

Riskreducerande kirurgi kvarstår därför som rekommendation för kvinnor som är BRCA1/2-bärare. För de kvinnor som ännu inte opererats uppmanas till årlig diskussion om riskreducerande kirurgi hos gynekolog. Det är mycket tveksamt om någon positiv effekt finns av upprepat serum CA 125 och vaginalt ultraljud fram till dess att riskreducerande salpingooforektomi (RRSO) genomförs.

### 6.3.2 Hereditära bröst- och ovarialcancersyndromet (HBOC)

Friska kvinnor med en patogen variant i BRCA1 eller BRCA2 ska erbjudas uppföljning avseende bröstcancer i enlighet med det nationella vårdprogrammet för bröstcancer.

BRCA-bärare ska erbjudas en regelbunden individuellt anpassad kontakt med gynekolog som kan ge information om riskreducerande salpingooforektomi och andra aktuella gynekologiska frågeställningar. Det är lämpligt att denna kontakt initieras vid cirka 30 års ålder.

Flera studier pågår om screening med gynekologisk undersökning inklusive vaginalt ultraljud och CA 125 av kvinnor med ärftligt ökad risk för äggstockscancer (ovarialcancer). För närvarande saknas evidens för att dessa kontroller minskar dödligheten i äggstockscancer och här rekommenderar vi därför i första hand SOEB för BRCA1- och BRCA2-mutationsbärare efter avslutat barnafödande (se även [avsnitt 6.3.5](#)) [51]. Det finns i dag ingen tillförlitlig evidens för att erbjuda bilateral salpingektomi i stället för SOEB, i syfte att förhindra för tidigt klimakterium; studier pågår emellertid [73, 74].

Kvinnor med påvisad ärftlig patogen variant i BRCA1 eller BRCA2 som behandlats för äggstockscancer bör erbjudas remiss för individuell bedömning för uppföljning av bröstet.

### 6.3.3 Lynchs syndrom

Kvinnor som bär en Lynch-associerad patogen variant rekommenderas uppföljning enligt nationellt dokument om ärftlig kolorektalcancer på [Svensk Förening för Medicinsk Genetik och Genomik](#).

Liksom för BRCA-bärare så rekommenderas vid Lynchs syndrom regelbunden individuellt anpassad kontakt med gynekolog som kan ge information om riskreducerande hysterektomi, salpingooforektomi och andra aktuella gynekologiska frågeställningar. Det är lämpligt att denna kontakt initieras vid 30–35 års ålder.

### 6.3.4 BRIP1, RAD51C och RAD51D

Då risken för äggstockscancer vid patogen variant i BRIP1, RAD51C och RAD51D är liten före 50 års ålder så rekommenderas inte några gynekologiska kontroller. I enlighet med resonemanget ovan så rekommenderas en individuellt anpassad kontakt med gynekolog som kan ge information om riskreducerande salpingooforektomi och andra aktuella gynekologiska frågeställningar.

### 6.3.5 Profylaktisk kirurgi vid hereditära bröst- och ovarialcancersyndromet (HBOC)

Profylaktisk bilateral salpingooforektomi (SOEB) är associerad med en 80 % relativ minskning av risken för äggstockscancer (ovarialcancer) hos BRCA1- eller BRCA2-bärare samt en 77 % minskning av den totala dödligheten [57]. En viss risk (cirka 5 %) att insjukna i primär peritonealcancer kvarstår. Denna risk är emellertid lägre än för kvinnor som inte genomgått SOEB [75, 76].

Profylaktisk SOEB rekommenderas för kvinnliga BRCA1- och BRCA2-bärare efter avslutad reproduktion, för BRCA1-bärare vid cirka 35–40 års ålder och för BRCA2-bärare vid cirka 40–50 års ålder (++++) [57]. Rekommendationen avseende riskreducerande salpingooforektomi gäller även efter kurativ bröstcancerbehandling (++++) för reduktion av incidens i äggstockscancer, (+++) för gynnsam effekt på total överlevnad [76].

Vid profylaktisk SOEB är det av största vikt att ägglarna tas bort eftersom dessa oftast är ursprunget för tumörutveckling. Det bör framgå på PAD-remissen till patologen, att operationen är profylaktisk och att patienten är BRCA-bärare. En noggrann histologisk undersökning av hela tuban ska göras. Det föreligger en risk på cirka 5 % för ockult cancer hos BRCA-bärare vid profylaktisk kirurgi. Vid påvisad manifest cancer bör reoperation ske av gynekologisk tumörkirurg. Om STIC påträffas vid profylaktisk salpingektomi, se [avsnitt 8.3 om STIC](#).

