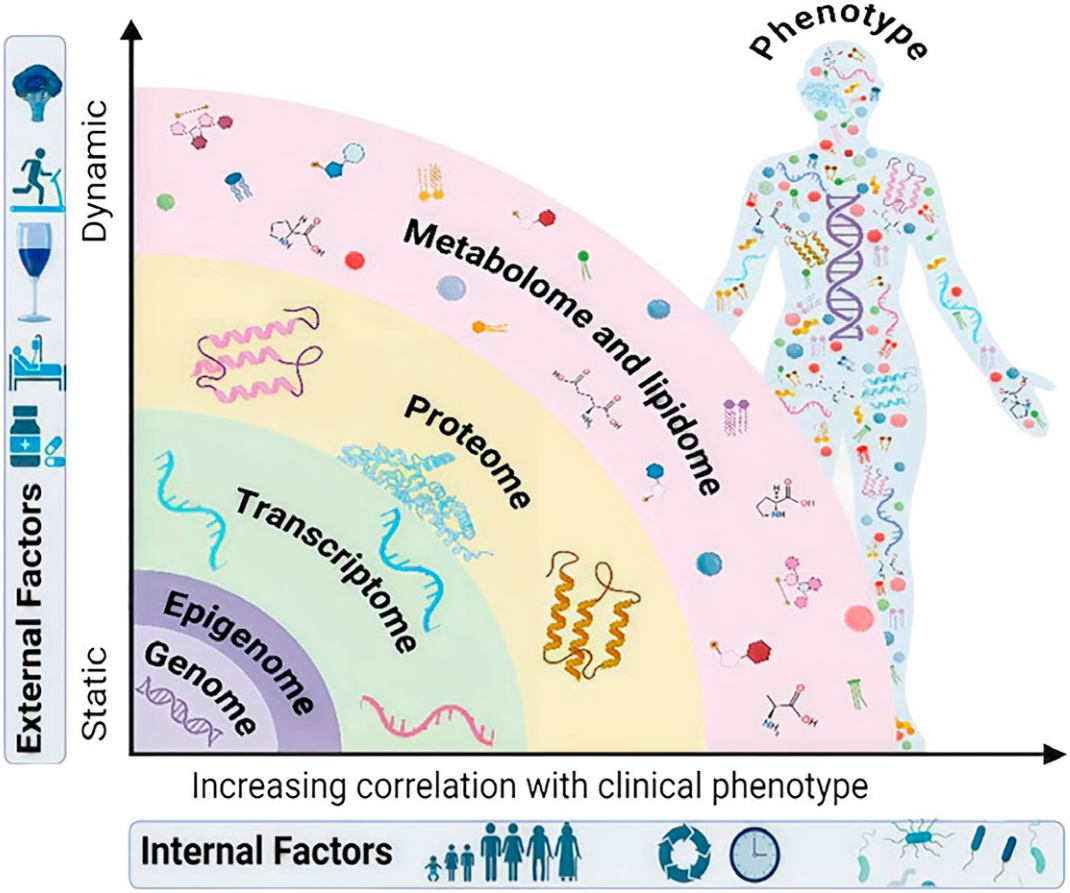
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Klinisk Biokemi i Norden



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Leder: Spør etter SKUP!

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Visste du at du som laboratorielege eller bioingeniør kan bidra til at flere pasientnære analyseinstrumenter blir undersøkt av en uavhengig instans før de tas i bruk?

Pasientnære analyser (point-of-care-testing; POCT) benyttes i økende grad for diagnostikk og oppfølging i helsevesenet, og anvendelsen er spådd å fortsette å øke kraftig i tiden fremover (1). Slike analyser kan gi raskere diagnostikk og behandling, avlaste sykehusene, og legge til rette for bedre desentraliserte helsetjenester. Samtidig er ikke alltid analysekvaliteten like god som for tradisjonelle laboratorieanalyser. Begrenset opplæring hos personellet som utfører testene - med påfølgende økt risiko for både preanalytiske, analytiske og postanalytiske feil - er også en utfordring ved bruk av pasientnære analyser.

For å redusere risikoen for feildiagnostikk og feilbehandling, er det derfor viktig å verifisere instrumentets analytiske kvalitet før bruk - dette er også forankret i ISO 15189:2022, som stiller krav til kvalitetsstyring av POCT. Faktorer som presisjon, riktighet, interferenser, brukervennlighet, deteksjonsgrenser, og pris bør vurderes. Helst bør en slik vurdering utføres av en leverandøruavhengig instans, men dette er tidkrevende, og bruker av utstyret har ikke alltid nødvendig kompetanse eller ressurser til å utføre denne typen utprøvninger.

Det er her SKUP kommer inn i bildet

SKUP (Skandinavisk utprøving av laboratorieutstyr for pasientnær analysering) har som formål å forbedre kvaliteten på pasientnær analysering ved hjelp av objektive og leverandøruavhengige utprøvinger av POCT-instrumenter. Organisasjonen ble stiftet i 1997, og er et samarbeid mellom kvalitetsorganene DEKS i Danmark, Equalis i Sverige, og Noklus i Norge.

Det høres jo supert ut å få utført uavhengige tester av nytt laboratorieutstyr man vurderer å anskaffe, så da er det vel bare å bestille en evaluering? Dessverre er det ikke så enkelt. SKUPs utprøvinger bestilles nemlig av instrumentprodusentene eller leverandørene selv; disse betaler også de direkte kostnadene ved utprøvingen. Det er dermed i stor grad opp til leverandørene hvilke instrumenter som blir evaluert. Noen har kanskje allerede gjennomført egne undersøkelser og ser liten nytte i å betale for enda en evaluering, andre vurderer muligens kostnadene som for høye i forhold til den forventede gevinsten. Uansett hva som forhindrer flere SKUP-utprøvinger, kan DU - kjære kollega - være med å bidra! Ved å etterspørre SKUP-evalueringer før man går til anskaffelse av nytt utstyr, kan vi trolig gjøre slike utprøvinger mer attraktive for instrumentleverandørene, og gi dem en grunn til å prioritere dem.

Allerede i 2017 tok SKUP til orde for at POCT-utprøvinger burde være fullfinansiert av offentlige helsemyndigheter, og at uavhengige tester av laboratorieutstyr som skal selges i Skandinavia burde være obligatorisk (2). Dessverre er det fortsatt ikke slik, men det betyr ikke at det ikke er noe vi kan gjøre: Neste gang en instrumentleverandør eller kliniker henvender seg til deg med spørsmål om anskaffelse av et nytt POCT-instrument, kan vi for eksempel spørre: «Finnes det en SKUP-rapport for dette instrumentet? Hvis ikke, har leverandøren vurdert å bestille en utprøving?».

Jo flere som etterspør uavhengig dokumentasjon, desto større blir forhåpentligvis motivasjonen for leverandørene til å få utstyret sitt evaluert. Slik kan vi bidra til bedre kvalitet, tryggere pasientnær diagnostikk, og mindre dobbeltarbeid ved sykehus og legekontorer.

Så gjør det til en vane: **Spør etter SKUP!**

Ressurser/kilder

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Mer informasjon og publiserte utprøvingsrapporter: www.skup.org

Chair´s column

Anna Linko-Parvinen
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To begin with, I wish to thank the NFKK Board for the honour of serving the Nordic community as the new chair of the NFKK. My journey in Nordic collaboration started in 2017, when I became chair of the Finnish Society of Clinical Chemistry. Almost a decade has passed since then, and many things have changed nationally, across the Nordic countries, in Europe and throughout the world. However, some things remain unchanged.

First, our shared Nordic values

I was born, and still live, in Åbo/Turku, Finland. My city has a long history and strong connections to Sweden, not least because it was once one of the largest cities in the Swedish realm. In many ways, not merely historically but also today, we in the Nordic countries belong to the same family. We share the same values and largely the same view on life. These values and the foundation they provide for great collaboration are something we should cherish.

Second, our history of collaboration

Reading about the history of the 80-year-old Finnish Society of Clinical Chemistry and browsing past issues of *Klinisk Biokemi i Norden*, with a little help from AI, reminds me that Nordic collaboration has been a driving force in laboratory medicine for decades. There have been major joint Nordic initiatives, ranging from reference intervals to education and professional development. These achievements demonstrate what we can accomplish by sharing knowledge and resources across national borders.

The NFKK courses are an example of this. I have had the privilege of participating in three of the four Nordic courses: the Nordic Course for MDs in Specialist Training, which I attended rather late in my specialisation; the Helene Course, where I served as a facilitator; and Quality Hike, which I helped organise. From what I have heard, the Arctic Experience scientific writing course is also highly recommended. Each course has its own purpose and target audience, and I warmly recommend them to my Nordic colleagues. Beyond their educational value, these courses provide opportunities to build professional networks and friendships that last well beyond the courses themselves.

The biennial Nordic Congress in Clinical Biochemistry can be described as legendary. Each congress has had a warm atmosphere with excellent speakers. Once more, I wish to thank the Danish organisers for this year's congress in Aarhus!

Third, looking towards the future

As Søren Kierkegaard wrote, "Life can only be understood backwards; but it must be lived forwards." Looking back at our shared history helps us appreciate what Nordic collaboration has achieved. However, we must also look ahead and consider how we can continue to develop our community and respond to the changing needs of laboratory medicine.

Our field is evolving rapidly, with new technologies, artificial intelligence and changing expectations for the contribution of laboratory medicine to patient care. I believe that Nordic collaboration offers us an excellent opportunity to address these developments together. In addition to the above-mentioned courses we have had and still have working groups focusing on specific topics. We wish to keep these groups active and also plan new initiatives around them.

I wish to thank Per Bjellerup for his excellent leadership as chair over the past years. I believe that every chair leaves a mark on the NFKK. My hope is that I, too, will be able to contribute something meaningful to the NFKK and the wider Nordic community. Lastly, if you, as a member of this society, have an idea you would like to share with your colleagues or a suggestion for collaboration, please feel free to contact me or any other board member! At this darkest time of the year, I wish you all warmth, happy holidays and crisp winter days!



TAK FOR EN VELLYKKET 40.

Nordiske Kongres i Klinisk Biokemi!

Den 40. Nordiske Kongres i Klinisk Biokemi er nu veloverstået, og vi vil gerne rette en stor og hjertelig tak til alle, der bidrog til at gøre kongressen i Aarhus til en mindeværdig begivenhed.

Med mere end 500 deltagere fra over 25 lande samlede kongressen kolleger, forskere, klinikere og repræsentanter fra industrien fra hele Norden og resten af verden. Det var en stor glæde at byde både velkendte ansigter og nye kolleger velkommen til nogle inspirerende dage med fokus på udviklingen inden for klinisk biokemi og laboratoriemedicin.

Det videnskabelige program var kongressens omdrejningspunkt og omfattede 27 videnskabelige sessioner og seks plenare foredrag. Vi var meget glade for den høje kvalitet i præsentationerne, de mange engagerede diskussioner og den livlige udveksling af erfaringer og idéer!

De Nordiske kongresser handler om meget mere end det videnskabelige program. Det er også her vi møder gamle og nye kolleger, styrker faglige netværk, skaber nye kontakter og nyder det særlige fællesskab, der kendetegner det nordiske miljø inden for klinisk biokemi. Vi håber, at de sociale arrangementer gav rig mulighed for netop dette.

Den efterfølgende evaluering af kongressen fremhævede det videnskabelige indhold og de sociale aktiviteter som fik meget positive tilbagemeldinger. Til gengæld var der blandede bemærkninger til kon-

gresfaciliteterne, som af mange blev oplevet som for beskedne og støjende. Det er godt at der er plads til forbedring til de næste kongresser!

En kongres af denne størrelse kan ikke gennemføres uden en stor indsats fra mange mennesker. Vi vil derfor gerne rette en særlig tak til alle foredragsholdere, sponsorer, udstillere, medlemmer af organisationskomitéen, frivillige og øvrige kolleger, der har arbejdet bag kulisserne og under selve kongressen. Jeres engagement og indsats var helt afgørende for, at kongressen blev en succes.

Afslutningsvis vil vi gerne takke alle jer, der rejste til Aarhus og deltog i den 40. Nordiske Kongres i Klinisk Biokemi. Vi håber at ses igen og glæder os allerede til at videreføre traditionen og mødes ved den 41. Nordiske Kongres i Klinisk Biokemi i Bergen i 2028!



DGI Huset Aarhus, stedet for kongressen.
Foto: Helle B. Hager.

På kongreskomiteens vegne



Holger Jon Møller, Chair of Scientific Committee.
Mie Samson, President of Congress.
Anne Winther Larsen. Birgitte Sandfeld Paulsen.



Størstedelen af kongresskomitéen samler kræfter før startskuddet. Foto: Anne-Birgitte Gram Blavnsfeldt.



Kongressens præsident, Mie Samson, klar til at åbne kongressen. Foto: Anne-Birgitte Gram Blavnsfeldt.



Fra Get Together på Centralværkstedet. Foto: Helle B. Hager.



Hovedsal. Foto: Per Bjellerup.



Vinderne af NFKK Young Researcher Award (NYRA): Fra venstre Joel Simrén, Jonas Ghouse, Lars Melholt Rasmussen (formand for priskomiteen) og Burak Arslan. Foto: Per Bjellerup.



Kongreskomitéen ved afslutningsceremonien. Foto: Margunn Bye Tøsdal.

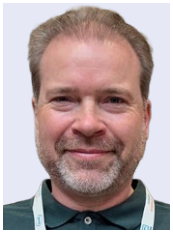
Reflektioner kring det nordiska mötet i Klinisk Kemi i Århus 2026 från några deltagare från Klinisk kemi, Karolinska, Stockholm



”Kongressen var väl organiserad. Programmet var varierat och intressant, med forskning i framkant inom klinisk kemi, till exempel Alzheimers blodbiomarkörer och integrering av AI i kliniken. Aarhus är en vacker stad och det var väldigt trevligt att ta en kvällspromenad. Kongressen hade också fina inslag för oss deltagare, som att dela ut en gåva till dem som presenterade samt ge ut vouchrar med fri entré till konstmuseet ARoS i staden.”, **Javier Perez**, *sjukhuskemist*.



”Programmet bjöd på en rad intressanta teman. Sektionen om lipidanalyser var givande, och man diskuterade där olika diagnostiska markörer vid hyperkolesterolermier, bl.a. framhölls att VLDL-kolesterol bör användas mera. Också en bra föreläsning om hyperlipidemier under graviditet och menopaus. Andra intressanta områden var användningen av masspektrometri vid diagnostik av myelom och diskussion om biomarkörer vid kroniska njursjukdomar. En annan programpunkt rörde sig om mikrovesiklar och deras framtida användbarhet. Bl.a. underströks att det fortfarande kommer att behövas mycket arbete innan mikrovesiklar kan etableras som kliniskt relevanta parametrar inom diagnostiken.”, **Gösta Eggertsen**, *prof emeritus, överläkare*.



”Århus är en trevlig och promenadvänlig stad, med bland annat ett charmigt restaurangstråk längs en kanal och ett växthus som var väl värt ett besök. Onsdagens båttur var också ett uppskattat inslag, med vackra vyer över staden. Vetenskapligt var det hög kvalitet på postrarna och framför allt utvecklingen inom LC-MS fångade mitt intresse, men även de många AI-relaterade inslagen och framtidsspaningen om tabulära AI-modeller.”, **Magnus Hansson**, *överläkare*.



”Kongressen inleddes starkt med Jens Juul Holsts föreläsning om upptäckten av inkretinerna, däribland GLP-1, och om hur ihärdighet och nyfikenhet kan leda till stora genombrott. Föreläsningarna visade också tydligt hur AI är på väg in i den kliniska vardagen, från diagnostiska algoritmer till validering och kvalitetskontroll. Konstmuseet ARoS var näst intill värt resan i sig, och galamiddagens servitörer, hämtade direkt från 1920-talet, var ett minnesvärt inslag. Mest givande var ändå samtalen med kollegor från hela Norden och Baltikum, där det visade sig att vi brottas med samma problem i våra laboratorier. Att jag dessutom vann konferensens quiz, och en LEGO Yoda-statyett, gjorde inte saken sämre.” **Gustav Hallén**, *specialistläkare*.



Taket på Aros konstmuseum. Foto: eyewave - stock.adobe.com

Notes from my first Nordic congress in clinical biochemistry

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In September, I had the pleasure of participating in my first Nordic Congress in Clinical Biochemistry, which happened to be the festive 40th. This was also my first ever visit to Aarhus. For my pastime, exploring the cosy and walkable host city was more than appropriate.

As a fairly new member of this community, I felt excitement and some nervousness, especially a few days before the event kicked off. The latter fortunately faded soon after the beginning of the congress, thanks also to meeting my fellow editors of KBN beforehand.

Though my inexperience still heightened my shyness when it came to mingling and connecting with international colleagues (which may remain a work in progress), I certainly did not leave the congress without new acquaintances. The atmosphere was warm all in all.

The subjects of the sessions were varied, stretching from biomarkers for, e.g., neurological diseases to artificial intelligence (AI). For someone mostly working within laboratory haematology, this set was a comprehensive introduction to some of the developments in the field, and I was grateful to hear about them first-hand from those involved. My notes included several questions for myself to dig deeper into. I count this as a sign of inspiring talks. I was also happy to notice how involved younger researchers were.

In the main lecture hall, I was a regular at the higher tables, which made writing notes, observing, and drinking nearly endless amounts of coffee enjoyable despite some conversation overheard from the adjacent exhibition hall. A detail that caught my eye in the audience whilst listening to the sessions was knitting; how wholesome, and why didn't I think of bringing my not-yet-finished handicrafts? So: note taken, though not in writing, for next time.



The exhibition hall from "a bird perspective".



My setting in the main hall (without the coffee).

Reisebrev fra Århus

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Som relativt nye leger i spesialisering var det spennende å delta på den nordiske kongressen i biokjemi i Århus i 2026. Det var mange kjente og ukjente ansikt, stor bredde i foredragene og gode sosiale arrangementer.

Det faglige nivået var høyt. Noe var det lett å bli engasjert i, mens andre ting utfordret oss som halvferske leger. Vi skal ikke påstå at vi fikk med oss alle detaljene, men det var inspirerende å få se hvor mye som foregår i faget og hvor ivrige kollegaer vi har. Denne kongressen var virkelig en god demonstrasjon av hva faget kan tilby.

Selv om det faglige ble prioritert høyt, var også menneskene et høydepunkt. Å bli kjent med flere yngre og eldre kollegaer fra forskjellige land er både gøy og viktig. Kongressen la godt til rette for dette med middager, joggeturer, yoga, cruise og, ikke minst, storslått festmiddag, rock-and-roll og dans.

I fremtiden håper vi at enda flere unge leger deltar på kongressen, slik at vi får bygget samhold og delt erfaringer. Hvem vet, kanskje står vi på scenen selv en dag?



Trygve og Helena. Foto: Helle B. Hager.



The Lorentz Eldjarn prize competition 2026

Recognition of outstanding scientific contributions in the Scandinavian Journal of Clinical and Laboratory Investigation

Jens Petter Berg
Managing Editor, Scandinavian Journal of Clinical and Laboratory Investigation
Professor, University of Oslo and Department of Medical Biochemistry, Oslo University Hospital



Background and Statutes of the Lorentz Eldjarn Prize

The Lorentz Eldjarn Prize competition was held for the seventh time during the 40th Nordic Congress in Clinical Biochemistry in Aarhus 15th-18th September 2026. The prize is funded by the Lorentz Eldjarn Fund, which was established through a generous donation from Professor Lorentz Eldjarn and his wife Torunn in 2009. The primary goal of the fund and the associated competition is to promote and stimulate high-impact Nordic publications in the Scandinavian Journal of Clinical and Laboratory Investigation (SJCLI), where Professor Eldjarn was Editor-in-Chief for 16 years.

Professor Lorentz Eldjarn (1920–2007) had several important roles in Nordic clinical chemistry and laboratory medicine. Throughout his remarkable career, he connected basic biochemical and biological sciences with clinical medicine. His engagement and dedication to Nordic collaboration has created long-lasting impact on academic excellence in our discipline.

According to the statutes of the competition, the prize is awarded every second year during the Nordic Congress in Clinical Biochemistry. The competition evaluates original research articles published in SJCLI

Overview of the 2026 award winners

Prize	Article title and authors	Journal reference
1st Prize (DKK 100,000)	The alcohol biomarker phosphatidylethanol (PEth) – test performance and experiences from routine analysis and external quality assessment. Anders Helander & Therese Hansson	Scand J Clin Lab Invest. 2023;83:424-431
2nd Prize (DKK 50,000)	Mild impairment of renal function (shrunken pore syndrome) is associated with increased risk of a future first-ever myocardial infarction in women. Elisabet Söderström, Ravna Blind, Patrik Wennberg, Jonas Andersson, Stefan Söderberg, Torbjörn K. Nilsson, Johan Hultdin	Scand J Clin Lab Invest. 2021;81:438-445
3rd Prize (DKK 30,000)	Platelet parameters and leukocyte morphology is altered in COVID-19 patients compared to non-COVID-19 patients with similar symptomatology. Anne Alnor, Maria Boysen Sandberg, Barbara Ella Toftanes, Pernille Just Vinholt	Scand J Clin Lab Invest. 2021;81:213-217

during the five years preceding the congress year. To be eligible for nomination, at least one of the article's authors must be engaged in activities related to medical biochemistry at an institution in a Nordic country. From the 20 most cited articles over the five-year period, a prize committee consisting of members of the SJCLI editors evaluates and nominates three finalist articles. The first authors of the nominated articles are invited to present their research during a seminar at the Nordic congress. The focus shall be on the scientific impact of their study and its influence on subsequent research.

For the 2026 competition, the Prize Committee was chaired by Managing Editor Jens Petter Berg (Norway) and comprised editor members Anna Linko-Parvinen (Finland), Elvar Theodorsson (Sweden), and Claus Vinter Bødker Hviid (Denmark). Based on scientific quality and the clarity of the presentations, the committee awarded the 1st, 2nd, and 3rd prizes consisting of DKK 100,000, DKK 50,000, and DKK 30,000, respectively, together with diplomas at the congress banquet 17th Sep 2026.



Summaries of the award-winning articles

1st prize: Phosphatidylethanol (PEth) measurement, quality assurance, and decision limits

Anders Helander and Therese Hansson. *The alcohol biomarker phosphatidylethanol (PEth) – test performance and experiences from routine analysis and external quality assessment. Scand J Clin Lab Invest. 2023;83:424-431.*

Phosphatidylethanol (PEth) comprises a group of phospholipid membrane molecules formed in red blood cell membranes via a transphosphatidylation reaction catalyzed by phospholipase D (PLD) in the presence of ethanol. Because ethanol is an absolute requirement for its formation, PEth exhibits near 100% specificity as a direct alcohol biomarker. Upon repeated alcohol exposure, PEth accumulates in blood cell membranes and is eliminated slowly during abstinence, with a half-life ranging between 4 and 10 days (median 6 days). Consequently, PEth reflects alcohol consumption over the preceding 2 to 6 weeks and fills a major diagnostic gap between short-term direct markers (breath/blood ethanol, urinary ethyl glucuronide) and non-specific indirect markers (CDT, GGT, MCV).

The review and analytical study by Helander and Hansson provide a comprehensive presentation of test performance, routine clinical implementation, and external quality assessment (EQA). While initial routine methods measured total PEth using HPLC with light-scattering detection, modern clinical laboratories regularly use liquid chromatography-tandem mass spectrometry (LC-MS/MS). LC-MS/MS can specifically quantify PEth 16:0/18:1 (containing palmitic acid and oleic acid), which represents the single predominant molecular subform in human blood and correlates tightly with total PEth concentration.

In 2013, a national Swedish harmonization initiative established standardized clinical decision limits for whole-blood PEth. Values below 0.05 µmol/L (~35 µg/L) indicate sobriety or low/sporadic consumption, whereas values exceeding 0.30 µmol/L (~210 µg/L) indicate regular, harmful alcohol consumption. Analysis of over 120,000 routine clinical samples from Kar-

olinska University Laboratory and Skåne University Hospital demonstrated a high degree of consistency in population distribution. Approximately 45–50 % of routine requests were <0.05 µmol/L, 23–24 % fell between 0.05–0.30 µmol/L, 16–19 % between 0.30–1.0 µmol/L, and 10–12 % exceeded 1.0 µmol/L with individual values occasionally exceeding 5 µmol/L.

An important contribution of the study is the evaluation of the Equalis EQA scheme for whole-blood PEth, which grew from six Swedish laboratories in 2013 to 56 participating laboratories across 13 countries in 2022. The inter-laboratory coefficient of variation (CV) improved from >20% in 2018 to ~12% in 2021–2022 across the analytical range of 0.05 to 3.3 µmol/L. The authors emphasize some key preanalytical considerations. EDTA whole blood stored at 4°C is stable for up to 3 weeks, but samples containing residual ethanol must not be stored at room temperature or frozen at -20°C. PLD remains active at -20°C and can synthesize *in vitro* PEth. The study establishes PEth 16:0/18:1 as a very valuable tool for addiction medicine, occupational health, transplant evaluation, and medico-legal assessments.



2nd prize: Shrunken pore syndrome and first-ever myocardial infarction in women

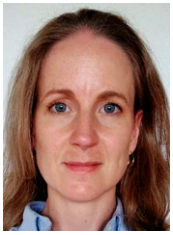
Elisabet Söderström et al. *Mild impairment of renal function (shrunken pore syndrome) is associated with increased risk of a future first-ever myocardial infarction in women. Scand J Clin Lab Invest. 2021;81:438-445.*

Renal function impairment is a well-established risk factor for cardiovascular disease (CVD). Shrunken Pore Syndrome (SPS) is a recently identified pathophysiological state characterized by a selective reduction in the pore size of the glomerular filtration membrane. In SPS, the filtration of medium-sized proteins (10–30 kDa, such as cystatin C) is selectively impaired, while the filtration of small solutes (such as creatinine, 113 Da) remains relatively preserved. Clinically, SPS has been defined by a reduced ratio between cystatin C-based estimated GFR and creatinine-based estimated GFR (eGFRcystatin C / eGFRcreatinine ratio < 0.60).

Söderström and colleagues conducted the first prospective study evaluating whether mild renal impairment defined by the eGFRratio predicts future first-ever myocardial infarction (MI) in individuals free from baseline cardiovascular disease. Using a nested case-referent design within the population-based Northern Sweden Health and Disease Study (NSHDS), the authors identified 545 subjects (29.0% women) who prospectively developed a first-ever MI over a median follow-up of 3.4–4.3 years compared with 1,054 individually matched referents.

The study revealed sex-specific differences in cardiovascular risk prediction. Among women, a lower eGFRcystatin C / eGFRcreatinine ratio was strongly and independently associated with an increased risk of future first-ever MI. In one model, each standard deviation (SD) increase in ln z-score of the eGFRratio conferred a 42% reduction in MI risk (odds ratio [OR] 0.58; 95% CI 0.34–0.99) after adjustment for traditional and biochemical risk factors, including Apolipoprotein B/A1 ratio, high-sensitivity CRP, total homocysteine, systolic blood pressure, body mass index (BMI), and diabetes. Women in the lowest quartile of the eGFRratio experienced significantly higher MI risk compared to those in the highest quartile.