SOEB som utförs premenopausalt kan även minska risken för bröstcancer bland BRCA-bärare, för djupare resonemang hänvisas till det nationella vårdprogrammet för bröstcancer, avsnitt 10.3.7.4.

Att samtidigt med profylaktisk SOEB överväga profylaktisk hysterektomi kan göras i enskilda fall vid tilläggsindikation men bör inte ske rutinmässigt [77].

#### 6.3.5.1 Profylaktisk kirurgi vid Lynchs syndrom

Den mest effektiva riskreducerande åtgärden är profylaktisk SOEB samt hysterektomi. Detta rekommenderas efter avslutat barnafödande, från cirka 35–40 års ålder [78, 79].



### 6.3.5.2 Profylaktisk kirurgi vid mutation i BRIP1, RAD51C och RAD51D

Risken för äggstockscancer vid patogen variant i BRIP1, RAD51C eller RAD51D motsvarar risken vid Lynchs syndrom i högre ålder. Särskilt kvinnliga bärare som också har en nära släkting med äggstockscancer kan därför överväga profylaktisk SOEB från 50–55 års ålder, eller tidigare om ungt insjuknande finns i familjen [53, 68, 72].

### 6.3.6 Hormonell antikonception och substitution

Att hamna i prematur menopaus, framför allt om kirurgi utförts före 40 års ålder, innebär en ökad risk för osteoporos, depression och nedsatt libido. I enlighet med det nationella vårdprogrammet för bröstcancer rekommenderas att kvinnor med BRCA1/2-mutation efter riskreducerande salpingooforektomi bör erbjudas HRT upp till cirka 50 års ålder, om det inte föreligger tidigare bröstcancerdiagnos. Kvinnor som genomgått riskreducerande salpingooforektomi efter tidigare östrogenreceptorpositiv bröstcancerdiagnos bör inte erbjudas HRT. Kunskapsläget avseende HRT är oklart efter behandling av östrogenreceptornegativ bröstcancer.

Flera studier och metaanalyser visar att p-pillerbruk minskar insjuknande i äggstockscancer med 30–50 % hos såväl kvinnor i den allmänna populationen som kvinnor med BRCA1- och BRCA2-mutation [80-82]. Huruvida risken för bröstcancer ökar hos mutationsbärare som använt p-piller är oklart och motstridiga resultat rapporteras i litteraturen [80, 83]. Risken för bröstcancer kan möjligen vara relaterad till den äldre typen av p-piller [81].

Vid Lynchs syndrom rekommenderas HRT efter profylaktisk SOEB och hysterektomi upp till cirka 50 års ålder, då man sett ökad dödlighet bland kvinnor med Lynchs syndrom som inte substitueras. Vad gäller minskad risk för äggstockscancer hos kvinnor med Lynchs syndrom i samband med p-piller så är detta oklart, men en skyddande effekt är sannolik [78, 82].



## KAPITEL 22

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## KAPITEL 24

# Vårdprogramgruppen

## 24.1 Vårdprogramgruppens sammansättning

Den nationella arbetsgruppen består av en representant per regionalt cancercentrum samt en ordförande som utsetts av RCC i samverkan. Gruppen har eftersträvat multiprofessionalitet med representanter för de olika vårdnivåer som är engagerade i patientens vårdflöde. Utöver detta har patientföreträdare deltagit.

## 24.2 Vårdprogramgruppens medlemmar

Christer Borgfeldt, ordförande, professor, överläkare, kvinnokliniken, Skånes Universitetssjukhus

Ulrika Ottander, docent, överläkare, Centrum för Obstetrik och Gynekologi

Kristina Aglund, överläkare, Cancercentrum

Eva Lundin, professor, överläkare, kliniken för klinisk patologi/cytologi  
samtliga vid Norrlands Universitetssjukhus

Karin Glimskär Ståhlberg, docent, överläkare, kvinnokliniken

Hanna Dahlstrand, docent, överläkare, onkologiska kliniken  
samtliga vid Akademiska sjukhuset

Håkan Geijer, professor, överläkare, bild- och funktionsmedicin

Per Ingverud, överläkare, bild- och funktionsmedicin  
samtliga vid Universitetssjukhuset Örebro