In contrast, no significant association between the eGFRratio and future MI was observed in men. However, men exhibited a significant positive association between eGFRcreatinine and future MI risk (OR 1.25 per SD increase, 95% CI 1.05–1.48), suggesting that glomerular hyperfiltration or altered muscle metabolism may play a distinct role in male cardiovascular pathophysiology. The authors conclude that the eGFRcystatin C / eGFRcreatinine ratio represents a simple, non-invasive, and powerful biomarker for identifying early subclinical atherosclerotic risk in female patients.



3rd prize: Hematological and morphological alterations in COVID-19 patients

Anne Alnor et al. *Platelet parameters and leukocyte morphology is altered in COVID-19 patients compared to non-COVID-19 patients with similar symptomatology. Scand J Clin Lab Invest. 2021;81:213-217.*

The clinical course of SARS-CoV-2 infection is remarkably heterogeneous, ranging from asymptomatic infection to severe acute respiratory distress syndrome (ARDS) and multi-organ failure. While automated complete blood counts are routinely performed during

acute hospital admissions, systematic evaluations comparing automated cell indices and microscopic peripheral blood smear morphology between COVID-19 patients and controls with identical clinical symptomatology had not been performed previously.

In this nested case-control study conducted at Odense University Hospital and Kolding Hospital in Denmark, Alnor and co-workers evaluated 74 RT-PCR-verified COVID-19 positive adult patients and 228 age- and gender-matched non-COVID-19 controls admitted with similar symptoms during the first pandemic wave. A prospective sub-study evaluated peripheral blood smear morphology across 27 COVID-19 positive patients and 18 non-COVID-19 controls using standardized automated digital microscopy (CellaVision DI-60).

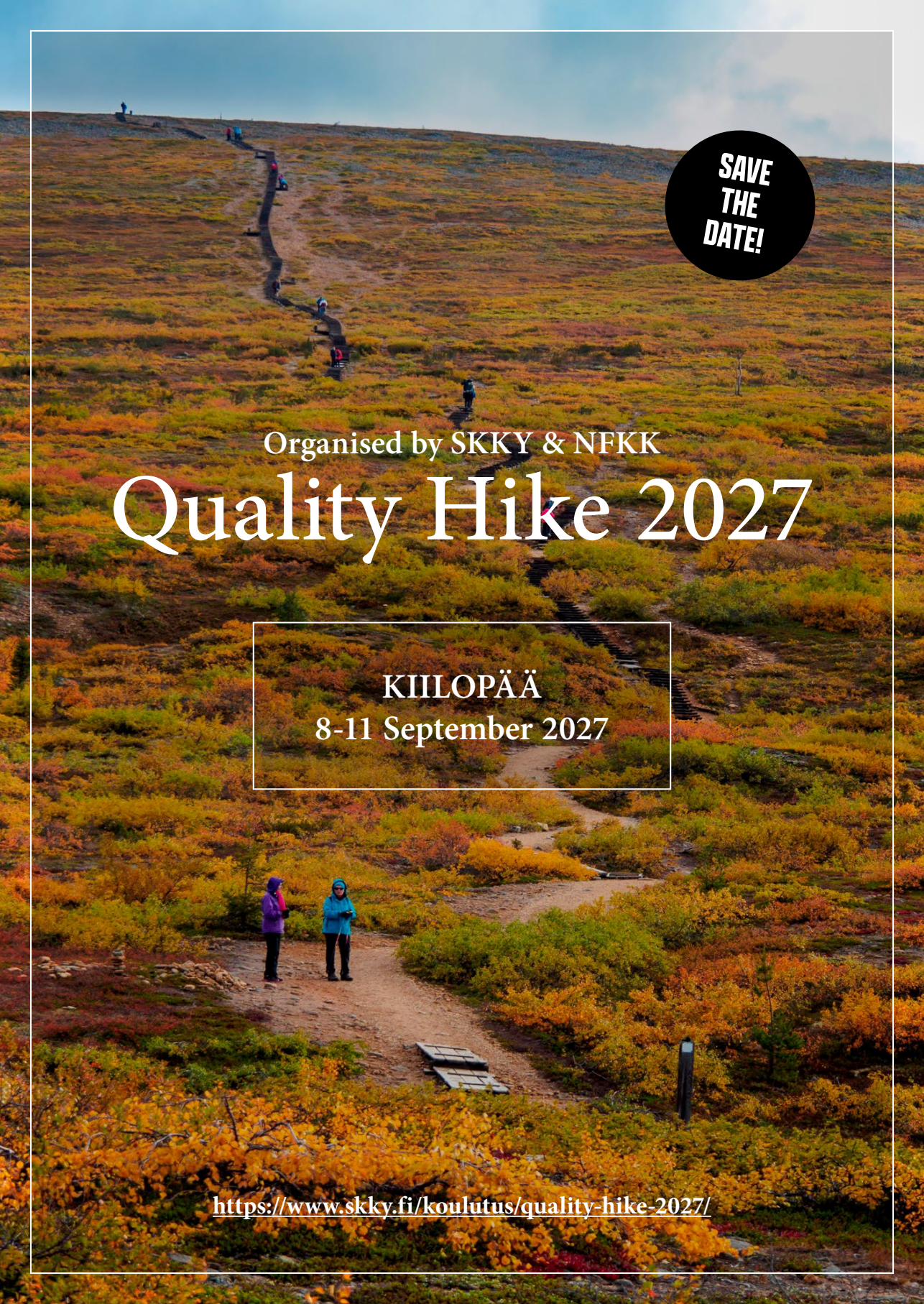
The study uncovered major disease-specific alterations in platelet indices and leukocyte morphology. COVID-19 patients exhibited significantly lower platelet counts (median 186 vs 237 × 10^9/L, $p < 0.001$) and plateletcrit, but significantly higher mean platelet volume (MPV: 10.6 vs 10.1 fL, $p = 0.002$), platelet distribution width (PDW: 12.2 vs 11.2 fL, $p = 0.003$), and platelet large cell ratio (PLCR: 29.4 vs 25.6%, $p = 0.002$) compared to non-COVID-19 controls. These findings are associated with accelerated platelet consumption and compensatory release of larger, hemostatically reactive young platelets and support a central role for platelet activation and microvascular thrombosis in COVID-19 pathophysiology.

All white blood cell (WBC) subpopulation counts were significantly lower in COVID-19 patients than in symptomatic controls. Severe COVID-19 disease (ICU admission or death) was marked by lymphopenia (0.72 vs 0.94 × 10^9/L, $p = 0.008$). On microscopic blood smear examination, only 14.8% of COVID-19 patients displayed a normal blood smear, compared to 83.3% of non-COVID-19 controls ($p < 0.001$). Neutrophil hypogranulation was striking in COVID-19 patients (70.4% vs 0%, $p < 0.001$) and was a direct morphological hallmark of neutrophil degranulation and hyperinflammation during acute viral infection.

Article summaries have been partly written by AI (gpt.uio.no).



The Eldjarn prize winners, from left Elisabeth Söderström, Anders Helander, Anne Alnor and the managing editor of SJCLI, Jens Petter Berg.



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Summary:

Quality assurance of pneumatic transportation systems – Nordic recommendations

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This is a summary of an article published in the Scandinavian Journal of Clinical and Laboratory Investigation (1).

Background

Pneumatic transportation systems (PTS) are used in hospitals to transport blood samples rapidly to the laboratory and thereby reduce turnaround times. However, PTS may cause preanalytical changes in blood samples, particularly haemolysis, and the changes depends on the characteristics of the individual transportation system. Consequently, PTS should be regarded as part of the preanalytical process and requires appropriate validation and continuous quality assurance (2–6).



To assess PTS practices in the Nordic countries, the Nordic preanalytical scientific working group (under NFKK) conducted surveys among clinical biochemistry laboratories in 2018 and 2024 (1). The questionnaire was based on the CLSI H18-A4 guideline and addressed the use, validation and quality control of PTS (7). Altogether, 85 laboratories responded in 2018 and 30 in 2024. Of these, 46 and 27 laboratories, respectively, reported using a PTS.

Not surprisingly, the two surveys demonstrated increasing integration of PTS with total laboratory automation. In 2018, most PTS lines ended in a waiting area, whereas by 2024 most samples were delivered directly to the analytical track or to a bulk sorter connected to it. Among the 2024 respondents, the proportion of all samples transported by PTS ranged from 5% to 95%, with a mean of 50%. Commonly, samples suspected to be especially sensitive to agitation during transport, including blood gases and viscoelastic coagulation tests, were restricted from PTS.

PTS validation had also become more common. The proportion of laboratories that had validated their PTS increased from 69% to 88%. In 2024, haemolysis index and potassium concentration were the most frequently used validation parameters. Other approaches included visible haemolysis, sample rejection rates and G-loggers. However, only 41% of respondents had validated all PTS lines. Furthermore, despite the increased use of validaation, continuous quality con-

trol remained uncommon. Only 10% reported using internal QC and 24% participated in an external quality assessment (EQA) programme.

Recommendations

Based on these findings, the Nordic preanalytical scientific working group recommends that all PTS lines should be validated before clinical use, with predefined quality requirements. G-loggers can be used during validation to characterise acceleration forces, or to localise problematic sections of the transportation system. For routine QC, haemolysis index or potassium values provide practical approaches for detecting changes in PTS performance. Laboratories should define QC performance requirements in advance as well as the actions to be taken when these requirements are not met. Combining different methods, such as haemolysis index monitoring and G-logger measurements, may provide additional information. Analyses susceptible to agitation, haemolysis or other preanalytical errors induced by the PTS should be locally validated before being transported by PTS (8–10).

Quality monitoring should preferably be continuous, automated and line-specific. Patient populations should also be considered because samples from certain groups, such as oncology and paediatric patients, may be particularly vulnerable to transportation-related effects. Participation in an EQA programme is strongly recommended to provide objective and comparable information on the performance of internal QC procedures and permitted PTS protocols.

Highlights

- **Validate every PTS line before clinical use**, using predefined quality requirements and preferably G-loggers as part of the validation.
- **Monitor PTS performance continuously**, using parameters such as haemolysis index or potassium values, with predefined QC limits and actions for QC failure.
- **Use G-loggers to identify problematic sections** when PTS-related preanalytical deviations are suspected.
- **Consider vulnerable samples and patient populations**, particularly when samples may be sensitive to agitation or haemolysis.
- **Participate in external quality assessment programmes** to obtain objective and comparable information on PTS performance.
- **Validate new or sensitive analyses locally before PTS transport**, as the effect of transportation depends on the characteristics of the individual PTS.

Artificial intelligence (ChatGPT) was used to aid in generating this summary from the original publication (1). AI did not generate any data or references.



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Clinical chemistry in the metabolomics era: Advancing diagnostics and precision medicine through comprehensive metabolomic profiling

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Summary
Metabolomics has become the annually most published omics technology (Figure 1). From a single drop of blood, global metabolomics can reveal an extraordinary wealth of biochemical information. As clinical chemists we have the mindset, expertise, experience, and opportunity to build on our established principles of standardization, harmonization, and quality assurance to transform this powerful technology from a predominantly research-based tool into a cornerstone of clinical diagnostics and precision medicine. Clinical genetics has already achieved a similar transformation with whole-genome sequencing. What is stopping us?

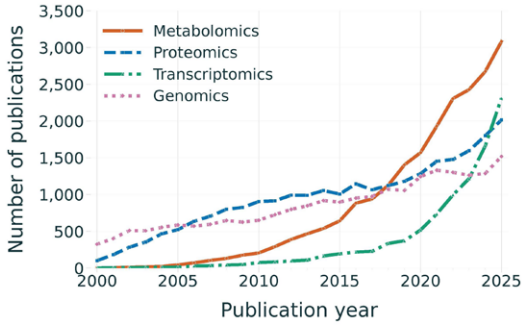


Figure 1. *Annual PubMed-indexed publications with "metabolomics," "proteomics," "transcriptomics," or "genomics" in the title. (Date for search November 5th, 2026. Presented using Excel and Python Matplotlib)*

1. The golden age of clinical chemistry – and a new chapter unfolding…

The 1950s and 1960s were defined a *Golden age of clinical chemistry* (1) due to technical innovations like electrophoresis, radioimmunoassays, improved blood collection systems and automated clinical chemistry analyzers that established the technological foundations of modern laboratory medicine. Multicomponent, simultaneous detection of numerous metabolites was described using gas chromatography and mass spectrometry (MS), primarily used for investigating inborn errors of metabolism (2). Subsequently, nuclear magnetic resonance (NMR) spectroscopy emerged, providing superior analytical and quantitative reproducibility, but with low sensitivity and fewer metabolites detected. Now, the wheel has gone full circle, with liquid chromatography coupled to high-resolution mass spectrometry (LC–HRMS) providing thousand-fold higher sensitivity than NMR and a coverage of thousands of metabolites in any type of human sample. This enables advanced diagnostics and a higher order of personalized medicine for virtually any medical field based on detailed biochemical snapshots of health and disease. The next golden age of clinical chemistry is ours to shape.

2. The biochemical universe of human health and disease – unveiled by metabolomics

Modern HRMS instruments can measure mass (i.e. "mass-to-charge ratio") of molecules with exceptional accuracy, within a few parts per million, and distinguish between compounds with very similar molecular masses

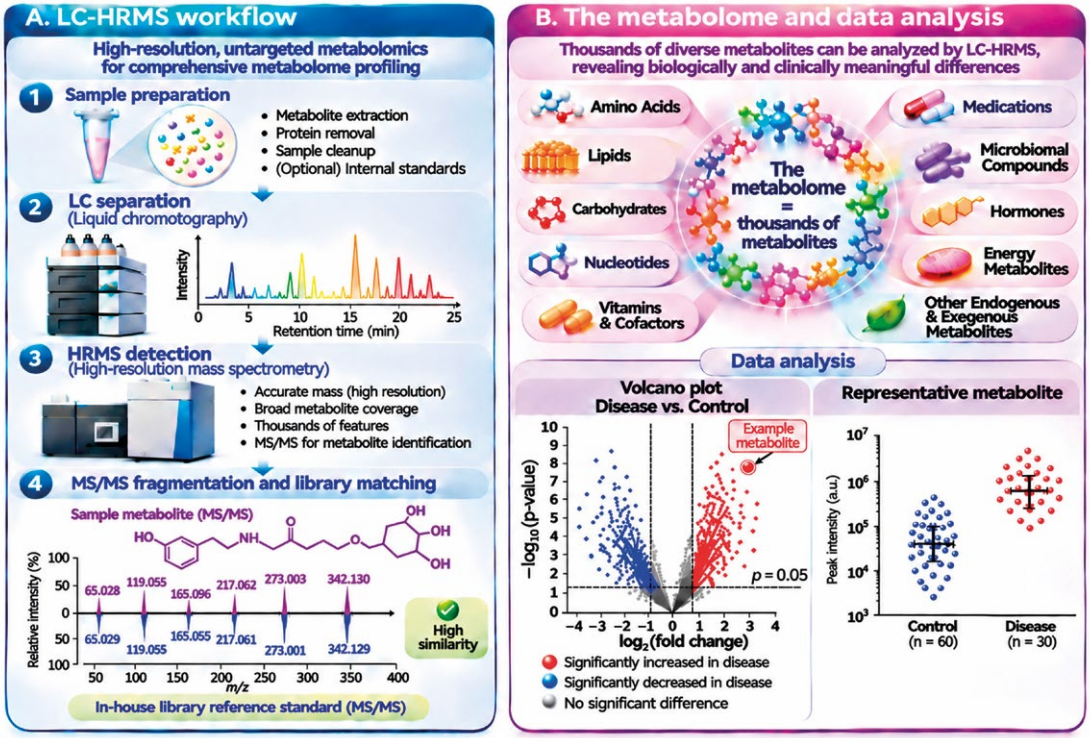


Figure 2. *Graphical illustration of metabolomics analysis comparing disease and control groups. (A) Sample preparation and liquid chromatography–high-resolution mass spectrometry (LC–HRMS) workflow, including MS/MS spectral matching. (B) Major classes of human metabolites (top). Volcano plot (bottom left) showing differences in metabolite abundance (log_2 fold change; x-axis) and statistical significance ($-log_{10}$ p-value; y-axis). A highlighted example metabolite is shown separately (bottom right), displaying MS peak intensities for individual control and disease samples. (Figure generated using ChatGPT. All illustrations, numerical values, and statistical results are illustrative rather than experimental.)*

(e.g. 100.000 vs. 100.001). This mass resolving power is thousandfold higher than for the standard MS instruments used in clinical chemistry routine. To enable the analysis of thousands of molecular compounds, MS is commonly combined with LC for separating metabolites in time according to their chemical properties, such as hydrophobicity. Consequently, different compounds reach the MS at different times (retention time), reducing interference between them. The MS ionizes the compounds and measures their mass-to-charge ratios. Subsequently, the metabolites are fragmented in a collision cell providing a characteristic molecular fingerprint that, together with accurate mass and chromatographic retention time, supports identification of individual compounds. The intensity of the signals provides information about their abundance, allowing relative quantification or, with appropriate calibration and internal standards, absolute quantification. Figure 2 illustrates the metabolomics workflow.

3. The dynamics and extraordinary informative power of the human metabolome

The metabolites constituting the human metabolome are low-molecular-weight compounds being substrates, intermediates and end products of the biochemical processes taking place in our bodies and microbiomes, and also comprise diet-, drug- and environmentally-derived compounds and their biotransformation products.

Metabolomics is the comprehensive study of these metabolites, including dynamic responses to physiological, pathological, environmental factors and therapeutic interventions. The metabolome presents a snapshot of the biochemical status at the time of sampling, reflecting health and disease status.

Whereas genomics is static and reveals genetic characteristics and disease susceptibility, transcriptomics reflects gene expression, and proteomics characterizes the proteins involved in cellular structure and func-

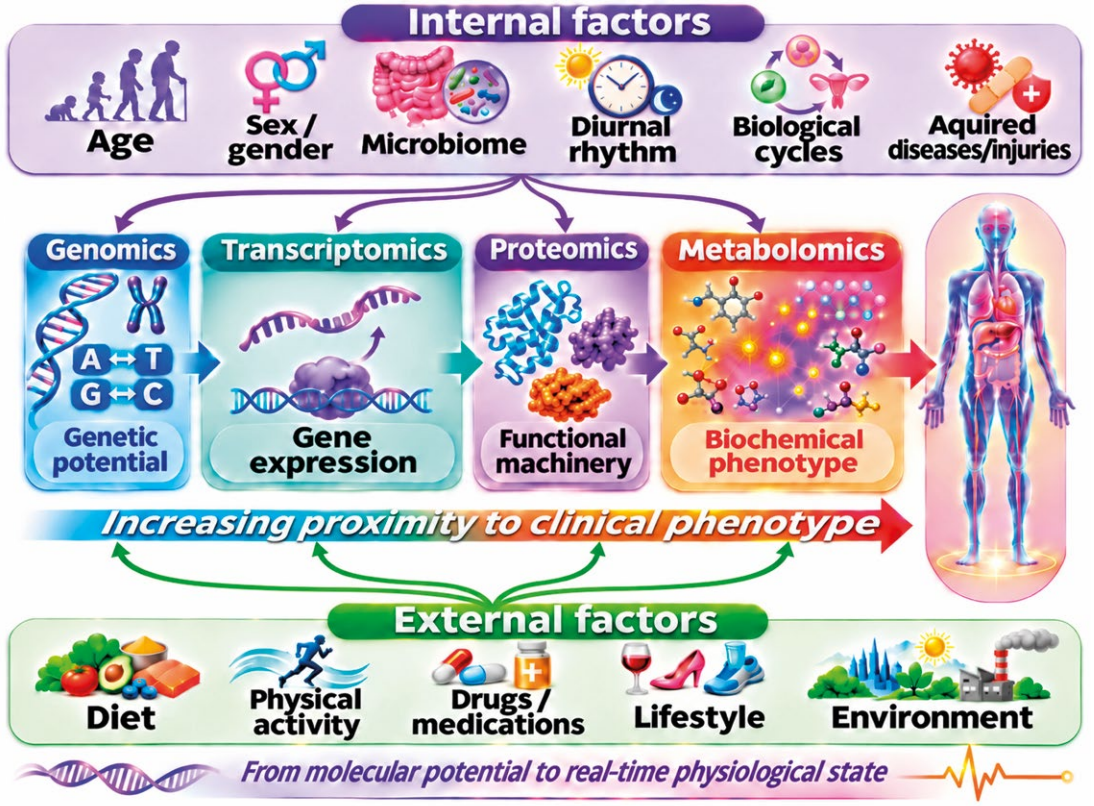


Figure 3. *The metabolome represents the end result of a cascade of biological and environmental events (Figure generated using ChatGPT)*

tion, metabolomics captures the dynamic biochemical phenotype resulting from the combined influences of genetics, age, lifestyle, microbiomes, environmental exposures, random events and injuries, disease, and treatment interventions. The metabolome thus most closely reflects the clinical phenotype (Figure 3).

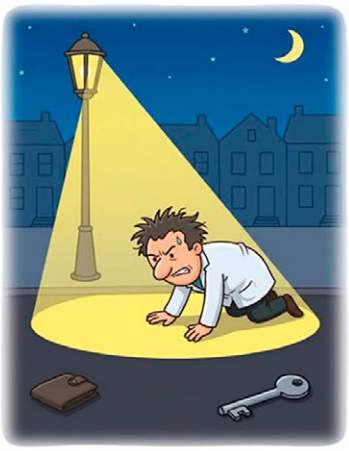
4. From targeted analyses to global discovery – and vice versa

Targeted analyses have traditionally formed the cornerstone of clinical chemistry, focusing on quantification of preselected metabolites. They represent valuable, but still selected glimpses into human metabolism and biochemistry. In contrast, global (or *untargeted*) metabolomics aims to see the whole picture and detect the broadest possible range of metabolites without restricting the analysis to predefined compounds (Figure 4). It is particularly valuable for comprehensive metabolic profiling, biomarker discovery, and hypothesis generation.

When global metabolomics reveal possible biomarkers and disease mechanisms, targeted analyses are subsequently developed to provide absolute quantification and test the hypothesis and provide cost-efficient analysis.

Vice versa, when targeted analyses find abnormalities pointing towards biochemical disturbances, global metabolomics can interrogate all the metabolites in the suspected pathway(s) and molecular networks confirming or challenging the suspected error and mechanism. Targeted and global metabolomics therefore complement each other, with different strengths and applications.

Commercially available kits for targeted metabolomics provide metabolite concentrations based on internal standards and single- or multipoint calibration. However, the accuracy of quantification varies between metabolites, depending on calibration procedure and availability of appropriate internal standards. In contrast, global metabolomics generally provides relative



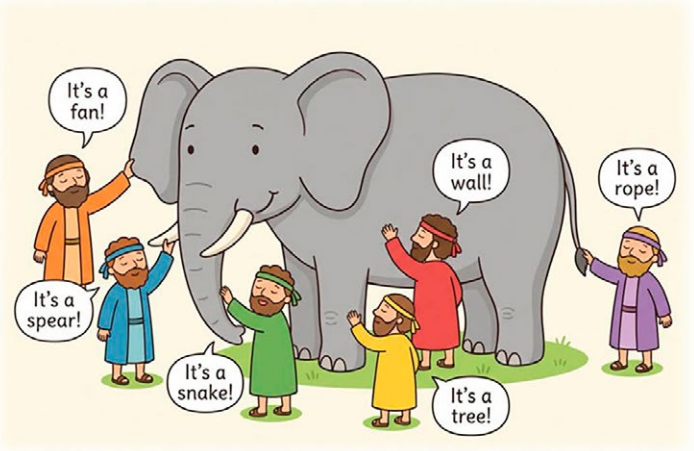


Figure 4. *Quiz: Why perform global/untargeted metabolomics analysis? (Illustrations generated using Google Gemini / Nano Banana 2.1)*

rather than absolute quantification, as individual calibration of the thousands of detected compounds is not practically feasible. Results are typically expressed as normalized signal intensities, allowing comparison of metabolite abundances between e.g. patient samples, control samples, and longitudinal samples from the same individual.

One of the greatest advantages of global metabolomics is its ability to capture information on a very broad range of metabolites, including compounds that are not uniquely identified or considered clinically relevant at the time of analysis. Importantly, all analytical data are stored digitally and can be revisited, reprocessed, and reinterpreted as new knowledge, biomarkers, or clinical questions emerge, without the need to reanalyze the original biological sample. This is conceptually similar to whole-genome sequencing, where previously acquired genomic data can be reexamined in light of novel discoveries or new clinical indications. In contrast, since targeted metabolomics is designed to only measure predefined metabolites, a wish for subsequent investigation of additional metabolites requires new analysis of the biological sample. Thus, only the global metabolomics approach can continue to provide new and clinically valuable information long after the original investigation.

5. Unlocking the potential of global metabolomics – a wealth of information

Clinical chemistry is founded on the principle that health and disease are reflected in human biochemistry, leaving metabolic signatures that can be investigated in biological samples. Global metabolomics offers unprecedented opportunities to explore these signatures and their significance across virtually all areas of biomedical research and clinical medicine:

- **Understanding human physiology:** Characterizing metabolic changes associated with e.g. aging, hormonal status, nutrition, physical activity, and lifestyle.
- **Understanding disease mechanisms:** Identifying disturbed metabolic pathways and biochemical processes underlying disease development and progression, potentially revealing new diagnostic biomarkers and therapeutic targets.
- **Biomarker discovery:** Identifying metabolites and metabolic signatures associated with disease, treatment responses, and clinical outcomes (3).
- **Disease risk assessment and prediction:** Identifying individuals at increased risk of developing diseases, enabling earlier preventive interventions.
- **Early detection and differential diagnoses:** Identifying disease-associated metabolomic abnormalities for early detection and confirmation, or exclusion, of suspected diagnoses.
- **Disease classification and patient stratification:** Identifying disease subtypes and disease severity and distinguishing between patients with similar presentation but different underlying pathophysiological mechanisms.
- **Prognostic assessment:** Predicting progression, complications, and clinical outcomes.
- **Personalized treatment:** Identifying known metab-

olomic signatures associated with treatment responsiveness, resistance, or susceptibility to adverse effects, and to identify novel potential therapeutic targets.

- **Monitoring disease activity and progression:** Tracking metabolomic changes over time to assess disease progression, remission, recovery, and recurrence.
- **Monitoring treatment response:** Evaluating therapeutic efficacy and treatment-related adverse effects to guide timely treatment adjustments, as biochemical responses precede clinical changes and those revealed by imaging.
- **Detection of drugs, toxins, and environmental exposures:** Identifying compounds and metabolites not included in existing targeted panels (4).
- **Detecting non-adherence to treatment:** Most medications and their metabolites are detected and provide evidence of recent drug exposure, and their presence or absence potentially uncover treatment non-adherence, enabling restoration of adherence.
- **Investigating diet–microbiome–host interactions:** Characterizing the metabolic effects of nutrition, gut microbiota, and environmental factors on health, disease, and treatment responses.
- **Functional interpretation of genetic findings:** Identifying biochemical consequences of genetic variants, interpreting variants of uncertain significance, and investigating their wider effects across interconnected metabolic pathways.
- **Equitable access to healthcare:** Dried blood spots (DBS) are suitable for metabolomics, and most metabolites remain stable after drying, allowing shipment at ambient temperatures, as already practiced in newborn screening. This enables home-based self-sampling and postal shipment to specialized laboratories, facilitating *Hospital at Home* functions and potentially expanding access to advanced diagnostics worldwide (5).(Figure 5).