Joseph Carlson, docent, överläkare, kliniken för klinisk patologi/cytologi

Sahar Salehi, medicine doktor, biträdande överläkare, patientområde bäckencancer, tema cancer

Elisabeth Hjerpe, medicine doktor, överläkare, patientområde bäckencancer, tema cancer

Angelique Flöter Rådestad, Docent, överläkare, patientområde ärftlig cancer, tema cancer  
samtliga vid Karolinska Universitetssjukhuset

Preben Kjølhede, professor, överläkare, kvinnokliniken

Gabriel Lindahl, doktorand, specialistläkare, onkologiska kliniken

Elisabeth Åvall Lundqvist, överläkare, professor onkologiska kliniken  
samtliga vid Universitetssjukhuset i Linköping

Pernilla Dahm-Kähler, docent, överläkare, kvinnokliniken

Maria Dimoula, överläkare, Jubileumskliniken

Henrik Leonhardt, medicine doktor, överläkare, radiologiska kliniken  
samtliga vid Sahlgrenska Universitetssjukhuset

Susanne Malander, medicine doktor, överläkare, VO Hematologi, patologi och strålningsfysik  
 Anna Måsbäck, medicine doktor, överläkare, [Laboratoriemedicin](#), [patologi](#)  
 Samtliga vid Skånes Universitetssjukhus, Lund

Carina Rundström, Samordnande kontaktsjuksköterska och processledare gynekologisk cancer  
 Regionalt cancercentrum Stockholm Gotland, Specialistsjuksköterska i cancervård  
 Karolinska Universitetssjukhuset

Margaretha Sundsten, representant för Gyncancerföreningarnas nationella samarbetsorganisation  
 Gynsam

Christian Staf, statistiker, stödjande RCC Väst  
 Elin Ljungqvist, nationell vårdprogramhandläggare/utvecklingsledare, stödjande RCC Väst  
 Malin Samuelsson, nationell vårdprogramhandläggare/utvecklingsledare, stödjande RCC Väst

### 24.3 Adjungerade författare

Ultraljud: Elisabeth Epstein, docent, överläkare, kvinnokliniken, Södersjukhuset, Stockholm

Fertilitet: Kenny Rodriguez-Wallberg, docent, överläkare, fertilitetsenheten, Karolinska  
 Universitetssjukhuset Huddinge

ERAS: Lena Wijk, medicine doktor, överläkare, kvinnokliniken, Universitetssjukhuset Örebro

Mutationsanalyser: Hans Ehrencrona, docent, överläkare, klinisk genetik, [Laboratoriemedicin](#),  
 Skånes Universitetssjukhus

### 24.4 Jäv och andra bindningar

Inga medlemmar i den nationella vårdprogramgruppen har pågående uppdrag som skulle kunna innebära jäv. Kopior av hela gruppens jävsdeklarationer, inklusive föreläsaruppdrag, går att få från Regionalt cancercentrum i Uppsala Örebro.

### 24.5 Vårdprogrammets förankring

Vårdprogrammet har utarbetats på uppdrag av RCC:s samverkansgrupp, vilken utsett professor och överläkare Christer Borgfeldt till vårdprogramgruppens ordförande.

I en remissrunda har nedanstående organisationer lämnat synpunkter på vårdprogrammets innehåll: Styrelsen för svensk sexologi, Region Halland, Region Skåne, Arbetsutskottet Region Skåne, RCC Norr, NAC 5 Håkan Dalvik, Närhälsan Vänersborg/Ljungskile, Medicinck rådgivare Primärvård VGR, SFOG, Anestesi och Intensivvård, Dietisternas Riksförbund, Sveriges arbetsterapeuter, Sjuksköterskor i cancervård, Svensk sjuksköterskeförening, SWEDPOS, Astra Zeneca, PNR RCC Väst, Nätverker mot gynekologisk cancer, Hanna Rapp, ÖL Kvinnokliniken Gävle Sjukhus, Nationella vårdprogrammet för cancerrehabilitering Gunnar Eckerdahl, Region Uppsala/Örebro, Gyncancerföreningen i Värmland, Svensk förening för medicinsk radiologi, SFPM, Sektionen för onkologisk och palliativ fysioterapi, Region Blekinge, Joy Ellis Mödravårdsöverläkare och medicinsk rådgivare Regiongemensam hälso- och sjukvård, VGR, Västra Götalandsregionen, Svensk kuratörsförening.