Together, these applications illustrate the huge potential of global metabolomics to advance our understanding of human health and disease and to expand the capabilities of laboratory medicine, particularly in diagnostics, patient monitoring, and precision medicine.

6. From analytical power to clinical reliability – overcoming the bottlenecks

The principal challenge is no longer simply the ability to detect large numbers of metabolites, but to ensure that the resulting measurements are reliable, comparable, biologically interpretable, and clinically useful.

6.1 Pre-analytical and analytical challenges

As specialists in clinical chemistry, we know how to address these challenges (6).

- **Pre-analytical variability:** Metabolomes are affected by diet, fasting, circadian rhythms, medication, phys-

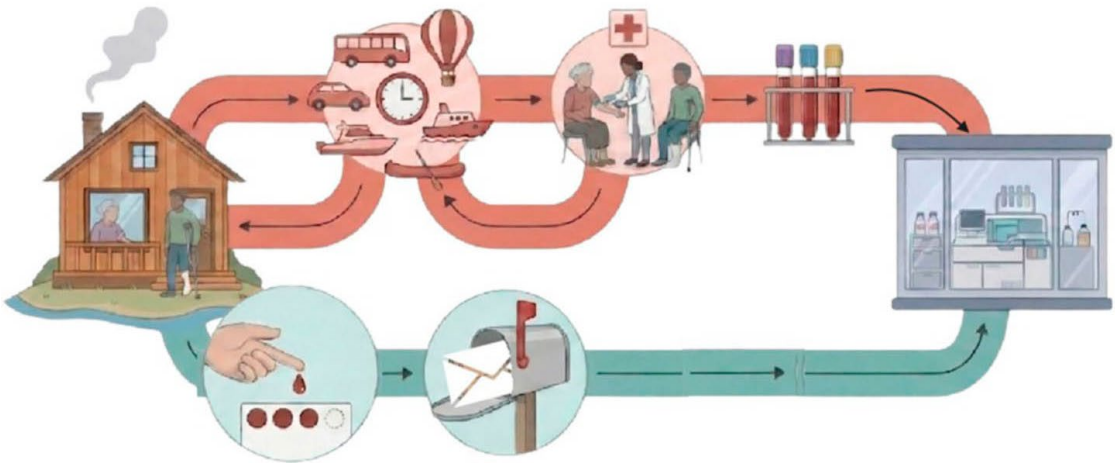


Figure 5. *Home-based self-sampling by finger prick, using dried blood spots (DBS) on filter paper or volumetric absorptive microsampling (VAMS) devices, followed by shipment via standard mail at ambient temperature, simplifies patient monitoring and supports Hospital at Home services.*

ical activity, sample collection and collection tubes, processing delays, storage temperature, freeze–thaw cycles and more. Adherence to standardized procedures and knowledge of analyte stability are essential.

- **Incomplete metabolome coverage:** No single extraction procedure, chromatographic method, or MS platform captures the entire metabolome. Improvements in instrumentations and methods and performing both metabolomics and lipidomics provide huge improvements.
- **Lipids are part of the metabolome, and lipidomics is a subgroup of metabolomics:** The substantial differences in physicochemical properties between polar and hydrophobic metabolites require different extraction and chromatography for optimal detection. Comprehensive global metabolomics therefore often combines conventional global metabolomics (7), covering polar and amphipathic compounds, including many lipids, with dedicated global lipidomics (8) to maximize total coverage.
- **Matrix effects and ion suppression:** Coeluting compounds can affect each other, altering signal intensities. Low-abundant metabolites might thus be masked by co-eluting, high-abundant compounds. New scan modes and acquisition technologies like enhanced dynamic range (eDR), and improved MS instrument performance reduce this problem.
- **Metabolite identification:** Thousands of compounds (referred to as "features" until identified) are detected using global metabolomics. Definitive identification requires exact mass, characteristic chromatographic retention time and fragmentation pattern in line with authentic reference standards of an in-house library analysed using the same instrumentation and analytical protocol. We have more than 1,000 biologically relevant chemical standards in our library and continuously add new ones.
- **Quantification:** Targeted methods use internal standards and single- or multipoint calibration enabling absolute concentrations, although with variable accuracy. Global analysis generally provides relative quantitation, complicating comparisons between analytical methods and laboratories.
- **Analytical drift and batch effects:** Changes in MS sensitivity, chromatography, ionization and signal responses must be normalized using e.g. internal standards, quality control samples, and reference materials for improving intra- and inter-batch comparability.

6.2 Quality assurance and harmonization

Although metabolomics has become the most published omics technology (Figure 1), reflecting the intense research activity, routine clinical applications of global metabolomics remain limited. Differences in sample preparation, chromatography, instrumentation, ionization, data processing, and calibration complicate comparability. Stable, metabolite-rich reference materials with demonstrated commutability are therefore needed to support long-term quality monitoring and interlaboratory comparisons and harmonization, even without certified concentrations for every metabolite (9).

Bioinformatics and data management including use of artificial intelligence (AI) are needed for standardizing and quality-assuring preprocessing of raw data, peak detection, alignment, normalization, annotation, statistical analysis, and more.

6.3 Challenges in translating metabolomics into clinical diagnostics

Demonstrating that individual metabolites or metabolomic signatures are associated with disease does not establish that they are clinically useful. A new test must also provide sufficiently reliable information to improve diagnosis, prognosis, treatment decisions or patient outcomes beyond what is already available (10).

Because metabolomic alterations often involve multiple interconnected pathways, comprehensive metabolomic profiles provide more clinically relevant information than individual metabolites alone. Therefore, reference datasets should encompass healthy individuals across a range of physiological conditions, addressing effects of e.g. fasting, nutrition, BMI, hormonal status, and physical activity as well as patients with well-characterized diseases and treatments (11). Establishing these healthy, disease and treatment-specific reference metabolomes, linked to detailed clinical information, will be important for clinical implementation. These datasets must be securely managed with appropriate privacy safeguards until they are anonymized. AI will be important in exploiting the full potential of comparing an individual patient profile (i.e. the “one sample approach”) with disease-specific metabolomic patterns, supporting more precise diagnosis, prognosis, and personalized treatment (12).

Since clinicians need clear conclusions, rather than a list of molecular signals, laboratories must determine

which findings are clinically relevant and how uncertainty should be communicated, for the metabolomics results to benefit patient management.

Routine implementation further requires validated procedures, integration with laboratory information systems, acceptable turnaround times, and sustainable financing. In Norway, diagnostic use of global metabolomics and lipidomics is publicly reimbursed through Helfo (the Norwegian Health Economics Administration). Used wisely, global metabolomics has the potential to deliver diagnostic and therapeutic benefits that far outweigh its costs. Nevertheless, targeted analyses should always be preferred whenever they can adequately meet clinical needs.

7. Where are we now – and 5 years from now?

We represent Section for metabolomics and lipidomics at Department of Medical Biochemistry at Oslo University Hospital (OUH), performing publicly funded diagnostics, and Core facility for global metabolomics and lipidomics at the Faculty of Medicine, University of Oslo, performing comprehensive metabolomics analyses on a non-profit, fee-for-service basis. (Figure 6).

We collaborate with the Technology and Innovation Clinic at OUH, Sykehuspartner Trust of the South-Eastern Norway Regional Health Authority providing information and communication technology and digital infrastructure, as well as one of the largest international developers and providers of AI, to establish AI tools for automating and quality assuring, with human check points, multiple manual processes as well as characterizing healthy, disease and treatment-response specific reference metabolomes for clinical decision support.

We also collaborate with one of the world-leading providers of LC-HRMS instrumentation and software doing clinical research for developing more sensitive, high-throughput clinical metabolomics services. We



Figure 6. *Our liquid chromatography–high-resolution mass spectrometry platform used for lipidomics. The authors flank three of the Clinical Metabolomics Scientists, from left to right, Sander Johannes Thorbjørnsen Guttorm, Barbora Pisklakova and Aleš Kvasnicka.*

will also soon partner with a producer and international distributor to provide the most diverse and clinically relevant, hospital population-based reference material for long-term quality control and harmonization between metabolomics labs worldwide.

Within the first few months of 2027, we will have expanded our LC-HRMS capacity from two to five platforms. Of these, one has been funded through a private donation, while two are provided through industry collaborations focused on developing high-throughput clinical metabolomics.

In 5 years from now, we hope – and believe - that that our work and collaboration with leading academic and industry partners will have resulted in a cost-effective, high-throughput clinical metabolomics service providing more precise diagnostics and profoundly better personalized medicine, and that clinical chemistry laboratories will provide and refine this service globally over the years to come.

8. Conclusion

Despite remarkable advances in analytical technologies, several challenges must be addressed to fully unlock the potential of metabolomics in clinical medicine. These include incomplete metabolome coverage, pre-analytical and analytical variability, metabolite identification and quantification, quality controls and reference materials for harmonization, rigorous validation of standardized methods, and appropriate reference metabolomes and tools for reliable interpretation and clinical utility.

Overcoming these challenges is essential to translate the extraordinary analytical capabilities of modern metabolomics into reliable and widely accessible clinical diagnostics and personalized medicine.

Transforming analytical innovations into clinically useful tools is at the very heart of clinical chemistry. This is what we do. Together, we can harness the full potential of metabolomics and shape the next golden age of clinical chemistry.

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Användning av patientmedianer för metodmonitorering exemplifierat med cyklosporin

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Kontinuerlig övervakning av analysmetoders prestanda är en central del av kvalitetssäkringen inom klinisk laboratoriemedicin. Traditionella kvalitetskontrollstrategier bygger främst på interna kontrollmaterial och externa kvalitetssäkringsprogram. Dessa är nödvändiga, men de har begränsningar: kontrollmaterial är inte alltid fullt kommutabla, och de speglar inte alltid hur metoden beter sig i verkliga patientprover. Detta har lett till ett växande intresse för patientbaserad kvalitetskontroll (PBQC), där statistiska mått beräknade direkt från patientresultat används som ett komplement till traditionella kontroller.

Ett av de mest robusta måtten inom PBQC är patientmedianen. Medianen är okänslig för extremvärden och påverkas mindre av skeva fördelningar än medelvärdet. I analyser där patientpopulationen är stor, stabil och där provtagningen sker regelbundet – exempelvis vid terapeutisk läkemedelsmonitorering – kan medianen fungera som en känslig indikator för långsam analytisk drift, metodskift eller förändringar i kalibrering.

Patientmedianer har visat sig särskilt användbara för läkemedel med relativt stabila koncentrationsmönster över tid, såsom cyklosporin, tacrolimus, levetiracetam och andra läkemedel som analyseras frekvent i klinisk vardag. När medianen följs över längre tidsperioder kan laboratoriet snabbt identifiera avvikelser som inte fångas av interna kontroller, exempelvis små systematiska förändringar vid byte av instrumentplattform, reagensbatch eller kalibreringsrutiner.

En viktig fördel med patientmedianer är att de bygger på verkliga patientprover, vilket innebär att de automatiskt tar hänsyn till matrixeffekter, biologisk variation och kliniska provtagningsmönster. Detta gör dem särskilt värdefulla i situationer där kontrollmaterial inte fullt ut efterliknar patientprovernas egenskaper. Medianbaserad monitorering kan dessutom utföras helt automatiskt i laboratoriets informationssystem, vilket gör metoden kostnadsfri, kontinuerlig och praktiskt genomförbar utan extra arbetsinsats.

Sammantaget erbjuder patientmedianer ett kraftfullt och praktiskt verktyg för att stärka laboratoriets kvalitetssystem. Genom att kombinera traditionell kvalitetskontroll med PBQC kan laboratorier uppnå en mer heltäckande övervakning av metodprestanda och säkerställa att analyserna förblir tillförlitliga över tid. Detta är särskilt viktigt för analyser där små förändringar i rapporterade värden kan få betydande kliniska konsekvenser.

Cyklosporin, ett exempel på användning av patientmedianer

Cyklosporin är ett av de mest betydelsefulla läkemedlen inom modern transplantationsmedicin. Sedan introduktionen på 1980talet har det revolutionerat möjligheten att förhindra avstötning av transplanterade organ och därmed förbättrat överlevnad och livskvalitet för patienter världen över. Samtidigt är cyklosporin ett läkemedel som kräver noggrann övervakning, eftersom dess terapeutiska fönster är smalt och dess farmakokinetik komplex. Denna studie undersöker hur cyklosporinkoncentrationer i blod har varierat över en period på 20 år, och hur patientbaserade statistiska mått, såsom medianer och percentiler, kan användas som ett robust verktyg för patientbaserad kvalitetskontroll (PBQC) i klinisk laboratoriemedicin.

Cyklosporin är en kalcineurinhämmare som används för att förhindra avstötning efter organtransplantationer, särskilt njur-, lever- och hjärttransplantationer. Läkemedlet används också vid vissa autoimmuna sjukdomar, såsom svår psoriasis, reumatoid artrit och

aplastisk anemi. Dess verkningsmekanism bygger på att hämma aktivering av Tlymfocyter, vilket är centralt i immunförsvarets reaktion mot främmande vävnad.

Cyklosporin binder till proteinet cyklofilin, och komplexet hämmar enzymet kalcineurin. När kalcineurin blockeras förhindras aktivering av transkriptionsfaktorn NFAT, vilket leder till minskad produktion av cytokiner som interleukin2. Detta dämpar immunsystemets förmåga att attackera det transplanterade organet.

Eftersom cyklosporin har ett mycket smalt intervall mellan effektiv och toxisk dos, är terapeutisk läkemedelsmonitorering (TDM) avgörande. För höga nivåer kan orsaka allvarliga biverkningar såsom njurtoxicitet, hypertoni, neurotoxicitet och dyslipidemi. För låga nivåer ökar risken för akut eller kronisk avstötning.

Material och metoder

Prover

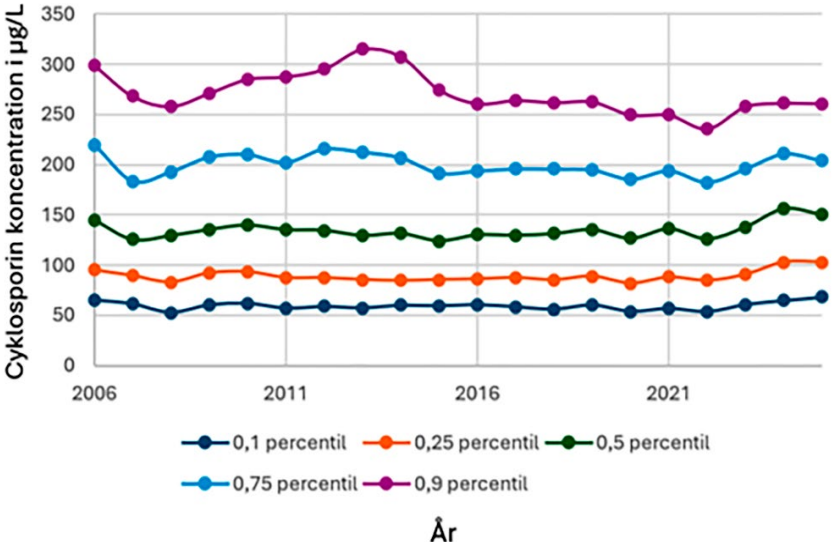
Rutinbeställningar av cyklosporin i blod vid Avdelningen för Klinisk Kemi och Farmakologi, Akademiska sjukhuset i Uppsala, inkluderades i studien. Helblodsprover samlades i EDTA-rör (367862, BD Vacutainer Systems, Plymouth, Storbritannien). Studieperioden sträckte sig från 1 januari 2006 till 31 december 2025. Totalt rapporterades 48 835 cyklosporinresultat under denna period. Proverna kom från 2027 individuella patienter som testats under en 20-årsperiod. Tillgängliga variabler inkluderade provtagningsdatum, patientens ålder i år, kön och uppmätt cyklosporinkoncentration. Ingen identifierbar patientinformation användes.

Analysinstrument

Cyklosporinkoncentrationer mättes initialt på Hitachi 912-analysatorn (Roche Diagnostics, Rotkreuz, Schweiz) med reagenser från Microgenics (100147, Fremont, CA, USA). I februari 2008 överfördes metoden till Architect-plattformen (Abbott Laboratories, Abbott Park, IL, USA) med samma reagens från Microgenics. Kalibrering och intern kvalitetskontroll utfördes enligt tillverkarens rekommendationer. Extern kvalitetskontroll tillhandahölls av UK NEQAS och analyserades månadsvis. Metodens variationskoefficient (CV) var 6,3 % vid 60 µg/L, 2,5 % vid 167 µg/L och 2,4 % vid 290 µg/L. I januari 2021 överfördes cyklosporinanalysen från Architect-plattformen till Cobas Pro e 801-analysatorn (Roche Diagnostics) med cyklosporinreagenser från samma tillverkare (07251246190, Roche Diagnostics). Före klinisk implementering validerades metodövergången enligt lokala laboratorieriktlinjer. Valideringen inkluderade jämförelse av patientprover analyserade på båda plattformarna, bedömning av bias och precision, verifiering av kalibreringsstabilitet samt utvärdering av det analytiska mätområdet. Den observerade överensstämmelsen mellan metoderna bedömdes



Foto: Henrik Alfthan.



***Figur 1**. Årliga patientbaserade medianvärden för serumkoncentrationer av cyklosporin samt utvalda percentilvärden under studieperioden.*

som kliniskt acceptabel, trots något lägre värden med Roche-metoden (cirka 7 %). Extern kvalitetskontroll utfördes månadsvis med material från LGC Standards (Wesel, Tyskland). Den totala CV var 3,3 % vid 75 µg/L, 2,6 % vid 165 µg/L och 7,8 % vid 408 µg/L.

Statistiska analyser

Patientpercentiler beräknades årsvis från alla rapporterade cyklosporinresultat. Deskriptiv statistik, inklusive medianer, kvartiler samt 10:e och 90:e percentiler, användes för att undersöka långtidstrender och variation över tid. Statistiska analyser utfördes med Excel 365 (Microsoft Corp., Seattle, WA, USA) och Statistica version 10 (TIBCO Software, Palo Alto, CA, USA).

Resultat

Förändringar i antal rapporterade cyklosporinresultat över tid

Det totala antalet förfrågningar under studieperioden var 48 835. Av dessa avsåg 20 920 kvinnliga patienter och 28 011 manliga patienter. En statistiskt signifikant negativ trend observerades för det årliga antalet analyser över 20-årsperioden, vilket indikerar en minskning av antalet analyser över tid från 2917 analyser år 2006 till 2177 analyser år 2025.

Förändringar i rapporterade cyklosporinresultat över tid

Medianvärdet för cyklosporin var 134,4 µg/L (kvinnor 134,1 µg/L och män 134,5 µg/L) för hela studieperioden. Det fanns mycket små könsskillnader för alla studerade percentiler från 10:e till 90:e percentilen. De lägre percentilerna (10:e, 25:e och 50:e) var stabila under perioden 2006–2025 med totala variationskoefficienter (CV) mellan 6 och 7 %. För den högsta percentilen varierade värdena mer, men den totala CV för årsvisa värden var fortfarande 7,6 % Figur 1).

Säsongsvariation i cyklosporinresultat

Juli är den huvudsakliga semestermånaden i Sverige. Juli var också den månad med lägst antal förfrågningar (Figur 2). Trots variationen i provvolym var cyklosporinresultaten stabila över året.

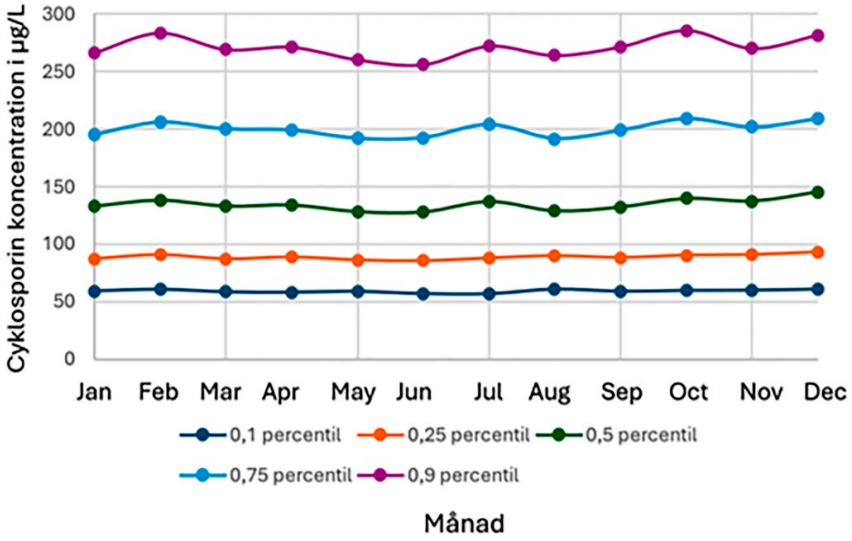
Metodjämförelse mellan Architect och Cobas

Metodjämförelsen bestod av 21 prover som täckte mätområdet 30–227 µg/L. Vid 100 µg/L var den genomsnittliga skillnaden 5,8 %, och den ökade till 8,4 % vid 250 µg/L.

Vi jämförde också analysresultat från de två åren före metodbytet (Architect) med de två åren efter bytet (Cobas). Medianen under Architect-perioden var 131,5 µg/L (95 % CI 128,7–134,5; n = 4809), medan den under Cobas-perioden var 130,0 µg/L (95 % CI 127,0–133,0; n = 4510). Det fanns inga statistiskt signifikanta skillnader mellan perioderna enligt Mann–Whitney-testet (p = 0,30).

Diskussion

I denna studie utvärderade vi långtidstrender i rutinmässiga cyklosporinkoncentrationer i blod över en 20-årsperiod med hjälp av patientbaserade statistiska



Figur 2. *Patientbaserade medianvärden för serumkoncentrationer av cyklosporin samt utvalda percentilvärden per månad under studieperioden.*

mått. Huvudfyndet är den anmärkningsvärda stabiliteten i patientmedianer och lägre percentiler, med minimal variation över tid, kön och metodövergångar. Dessa observationer tyder på hög konsistens i både analytisk prestanda och kliniska doseringsrutiner.

Den övergripande medianen på cirka 134 µg/L förblev i stort sett oförändrad under hela studieperioden. Detta värde överensstämmer väl med vanliga målintervall för underhållsbehandling vid organtransplantation, särskilt hos njurtransplanterade patienter flera månader till år efter transplantation. Avsaknaden av kliniskt betydelsefulla könsskillnader stödjer ytterligare robustheten i de observerade populationstrenderna.

De lägre percentilerna (10:e och 25:e) samt medianen visade minst variation över tid, med CV mellan 6 % och 7 %. Detta är anmärkningsvärt med tanke på cyklosporins betydande farmakokinetiska variation samt förändringar i transplantationspraxis och behandlingsriktlinjer över två decennier. Stabiliteten tyder på att patientbaserad statistik är effektiv för att övervaka långsiktig analytisk konsistens och upptäcka större förändringar i klinisk praxis.

De högre percentilerna varierade mer, vilket är förväntat eftersom dessa värden oftare speglar kliniska specialfall såsom tidig post-transplantationsfas, hög immunologisk risk, läkemedelsinteraktioner eller bristande följsamhet. Trots detta hade även 90:e percentilen en CV under 8 %, vilket visar att extrema värden inte ökade över tid.

Viktigt är att inga systematiska förändringar i patientpercentiler observerades efter metodbyten, trots en dokumenterad negativ bias på cirka 7 % med Cobas-metoden. Detta tyder på att kalibrering och validering vid metodövergångar var tillräckliga för att bibehålla kontinuitet i rapporterade resultat. Medianbaserad monitorering verkar därmed vara robust mot måttlig analytisk bias. Den ringa nedgången i årliga förfrågningar speglar sannolikt förändringar i klinisk praxis, inklusive ökad användning av tacrolimus och mer individualiserad monitorering av stabila transplantationspatienter. Minskningen påverkade dock inte koncentrationsfördelningarna.

Ur kvalitetssäkringsperspektiv har patientbaserad realtidskontroll fått ökad uppmärksamhet, särskilt för analyser där kommutabla kontrollmaterial är begränsade. Denna studie visar att patientmedianer och percentiler är ett kraftfullt komplement till traditionell kvalitetskontroll, särskilt för långsiktig övervakning.

Sammanfattningsvis visar studien att cyklosporinkoncentrationer varit stabila på populationsnivå över två decennier. Patientmedianer och percentiler är därmed ett enkelt, robust och informativt komplement till konventionell kvalitetskontroll och särskilt värdefulla för långsiktig metodövervakning i kliniska laboratorier.

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En berättelse om kemisten Otto Folin

Anders Larsson



Biokemisten Otto Folin är känd för att ha gjort biokemiska analyser noggranna, kvantitativa och kliniskt användbara, och räknas som en av grundarna av medicinsk biokemi. Han utvecklade precisa metoder för att mäta ämnen i blod och urin (t.ex. urea, kreatinin och urat).

Otto Folin, (1867-1934, var en svensk-amerikansk biokemist. Folin föddes i Åseda i Sverige som ett av tolv barn, varav endast fem överlevde till vuxen ålder. Föreldrarna var garvaren Nils Folin och barnmorskan Eva Folin. I augusti 1882 emigrerade han till Minnesota i USA där han bodde hos sin bror. Efter utbildning på University of Minnesota studerade han på University of Chicago och blev filosofie doktor med en avhandling om uretaner 1896. Samma år återvände han till Sverige och studerade på Uppsala universitet under professor Olof Hammarsten.

Folin återvände till USA 1898 och blev biträdande professor i biokemi vid Harvard Medical Scholl 1907, samt professor där 1909. I laboratorierna vid Harvard Medical School gjorde han sina största upptäckter. Det var där han började formulera sin vision: att människans inre kemi skulle kunna mätas med precision – även i små mängder blod och urin.

Vid den tiden krävde analyserna stora provvolymer, var oprecisa och gav ofta motsägelsefulla resultat. Folin såg detta som ett problem som kunde lösas med bättre kemi. Han utvecklade därför mikrometoder – tekniker som gjorde det möjligt att analysera mycket små mängder biologiskt material med hög noggrannhet. Man skall dock vara medveten om att det som på den tiden var mikrometoder skulle idag uppfattas som stora provvolymer. Ännu på 60-talet så använde man hinkar för att fylla på analysreagens till autokemist instrumenten. Detta var revolutionerande. Plötsligt kunde läkare få tillförlitliga värden på ämnen som urea, kreatinin och glukos, även hos svårt sjuka patienter där provmaterial var begränsat.

Tillsammans med sin kollega Hsien Wu utvecklade Folin en metod för att mäta glukos i blod – Folin–Wumetoden. Den blev snabbt standard i laboratorier världen över. Den byggde på en färgreaktion där glukos reducerade kopparjoner, vilket skapade en mätbar färgförändring. Detta gjorde blodsockermätning mer exakt än någonsin tidigare.

Folin var också fascinerad av hur kemiska reaktioner kunde uttryckas i färg. Tillsammans med Ciocalteu utvecklade han Folin–Ciocalteureagenset, ett blåfärgat reagens som reagerar med fenoler och proteiner. Det är ett av de mest långlivade kemiska verktygen i laboratorievärlden.

En av Folins mest betydelsefulla vetenskapliga insikter var upptäckten av kreatinfosfat i muskler – en energirik förening som fungerar som snabb energireserv. Detta lade grunden för modern förståelse av muskelmetabolism och energilagring.

Folin utvecklade också standardiserade metoder för att mäta:

- kreatinin
- urea
- urat
- ammoniak

Dessa analyser möjliggjorde en betydeligt tidigare diagnostik av njursjukdomar jamfört med tidigare metoder.

Som professor vid Harvard utbildade Folin en generation forskare som senare själva blev Nobelpristagare, bland annat George Hitchings och James B. Sumner. När Otto Folin avled 1934 hade hans arbete lagt grunden för den moderna kliniska kemin. Folin bidrog också till utveckling av proteinbestämning enligt Folin som ingår i den klassiska Lowry metoden (1), där Folin–Ciocalteu reagenset används som färgreagens för att kvantifiera proteiner. Metoden är en av de mest använda och känsliga proteinbestämningsmetoderna och bygger på två kemiska reaktioner som tillsammans ger en blå färg proportionell mot proteinkoncentrationen. Artikeln har mer än 350 000 citeringar och är en av de mest citerade arbetena någonsin.

1. Lowry OH et al. Protein measurement with the Folin phenol reagent. J Biol Chem 1951;193:265-75.



Illustrationen genererades med Copilot och är avsedd att föreställa en arbetsbänk från Folins tid.

PhD: Iron deficiency biomarkers and hepcidin isoforms in chronic obstructive pulmonary disease

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Short summary
Iron deficiency is common in patients with chronic obstructive pulmonary disease (COPD), but prevalence estimates vary considerably depending on the biomarker used to define iron deficiency. In this thesis, iron deficiency was more prevalent in COPD than in individuals without COPD, particularly among patients with more severe disease. Different biomarkers identified different patient as iron deficient, highlighting the challenges of assessing iron status in COPD. We also investigated hepcidin isoforms and found associations with iron stores and inflammation, but no clear evidence that hepcidin explains the increased prevalence of iron deficiency in COPD.

Why study iron deficiency in COPD?

COPD is a heterogeneous disease characterized not only by airflow limitation but also by systemic manifestations and comorbidities. Several features of COPD may increase the risk of iron deficiency.

First, hypoxaemia, defined as reduced oxygenation of the blood, stimulates erythropoiesis and thereby increases iron demand. Second, chronic inflammation may impair intestinal iron absorption and iron mobilization through increased production of the iron-regulatory hormone hepcidin. Together, these mechanisms create a setting in which iron deficiency is both common and clinically relevant.

Iron deficiency has attracted increasing attention because it may contribute to symptoms such as fatigue, dyspnoea, and reduced exercise capacity, even in the absence of anaemia. However, assessment of iron status is challenging in COPD because commonly used biomarkers are influenced by inflammation.

The overall aim of this thesis was to improve our understanding of biomarker-defined iron deficiency in COPD. In addition, we investigated circulating hepcidin isoforms in individuals with and without COPD to explore whether altered hepcidin regulation might contribute to iron deficiency in this patient population.

Study populations and biomarkers

The work was based on two Norwegian cohorts.

- The Long-term Outcome of Oxygen Supplementation on Cardiovascular Co-morbidity, Anaemia and Psychological Distress in COPD (LTOT) study included 84 patients with stable COPD and 59 participants from the general population. Twenty-one COPD patients had chronic hypoxaemic respiratory failure and were candidates for long-term oxygen therapy.
- The Norwegian Early Lung Cancer Screening (TIDL) study included 1,002 individuals with a history of heavy smoking, of whom 232 had COPD.

Iron deficiency was assessed using three definitions commonly applied in clinical practice and research on chronic inflammatory diseases (1-4).

1. Ferritin < cutoff (<30 µg/L in absence of inflammation (defined as CRP < 5 mg/L), or <100 µg/L in the presence of inflammation).
2. Transferrin saturation (TSat) <20%
3. A combined criterion requiring both low ferritin and low TSat

The ferritin-based definitions do not account for differences between analytical methods. As ferritin concentrations may vary between assays, the use of fixed decision limits can influence prevalence estimates and limit comparability between studies and laboratories.

Iron deficiency is common, but prevalence depends on the biomarker

One of the most striking findings was the large variation in prevalence of iron deficiency depending on the biomarker used.

Among patients with COPD, prevalence ranged from approximately 15% to 70% in the LTOT study and from about 5% to 30% in the TIDL study (5-6). In participants without COPD, prevalence was lower, ranging from approximately 2% to 20%.

Importantly, the biomarkers did not identify the same individuals as iron deficient. Only partial overlap was observed between ferritin < cutoff and TSat <20%, suggesting that these biomarkers reflect different aspects of iron metabolism, such as iron stores and circulating iron availability, rather than a single underlying condition (Figure 1).

Associations with clinical and biochemical characteristics also varied between biomarkers and study populations. Iron deficiency was generally more common in patients with more severe COPD.

These findings highlight a major challenge in both clinical practice and research: the absence of a universally accepted biomarker for assessing iron status in COPD.

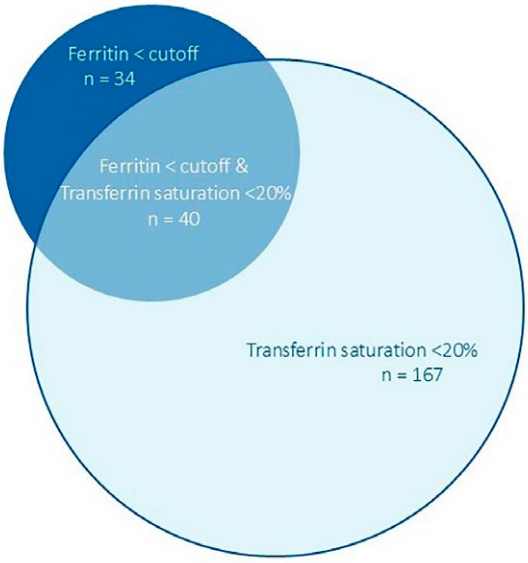


Figure 1. *Overlap between iron deficiency biomarkers in participants in the TIDL study. Ferritin < cutoff: <30 µg/L if CRP <5 mg/L, <100 µg/L if CRP ≥ 5 mg/L.*

Anaemia was uncommon, but elevated haemoglobin occurred in hypoxaemic COPD

Despite the high prevalence of iron deficiency, anaemia was relatively uncommon. In both study populations, anaemia was present in around 8% of participants, regardless of COPD status.

In contrast, elevated haemoglobin concentrations were frequently observed among patients with untreated chronic hypoxaemia. In the LTOT cohort, about one-third of these patients had elevated haemoglobin levels, compared with only a few percent of non-hypoxaemic COPD patients and none of the participants without COPD.

This finding illustrates how chronic hypoxaemia stimulates erythropoiesis, leading to increased haemoglobin concentrations. Adequate iron availability is essential for this adaptive response. Failure to increase haemoglobin levels in the presence of hypoxaemia may therefore indicate inadequate iron availability for an appropriate erythropoietic response.

What can hepcidin tell us?

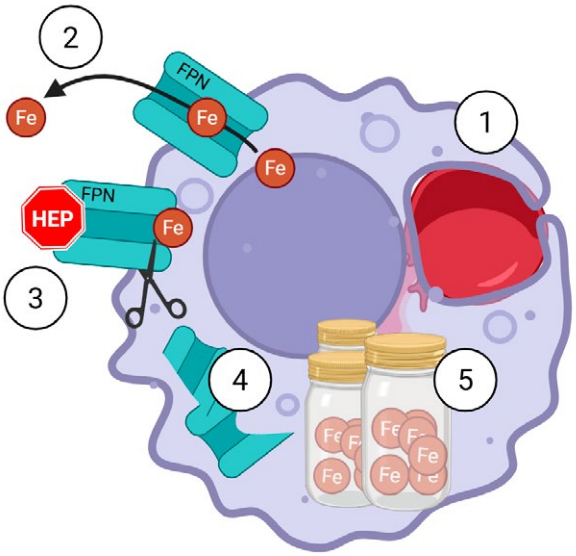
Hepcidin is the principal regulator of systemic iron homeostasis and has therefore been proposed as a potential link between chronic inflammation and iron deficiency in COPD.

Serum concentrations of four hepcidin isoforms (Hep25, Hep24, Hep22 and Hep20) were measured by liquid chromatography-high resolution mass spectrometry (LC-HRMS). All four isoforms were frequently detected in participants with and without COPD (7).

Although no significant differences in hepcidin concentrations were observed between COPD and non-COPD participants in the LTOT study, variability was greater among COPD patients. Hep25 was by far the predominant isoform, with concentrations 10 to 20 times higher than those of the other isoforms.

All isoforms were associated with ferritin and C-reactive protein, supporting links with both iron stores and inflammation. Hep25 and Hep24 were additionally associated with TSat, while Hep24 was associated with albuminuria.

While our findings suggest that hepcidin analyses do not explain the increased prevalence of iron deficiency in COPD, measuring hepcidin may still improve our understanding of unexplained disturbances in iron metabolism and poor responses to iron supplementation in both COPD and non-COPD populations.



Macrophages acquire iron through phagocytosing senescent erythrocytes (1). Iron (Fe) is exported through ferroportin (FPN)(2). When Hepcidin (HEP) binds to FPN (3), FPN is degraded (4). Instead of being exported, iron is stored in "containers" such as ferritin (5). Created by Ingrid M. Hardang using BioRender.com.

Clinical implications

This thesis demonstrates that iron deficiency is more common in COPD than in individuals without COPD and that its prevalence appears to increase with disease severity. At the same time, the findings show that the choice of biomarker has a major impact on both prevalence estimates and the identification of affected individuals.

A major limitation of the work is the absence of a gold standard for iron deficiency. Consequently, it remains uncertain which biomarker best reflects iron status in patients with COPD.

Furthermore, interpretation of ferritin-based criteria is complicated not only by inflammation but also by analytical differences between ferritin assays, a factor that is rarely considered when universal decision limits are applied.

For clinical biochemists and clinicians alike, this raises an important question: which biomarker best identifies clinically relevant iron deficiency in COPD? At present, there is no clear answer. Bone marrow iron staining remains the reference method for assessing iron stores, but its invasive nature limits clinical applicability and makes it unsuitable for large-scale studies. Alternative approaches to validating iron biomarkers in COPD are therefore needed. For instance, as iron deficiency may lead not only to anaemia but also to impairments in measurable functions such as exercise capacity, researchers could investigate the relationship between exercise capacity and specific iron deficiency biomarkers.

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>
> Fleming et al. Clin Chem Lab Med 2019

> *"Our study revealed five cases where the only means for diagnosis or detecting disease progression was FLC analysis, and where only one (Freelite from Binding Site) of the two parallel applied methods served to detect the relevant changes"*
>
> Veskovski et al. eJHaem 2024

> *"Based on our findings, the three different FLC assays should not be used interchangeably"*
>
> Schieferdecker et al. Blood Cancer Journal 2020

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Jämförelse av placental growth factor analyserat under tidig graviditet med 3 olika metoder

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Introduktion

Preeklampsi förekommer vid cirka 3–5 % av alla graviditeter i Skandinavien och är en av de viktigaste orsakerna till sjuklighet och dödlighet hos både mödrar och barn. Identifiering av kvinnor med ökad risk är viktig eftersom tidig upptäckt av preeklampsi har visats gynna både modern och det ofödda barnet. Dessutom kan profylaktisk behandling med acetylsalicylsyra som påbörjas tidigt under graviditeten hos högriskkvinnor minska förekomsten av preeklampsi med upp till 40 %.

Flera multivariabla modeller har utvecklats för att förutsäga preeklampsi. Dessa bygger på kombinationer av maternella karakteristika, medicinsk anamnes, biofysiska undersökningar och blodbaserade biomarkörer. En av de viktigaste blodbaserade biomarkörerna är placental growth factor (PlGF). PlGF uttrycks huvudsakligen i placenta och navelsträng under normala graviditeter och tillhör VEGF-familjen, som är involverad i angiogenes samt utveckling och underhåll av blod- och lymfkärlens endotel. Proteinet består av 149 aminosyror och binder både till membranbundet och lösligt VEGF-receptor 1 (VEGFR1), även kallat sFlt-1.

Vid normala graviditeter ökar nivåerna av PlGF successivt och når sina högsta värden mellan vecka 27 och 30. Därefter sjunker koncentrationerna. Kvinnor som senare utvecklar preeklampsi eller intrauterin tillväxthämning uppvisar redan under första trimestern lägre cirkulerande nivåer av PlGF.

PlGF ingår idag i flera olika modeller för prediktion av preeklampsi bla i England och Danmark. Eftersom det saknas en internationellt accepterad kalibrator för PlGF kan dock olika laboratorieplattformar ge olika analysresultat. Ett internationellt referensmaterial skulle kunna minska eller eliminera dessa skillnader.

De huvudsakliga tillverkarna av immunologiska PlGF-analyser för klinisk användning är idag PerkinElmer, Roche Diagnostics och Thermo Fisher Scientific.

Syftet med studien var att jämföra resultaten från dessa analysplattformar för bestämning av cirkulerande PlGF-koncentrationer samt att utveckla ekvationer för omräkning mellan de olika metoderna.

Material och metoder

Studien var en observationsstudie där tre olika metoder för analys av PlGF jämfördes med hjälp av serumprover från 150 gravida kvinnor. Kvinnorna rekryterades inom ramen för IMPACT-studien mellan november 2018 och november 2020. Inklusion skedde under graviditetsvecka 11+0 till 13+6.

Serumprover valdes slumpmässigt från IMPACT-kohorten och förvarades frysta vid −80 °C. Inför PlGF-analys tinades proverna, blandades och centrifugerades vid 2400 g i 10 minuter. Därefter delades varje prov upp i tre aliquoter som åter frystes inför transport till respektive laboratorium. Proverna skickades till Stockholm, Uppsala och Göteborg där analyser utfördes med PerkinElmers, Roche Diagnostics respektive Thermo Fishers plattformar. Tiden från transport till analys var mindre än två månader.

I Stockholm användes Delfia Express® Model 6000–0010 från PerkinElmer. I Uppsala användes Roche Diagnostics cobas e601-analysator. I Göteborg användes BRAHMS PlGF plus KRYPTOR från Thermo Fisher Scientific.

Metodjämförelserna utfördes med Deming-regressionsanalys i programvaran Method Validator. De tre metoderna jämfördes parvis med både linjär regression och Deming-analys.

Resultat

Populationens allmänna karakteristika

De inkluderade kvinnorna rekryterades inom IMPACT-studien, som är en populationsbaserad

kohort. Samtliga kvinnor var bosatta i Uppsala län som har en prevalensen av preeklampsi på cirka 3 %.

Jämförelse mellan PerkinElmer och Roche Diagnostics

Roche Diagnostics-metoden gav ungefär 80 % högre PlGF-värden än PerkinElmer-metoden. Korrelationskoefficienten mellan metoderna var hög (r = 0,966).

Deming-regressionen beskrevs av följande ekvation:
PlGF_PerkinElmer = 0,553 × PlGF_Roche − 1,112
(95 % KI för lutningen: 0,518–0,588; 95 % KI för interceptet: −2,773 till 0,550).
Medelskillnaden mellan metoderna var −24,6 ng/L (95 % KI: −26,4 till −22,8).

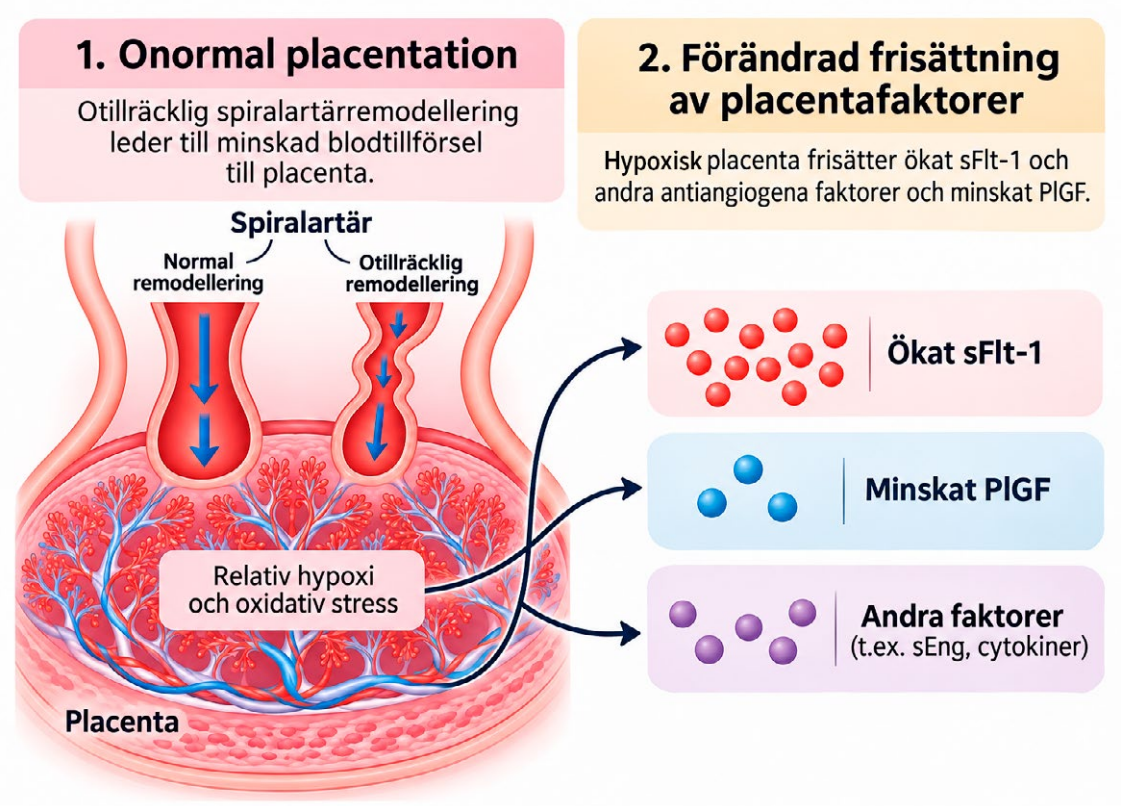
Omräknat i motsatt riktning erhölls:
PlGF_Roche = 1,809 × PlGF_PerkinElmer + 2,010
(95 % KI för lutningen: 1,694–1,923; 95 % KI för interceptet: −0,877 till 4,897).
Medelskillnaden var då 24,6 ng/L (95 % KI: 22,8–26,4).

Jämförelse mellan PerkinElmer och Thermo Fisher

Thermo Fisher-metoden gav ungefär 50 % högre PlGF-värden än PerkinElmer-metoden. Korrelationskoefficienten var 0,945.

Deming-regressionen beskrevs av:
PlGF_PerkinElmer = 0,673 × PlGF_ThermoFisher − 0,199
(95 % KI för lutningen: 0,618–0,729; 95 % KI för interceptet: −2,292 till 1,894).
Medelskillnaden mellan metoderna var −13,8 ng/L (95 % KI: −15,1 till −12,6).

Omräknat i motsatt riktning erhölls:
PlGF_ThermoFisher = 1,485 × PlGF_PerkinElmer + 0,296
(95 % KI för lutningen: 1,363–1,607; 95 % KI för interceptet: −2,784 till 3,375).
Medelskillnaden var 13,8 ng/L (95 % KI: 12,6–15,1).



Placentära mekanismer vid preeklampsi. Figuren har genererats med Copilot.

Jämförelse mellan Roche Diagnostics och Thermo Fisher

Roche Diagnostics-metoden gav ungefär 24 % högre värden än Thermo Fisher-metoden. Korrelationskoefficienten var 0,937.

Deming-regressionen beskrevs av:
PlGF_Roche = 1,237 × PlGF_ThermoFisher + 0,840
(95 % KI för lutningen: 1,113–1,361; 95 % KI för interceptet: −3,684 till 5,363).
Medelskillnaden var 10,8 ng/L (95 % KI: 9,4–12,1).

Omräknat åt andra hållet erhölls:
PlGF_ThermoFisher = 0,808 × PlGF_Roche − 0,679
(95 % KI för lutningen: 0,726–0,891; 95 % KI för interceptet: −4,456 till 3,099).
Medelskillnaden var −10,8 ng/L (95 % KI: −12,1 till −9,4).

Diskussion

Valet av PlGF-metod har en tydlig påverkan på de erhållna PlGF-resultaten. Samtidigt var korrelationskoefficienterna höga för samtliga metodjämförelser, vilket indikerar att det kan vara möjligt att omräkna resultaten och därmed möjliggöra jämförelser och sammanslagning av data genererade med de tre analyser som studerats.

PlGF används idag i flera prediktionsmodeller för preeklampsi, inklusive den modell som utvecklats av Fetal Medicine Foundation i England. Samtliga de tre analysplattformarna som undersöktes i denna studie används kliniskt.

En tidigare studie som undersökte gravida kvinnor med preeklampsi eller HELLP-syndrom visade skillnader i kalibrering mellan Roche Diagnostics och Thermo Fisher vid sen graviditet. En potentiell förklaring är att sFlt-1 binder till PlGF och därmed kan maskera de epitoper som antikropparna i analyserna ska känna igen. Eftersom sFlt-1-koncentrationerna ökar kraftigt under graviditeten varierar förhållandet mellan PlGF och sFlt-1 beroende på graviditetsvecka och förekomst av preeklampsi. Beroende på antikropparnas bindningsställen kan detta påverka resultaten olika mycket i olika analyser.

Ytterligare en faktor som försvårar jämförelser mellan olika graviditetsstadier är att PlGF är glykosylerat. Glykosylering är den vanligaste posttranslationella modifieringen av proteiner och kan påverka antikroppsbindningen i immunologiska analyser. Tidigare studier har visat att graden av glykosylering förändras under graviditetens gång, vilket innebär att överensstämmelsen mellan olika immunoassays kan variera mellan olika graviditetsveckor.

Sedan 2022 rekommenderas PlGF som biomarkör i flera internationella riktlinjer för handläggning av preeklampsi. Med hänsyn till svarstid, kostnad och det stora antal analyser som skulle krävas inom nationella screeningprogram är det sannolikt att analyserna även fortsättningsvis kommer att utföras på regional nivå. Valet av instrument kommer då att vara beroende av den lokala laboratorieutrustningen. Därför finns ett behov av att kunna omvandla resultat mellan olika analysplattformar för att möjliggöra jämförbara beslutsgränser och riskbedömningar.

För att kunna använda resultat från olika tillverkare är det viktigt att känna till skillnaderna i lutningar och intercept mellan metoderna. Detta är också betydelsefullt vid tolkning av vetenskapliga studier där olika PlGF-metoder har använts. De regressionsformler som presenteras i denna studie kan användas för direkt översättning mellan de vanligaste PlGF-plattformarna och därmed underlätta integreringen av PlGF i kliniska prediktionsmodeller.

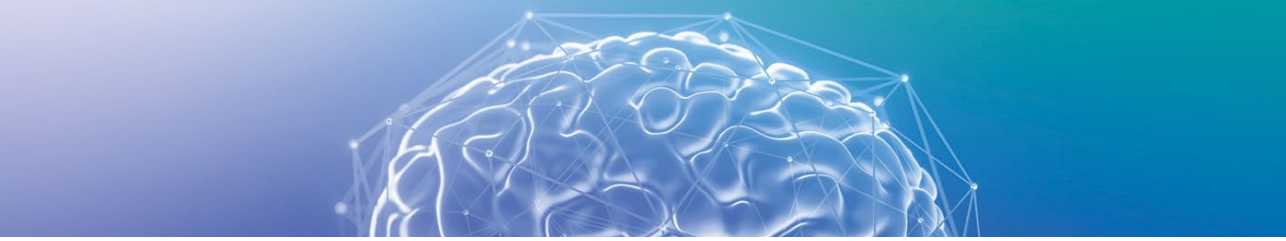
Skillnaderna i kalibrering mellan metoderna kan ibland leda till felaktiga slutsatser. Ett internationellt referensmaterial för PlGF skulle därför vara värdefullt för att minska metodskillnaderna. Framtagandet av ett sådant referensmaterial kan dock ta många år, och fram till dess behöver omräkningsekvationer användas när olika analysmetoder ingår i prediktionsalgoritmer.

Slutsats

Resultaten visar att de kommersiella PlGF-plattformarna från PerkinElmer, Roche Diagnostics och Thermo Fisher Scientific har olika kalibreringar vid bestämning av cirkulerande PlGF-koncentrationer. Trots dessa skillnader uppvisar metoderna en god överensstämmelse, vilket innebär att resultaten kan omräknas och jämföras mellan plattformarna.

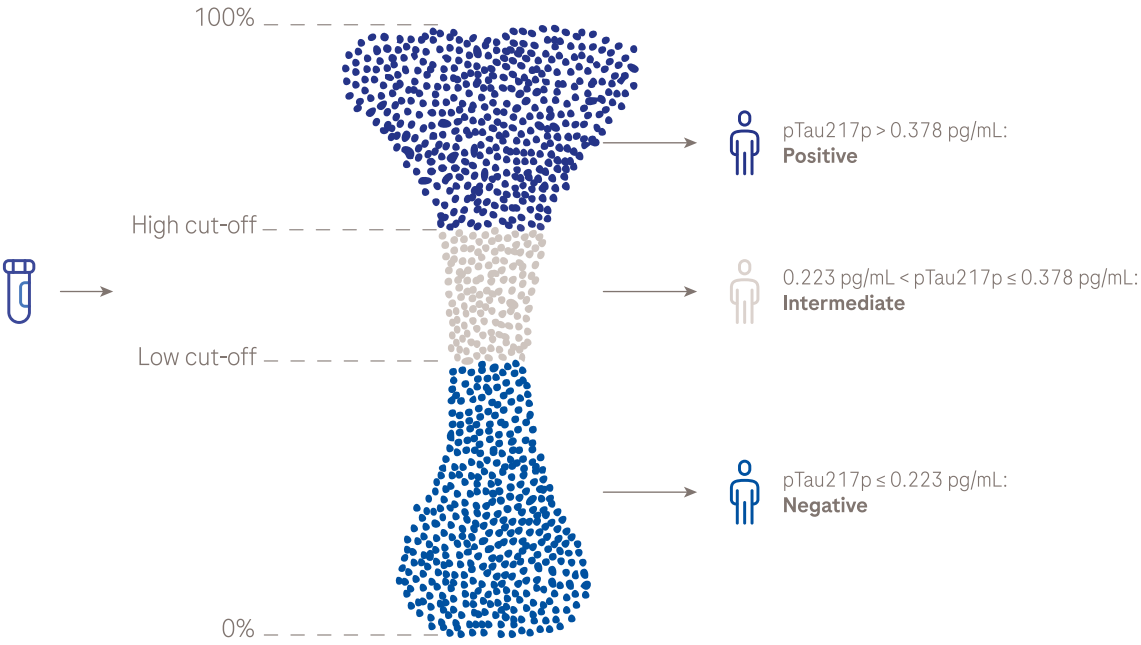
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